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Contents

Volume 06 Number 02 July - December 2012

1. Cresol from Toluene Metabolites in Head Space of Urine And Blood From Glue Sniffing Activity Using Gas Chromatography- Mass Spectrophotometry 1
   Abdul Rahim Yacob, Mohd. Raizul Zainalibdin, Nazirah Said
2. Profile and Pattern of Suicidal and Homicidal Burn Victims at a Tertiary Care Hospital in Northern India 06
   Abhishek Singh, Rajesh DR, Balbir Kaur, Anu Bhardwaj
3. Alkaptonuric Ochronosis : A Case Report 10
   Vishali Kotwal, Amit Thakur
4. Age Estimation in Human Adults by Root Dentin Transparency 13
   Banu V, Shivaprasad S, Ashok L
5. An Analytical Study of Deaths Due to Hanging in Warangal Area, Andhra Pradesh 16
   Bharathi Rama Rao, Chand Basha V, Sudhakar Reddy K
6. Importance of Dental Age and Skeletal Age in Forensic Sciences for the Assessment of Pediatric Growth and Development 20
   Gupta B., Sudha P., Anegundi R., Indushekar Kr
7. Dermatoglyphics-A New Diagnostic Tool in Detection of Dental Caries in Children 24
   Padma K Bhat, Bhumika K Badiyani, Aruna C N
8. Study of Pattern of Injuries in Victims of Fatal Road Trafic Accidents in Nepal 29
   Birendra Kumar Mandal, Abhishek Karn, Amshu Pradhan, Shivendra Jha
9. Cheiloscopy: Detection and Development of Latent Lip Prints 34
   Vanishree. N, Chaithra. V, Amandeep Pabbla
10. A Comprehensive Medico-Legal Analysis of Paediatric Deaths at Osmania General Hospital Hyderabad 40
    J.Srinivas, V.Chand Basha, K.Sudhakar Reddy
11. Autopsy Study of Cut Throat Cases Brought to Morgue of Sion Hospital, Mumbai- A three Year Study 44
    Dhiraj Buchade, Hemant Kukde, Rajesh Dere, Ramesh Savardekar, Nilesh Devraj, Amol Maiyyar
12. A Study of Thoraco-abdominal Trauma in Fatal Road Traffic Accidents in Aurangabad 47
    M.R. Lamb, J.R. Shinde, R.M.Tandle, N.M.Varma
13. An Analytical Study of Causes of Death in Fall from Height Cases from Gandhi Medical College & Hospital Mortuary, Hyderabad from 2006 to 2008 year 51
    K.V. Ramesh, V. Jayashankar
14. A Study of Pattern of Fatal Head Injury at J.N.M.C. Hospital, Aligarh 53
    Mohd Kaleem Khan, Afzal Haroon, Shaukat Arif Hanif, Munawwar Husain
15. Stature Estimation from Skull and Facial Anthropometry in Central Indian Population 57
    Kanchankumar P. Wankhede, Madhukar P. Parchand

16. Pulmonary Candidiasis: Incidental Cause of Death Made on Autopsy 61
Kanwardeep Kaur Jhajj, Paramjit Kaur, Samarjit Kaur Sandhu, Sarita Nibhoria

17. Analysis of Homicidal patterns in Bangalore City, India 64
Karthik SK, Balaji PA, Syed Sadat Ali, Jayaprakash G, Mohan Velu J, Mallikarjun S Ballur

18. A Radiological Study of Epiphyseal Union of Knee Joint for Age Estimation 68
Nemade KS, Kamdi NY, Meshram MM, Parchand MP

19. Cutaneous Manifestations of Spinal Dysraphism in an Adult Male - A Case Report 73
M. Babu, T.C. Anjanamma, B.Venkateswarulu, K.P. Varalakshmi, N. Prabhakar Rao

20. Expression of Dengue Antibodies in Hemolysed Blood Sample in Autopsy - A Case Report 76
M. Babu, B.Venkateswarulu, K.P. Varalakshmi, N. Prabhakar Rao, M. Neeraja

21. Estimation of the Time of Death by Evaluating Histological Changes in the Pulp 80
Madhuri Gawande, Minal Chaudhary, Anita Das

22. Case report - Unusual Cases of Late Deaths in Hanging 83
M.E. Bansude, R.V. Kachare, C.R. Dode

23. Variations in Length of Facial Nerve in the Segments of Temporal Bone of Male Cadavers in South Indian Region 86
Makandar UK, Kulkarni PR

24. Morphometrical Study of Mitral Valve 90
Kulkarni P.R., Makandar UK

25. A Comparative Study of Injuries of Lower Extremity in Junior Foot Ball Players in Both Sexes 94
Makandar UK, Kulkarni PR

26. Incidence and Patterns of Skull Fractures in Accidental Deaths at Davanagere, Karnataka 99
Manish K, Gurudatta S Pawar, Rajesh Sangram, Jyothi N S

27. Study of Morphological Changes in Endometrium in Cases of Abnormal Uterine Bleeding and its Correlation with Age, Clinical Diagnosis and Bleeding Pattern 103
Manish Kumar Singhal, Anshu Gupta, Shivani Kalhan, Onis Singhal, Viplesh Kaur, Veena K Sharma, Sharmila Dudani

28. Sewer Gas Poisoning: A Case Report 108
Maroti D Dake, M B Shrigiriwar

29. Estimation of Stature from Head Height in Hyderabad 112
Mohammed Habeebuddin Shaji

30. Credibility of Pubic Length, Ischium Length and Ischio-pubic Index in Identification of Gender 115
Shreekrishna H.K., Nagesh Kuppast, Vinoth, Anand Mugadlimath, Udaykumar C. Nuchhi

31. A Study of Changing Trends and Patterns of Poisoning at Bangalore 119
Naveen Kumar T., Jagannatha S.R., Ananda K

32. Assessment of Sexual Dimorphism in Permanent Canines among Different Indian Ethnic Groups - A comparative study 123
Nidhi Dhakar, Mahesh Jain, Swati Saawarn, Nisheeth Saawarn, Madhusudan AS

33. Hospital Waste Management: Indian Perspective 127
Nishat Ahmed Sheikh

34. Medical Certification of Cause of Death at General Hospital Nalgonda 130
Nishat Ahmed Sheikh, Poonam Naik, Abhay Nirgude

35. Cheiloscopy: A Tool for Solving Crime and Identification 133
Nishat Ahmed Sheikh, Pradeep S. Londhe

36. Evaluation of Blood Ethanol and Opium Level in Non Survived Drivers Due to Traffic Accidents 136
Amir Masoud Hashemian, Hamid Kariman, Omid Mehrpour, Nastaran Eizadi-Mood

37. Death Due to Diffuse Axonal Injury: A Case Report 140
Pradeep K Saralaya, N.T. Satish, S. Harish

38. Investigating the Force Relative to Blood Stain Size and Pattern 144
Raghuraghavendra Sundarrajan, Rahul Pathak

39. Analysis of Body Fluids by Head Space-Gas Chromatography (HS-GC) in Illicit Liquor Poisoning 150
Rawat B.R.

40. Sexual dimorphism of human femur in Western India 155
Ruta N. Ramteerthakar, R. A .Joshi

41. Medico Legal Autopsies of Custodial Deaths in Mumbai Region - A two Years Prospective Study 159
Sachin S. Sonawane, S.D.Nanandkar

42. Aberrations of Human Gender Identification by Amelogenin Test in Libyans (Benghazi City) 163
Samir Elmrghni, Michael Shaw, Nadir, Ali, Ron A Dixon, D. Ross Williams

43. Effect of Smoking on Plasma Homocysteine Level in Some Libyans as a Risk Factor for Cardiovascular Disease ... 167
Samir Elmrghni, Mahmud Kaddora

44. Sodium Dichromate Poisoning - A case Report 171
Sanjay Sukumar, Udit B Das

45. Age Estimation by Permanent Teeth Eruption Among School Children in Madurai (South India) 174
Sajeev Slater, Senthil Vel

46. Bilateral Optic Neuritis Following Ressell's Viper's Bite-A Rare Complication 178
S.C.Aundhakar, Sanket K. Mahajan, Makarand B. Mane, Swapnil S. Arsekar

47. Palm Length - A Predictor of Stature in Indian Population 181
Srinivasa Peddy.P, Asha K.R., Shashidhar C Mestri, Lakshmi Prabha Subhash

48. Crime, Society and Role of Forensic Medicine 184
Subodh K. Singh

49. Sudden Death Due to Pulmonary Embolism: Two Case Reports 188
Sanjay Sukumar, Udit B. Das

50. Use of Carpal Bones as Skeletal Maturity Indicators in Indian Pediatric Age Group 191
Sumit T. Patil, Madhukar P. Parchand, M. M. Meshram

51. Age Estimation Radiologically from Epiphyseal Union at Wrist Joint Among Subjects in Davanagere District 195
Sunil S Kadam, Viswanathan K G

52. A Study of Burn Deaths in North Karnataka 199
Sunil P. Tapse, Vinay B. Shetty, Anil D. Jinturkar

53. Variations in ABH Antigenic Stability of Dried Bloodstains from Different Surfaces with Age 202
S. K. Mishra, A.S. Yadav

54. Ossification of Bones of the Knee Joint in 17 to 20 Years of Age in South Indian Population 205
Talwad.R.V.1, Makandar UK

55. Evaluation of Dowry Related Crimes in Bijapur City 209
Nuchhi UC1, Gannur DG, K. Yoganarasimha

56. An Autopsy Study of Sudden Natural Deaths in Victoria Hospital, Bowring and Lady Curzon Hospitals, Bangalore 214
Y. Udayashankar

57. Role of Nature of Anions in the Toxicities of Various Lead Salts Lead Nitrate, Lead Sulphate & Lead Acetate on the Different Organs of Female Rats 220
Vaneet Dhir

58. Dental Profiling- A Boon for the Forensic World 225
Vanishree. N, Jeswin. J, Madhusudan

59. Aluminum Phosphide Poisoning: An Agent of Sure Death 231
Varun Vijay Mahajan, Iesha Pargal

60. Meckel Gruber Syndrome- A Case Report 236
Vardendra Kulkarni, Seema Bijjaragi, Satish Belagatti, Sunil S Kadam

61. Clinical Profile and Outcome of Organophosphorus Poisoning at Tertiary Care Centre in Western Maharashtra ... 239
Virendra C. Patil, Harsha V. Patil, Vaibhav Agrawal, Satish Tryambake, Swapana Kore

62. Current Status of Hair Analysis in Drug Detection 244
Vivek Sharma, Shalini Sharma, Geetanjali Sharma, Luv Sharma

63. Study of Pattern of Head Injury in Drivers of Two Wheeler Auto Vehicle Accidents 248
Pragnesh Parmar, Mohammed Ziyauddin G Saiyed, Pratik R Patel

64. A Study of Post-Mortem Histopathological Changes in Septaecemia Due to Burns 253
Yogiraj V, Vinayaka Prasanna K, Chaithanya R

65. Study of Pattern of Injuries in Fatal Railway Accidents 257
NT Satish, S Harish, Girish Chandra YP

66. Determination of Sex from Clavicle in Vidarbha Region of Maharashtra 262
Trupti K Balvir, MSM Bashir, AS Rahule, Badwaik P

Cresol from Toluene Metabolites in Head Space of Urine And Blood From Glue Sniffing Activity Using Gas Chromatography- Mass Spectrophotometry

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ABSTRACT

Glue sniffing has been an addiction problem to countries all over the world since late nineteenth century. Back then, it is the adults who are mostly abusers but recently, the trend have shifted towards younger generations. More teenagers have been recorded to be glue sniffers. Most literatures indicated that it is the volatile solvent present in glue, the substance that gives the abusers 'high' or the intoxicating effects that they pursue. To study the metabolites of these substances, blood and urine samples from positive glue abusers obtained from local agencies were analyzed. The results showed that there were significant amount of *para*- cresol in the head space which is one of toluene's metabolites.

Key words: *Glue, Toluene, Gas Chromatography-Mass Spectroscopy Detector, Head Space*

INTRODUCTION

History recorded that since the late nineteenth century, glue sniffing has been an addiction problem. Back then, the abusers were mostly adults but as of late, growing number of children and teenagers are found to be abusing the volatile substance found in glue (Cairney *et al*., 2002; Lubman *et al*., 2006). For instance, a nationwide survey conducted in Korea found that more than 90% of male teenagers while over 60% female teenagers have at least once sniffed glue in their lifetime (Bobae *et. al*., 2011). The volatile substances give an intoxicating or 'high' feeling effect to the abusers and this feeling of euphoria is the cause of the addiction (Flanagan and Ives, 1994). In some respects, solvent in glue is even more abused than drugs. This is because glue is generally easier to get and cheaper than drugs (Arif and Navaratnam, 1988).

In Malaysia, the abuse of organic volatile solvents has been observed since the early 1980s. The problem of solvent abuse is predominant in East Malaysia (i.e., Sabah and Sarawak) and in Johor (Navaratnam et. al, 1988). The exact number of glue sniffers is not available as solvent abuse is considered as an alternative to drugs, thus it is not reported to the national registry of abusers in Malaysia (Yacob and Zainalibdin, 2010). The volatile solvent in glue has a long term negative health effect when inhaled. These substances pose dangerous after effects on the central nervous system and the normal developments of psychological, emotional and neurobiological of the individual abusing it (Balster, 1998; Kurtzman *et al*., 2001; Bowen *et al*., 2006; Lubman *et al*., 2006).

As the solvents in the glue inhaled are volatile substances, head space method may be the best method in 'capturing' what exactly is the glue sniffers are inhaling. In this study, the most commonly abused glue types are identified and the most abundant volatile substance contained in the glue samples are determined using gas spectrometer mass spectrometry using head space method. Blood and urine samples of glue sniffers are also being studied via the same method. This project has considerable potential and the positive impact of its findings to Malaysia and the society can be high. The results of this project will go a long way towards helping the Royal Police and National Anti-Drug agency to prevent this problem from becoming worse.

EXPERIMENTAL

Instrumentation

GC-MSD head space utilized 10 µL head space Hamilton syringe from Agilent Technologies,

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USA with a HP-5 capillary column of dimension 30 m × 0.30 µm x 0.30 mm brand J&W Scientific, USA. An isothermal condition employed with 65ºC for 45 minutes following an initial oven temperature at 150ºC. The injector temperature was set at 150ºC and detector temperature at 200ºC and helium flow rate of 0.7 mL/min (Saad and Pakiam, 2005).

Blood and urine samples

Blood and urine samples were kindly supplied by the Pathologist Lab, Forensic Division, Sultanah Aminah Hospital. The samples from the lab ranges from cases of glue sniffing, samples from accident cases where the driver's impairment was caused by toluene inhalation, crime (usually violent) where toluene may be related to behavior of the individuals involved, and cases in which the presence of toluene in blood and urine has some relationship to the cause of death or the general health of the person (Serap *et al*., 2001).

Urine samples of a secondary school's students that were intended for glue sniffing screening were obtained with the co-operation of the National Anti Drug Agency. Urine and blood sample preparations were conducted by the National Anti Drug Agency and Pathologist Lab, Forensic Division, Sultanah Aminah Hospital. All of the urine samples obtained was screened using Glue Sniffing Kit (G.S. Kit) (Yacob and Zainalibdin, 2010) prior to confirmation test using GC-MS with head space method.

Preparation of Toluene Standard

1 mL of toluene 99.7% was pipetted into a 100 mL volumetric flask and then made up to the mark with dichloromethane 99.7%. Toluene standard was sonicated in ultrasonic water bath.

Sample Preparation

The urine samples were added with sodium fluoride as preservative while the blood sample was added with sodium citrate as preservative and anti coagulation. Blood and urine specimens were stored at 0-5°C.

10 mL of urine or blood sample was placed in a head space bottle. The samples were heated in the | oven at 100ºC for ½ hour. The headspace was withdrawn with a headspace syringe and injected at the GC-MSD.

RESULTS AND DISCUSSIONS

Toluene metabolite results

GC-MS with headspace method was used for qualitative analysis for determination of toluene metabolites in this research. All the samples were analyzed by this method and the results are showed in Figure 1 to Figure 11.

The retention time for *para*-cresol was recorded at 15.9 minutes, N-N-dimethyl-Benzeethanamine at 18.1 minutes and N-(methylbenzoyl)-methyl ester-glycine at 13.3 minutes. Table 2 below shows the tabulated results for Gas Chromatography Mass Spectrometry

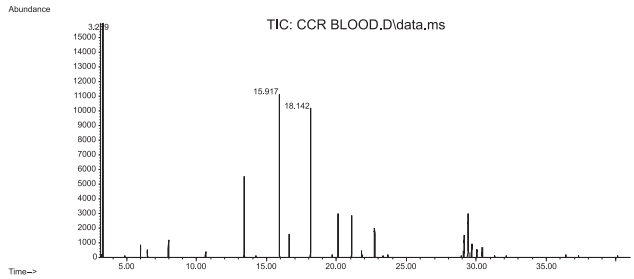

Fig. 1. GC-MS Head Space chromatogram for sample Blood 1.

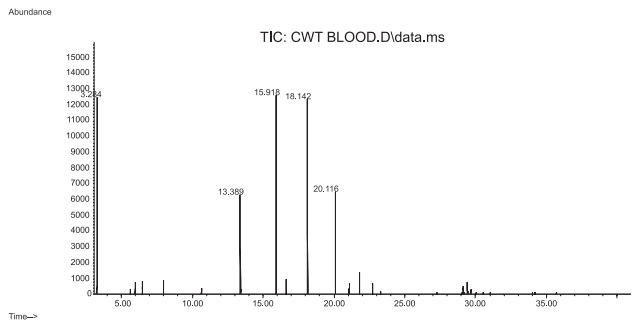

Fig. 2. GC-MS Head Space chromatogram for sample Blood 2.

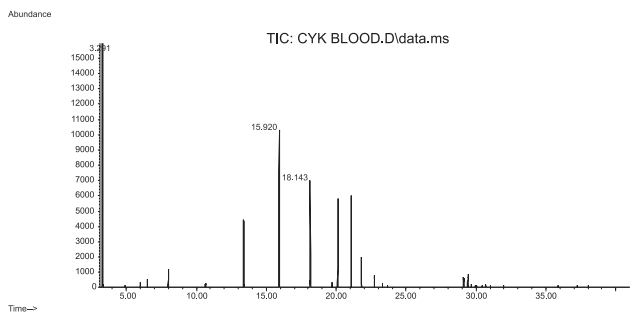

Fig. 3. GC-MS Head Space chromatogram for sample Blood 3

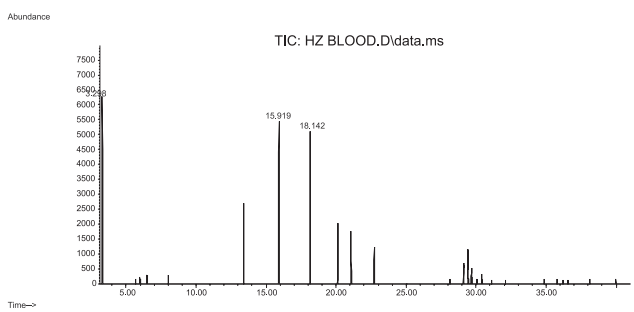

Fig. 4. GC-MS Head Space chromatogram for sample Blood 3

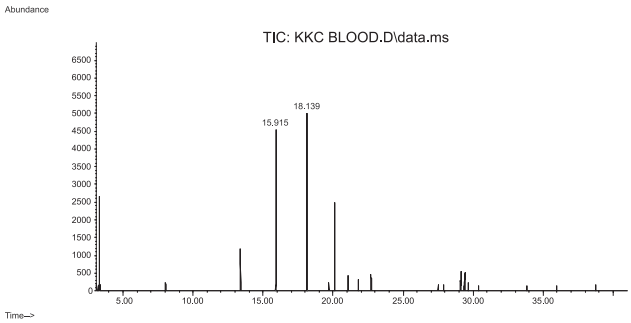

Fig. 5. GC-MS Head Space chromatogram for sample Blood 5

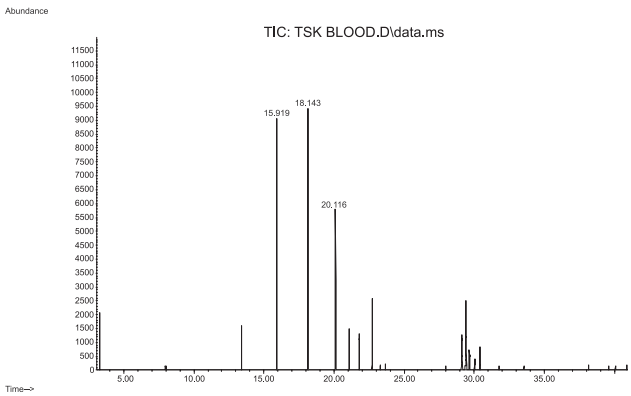

Fig. 6. GC-MS Head Space chromatogram for sample Blood 6

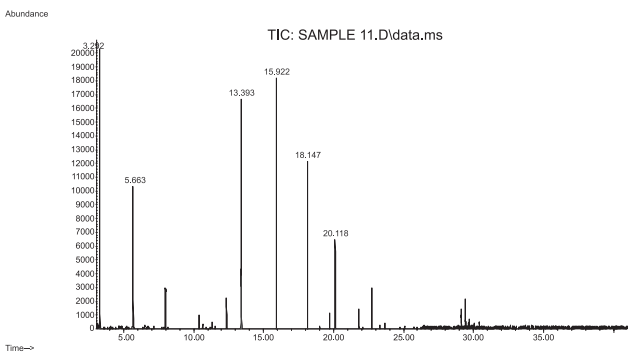

Fig. 7. GC-MS Head Space chromatogram for sample Urine 1

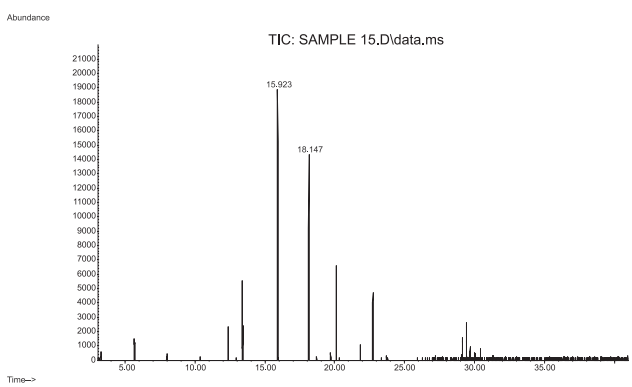

Fig. 8. GC-MS Head Space chromatogram for sample Urine 2

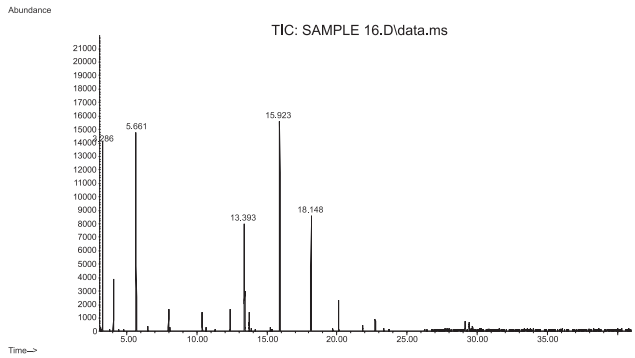

Fig. 9. GC-MS Head Space chromatogram for sample Urine 3

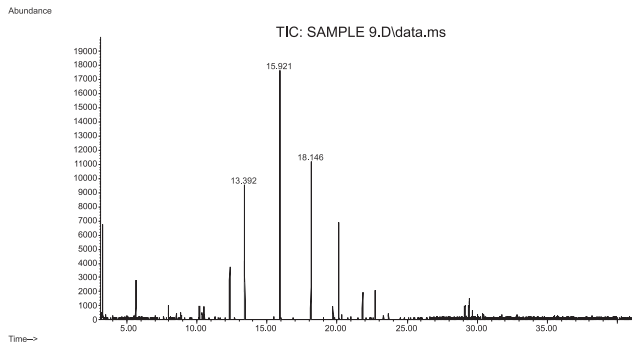

Fig. 10. GC-MS Head Space chromatogram for sample Urine 4

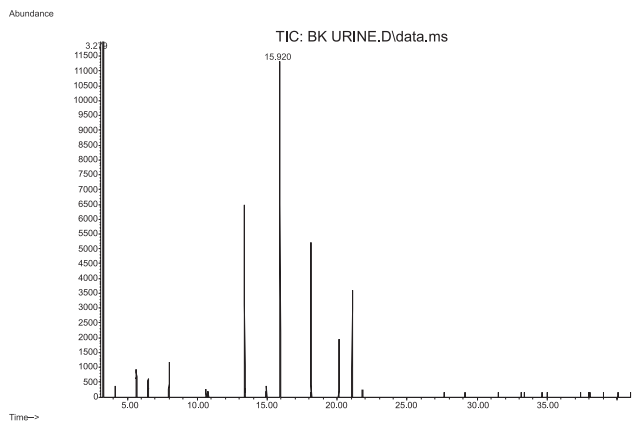

Fig. 11. GC-MS Head Space chromatogram for sample Urine 5

(GC-MS) with head space method of all the samples together with its respective probability.

Table 2. Result of Gas Chromatography Mass Spectrometry (GC-MS) with head space method of toluene metabolites in urine and blood.

Sample Name	Compound found	Probability
Blood 1	N-N-dimethyl-Benzeethanamine	89
	p-cresol	87
Blood 2	N-N-dimethyl-Benzeethanamine	88
	p-cresol	89
Blood 3	N-N-dimethyl-Benzeethanamine	86
	p-cresol	87
Blood 4	N-N-dimethyl-Benzeethanamine	89
	p-cresol	87
Blood 5	N-N-dimethyl-Benzeethanamine	86
	p-cresol	85
Blood 6	N-N-dimethyl-Benzeethanamine	87
	p-cresol	88
	N-(methylbenzoyl)-methyl ester-glycine	88
Urine 1	N-N-dimethyl-Benzeethanamine	89
	p-cresol	87
	N-(methylbenzoyl)-methyl ester-glycine	90
Urine 2	N-N-dimethyl-Benzeethanamine	86
	p-cresol	85
Urine 3	N-N-dimethyl-Benzeethanamine	90
	p-cresol	87
	N-(methylbenzoyl)-methyl ester-glycine	89
Urine 4	N-N-dimethyl-Benzeethanamine	86
	p-cresol	87
	N-(methylbenzoyl)-methyl ester-glycine	88
Urine 5	*p*-cresol	86

From the results shown, all blood samples 1, 2, 3, 4, 5, and 6 and urine samples 1, 2, 3, 4 and 5 contained *p*-cresol while all of the blood samples and urine samples except for urine sample 5 had N-N-dimethyl-Benzeethanamine. Blood sample 6 and urine samples 1, 3 and 4 showed presence of N-(methylbenzoyl)-methyl ester-glycine. In this case, the N-(methylbenzoyl)-methyl ester-glycine has a similar structure as a hippuric acid which in turn indicates that these samples contain high concentration of hippuric acid. Hippuric acid, as has been showed in previous study (Yacob and Zinalibdin, 2009) to be the major metabolites of toluene in dissolved form found in blood and urine. Hippuric acid however was also the metabolites from benzoic acid a preservative obtain in food sample that will give a false result from glue sniffing activity.

p-cresol on the other hand was detected in all of the head space of the urine and blood samples. Yamazaki *et al*., (1990) reported that 0.4-1.1 % *p*- and *o*-cresol were produced when an individual is exposed to toluene. If the amount of cresol can be determine qualitatively using GC-MS head space, thus, it can be conclusively indicate glue sniffing activity.

The results from this study is coherent with previous studies that showed toluene when administered into the human body, undergo several steps of metabolism which results in the production of hippuric acid, *ortho*-, *para*- and *meta*-cresol in urine and blood (Chapman *et al*., 1990; Kawamoto *et al*., 1994; Tassaneeyakul *et al*., 1996; Wilkins-Haug, 1997;Agency for Toxic Substances and Disease Registry, 2000). Figures 12 below show the metabolism pathways of toluene that has been proven in previous studies and is supported by the findings of the current research. The results from this study also prove that in the head space of blood and urine of glue sniffers, *para*-cresol is abundant. *Meta*- and *ortho*-cresol's was not detected most probably due to its amount too insignificant to detect using this method.

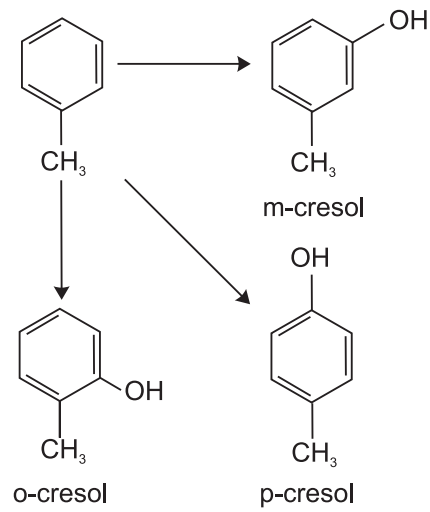

Fig. 12. Toluene metabolism to *ortho*-, *para*- and *meta*-cresol pathway.

From the results shown, it is proven that GC-MS with head space method is an efficient way of detecting volatile solvents abused in glue and its metabolites in blood and urine.

CONCLUSION

Toluene is used in industry for the synthesis of the chemical compound and as the solvent for paint, print ink and adhesive. In a previous experiment, toluene was showed to be the major component in head space of commercial glue. Subsequently, in the current study, *para*-cresol was found to be the most abundant metabolite of toluene in blood and urine of glue sniffers. GC-MS with head space method has proven to be an easier confirmation test in comparison to GC-MS as the analytes are volatile compounds.

REFERENCES

1. Arif, A.E. and Navaratnam V., eds. Abuse of Volatile Solvents and Inhalants: Papers Presented at a WHO Advisory Meeting. International Monograph Series No. 1. Penang, Malaysia: Drug Research Centre, Universiti Sains Malaysia, 1988. pp. 16-23.

2. Nicholas K, Zili Sloboda, Maria De La Rosa. (1995). Epidemiology of Inhalant Abuse: An International Perspective, U.S Department of Health and Human Service, Public Health Service, National Institute of Health. Journal of Inhalant Abuse, Page 2-3.
3. Sharifah, M.T.F. "Report on the Inhalant Abuse Problem in Sarawak, East Malaysia." Paper presented at the Twelfth IFNGO Conference, Singapore, November 12-16, 1990.
4. Saad S. and Pakiam C. (2005). Screening for Volatile Solvents in Blood by Headspace Gas Chromatography. Department of Chemistry Method, JKM F0540. Pg 1-5.
5. Serap A.A, Pembe O. Yusuf K, Huseyin K. and Suheyla E. (2001). Medicolegal Aspects of Blood-Urine Toluene and Urinary Ortho-Cresol Concentration in Toluene Exposure Turk J Med Sci. 31, 415-419.
6. Yacob, A.R. Zainalibdin, M.R. (2009). Characterization using instrumentation of local glue related glue sniffing activity. Indian Journal of Forensic Medicine and Toxicology. Vol 3 (2): 58-64.
7. Yacob, A.R. and Zainalibdin, M.R.(2010). G.S. Kit: Mobile test kit for glue sniffing screening. Advanced Management Science. 399-402.
8. Yamazaki K., Tanaka E. and Misawa S. (1992). Urinary ortho-cresol Concentrations as an Indicator of Toluene Inhalation in Glue Sniffer J For Sci 32, 215-223.
9. Flanagan, R.J. and Ives, R.J. (1994). Volatile substance abuse. Bull Narc. 46, 49-78.
10. Bobae, K, Siwon, K, Sosun, K, Dong-Kye, L., Yu-Jin, P., Myung-Duck, K, Jae-Shin, L, and Suhkmann, K, (2011). 1H NMR spectroscopic identification of a glue sniffing biomarker. Forensic Science International. 209, 120-125.
11. Agency for Toxic Substances and Disease Registry. (2000).Toxicological profile for toluene. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
12. Wilkins-Haug, L. (). Teratogen update: toluene. Teratology. 55 (2): 145-151.
13. Tassaneeyakul, W., Birkett, D.J., Edwards, J.W., Veronese, M.E., Tassaneeyakul, W., Tukey, R.H. and Miners, J.O. (1996). Human cytochrome P450 isoform specificity in the regioselective metabolism of toluene and o-, m- and p-xylene. Journal of Pharmacology and Experimental Therapeutics 276 (1): 101-108.
14. Chapman, D.E., Moore, T.J., Michener, S.R. and Powis, G. (1990). Metabolism and covalent binding of [14C]toluene by human and rat liver microsomal fractions and liver slices. Drug Metabolism and Disposition 18 (6): 929-936.
15. Kawamoto, T Matsuno, K, Kodama, Y, Murata, K., Matsuda, S. (1994). ALDH2 polymorphism and biological monitoring of toluene. Archives of Environmental Health 49 (5): 332-336.

Profile and Pattern of Suicidal and Homicidal Burn Victims at a Tertiary Care Hospital in Northern India

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ABSTRACT

Aim : To determine the demographic and socio-cultural factors, pattern and risk factors of the burn cases. An additional objective was to assess the factors affecting outcomes of burn injury cases.

Methods : The present prospective epidemiological study was carried out among suicide and homicidal burn victims seeking care at Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR), Mullana between May 1st 2010 to December 31st 2011. After obtaining an informed consent, the required data was collected using a pre-designed and pre-tested proforma.

Results : Female patients (55.91%) out numbered the male patients (44.09%). Majority of the patients had primary level of education (31.71%) and most of females (35.43%) were married for less than 5 years. A cooking appliance was the source of burn in more than 50% of the females. 81.69% of the suicidal cases and 57.78% of the homicidal cases succumbed to burns. 51.07% died who were doused with water as an immediate response. Among the victims who responded by shouting or running, 44.44% died and 36.11% recovered

Conclusion : The results of this study clearly highlighted the specific epidemiological features of burns patients in our area, and this should provide the necessary information to develop adequate burn prevention programmes, thereby reducing the frequency of burns and burn-related deaths.

Key words: *Epidemiology, Burns, Homicide, Suicide, TBSA.*

INTRODUCTION

Burns is a serious trauma and it destroys many hopes and dreams for ever. Apart from high numbers of deaths, the pain, suffering and agony of burn survivors are immeasurable. Injury due to burns arising from unintentional incidents (accidents) and intentional incidents (suicide or homicide) is one of the leading causes of death and disability globally.[1] Burn injuries rank among the most severe types of injuries suffered by the human body with an attendant high mortality and morbidity rate.[2] The consequences of burns are protean and in most cases devastating for the patient.

The magnitude of burn related fatalities in India is alarming. An estimate of burn injury related deaths in 2010 was 24414 in India.[3] However this is an underestimate as not all such deaths are reported. Number of deaths due to burns to be 1,63,000 based on estimates, while the official reported deaths were 22,000 in the 2001.[4] An estimated 1,84,000 persons died of burn injuries in the countries of South East Asia Region (SEAR) in 2002 with 6.55 million disability adjusted life years (DALY) lost due to burns.[5]

As the etiologic factors of burn injuries vary considerably from place to place careful analysis of the epidemiological features is needed to plan the prevention modalities. No studies have been carried out in the region to know the various epidemiological factors responsible for burns. Hence present study was conducted with an objective to determine the demographic and socio-cultural factors, pattern and risk factors of the burn cases. An additional objective was to assess the factors affecting outcomes of burn injury cases.

MATERIALS AND METHODS

The present prospective study was conducted was carried out at Maharishi Markandeshwar Institute of Medical Sciences And Research (MMIMSR), Mullana (Ambala). All suicidal and homicidal burn victims admitted in hospital from May 1st 2010 to December

31st 2011 formed the study subjects. For the purpose of the study, the term burn injury was defined as a body lesion due to an external cause, either intentional (homicidal or suicidal) or unintentional (accidental) resulting from a sudden exposure to energy (mechanical, electrical, thermal, chemical, or radiant) generated by agent host interaction.[6]

After obtaining an informed consent, the data regarding demographic and socio cultural variables; types, modes, causes, risk factors; and factors affecting outcome was collected using a detailed pre-designed and pre-tested proforma from all the burn injuries admitted in the hospital during the study period. The data was obtained by questionnaire-interview with the patients themselves if they were mature and well enough to answer the questions. In the case of younger children or patients who were not well enough as a result of severe burn injury, the data was obtained from close relatives. Outcome of interest was captured using personal history and/or inquest report and/or dying declaration whichever was applicable.

The collected data was entered in Microsoft Excel. Coding of the variables was done. SPSS version 11.5 was used for analysis. Interpretation of the collected data was done by using appropriate statistical methods like percentage, proportions, bar and line diagrams. Chi square (x2) test was applied to test significance wherever applicable. Total burn surface area (TBSA)

RESULTS

Out of 611 burn cases admitted in the hospital during the study period, 78 (12.77%) were suicidal cases and 49 (8.02%) were homicidal cases. Accidental burn cases were not included in the analysis.

A. Socio-demographic characteristics of the burn injury cases

Out of total 127 suicidal and homicidal cases, 56 (44.09%) were males and remaining 71 (55.91%) were females. Majority of victims 84 (66.14%) were in age group of 15-44 years. Most 112 (88.18%) of the victims were Hindus. Of the 127 victims interviewed 106 were married accounting for 83.46% of the victims. 39 victims (31.71%) had a primary level of education while 19.68% of the victims were illiterates. Maximum number of burn cases were in housewives (63.78%) followed by male unskilled workers (40.94%) and male skilled workers (27.56%). Out of total 3.94% of cases were observed in unemployed followed by professionals and business people. Majority of the females (35.43%) were married for less than 5 years whereas majority of the males (44.88%) were married for more than 10 years.

B. Pattern of burn injuries

Month wise pattern of burn injury cases

On analysing distribution of burn cases based on monthly admission it was observed that maximum number of admissions (18.11%) were in November 2010 followed by 11.02% in December 2010. Minimum number of admissions were in month of July 2011.

Time of burn injury

Majority (31.49%) of burn injuries occurred between 4 pm to 8 pm. 18.11% burns occurred between 8 am to 12 noon and 16.53% between 8 pm to 12 midnight. ($P<0.05$)

Location of Burns

Maximum number (93.70%) of burn injuries in females occurred at home compared to 4.72% burn injuries occurring outdoors whereas, in males 58.27% sustained burns at home and 40.16% outdoors. Out of the total domestic burns, majority (74.80%) occurred in the kitchen / kitchen cum living room / kitchen cum bathroom, followed by (12.59%) occurring in the bedroom / bathroom. ($P<0.05$)

Risk factors for burn injuries

Majority of suicidal cases, 51.61% in males and 65.96% in females were due to marital or family dispute. Failure in affair contributed to 10.64% of suicidal cases in females. Majority of the homicidal cases (72%) in males were due to marital or financial dispute followed by 20% due to a property dispute. (Table 1)

Table 1. Distribution of subjects according to the circumstances for burn injury

Distribution of suicidal cases according to the circumstance for burn injury			
Circumstance	**Male**	**Female**	**Total**
Torture for dowry	00 (0.00)	1 (2.13)	1 (2.13)
Family or marital dispute	16 (51.61)	31 (65.96)	47 (117.57)
Debt	6 (19.35)	00 (0.00)	6 (19.35)
Failure in affair	2 (6.45)	5 (10.64)	7 (17.09)
Verbal abuse by parents	3 (9.68)	2 (4.26)	5 (13.94)
Attempt to rape	00 (0.00)	1 (2.13)	1 (2.13)
Depression	1 (3.23)	3 (6.38)	4 (9.61)
Others	3 (9.68)	4 (8.50)	7 (18.18)
Distribution of homicidal cases according to the circumstance for burn injury			
Dowry	00 (0.00)	4 (16.67)	4 (16.67)
Marital or financial dispute	18 (72.00)	17 (70.83)	35 (142.83)
Property dispute	5 (20.00)	2 (8.33)	7 (28.33)
Others	2 (8.00)	1 (4.17)	3 (12.7)

Types and source of the burn injury cases

Our study revealed that 89.73% of the females and 70.87% of the males sustained flame burns. Cooking appliances were responsible for 52.76% and 17.32% of the burn cases in females and males respectively.

Scalds were responsible for 11.81 % in males and 7.87% in females. Electrical injuries were more in male patients (14.96 %) than in female patients (3.94%).

C. Factors affecting outcomes of burn injury cases

In the present study, it was observed that majority of both suicidal cases (60.26%) and homicidal cases (34.71%) had TBSA more than 80%. (p< 0.001) (Table 2)

Table 2. Distribution of subjects according to mode and TBSA.

TBSA	MODE OF BURN	
	Suicidal	Homicidal
	Number (%)	Number (%)
< 19 %	0 (00.00)	7 (14.28)
20-39	9 (11.54)	15 (30.61)
40-59	11 (14.10)	3 (6.12)
60-79	11 (14.10)	7 (14.28)
>80 %	47 (60.26)	17 (34.71)
Total	78 (100.00)	49 (100.00)
p< 0.001		

It was also observed that 81.69% of the suicidal cases and 57.78% of the homicidal cases finally resulted in death. (Table 3)

Table 3. Distribution burn patients according to the mode of burn and outcome*

Outcome	Suicidal	Homicidal
	Number (%)	Number (%)
Recovery	6 (8.45)	10 (22.22)
Residual disability	7 (9.86)	9 (20.00)
Death	58 (81.69)	26 (57.78)
Total	71 (100.00)	45 (100.00)

*Against Medical Advice cases excluded. (p< 0.001)

It was also observed that 51.07% died who were doused with water as an immediate response. 58.33% of the cases where ignited clothes were removed also had recovery. Among the victims who responded by shouting or running, 44.44% died and 36.11% recovered. (Table 4)

DISCUSSION

The epidemiology of burns vary from place to place being a function of the level of civilization, industrialization and culture among other things. Also the lack of uniformity between methodologies in addition to the existence of a plethora of variables and differences in the periods of study, makes any comparison with other studies difficult.[7]

In the present study, more than 50% of the cases were females. This finding is similar to studies undertaken in various hospitals all over India.[8-10] Studies conducted in Turkey and Singapore revealed that males were more affected than females in burn injuries.[11,12] This could perhaps be due to higher incidence of industrial and recreational burns among males in such countries.

The present study showed that majority of the burn patients were Hindus (88.18%) which is similar to another Indian study where 75.2% of the victims were Hindus.[13] Victims having primary education were more (31.71%) when compared with those with illiterates. Current finding is in contrast with other studies conducted in Pune[10] and Madras[14] where majority were illiterates.

Our study revealed that majority of the female patients were married for less than 5 years and majority of the males were married for more than 10 years. Another study8 also revealed similar findings. This could be explained by the fact that newly married girls are inexperienced in handling cooking appliances and also have to bear harassment by in-laws and husbands for the sake of dowry. In the case of males, majority were married for more than 10 years. The reason could be responsibilities of family life and social problems like debt and alcoholism.

Regarding location of burns, 93.7% of burn injuries in females occurred at home compared to 4.72% occurring outdoors. Reports from industrialized countries indicate that domestic burns are also the

Table 4. Distribution of burn patients according to immediate response* shown and the outcome.

Immediate Response	Recovery		Residual Disability		Death		Total	
	No.	%	No.	%	No.	%	No.	%
Dousing with water	11	23.40%	12	25.53%	24	51.07%	47	100%
Dousing with cloth /blanket etc	9	33.33%	7	25.93%	11	40.74%	27	100%
Application of any material	6	75.0%	0	00.0%	2	25.0%	8	100%
Removal of ignited clothes	7	58.33%	1	8.33%	4	33.33%	12	100%
Pulled out of blaze	0	00.0%	1	50.0%	1	50.0%	2	100%
Removal of electric wire by stick	0	00.0%	2	66.67%	1	33.33%	3	100%
Responded by shouting / running	13	36.11%	7	19.44%	16	44.44%	36	100%
Others	1	50.0%	0	00.0%	1	50.0%	2	100%

Multiple responses*

commonest as reported in England.[15] Majority of the domestic burns (77.8%) occurred in the kitchen or kitchen cum living room. This finding is similar to another study where 83% of the domestic burns occurred in the kitchen.[10] All these findings indicate that the kitchen is a danger area in every home where there are unsafe cooking appliances and there is a need for education in this respect.

The present study revealed that 23.4% of the burn victims were doused with water as a immediate response. In contrast, in another Indian study, it was noted that response was adjudged appropriate (Ex stop, dop and roll or pour water) in only 4.6% of the case and inappropriate (running, trying to put out fire by hands, applying oil, ink) in the remaining 95.4%.[10] out of the victims that were doused with water as an immediate response, 51.6% died. These findings suggest that despite dousing with water, majority have died, due to extensive burns. Removal of ignited clothes has shown a better outcome.

The present study showed that majority of the males (51.61%) attempted suicide because of marital or family dispute. Marriage has been found to have both a protective and stressful role in the etiology of suicide. Depression has been identified among those with fatal and attempted suicides, varying from 14% - 67% in various studies.[16,17] Majority of the homicidal burns (72.0%) in males were again due to marital or family dispute followed by 20% due to a property dispute. Social status, dependence, responsibility and issues with regard to family have also been found to be influencing factors.[16]

CONCLUSION

The results of this study clearly highlighted the specific epidemiological features of burns patients in our area, and this should provide the necessary information to develop adequate burn prevention programmes, thereby reducing the frequency of burns and burn-related deaths.

REFERENCES

1. Attia AF, Sherif AA, Mandil AM, Massoud NM, Arafa MA, Mervat W et al. Epidemiological and sociocultural study of burn patients in Alexandria, Egypt. Eastern Mediterranean Health Journal 1997; 3(3): 452-461.
2. MacArthur JD, Moore FD. Epidemiology of burns. JAMA 1975; 231(3): 259-263.
3. Accidental Deaths and Suicides in India 2010, National Crime Records Bureau. Ministry of Home Affairs. Government of India, New Delhi.
4. G. Gururaj, Injury & Violence in India: Facts and Figures National Institute of Mental Health and Neuro Sciences, Publication no 82, Bangalore. Available from: URL: www.nimhans.kar.nic.in/epidemiology/bisp/ivi2011.pdf (Cited on 27.04.2012)
5. Park K. Text Book of Preventive and Social Medicine, 18th Ed. Jabalpur: Banarasidas, Bhanot Publishers; 2005.
6. Thacker SB, Mackenzie EJ. Injury prevention and control. Epidemiological review 2003; 25: 1-2. Baltimore, Maryland, USA: Oxford University Press, John Hopkins Bloomberg School of Public Health.
7. Obalanji JK, Oginni FO, Bankole JO, Olaside AA. A ten - year review of burn cases seen in a Nigerian Teaching Hospital. Journal of Burns and Surgical Wound Care 2003;2(1):1. Available from: URL: http://www.journalofburns.com. (Cited on 27.04.2012)
8. Gupata RK, Srivastava AK. Study of fatal burn cases in Kanpur, India. Forensic Science International 1988; 37: 81-89.
9. Sidhu DS, Sodi GS, Bannerjee AK. Mortality profile in trauma victims. JIMA 1993; 91(1): 16-8.
10. Singh MV, Ganguli SK, Aiyanna BM. A study of epidemiological aspects of burn injuries. Medical Journal of Armed Forces in India Oct 1996; 52(4): 229-232.
11. Stuart PP. Burn epidemiology in the Brisbane and Queensland area. Burns 2005; 31S: S27-S31.
12. Coruh A, Gunay GK, Esmaoglu A. A Seven - year burn unit experience in Kayseri, Turkey: 1996-2002. J Burn Care Rehabil 2005; 26: 79-84.
13. Subrahmanyam M, Joshi AV. Analysis of burn injuries treated during a one year period at a district hospital in India. Annals of burns and Fire Disasters June 2003; 16(2): 74-76.
14. Jayaraman V, Ramakrishnan MK, Davies MR. Burns in Madras, India: an analysis of 1368 patients in 1 year. Burns Aug 1993; 19(4): 339-344.
15. Cason JS. Treatment of Burns, 1st Ed. New York: Harper and Row Publication: 1981.
16. Gururaj G. Injuries in India - A national perspective. Burden of disease in India, National Commission on macroeconomics and health. New Delhi: MOHFW, Govt. of India; 2005.
17. Pham TN, King JR, Palmieri T, Greenhalgh DG. Predisposing factors for self-inflicted burns. J Burn Care Rehabil 2003; 24: 223-227.

Alkaptonuric Ochronosis : A Case Report

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ABSTRACT

A fifty year old female presented with bluish black discoloration of body tissues and low backache. There was visible bluish black dislocation of sclera and pinna. Patient had extensive involvement of spine with degenerative changes in knee joint.

Key words : *Bluish Black Discoloration, Sclera, Pinna.*

INTRODUCTION

Ochronosis is a rare disorder of metabolism and very few cases of this condition have been reported in India. A case of ochronosis is reported for its rarity and extensive involvement of body tissues and spine.

CASE REPORT

A 50-year-old female presented to our outpatient department with progressive bluish black discoloration of body tissues. Patient reported that she had noticed the bluish black discoloration few years back and it has progressed since then. Patient also complained of stiff back with low back pain.

On examination there was bluish black discoloration of sclera, pinna, eyelids, forehead, cheeks, axillae, nail beds, buccal mucosa and tendons. The patient has slight kyphosis in the thoracic spine with obliteration of lumbar lordosis, straight leg raising test was postive at about 700. There was marked discoloration of both sclera, with pinna of both ears felt stiffer and thicker than normal (Fig.1 & 3).

Laboratory investigations revealed routine urine examination to be normal but positive for homogentisic acid. Urine turned black on exposure to air and addition of sodium hydroxide accelerated the process. On addition of Benedict's reagent to a sample of urine, there was greenish brown precipitate and brownish black supernatant (Fig.2). Other investigations like hemoglobin, ESR, blood urea, uric acid , serum calcium and phosphorus, serum alkaline phosphate were within normal limits. HLA-B-27 was negative.

X-ray of the lumbosacral spine showed slight scoliosis, with disc space calcification and bony ankylosis of lumbar spine. The cervical spine also showed diffuse calcification with secondary degenerative changes (Fig. 4.).There was osteoporosis of vertebral bodies. In the knee joints the joint space was narrowed with marginal osteophytes. Although there is no specific treatment for this condition, the patient was put on ascorbic acid, analgesics and was advised to cut down food rich in tyrosine like cow and buffalo milk and corn.

DISCUSSION

Ochronosis is the bluish black discoloration of certain tissues, such as the ear cartilage and the ocular tissue, seen with alkaptonuria, a rare autosomal recessive metabolic disorder caused by deficiency of homogentisic acid oxidase, the only enzyme capable of catabolizing homogentisic and . There is defect in biochemical pathway by which phenylalanine and tyrosine are normally degraded into fumaric acid and acetoacetic acid. As a result there is accumulation and deposition of HGA in cartilage causing the pigmentation. The affected connective tissue becomes weak and brittle with time; leading to chronic inflammation, degeneration and osteoarthritis.[9] Exogenous ochronosis has been seen after exposure to noxious substances, including phenol, trinitrophenol, benzene,and hydroquinone.

Ochronosis is often recognized at birth when parents note discoloration of urine. Nevertheless, many patients with this metabolic disorder are symptomless until ochronotic changes occur with

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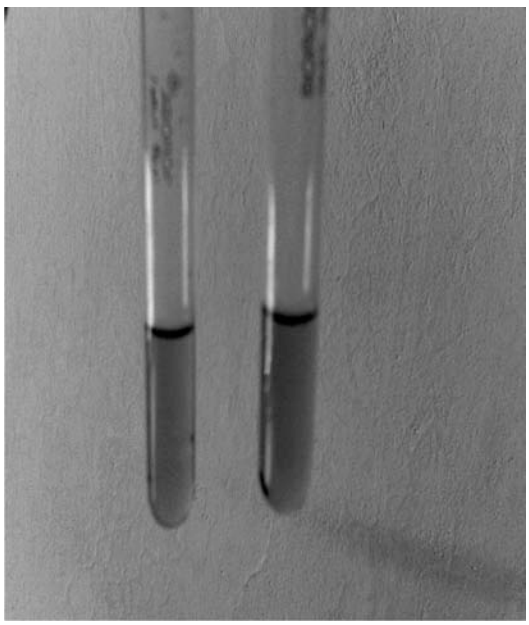
LEGENDS

Fig. 1. Bluish-black discoloration of pinna



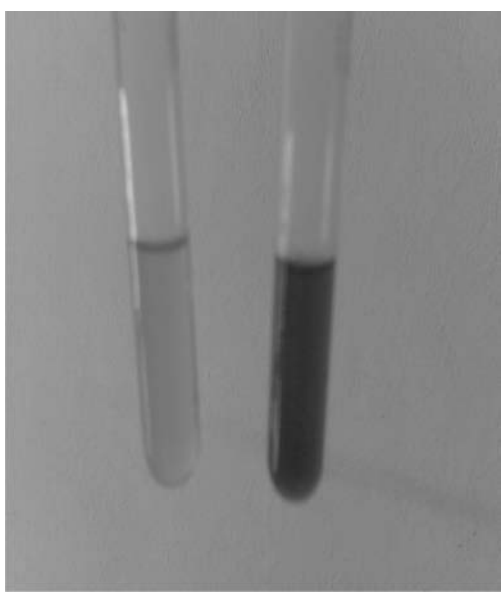


Fig. 2. Urine discoloration after adding Benedict's reagent

Fig. 3. Bluish-black discoloration of pinna

Fig. 4. Cervical spine showing calcification and degenerative changes

bluish black pigmented patches in the sclera developing in patients aged 30-39 years.[7] The fourth decade often marks the onset of thickening and bluish-black discoloration of ear cartilage. Other body locations affected are eyelids, forehead, cheeks, axillae, genital region, nail beds, buccal mucosa, larynx,

tympanic ear drum and the tendons. Ochronotic arthropathy develops later with arthritic symptoms.[6,8]

Different authors have reported the involvement of different symptoms in the body. Palmoplantar pigmentation associated with blunt pigmented patches over the sclera of the eyes have been reported by Vijaikumar et. al.[1] Gulati as well Shetty et. al reported alkaptonuric spondylitis associated with bladder stone and calcification of pinnae.[2,3,5] Cardiovascular involvement with affection of heart valves demonstrated on echocardiography was reported by Kocyigit et. al.[4]

Although no present medical treatment is available for ochronosis, genetic advances offer hope that corrective measures are forthcoming. Some have advocated diets low in tyrosine and phenylalanine, there by reducing the toxic by product HGA. Additionally, a diet high in vitamin C might prevent oxidation of homogentisic acid. One possible hope is that nitisinone proves effective. The US Food and Drug Administration has approved this drug for the treatment of tyrosinemia type 1. It significantly lowers the urinary excretion of HGA by inhibiting 4-hydrophenylpyruvate dioxygenase and would reduce HGA accumulation. Testing presently is assessing safety and long term results. Ochronotic arthropathy is treated with physiotherapy, analgesic, rest and prosthetic joint replacement when necessary.

REFERENCES

1. Vijai Kumar M, Thappa DM, Srikanth S, Sethuraman G, Nadarajan S. Alkaptonuric ochronosis presenting as palmoplantar pigmentation. Clin Exp Dermatol 2000; 25[4]: 305-307.
2. Shetty B.S.S. and Jayaprakash M.R. Alkaptonuria and ochronotic arthritis. Ind J Orthopaedics.1980; 14[2] : 231-232.
3. Gulati A.K. Alkaptonuric arthritis- a case report. Ind J Orthopaedics 1986 ; 20[1] : 63-65.
4. Kocyigit H, Gurgan A ,Terzioglu R, Gurgan U. Clinical, radiographic and echocardiographic findings in a patient with ochronosis. Clin Rhematol 1998; 17[5] : 403-406.
5. Stiehl P, Klinger K M. Joint effusion findings in alkaptonuric arthropathy[ochronosis]. J Rheumatol 1994 ; 53[3]: 150-154.
6. Verma SB. Early detection of alkaptonuria. Indian J Dermatol Venerol Leprol 2005; 71: 189-191.
7. Mishra V, Ranganath LR. Pigmented sclera: a diagnostic challenge. Post Grad Med J 2004; 480: 491.
8. Van Offel JF, De Clerck LS, Francx IM, Stevens WJ. The clinical manifestations of ochronosis : a review. Acta clin Belg 1995; 50 : 358-362.
9. Mannoni A et. al. Alkaptonuria, ochronosis and ochronotic arthropathy. Semin Arthritis Rheum 2004; 33 : 239-248.

Age Estimation in Human Adults by Root Dentin Transparency

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ABSTRACT

Estimating age from teeth is generally reliable as they are naturally preserved long after all the tissues have disintegrated. Main aim of the study was to estimate the age of an adult individual based on the relation between age and root dentin transparency. The study group comprised of 120 single rooted extracted teeth free from caries, restoration, trauma in the age range of 30-69years of both sexes divided into 4 age groups. Teeth were sectioned labiolingually to 400µ thickness and root dentin transparency was measured under stereomicroscope by means of sliding caliper and the results were statistically analyzed. Pearson's correlation coefficient showed high level of correlation of 0.79 between age and root dentin transparency. Step wise multiple regression yielded linear regression formulae to predict the chronological age .The full model explained 62% of total variance. Length of root dentin transparency is closely correlated with age and can be used to estimate chronological age of an adult individual.

Key words : *Age Estimation; Single Rooted Teeth; Root Dentin Transparency.*

INTRODUCTION

All humans have an identity in life. The identification of living or deceased persons using the unique traits and characteristics of the teeth and jaws is a cornerstone of forensic science.[1] Age is one of the important criteria for identification both in living persons and dead bodies.[2, 3]

Estimating age from teeth is generally reliable as they are naturally preserved long after all the tissues have disintegrated.[4] Being the hardest substances in the human body and sometimes may be the only method available to identify the decedent. They can survive most of the insults and consequences encountered at death and during decomposition.[1] It is being used in cases where a dead body or part of the body has to be identified.[5]

In adults several methods exist for age assessment. It is usually based on regressive changes in dental tissues such as enamel, dentin, cementum and periodontal attachment.[6-9] The length of the transparent dentine relative to the length of the root has been used in forensic dentistry to aid in the estimation of person's age.[10] It follows a regular pattern from the apex of the tooth to crownwards and hence is used in forensic work to estimate the age of the patient.[11]

Translucent zone of the tooth root is considered to be less influenced by pathological processes and environmental factors than other age related changes. Increase in the size of the apical zone of translucent dentine in human teeth has been used for age assessment either as one of several regressive changes related to age or as the sole indicator of age.[12]

The main aim of the study was to estimate the age of an adult individual based on relationship between age and measurement of length of transparent root dentin and with an objective to assess the precision and reliability of age estimation, whether this study could be applied in estimation of age beyond 25 years.

MATERIAL AND METHODS

120 single rooted extracted teeth free from caries, restoration and trauma to tooth before and after extraction, and curved roots between age groups of 30-69 years, with equal sex distribution were collected with the written consent of the patient within a time frame of 2 years. The reasons for teeth extractions were either mobile tooth and indication for total extraction. The chronological age was calculated by subtracting date of birth from the date of extraction of tooth. The study was conducted in Bapuji Dental College and Hospital, Karnataka, India.

Soft tissue was removed from the root surface and teeth were disinfected in 10% neutral formaldehyde solution for 24 hours. The teeth were then thoroughly washed under running water. Later the teeth were mounted on self cure resin and labiolingual sections

of 400µ thickness were made exactly through the apex using Hard Tissue Microtome (Leica Sp 1600). The sectioned teeth were observed under stereomicroscope (Leica Wild M3Z) under 10x under a constant light source of 6V, 20W quartz halogen bulb.

The length of transparent root dentin was measured from apex of root in coronal direction to borderline between transparent and opaque dentin by means of plastic sliding caliper. If the junction between transparent and opaque dentin was even in horizontal line on both labial and lingual side, then only one measurement (TL) was taken and when junction on both side was not depicted as relatively even horizontal line, both minimal (TL_1) and maximal (TL_2) extension of transparency was recorded and mean of both minimal (TL_1) and maximal (TL_2) measurement was taken. The distinct zone of cementum which appeared opaque, yellow in colour at the tip was not included in transparent root length. All measurements were recorded in millimeters.

All these data were analysed using SPSS and Minitab soft ware programs (version 13).

RESULTS

Data of known and estimated age was compared using Pearson's correlation coefficient and regression analysis.

There was no statistically significant difference between the genders. (Table 1)

Table 1. Root dentin transparency for males, females and total sample in different age group

Age Group	Males (60)	Females (60)	Total (120)
30-39 Yrs	0.00-1.75 mm	0.00-1.50 mm	0.00-1.75 mm
40-49 Yrs	1.00-4.00 mm	0.50-4.50 mm	0.50-4.50 mm
50-59 Yrs	2.00-8.50 mm	1.50-6.50 mm	1.50-8.50 mm
60-69 Yrs	4.00-11.50 mm	3.00-11.00 mm	3.00-11.50 mm
Total 30-69 Yrs	0-11.5 mm	0-11.0 mm	0.00-11.50 mm
Mean ±SD	3.96 ± 3.05	3.24 ± 2.52	3.60 ± 2.81

Male Vs Female: t- 1.41, p- 0.16, Significance: NS

t student (unpaired) t test
$p < 0.05$ significant
$p > 0.05$ Not significant

Pearson's correlation coefficient between age and root dentin transparency showed that length of apical root dentin transparency was strongly correlated with age and all correlation coefficients were highly significant (Table II). In total of 120 sample Pearson's correlation coefficient was +0.79 with a 'p' value < 0.001 which was statistically significant.

Table II. Correlation of root dentin transparency

Group	Pearson's correlation coefficient (r)	p	Significance
Males (60)	+0.82	< 0.001	HS
Females (60)	+0.75	< 0.001	HS
Total (120)	+0.79	< 0.001	HS

Pearson's correlation coefficient (r value)
$p < 0.05$, $p < 0.01$ significant
$p > 0.05$ not significant

In whole 120 sample regression coefficient was found to be 3.07, standard error of 6.7 with explained variance (R^2) of 62%. The regression model utilizing Root Dentin Transparency yielded the following linear regression formula to predict the chronological age.

$$\text{Age} = 38.4 + 3.07(\text{RDT})$$

The median age of the absolute values of the total 120 sample between actual and predicted age was 5.64 years and the predicted age was more precise between 40-49 years.

DISCUSSION

In the present study 120 single rooted extracted teeth were examined and results showed that gender has no significant influence on age estimation when root dentin transparency which was similar to the previous studies conducted by Lamendin H et al (1992),[13] Foti B et al (2001).[14]

Pearson's correlation coefficient between age and root dentin transparency is 0.79 in our study which was in consistent with all the earlier studies conducted by Miles, A.E.W (1963),[15] Bang G., Ramm E (1970),[16] Azaz B et al (1977)[17] Whittaker. D.K et al (1996)[18] Gretel Gonzalez-Colmenares (2007),[19] except for study conducted on Foti B et al (2001).[14] This difference can be attributed more number of samples in our study and the thickness of 400µm and the measurement by using sliding caliper and observing transparency under stereomicroscope of 10X magnification in our study, where as the measurement was carried out using a square caliper amd millimeter ruler through direct observation without magnification.

In the total 120 sample of single rooted extracted teeth, the median age of the absolute values of the residuals (actual age minus predicted age) was 5.64 years with a standard error of estimate of 6.7 years, which was in contrast with a study conducted on Douglas H. (2008).[20]

Regression model in our study explained 62% of total variance which was in contrast with a study conducted by Prince DA (2002),[21] while 38% and 33%

of total variance was obtained in studies conducted by Gustafson G (1950),[22] Miles.A.E.W (1963),[15] Lamendin H et al (1992).[13] This difference can be attributed to change in source of illumination of transparency, change in the pattern of patient selection and geographic distribution of the population; the present study was done in the south Indian population.

In our study age estimation was precise in the age range of 40-60yrs, even though there was overestimation of age in younger age group and underestimation in older age group. From the results it can be concluded that length of root dentin transparency can be used to estimate chronological age of an adult individual.

Future research should aim at involving larger sample, including not only age and gender but also race, culture and other parameters like attrition, cementum deposition, periodontosis, secondary dentin and root resorption.

REFERENCES

1. David Sweet: Why a dentist for identification? Dental Clinics of North America: April: 2001: 45(2); 237-251.
2. Patel D.J, Kumar S, Sheikh M.I. Age estimation in 70 cases by applying gustafson's method on incisors and canines. J Forensic Med & Toxicol: 2007; vol.24(2): 32-35
3. Valenzuela A, Mandojana J.M, Valenzuela M: Multiple regression models for age estimation by assessment of morphologic dental changes according to teeth source. Am. J Forensic Med Pathol: 2002; 23(4): 386-389
4. Balwant Rai, Dhattarwal S.K, Anand S.C, Simmi Kharb: Modification in gustafson's method: Age estimation. Medico Legal Update: Vol 7, No.1 (2007-01- 2007-03)
5. Ajmal M, Mody B, Kumar G. Age estimation using three established methods. A study on Indian population. Forensic Sci Int. 2001: 122: 150-154
6. Solheim T. Dental color as indicator of age. Gerodontics. 1988: 4: 114-118
7. Cameriere R, Ferrante L, Belcastro MG, Bonfiglioli B, Rastelli E and Cingolani M. Age estimation by pulp/tooth ratio in canines by peri-Apical Xrays. J Forensic Sci 2007; 52(1): 166-170.
8. Ohtani S. Studies on age estimation using racemization of aspartic acid in cementum. J Forensic Sci 1995; 40:805-807.
9. Ohtani S, Yamamoto K. Estimation of age from a tooth by means of racemization of an amino acid, especially aspartic acid- comparison of enamel and dentin. J Forensic Sci 1992; 37 (4): 1061-1067.
10. Johnson C.C. Transparent dentine in age estimation. Oral Surg 1968; 25: 834-838.
11. Adams D. Age changes in oral structures. Dental Update. 1991; 18(1): 15-17.
12. Solheim T. Dental root translucency as an indicator of age. Scand J Dent Res. 1989; 97: 189-197.
13. Lamendin H, Baccino E, Humbert JF, Tavernier JC, Nossintchouk RM, Zerilli A. A simple technique for age estimation in adult corpses: the two criteria dental method. J Forensic Sci 1992; 37: 1373-1379.
14. Foti B, Adalain P, Signoli M, Ardagna Y, Dutour O, Leonetti G. Limits of the Lamendin method in age determination. Forensic Sci Int 2001;122: 101-106.
15. Miles, A.E.W., Dentition in the estimation of age. J.Dent. Res. Suppl 1963: 42(1), 255-263.
16. Bang G., Ramm E. Determination of age in humans from root dentin transparency. Acta Odontol Scand 1970; 28: 3-35.
17. Azaz B, Michaeli Y, Nitzan D. Aging of tissues of the roots of nonfunctional human teeth (impacted canines). Oral Surg Oral Med Oral Pathol:1977: 43(4): 572-578.
18. Whittaker, D.K., Bakri, M.M., Racial variations in the extent of tooth root translucency in ageing individuals. Archs Oral. Biol. 1996: 41, 15-19.
19. Gretel Gonzalez-Colmenares, Miguel C. Botella-Lopez, Gregorio Moreno-Rueda, and Juan R. Fernandez-Cardenete. Age Estimation by a Dental Method: A Comparison of Lamendin's and Prince & Ubelaker's Technique. J Forensic Sci, September 2007, Vol. 52, No.5: 1156-60
20. Douglas H., Ubelaker, 1 Ph.D. and Roberto C. Parra. Application of Three Dental Methods of Adult Age Estimation from Intact Single Rooted Teeth to a Peruvian Sample. J Forensic Sci, May 2008, Vol. 53, No. 3: 608-611.
21. Prince DA, Ubelaker DH. Application of Lamendin's adult dental aging technique to a diverse skeletal sample. J Forensic Sci 2002; 47(1): 107-116.
22. Gustafson G. Age determinations on teeth. J.Am.Dent.Assoc 1950; 41: 45-54.

An Analytical Study of Deaths Due to Hanging in Warangal Area, Andhra Pradesh

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ABSTRACT

Death due to hanging is not an uncommon cause in the present day. Suicidal manner is more common in hanging deaths. The tendency towards suicidal hanging is found to be higher because of the easy availability of the ligature material. Deaths due to suicidal hanging are common in the Warangal area of Andhra Pradesh. Their magnitude is quite significant among the total number of deaths subjected to postmortem examinations. Hence it is necessary to analyze these deaths, in relation to their manner of occurrence. As a part of this study it is mandatory to know the different factors influencing suicide. Suicidal hangings are more common than others (95%). The number of deaths due to suicidal hanging is increasing in the society; the Male to Female ratio being unaltered at 2:1 and the common Age group of occurrence being 21-40 years Victims are usually from rural setup with low social economic background. Illiteracy is one of the main cause of deaths due to suicidal hanging (76%). The percentage of hanging deaths is found to be higher in married women (76%) than in the others.

Key words : *Hanging, Suicide, Age, Sex distribution & Socio-economic status.*

INTRODUCTION

Death occurring due to hanging is not an uncommon cause in the present day. Suicidal manner is more common in hanging deaths. The tendency towards suicidal hanging is found to be higher because of the easy availability of the ligature material. Any long flexible or rigid material can be used for hanging. Rarely, death due to hanging could be accidental. Homicidal hanging is also another cause. Deaths due to Hanging are common occurrences in the Warangal area of Andhra Pradesh. Their magnitude is quite significant compared to the total number of deaths subjected to postmortem examinations. Hence it is necessary to analyze these deaths, in relation to their manner of occurrence. As a part of this study it is mandatory to know the different factors influencing suicide as their number is quite significant. Sometimes the hanging deaths are associated with other methods, in which case we call it a complex suicide[1].

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MATERIAL AND METHODS

The present analytical study is made on those dead bodies which are subjected to Post-mortem examinations in the mortuary of Department of Forensic Medicine, Kakatiya Medical College, Warangal.

RESULTS

Table 1. Total no of autopsies

Year	Total No of Autopsies conducted in mortuary	Total No of Hanging Deaths	Percentage
2006	1536	96	6.25 %
2007	1596	102	6.39 %
2008	1601	108	6.74 %

DISCUSSION

Table No. 1 The total number of Postmortem examinations conducted in the mortuary of Kakatiya Medical College, Warangal is increasing by the day. Their number was 1536 in the calendar year of 2006; 1596 in the year 2007 and 1601 in 2008.

Table No. 2 For the present study 100 cases of hanging are selected from June 2007 to May 2008. Among them 67% are males and 37% are females. The

male to female ratio is 2: 1[3, 4,7]. The most vulnerable age group for hanging is found to be between 21 and 40 years with an incidence of 47%[2, 4-7]. The next vulnerable age group is 41 to 60 years in which 30% people died of suicidal hanging. In the younger individuals of less than 20 years of age, there are considerable numbers of deaths, amounting to 19%. In the older age group i.e. 61 years of age and above, the incidence came down to 4%.

Table No. 3 Most of the victims are from low socio-economic status (65%); middle class victims occupied the next strata with 25% of deaths and the least being the higher income group with 10% of the deaths. This indicates the inclination of people from low socio-economic group towards suicides; hanging in particular [3,8,9].

Table No. 4 The incidence of hanging deaths was found to be higher in rural areas (54%) which have out-numbered the Sub-urban areas (24%) and urban area (22%).

Table No. 5 Married women are more inclined towards suicidal hanging. The cross sectional incidence of married women committing suicide by hanging being 23 (69.69%)1 out of total 33 deaths due to hanging that have occurred in females. In comparison the incidence of deaths due to suicidal hanging in married men has been found to be 46 (68.7%) out of total 67 deaths due to hanging that have occurred in males. Most of the married victims in whom death has occurred due to suicidal hanging were found to be in the younger age group[1,2]. Out of the rest, 13 victims were unmarried, 8 widowed and 10 divorced/ living separately from their spouses.

Table No. 6 Education also plays an important role in un-natural deaths like hanging. 76% of victims were found to be illiterates. It conveys a message that, illiteracy is one of the major components which precipitates the incidence of suicides [6,8].

Table No. 7 The above hanging deaths are analyzed based on the Inquest, Personal enquiry and other available information regarding the manner of death. It showed that 95% of cases were suicides, one is accidental death and in four, the manner could not be ascertained because of the suspicion of homicide. The reason for the Accidental hanging death in a 6 years old male child was getting caught in the rungs of a ladder. The allegation of homicidal hanging was because of earlier assaults on the victim. It emphasises that most of the hanging deaths are suicidal in nature[5,7].

CONCLUSION

The present study is done to analyze those deaths which are caused due to hanging, which are subjected to autopsy in the mortuary of Kakatiya Medical College, Warangal during the period June 2007 to May 2008.

1. The Most vulnerable Sex is Male and the male: female ratio is 2:1[3,4,7].
2. The Most vulnerable age group is 21 to 40 (47%) followed by the age groups 41-60 (30%),less than 21years (19%) and above 61 years(4%) as seen in the present study[2,4-7].
3. Suicidal hanging (95%) is more common than other types of hanging. Once again it emphasises that hanging is suicidal, unless proved otherwise[5-7].
4. The victim is from rural setup and is from low socio economic group in many deaths[3,8,9].
5. Illiteracy (76%), is one of the major factor which cannot be ignored, as a cause of hanging deaths[6,8].
6. Married Women (76%) are committing suicides by hanging more frequently than other Women[1, 2].

SUGGESTIONS

1. The victims of suicide and their behavior have to be studied in detail to prevent suicidal deaths at least in future.
2. Proper education of the society is needed to reduce the menace of suicides, including hangings.
3. Scenes showing hangings must be banned in media i.e. in cinema, news papers and TV channels, as they provoke the underlying thought in them to commit suicide[10].
4. Every failed attempt of suicide must be counseled properly to discourage the victim from further attempting suicide.
5. Children must be watched carefully especially those in the pre-adolescent age group to prevent some of the deaths which occur accidentally due to hanging by playing equipment.

REFERENCES

1. Demirci S, Hakan Dogan K, Erkol Z, Deniz I. A series of complex suicide. Am J Forensic Med Pathol. 2009 Jun; 30(2): 152-154.
2. Bastia BK, Kar N. 'A psychological autopsy study of suicidal hanging from Cuttack, India: focus on stressful life situations' 'Arch Suicide Res. 2009; 13(1): 100-104.

Table 2. Age and Sex distribution

< Sex / Age	≤ 10 yrs	11 - 20 yrs	21 - 30 yrs	31 - 40 yrs	41 - 50 yrs	51 - 60 yrs	61 - 70 yrs	> 71 yrs	Total
Male	1	6	11	22	15	9	2	1	67
Female	0	12	9	5	3	3	1	0	33
Total	1	18	20	27	18	12	3	1	100

Table 3. Socio-economic status

Socio-economic status	<10 yrs		11- 20 yrs		21-30 yrs		31- 40 yrs		41- 50 yrs		51-60 yrs		61-70 yrs		> 71 yrs		Total	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Low socio economic	1	0	4	9	6	8	16	2	9	2	5	2	0	0	1	0	42	23
Middle class	0	0	1	2	3	0	5	3	5	1	2	1	1	1	0	0	17	8
Upper socioeconomic	0	0	1	1	2	1	1	0	1	0	2	0	1	0	0	0	8	2

Table 4. Habitat

Habitat	<10 yrs		11- 20 yrs		21-30 yrs		31- 40 yrs		41- 50 yrs		51-60 yrs		61-70 yrs		> 71 yrs		Total	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Rural	1	0	4	7	4	5	14	2	7	1	3	2	2	1	1	0	36	18
Sub - Urban	0	0	1	2	3	2	4	2	4	1	4	1	0	0	0	0	16	8
Urban	0	0	1	3	4	2	4	1	4	1	2	0	0	0	0	0	15	7

Table 5. Marital status

Marital Status	<10 yrs		11- 20 yrs		21-30 yrs		31- 40 yrs		41- 50 yrs		51-60 yrs		61-70 yrs		> 71 yrs		Total	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Married	0	0	1	9	8	6	19	5	11	2	6	1	1	0	0	0	46	23
Unmarried	1	0	5	3	3	1	0	0	0	0	0	0	0	0	0	0	9	4
Widowed	0	0	0	0	0	0	0	0	1	0	2	2	1	1	1	0	5	3
Divorced/Separated	0	0	0	0	0	2	3	0	3	1	1	0	0	0	0	0	7	3

Table 6. Educational status

Educational Status	<10 yrs		11- 20 yrs		21-30 yrs		31- 40 yrs		41- 50 yrs		51-60 yrs		61-70 yrs		> 71 yrs		Total	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Illiterate	1	0	4	10	8	6	17	2	11	2	8	3	2	1	1	0	52	24
Primary	0	0	1	2	2	1	3	2	3	1	1	0	0	0	0	0	10	6
Secondary	0	0	1	0	1	2	2	1	1	0	0	0	0	0	0	0	5	3
Graduation	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Post Graduation	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Table 7. Manner of death

Manner of Death	<10 yrs		11- 20 yrs		21-30 yrs		31- 40 yrs		41- 50 yrs		51-60 yrs		61-70 yrs		> 71 yrs		Total	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Suicide	0	0	5	11	11	9	22	4	14	3	9	3	2	1	1	0	64	31
Homicide	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Accident	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
Unknown	0	0	1	1	0	0	0	1	1	0	0	0	0	0	0	0	2	2

3. Uzün I, Büyük Y, Gürpinar K.Suicidal hanging: fatalities in Istanbul retrospective analysis of 761 autopsy cases. J Forensic Leg Med. 2007 Oct; 14(7): 406-409. Epub 2007 Mar 26.
4. Sharma BR, Singh VP, Harish D. 'Neck structure injuries in Hanging-comparing retrospective and prospective studies'. 'Med Sci Law. 2005 Oct; 45(4): 321-330.
5. Meel BL - 'A study on the incidence of suicide by hanging in the sub-region of Transkei, South Africa'. 'J Clin Forensic Med. 2003 Sep; 10(3): 153-157.
6. Mohanty S, Sahu G, Mohanty MK, Patnaik M. 'Suicide in India: a four year retrospective study'. 'J Forensic Leg Med. 2007 May; 14(4): 185-189'.
7. Kanchan T, Menezes RG. 'Suicidal hanging in Manipal, South India - victim profile and gender differences'. 'J Forensic Leg Med. 2008 Nov; 15(8): 493-496'.
8. Osama Mohammed Al Madni PhD, Magdy Abdel Azim Kharoshah MD. Hanging deaths in Dammam, Kingdom of Saudi Arabia Journal of Forensic and Legal MedicineVolume 17, Issue 5, July 2010.
9. Inoue K, Tanii H, Abe S, Okazaki Y, Nata M, Fukunaga T. 'Suicide in Mie Prefecture, Japan, since 1990'. 'West Indian Med J. 2008 Jan; 57(1): 58-62'.
10. Ghanizadeh A. 'Copycat suicidal attempt by a 7 year old boy after watching homicidal behavior in media: a case report'. 'Cases Journal. 2009 Jan 12; 2(1): 43'.

Importance of Dental Age and Skeletal Age in Forensic Sciences for the Assessment of Pediatric Growth and Development

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ABSTRACT

Age determination plays an important aspect in the clinical practice of Paediatric medicine, Endocrinology, Paediatric Dentistry and Orthodontics. It also plays an important role in Forensic medicine not only in identification of bodies but also in connection with crimes and accidents. It has also helped in determining the nutritional status of children in various public health programmes The study was carried out at the Department of Paediatric Dentistry, SDM College of Dental Sciences, Dharwad on 250 children aged 5-15 years. This led to the conclusion that the Moorees method underestimates the dental age in Hubli-Dharwad (Northern Part of Karnataka State, South India) children therefore certain modifications or newer methods should be devised which are sensitive to the present population.

Key words : *Dental age, Skeletal age, Fishman, Moorees.*

INTRODUCTION

Age determination plays an important aspect in the clinical practice of Pediatric medicine, Endocrinology, Pediatric Dentistry and Orthodontics. It also plays a very important role in Forensic medicine not only in identification of bodies but also in connection with crimes and accidents1. It has also helped in determining the nutritional status of children in various public health programs[2]. The concept of biological age is believed to be more conclusive since it is based upon the degree of maturation of different tissue systems. Historically, several biological ages have been contemplated viz, skeletal age, dental age, morphological age, secondary sexual age etc. But, what is important is the fact that one expression cannot denote the complexity of growth and development[3,6]. In this study an attempt was made to evaluate the chronological and also to co-relate the same with the dental age by Moorees method in school going children of Hubli-Dharwad city which falls in the northern belt of Karnataka state of South India.

The present study was conducted in the Department of Paediatric Dentistry and Preventive Dentistry, SDM College of Dental Sciences, Hubli-Dharwad with the following objectives:

- Determining the dental developmental stages in the children of Hubli-Dharwad cities.
- Determining the skeletal developmental stages in the same sample.
- Testing the applicability of the Moorees criteria for maturity score in dental age assessment in Hubli-Dharwad children.
- Comparing and correlating the obtained dental age with the respective skeletal ages.

MATERIAL AND METHODS

The present study was conducted at the Department of Pediatric Dentistry, SDM College of Dental Sciences, Hubli-Dharwad.

The study group comprised of 250 children in the age group 5 to 15 years visiting the Department of Pediatric Dentistry and Department of Orthodontics. 100 Hand wrist radiographs were taken in the age group of 9-15 years (50 boys and 50 girls) from the same sample.

The children were selected devoid of any growth disorder, with the presence of all the permanent teeth either erupted or in the process of eruption except 3rd molars. The chronological age was recorded by the parent's information.

To determine the dental developmental stages, panoramic radiographs obtained from the Villa Systemi Medicali and the exposure setting of 65kv/3mA, for a period of 3 seconds were utilized according to Moorees method respectively. Hand and Wrist radiographs were obtained for the Fishman's method for the skeletal age assessment, with X-ray machine Siemens, and exposure settings 45kv/40mA for 2 seconds. Magnification was of no importance, since relative measurements and not the absolute measurements were considered. As the radiographs were taken by one single technician, any technical or operational errors were considered to be minimal.

The x-rays obtained were coded by a non-investigator in order to avoid bias during scoring of radiographs. The character of I and II (Roman numeral) denoted sex i.e. boy or girl. The second character was a serial number ranging from 1 to 250 assigned randomly to the radiograph.

Both the Orthopantogram and Hand wrist radiograph was given the same code for one child. Eight Orthopantogram were assessed daily to minimize the intraexaminer and interexaminer fatigue and variability to measure the teeth matching the scores.

The materials used were

5"×12" Kodak (OPG films), 8"×10" Fuji (Hand wrist Radiograph;) X-ray viewer, Orthopantogram machine, Mouth mirror, Straight probe.

Chronological Age

The chronological age was assessed on the basis of the parents' information and date of birth in the school records.

Fishmans Method 7

The radiographic evaluation of skeletal maturation was done by Fishman's Method based on four stages of bone maturation; all found at six anatomical sites located in the thumb, third finger, fifth finger and radius.

Fig. 1. Six Anatomical Sites

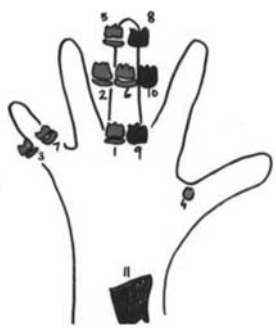

Fig. 2. Skeletal Maturity Indicators

Moorees Method 8

Provides norms of the formation of ten permanent teeth, namely, the maxillary incisors and all eight mandibular teeth on the basis of fourteen arbitrarily selected stages of tooth development.

Stages in single rooted tooth

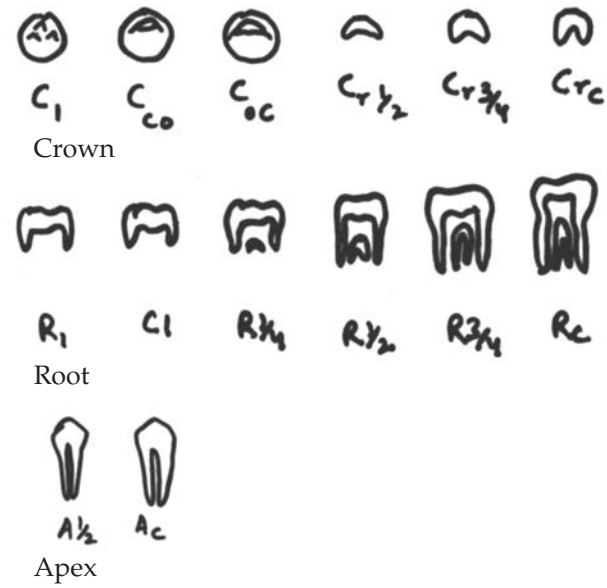

Fig. 3. Stages in a single rooted tooth

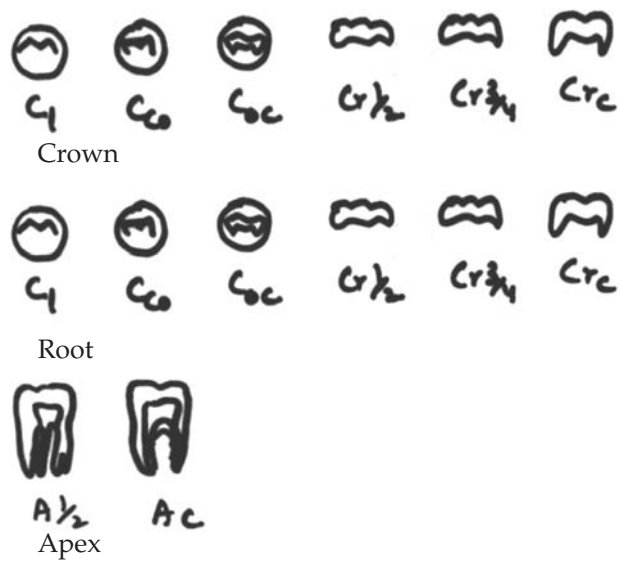

Fig. 4. Stages in multirooted Tooth

Fig. 6. Tooth Formation Stages And Their Coded Symbols

Initial cusp formation	Ci
Coalescence of cusps	Cco
Cusp outline complete	Coc
Crown ½ complete	Cr. ½
Crown ¾ complete	Cr. 1/4
Crown complete	Cr.c
Initial root formation	R
Initial cleft formation	Cl.l
Root length 1/4	R1/4
Root length ½	R½
Root length ¾	R¾
Root length complete	Rc
Apex½ closed	A½
Apical closure complete	Ac

The tooth stages are plotted on the interpolation graph and the mean alveolar emergence or dental age can be calculated

Fig. 7. Orthopantograph of a patient

RESULTS

In the present investigation, an attempt has been made to assess dental age, skeletal age, or chronological age of 250 children of Hubli-Dharwad, who otherwise were normal in all the aspect of growth and development were selected for the study. All these groups were equally divided into 125 boys and 125 girls further classified as high, medium and low (42, 42 and 41) in boys and girls respectively.The observations have been subjected for student -t-test, ANOVA, Karl Pearson and Multiple Linear Regression test.

Table I. Comparison of different types of age of females

Variable	Mean	SD	t-value	p-value	Signi
Chrono	12.5	1.8511	3.4034	<0.01	S
Skeletal	11.09	0.9746			
Chrono age	12.52	1.8611	5.1326	<0.01	S
Dental Age	9.76	1.9446			
Skeletal Age	11.09	0.9746	3.0618	<0.01	S
Dental Age	9.76	1.9446			

Table II. Comparison of different types of age of males

Variable	Mean	SD	t-value	p-value	Signi
Chrono	13.18	1.83	0.7465	<0.05	NS
Skeletal	12.83	1.42			
Chrono age	13.18	1.83	5.5312	<0.01	S
Dental Age	10.36	1.77			
Skeletal Age	12.83	1.42	5.4349	<0.01	S
Dental Age	10.36	1.77			

Table III. Comparison of different types of age of total (males + female)

Variable	Mean	SD	t-value	p-value	Signi
Chrono	13.18	1.83	0.7465	<0.05	NS
Skeletal	12.83	1.42			
Chrono age	13.18	1.83	5.5312	<0.01	S
Dental Age	10.36	1.77			
Skeletal Age	12.83	1.42	5.4349	<0.01	S
Dental Age	10.36	1.77			

Quentessential of the Results: (Tables I to III)

1. The Dental Age of Hubli-Dharwad child was under estimated by Moorees method.
2. An under estimation of the Dental age of Females was more than males by Moorees Method.
3. The mean of the chronological age and the skeletal age showed less significant difference when compared to the dental age in the male children.
4. The Skeletal Age is more correlated to the chronological age than the dental age in Hubli-Dharwad children.

DISCUSSION AND CLINICAL IMPLICATIONS OF THE STUDY

Panoramic radiographs have been used because they are easier to make than the intra oral radiographs in young children and also give less amount of radiation when compared to full mouth radiography (Mc Donald, 1969)

The dental age obtained by Moorrees method underestimated the age of Hubli-Dharwad children. This is in accordance to that reported by Marks (1992) .Nystrom et al. suggested that differences in overall

dental maturity exists not only between nations but also between groups of children in a nation with a relative homogenous population.[9,12]

The difference of the mean of the skeletal ages of the girls was statistically significant. Steel (1965)[13], Gyulavari O, (1966)[14], Lacey (1973)[15], Anderson (1976)[16],. Tanner (1975)[17], Patterson (1984)[18] have reported lower correlations between skeletal and dental development. A high correlation between dental and skeletal ages has been observed in Gulati et al[19] (r = 0.98 in boys and r = 0.93 in girls), Lamons and Gray[5] (r = 0.93 in boys and r = 0.88 in girls) and Sierra[20] (r = 0.82) Prabhakar[21] (r = 0.89). Lautersteins (1961)[22] suggested that girls in growth and development are more advanced in many respects and the ossification appear earlier. It is however contradictory to Pryor (1907)[23] who suggested the skeletal precocity of girls as compared to boys.

Clinical Implications of the study

As for the purpose of age assessment, this study again proves the fact that knowledge of only the chronological age is often inaccurate. The concept of biological age was again showed to be more conclusive since it is based upon the degree of maturation of different tissue systems however it makes little clinical sense to rely on one biological indicator to arrive at a concrete conclusion regarding the dynamics of child growth and development.

CONCLUSION

From this study it could be inferred that ethnic variations and racial differences suggest new criteria's and new grading scores should be formulated for the particular population on which they have to be applied for the Forensic Sciences.

REFERENCES

1. Gustafson G., Koch G. Age estimation upto 16 years based on dental development. Odontol Revy; 1974; 25: 297-296.
2. Voors The use of dental age in studies of nutrition in children. Documenta Med Geogr Trop 1957; 9: 137-148 cited from Brook and Barker - Eruption of teeth among the racial groups of eastern New Guinea: A correlation of tooth eruption with calendar age. Archs Oral Biol, 1972; 17: 751-759.
3. Bjork A. Variations in the growth pattern of human mandible: Longitudinal radiographic study by the implant method. J Dent Res., 1963; 42: 400-411.
4. Bjork A. and Helm I. Prediction of the age of maximum pubertal growth in body height. Angle Orthod, 1967; 37: 134-143.
5. Lamons and Gray. A study of the relationship between tooth eruption age, skeletal development age and chronological age in sixty one Atlanta Children. Am J Orthodont 1958; 44: 687-691.
6. Lauterstein and Stokie. A cross sectional study in dental development and skeletal age. JADA 1961; 62: 161-167.
7. 1502 Fishman Radiographic evaluation of skeletal maturation. Angle Orthod. 1982; 52: 88-112.
8. Moorees CFA, Fanning F.A. and Hunt Age variation formation stages for 10 permanent teeth. J. Dent. Res. 1963; 1490
9. Nystrom M., Haataja J, Kataja M. Dental maturity in Finnish children estimated from the development of seven permanent mandibular teeth. Acta Odontol Scand 1986; 44: 193-198.
10. Marks S. Mappes, Edward F. Harris, Rolf Behrents. An example of regional variation in the tempos of tooth mineralization and hand wrist ossification. Am. J. Orthod. Dentofac. Orthop 1992; 101: 145-151.
11. Nystrom M., Haataja J, Kataja M. Dental maturity in Finnish children estimated from the development of seven permanent mandibular teeth. Acta Odontol Scand 1986; 44: 193-198.
12. Steel G.H. The relation between dental maturation and physiological maturity. Dent. Proc. 1965; 16: 23-34.
13. Gyulavari Dental and skeletal development of children with low birth weight. Acta Pediatr Hung. 1966; 7: 301-310, 1966.
14. Lacey KA. Relationship between bone age and dental development. Lancet, 1973; 2: 736-737.
15. Anderson DL, Thompson GW and Popovitch F. Age of attainment of mineralization stages of the permanent dentition. J Forensic Sci. 1976; 21: 191-200.
16. Tanner J.M. and Whitehouse R.H. Assessment of skeletal maturation and prediction of adult height TW 2 method Academic Press, London, 1975.
17. Patterson DK, Demirjian A, Tanguay R, Buschang Ph. Third molar formation and skeletal maturity among French Canadian children. Am. J. Phys Anthropol 1984; 63: 202.
18. Gulati A., Taneja JR, Chopra SL and Madan S Interrelationship between dental, skeletal and chronological ages in well nourished and malnourished children. J. Indian Soc. Pedo. Prev. Dent. 1990; 8: 19-23.
19. Alicia Sierra Assessment of dental and skeletal maturity. Angle Orthod., 1987; 57: 194-207.
20. Prabhakar A.R., Panda A.K., Raju O.S. Applicability of Demirjians method of age assessment in children of Davengere. J. Indian Soc. Pedo Prev Dent. 2002; 20: 2: 54-62.
21. Lauterstein and Stokie A cross sectional study in dental development and skeletal age. JADA 1961; 62: 161-167.
22. Pryor JW The hereditary nature of variation in the ossification of bones. Anat. Rec. 1907; 1: 84-88.

Dermatoglyphics-A New Diagnostic Tool in Detection of Dental Caries in Children

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ABSTRACT

Introduction: Dermatoglyphics is the study of dermal ridge configurations on palmar and plantar surfaces of hands and feet. Dermal ridges and craniofacial structures are both formed during 6-7th week of intra-uterine life, therefore hereditary and environmental factors leading to Dental caries may also cause peculiarities in fingerprint patterns.

Objective: This study compares and evaluates the dermatoglyphic peculiarities and caries experience of Deaf and mute children with those of healthy children .

Design: A total of 200 school children aged 6-16 years were selected using purposive and simple random sampling.Their fingerprints were recorded with duplicating ink and caries experience was clinically assessed by dmft/DMFT index.

Results: Mann-Whitney test revealed statistical association between whorl patterns and loop patters in caries and caries free group (P<0.001).However, no overall statistical association was observed between fingerprint patterns between deaf and mute children and healthy children (P<0.001). Caries experience was higher amongst the deaf and mute children (P<0.001).

Conclusion: Dermatoglyphics could be an appropriate method to explore the possibility of a non-invasive and an early predictor for dental caries and hearing impairment in children so as to initiate the preventive oral health measures at an early age.

Key words : *Fall, Height, Head Injury, Fracture, Intracranial Hemorrhage*

INTRODUCTION

The study of the human hand has always been fascinating, not only to anthropologists and physicians, but also to psychologists, writers, painters, sages and chiromancers. It was in 1926 that Cummins introduced the term "Dermatolgyphics". It is the term applied to the study of the topography of the little epidermal ridges that run parallel to one another on the volar surface of the hand and feet[1]. It refers to the friction ridge formations which appear on the palms of the hands and soles of the feet which begin to appear during the third and fourth month of foetal development.Dental caries has a high prevalence worldwide. Its etiology is complex and multifactorial,however the question of a possible true genetic predisposition toward dental caries has piqued the minds of dental investigators for decades.[2,3] The basis of considering dermatoglyphic patterns as genetic marker for dental caries is that the primary palate develops during 6-13th week of intrauterine life. Epithelium of primary palate as well as finger buds develop from the same site and are of ectodermal origin.[4] The other point which needs to be mentioned is epithelium of finger buds as well as enamel have ectodermal origin, and both develop at the same time of intrauterine life.[5,6] Similiarly Development of dermal ridges and congenital deafness seems to be interlinked as they develop at around the same time. It is estimated

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that about 50% of cases of childhood hearing impairment of moderate to profound degree are genetically determined.[7] Studies have also shown that caries has been high in the deaf and mute children.Dental treatment is the greatest unattended health need of these children.[8,11] Thus when combined with other clinical and investigative features dermatoglyphics can serve to strengthen as a diagnostic tool.The purpose of the study was

1) To find correlation between dermatoglyphic pattern and caries in deaf and mute children.
2) To find correlation between dermatoglyphic pattern and caries in healthy children.
3) To associate relation in between the above two objectives.

MATERIAL AND METHODS

PARTICIPANTS

1 Deaf and mute school and 1 Bangalore urban school were selected using purposive sampling method. In each school,100 school children(50 children having caries and 50 not having caries) in the age group 6-16 yrs were selected using simple random sampling method.Consent from parents/guardians was obtained before the start of the study.

Children with skin disorders;trauma to fingertips;uncoperative children and children whose parents/guardians didn't give consent were excluded from the study.

PROCEDURE

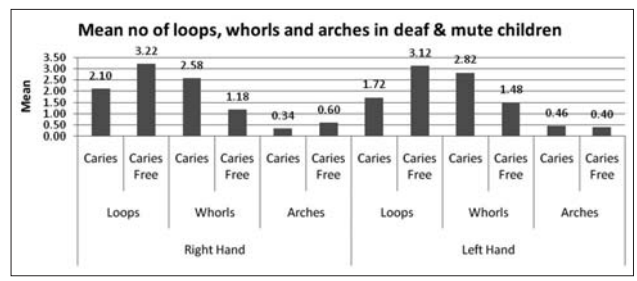
Dermatoglyphic patterns of all 10 palmar digits were recorded using Cummins and Midlo(1943)method. The hands were cleaned with soap and water and then scrubbed thoroughly with an antiseptic lotion (Savlon) and allowed to dry. This was done to enhance the quality of the dermatoglyphic prints, by removing sweat, oil or dirt from the skin surface.The student's right palm was pressed in the ink pad followed by pressing it firmly against the bond paper 2-3 times. since the second or third recording was satisfactory and readable.Same procedure was repeated for the left hand. In this way, a total of 2000 digital prints(dermatoglyphic patterns) were obtained. The obtained dermatoglyphic patterns were assessed for finger tips i.e Presence of arches, loops and whorls with the help of a magnifying glass (10x). Caries experience of deaf and mute children and healthy children was measured using dmft/DMFT Index.

STATISTICAL METHODS

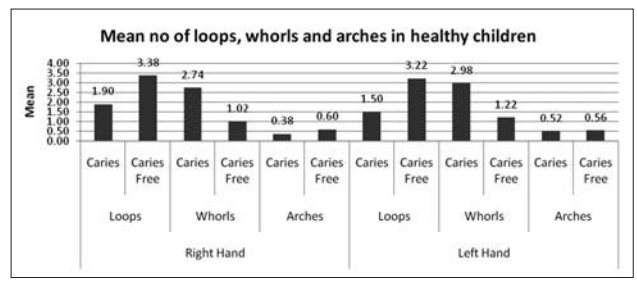
The data collected was subjected to statistical analysis. Mann-Whitney test was used to determine the comparisons between the healthy and deaf and mute children using SPSS Software version 13.

RESULTS

Results showed there was significant change in the dermatoglyphic pattern between the Caries and the caries free group in deaf and mute children and also in healthy children.The frequency of whorls were found to be more in caries group and the frequency of loops more in caries free group.Statistically significant difference was observed between caries and caries free group with respect to loops ($P<0.01$) and whorls ($P<0.001$) in right hand . No significant difference is observed with respect to arches between caries and caries free group in right hand ($P>0.05$) .Similarly in the left hand, no significant difference was observed between caries and caries free group with respect to loops ($P<0.001$) and whorls ($P<0.001$) and arches ($P>0.05$).(Graph 1 and 2) No statistically significant difference is observed between deaf & mute children with caries and healthy children having caries and not having caries with respect to loops, whorls and arches in right hand as well as left hand ($P>0.05$). (Table 1 and 2). The mean dmft score for the 6-12 years old was 5.56 in Deaf and mute children and 3.13 in healthy children. The mean DMFT score for the 6-12years old was 4 in Deaf and mute children and 2.25 in healthy children. (Table 3 and 4). The mean DMFT score for 13-16 years old was 4.58 for the Deaf and mute children and 2.23 in healthy children(Table 5).The difference between the dmft/DMFT scores was statistically significant,thus showing caries experience was higher amongst the deaf and mute children. (Table 3,4,5)



Graph 1. Comparison of Loops, Whorls and Arches between Caries and Caries Free groups in Deaf and Mute Children:



Graph 2. Comparison of Loops, Whorls and Arches between Caries and Caries Free groups in Healthy Children:

Table 1. Comparison of Loops, Whorls and Arches between Deaf & Mute and Healthy Children having Caries:

Hand	Parameter	Category	Mean	Std dev	Median	Mean difference	Z	P-Value
Right Hand	**Loops**	Deaf & Mute	2.10	1.54	2	0.200	-0.746	0.455
		Healthy	1.90	1.63	2			
	Whorls	Deaf & Mute	2.58	1.74	3	-0.160	-0.505	0.614
		Healthy	2.74	1.84	3			
	Arches	Deaf & Mute	0.34	0.75	0	-0.040	-0.425	0.671
		Healthy	0.38	0.75	0			
Left Hand	**Loops**	Deaf & Mute	1.72	1.57	1	0.220	-0.853	0.394
		Healthy	1.50	1.53	1			
	Whorls	Deaf & Mute	2.82	1.65	3	-0.160	-1.000	1.000
		Healthy	2.98	1.66	3			
	Arches	Deaf & Mute	0.46	1.05	0	-0.060	-0.438	0.662
		Healthy	0.52	1.07	0			

Table 2. Comparison of Loops, Whorls and Arches between Deaf & Mute and Healthy Children without Caries:

Hand	Parameter	Category	Mean	Std dev	Median	Mean difference	Z	P-Value
Right Hand	**Loops**	Deaf & Mute	3.22	1.53	4	-0.160	-0.458	0.647
		Healthy	3.38	1.46	4			
	Whorls	Deaf & Mute	1.18	1.34	1	0.160	-0.397	0.691
		Healthy	1.02	1.17	1			
	Arches	Deaf & Mute	0.60	1.05	0	0.000	—	—
		Healthy	0.60	1.05	0			
Left Hand	**Loops**	Deaf & Mute	3.12	1.45	4	-0.100	-0.238	0.812
		Healthy	3.22	1.30	4			
	Whorls	Deaf & Mute	1.48	1.31	1	0.260	-0.999	0.318
		Healthy	1.22	1.23	1			
	Arches	Deaf & Mute	0.40	0.64	0	-0.160	-0.793	0.428
		Healthy	0.56	0.84	0			

Table 3. Comparison of dmft scores between Deaf & Mute and Healthy Children.(6 years-12 years)

Group	Mean	Std dev	Median	Mean difference	Z	P-Value
Deaf & Mute	5.56	1.52	4.5	2.430	10.451	<0.001
Normal	3.13	1.26	2.5			

Table 4. Comparison of DMFT scores between Deaf & Mute and Healthy Children.(6 years-12 years)

Group	Mean	Std dev	Median	Mean difference	Z	P-Value
Deaf & Mute	4	1.27	4	1.9	9.880	<0.001
Normal	2.5	0.92	2			

Table 5. Comparison of DMFT scores between Deaf & Mute and Healthy Children.(13 years-16 years)

Group	Mean	Std dev	Median	Mean difference	Z	P-Value
Deaf & Mute	4.58	1.03	4	2.350	10.430	<0.001
Normal	2.23	0.86	2			

DISCUSSION

Dermatoglyphics has been used to unveil oral diseases like Dental caries,oral cancer,bruxism, malocclusion, anomalies of teeth,cleft lip,cleft palate,periodontal disease,dental fluorosis and also in unveiling truth with forensic odontology.[4,5,12,23].

In this study,the frequency of whorls were found to be more in caries group and the frequency of loops more in caries free group group, Thesse obtained results are in line with a study done by Metin Atasu in 1998 among Dental students in Marmara University, Turkey[12]. Simliarly a study done by Sharma A and Somani R in 2009 among 3-6 years old children showed decreased frequency of loops in caries group compared to caries free group.A study was conducted at Dayanand Medical College and Hospital, Punjab,to compare the dermatoglyphic patterns in established congenitally deaf cases with that of control healthy individuals[7]. It was found that the frequency of whorls was more in deaf and mute group. However our study showed no statistically significant difference is between deaf & mute children and healthy children with caries and without caries with respect to whorls, loops and arches of both hands.

In this study dmft/DMFT is higher amongst deaf and mute children. Deaf and mute children with disabilities have higher caries experience than healthy children. The mean dmft score for deciduous dentition in the 6-12 years old was 5.56 in Deaf and mute children and 3.13 in healthy children. The mean DMFT score for mixed dentition in the 6-12years old was 4 in Deaf and mute children and 2.25 in healthy children. (Table 3 and 4) and the mean DMFT score

for permanent dentition in 13-16 years old was 4.58 for the Deaf and mute children and 2.23 in healthy children(Table 5).These results were parallel to a study done in 1996 by Behjat Almolook Ajami,Mahboobeh Shabzendedar,Yar Ali Rezay ,Mohammad Asgary in 13 special schools of Iran it was observed that The caries prevalence in the 6-7-year-old deaf children with a mean dmft score of 7.35.The caries prevalence in 11-12-year-old deaf children was 93% with a mean DMFT of 5.12[11]. Another study conducted by Z Al-Qahtani and A H Wyne in the 6-7-year-old and 11-12-year-old blind, deaf and mentally retarded female children registered with the Presidency of Girls' Education schools in Riyadh,showed the caries prevalence in deaf 6-7-year-olds was 95.7% with a mean dmft score of 7.35. The caries prevalence in 11-12-year-old deaf children was 93% with a mean DMFT of 5.12 thus proving that the Caries experience and severity in deaf and mute children was high[10]. A study of handicapped children attending special schools in Birmingham, UK showed mean DMFT as 1.76 in 11-12 year old children which showed that children with impaired hearing and communication problems had better oral hygiene than other handicapped children[24]. Caries prevalence amongst handicapped children of South Canara district in Karnataka showed the mean deft score in the primary dentition was found to be 3.06 ± 3.14. The mean deft and DMFT in the mixed dentition were 3.32 ± 2.82 and 1.14 ± 1.08 respectively and the mean DMFT in the permanent dentition was 4.51 ± 3.17[25]. A study was conducted to know the dentition status and treatment needs deaf and mute children in Udaipur,India which showed that the mean dmft in children of 5-8 year age group was 2.17, in 9-12 year age group it was 1.59 and in 13-17 year age group it was 0.16.Similiarly the DMFT score in 5-8 year age group was 0.50,in 9-12 year age group it was 1.76 and in 13-17 year age group it was 2.95[8]. A study showing similar results was conducted among disabled children and young adults in Kuwait showed dmft in 3-12 years old children was 5.3 and DMFT was 5 indicating the caries experience was higher among the disabled population than among the healthy children.[26]

RECOMMENDATIONS

We recommend more comparative studies in different geographical areas to identify the dermatoglyphic patterns in patients with hearing impairment in various conditions since the dental health of handicapped children should be brought up to, and maintained at, the level of that provided for healthy children.

CONCLUSION

Dermatoglyphics can prove to be a useful and cost-effective tool for preliminary investigations into conditions with a suspected genetic base. There is a definite correlation between dermatoglyphics and Dental caries.A statistically significant correlation was found in relation to the increased frequency of the whorls in deaf and mute and healthy children having caries. It can serve to strengthen the diagnostic impression of the disease right from an early age and preventive oral health measures can be obtained. In addition, the oral hygiene habits of individuals with disabilities can be improved by close monitoring and periodic dental check-ups. The current status of dermatoglyphics claims a very high degree of accuracy in the diagnosis and prognosis towards oral diseases.Although dermatoglyphic examination is technique sensitive,but once applied,can give new dimension and reliable parameter to Dental Science.

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REFERENCES

1. Naffah J Dermatoglyphic analysis: anthropological and medical aspects. Bull N Y Acad Med. Oct 1977; 53(8): 681-692.
2. Ahmed RH, Aref MI, Hassan RM, Mohammed NR Dermatoglyphic Study on Patients with Dental Caries Who Wearing Dental Fillings and its Correlation to Apoptosis that Induced by Using Dental Fillings Nature and Science 2010; 8(10): 54-57.
3. Tyagi R,Khuller N, Sharma A, Khatri A Genetic Basis of Dental Disorders: A Review J Oral Health Comm Dent Oct 2008; 2(3): 55-56.
4. Mathew L, Hegde AM, Rai K Dermatoglyphic peculiarities in children with oral clefts. J Indian Soc Pedod Prev Dent. 2005 Oct-Dec; 23(4): 179-182.
5. Sharma A, Somani R. Dermatoglyphic interpretation of dental caries and in correlation to salivary bacteria interactions: an in vivo study. J Indian Soc Pedod Prev Dent. 2009 Jan-Mar; 27(1): 17-21.
6. Kiran K, Rai K, Hegde AM. Dermatoglyphics as a noninvasive diagnostic tool in predicting mental retardation J. Int Oral Health 2010; 2(1): 95-100

7. Sharma A, Singh P, Sood V Palmar And Digital Dermatoglyphics In Congenitally Deaf Subjects Journal of The Punjab Academy of Forensic Medicine & Toxicology 2007: 7(1)10.
8. Jain M, Mathur A, Kumar S, Dagli RJ, Duraiswamy P, Kulkarni S Dentition status and treatment needs among children with impaired hearing attending a special school for the deaf and mute in Udaipur, India J Oral Sci. 2008 Jun; 50(2): 161-165.
9. Gupta DP, Chowdhury R, Sarkar S Prevalence of dental caries in handicapped children of Calcutta J Indian Soc Pedod Prev Dent. 1996 Mar; 14(1): 23-7.
10. Al-Qahtani Z, Wyne AH Caries experience and oral hygiene status of blind, deaf and mentally retarded female children in Riyadh, Saudi Arabia. Odontostomatol Trop. 2004 Mar; 27(105): 37-40.
11. Ajami BA,Shabzendedar M, Rezay YA, Asgary M Dental Treatment Needs of children with Disabilities JODDD 2007 1(2) 93-98.
12. Atasu M Dermatoglyphic findings in dental caries: a preliminary report. J Clin Pediatr Dent. 1998 Winter; 22(2): 147-149.
13. Singh N, Sharma A, Goel D, Singhal A, Khera T,Sharma A Is dermatoglyphics a reliable criteria for quantifying oral and systemic diseases- A review Indian Journal of Forensic Medicine & Toxicology 2010 4(2): 41-44.
14. Sharma A, Palvi, Kapoor D Dermatoglyphics, Dentistry and Diagnosis– A Review Baba Farid University Dental Journal 2010 1(2): 45-48.
15. Tikare S, Rajesh G, Prasad KW, Thippeswamy V, Javali SB Dermatoglyphics— a marker for malocclusion? Int Dent J. 2010 Aug; 60(4): 300-304.
16. Reddy S, Prabhakar AR, Reddy VV A dermatoglyphic predictive and comparative study of Class I, Class II, div. 1, div. 2 and Class III malocclusions. J Indian Soc Pedod Prev Dent. 1997 Mar; 15(1): 13-9.
17. Kargül B, Alcan T, Kabalay U, Atasu M. Hypohidrotic ectodermal dysplasia: dental, clinical, genetic and dermatoglyphic findings of three cases. J Clin Pediatr Dent. 2001 Fall; 26(1): 5-12
18. Atasu M, Biren S.Ellis-van Creveld syndrome: dental, clinical, genetic and dermatoglyphic findings of a case. J Clin Pediatr Dent. 2000 Winter; 24(2): 141-145.
19. Atasu M, Cimilli H. Fusion of the permanent maxillary right incisor to a supernumerary tooth in association with a gemination of permanent maxillary left central incisor: a dental, genetic and dermatoglyphic study.J Clin Pediatr Dent. 2000 Summer; 24(4): 329-333.
20. Yilmaz S, Atasu M, Kuru B A genetic and dermatoglyphic study on periodontitis. J Marmara Univ Dent Fac. 1993 Sep; 1(4): 297-306.
21. Atasu M, Ozbayrak S, Eryilmaz A.Generalized microdontia and associated anomalies: a clinical, genetic, radiologic and dermatoglyphic study. J Clin Pediatr Dent. 1996 Winter; 20(2): 161-72.
22. Polat MH, Azak A, Evlioglu G, Malkondu OK, Atasu M The relation of bruxism and dermatoglyphics. J Clin Pediatr Dent. 2000 Spring; 24(3): 191-194.
23. Kobayashi ET, Maruyama Y, Kobayashi K.A longitudinal evaluation of craniofacial growth in a patient with Kabuki make-up syndrome: a case report. Eur J Orthod. 2001 Apr; 23(2): 205-213.
24. Shaw L, Maclaurin ET, Foster TD. Dental study of handicapped children attending special schools in Birmingham, UK.Community Dent Oral Epidemiol. 1986 Feb; 14(1): 24-27.
25. Rao DB, Hegde AM, Munshi AK. Caries prevalence amongst handicapped children of South Canara district, Karnataka. J Indian Soc Pedod Prev Dent. 2001 Jun; 19(2): 67-73.
26. Shyama M, Al-Mutawa SA, Morris RE, Sugathan T, Honkala E. Dental caries experience of disabled children and young adults in Kuwait.Community Dent Health. 2001 Sep; 18(3): 181-186.

Study of Pattern of Injuries in Victims of Fatal Road Trafic Accidents in Nepal

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ABSTRACT

During one year study period medico-legal autopsies were conducted on 150 cases of fatal road traffic accident (RTA) victims at the mortuary of B.P.Koirala Institute of Health Sciences, Dharan, Nepal. Among age group 21-30 years, highest number (29.3%) of road traffic fatalities occurs. Male accounted for 76.7% of RTA deaths, with over three fourth that of female. About 3/4th of the fatal accidents took place on highways (74.7%) followed by on city road (12%). Nearly half of the victims (45.3%) came from rural areas. Majority of the victims were pedestrians (39.3%) followed by motorcyclists (26.7%). Among the victims in whom impact injuries could be identified at autopsy 61.3% of victims sustained secondary injuries followed by secondary impact injuries 58.7%. In majority of the cases, the site of initial impact of the responsible vehicle was frontal (31.3%) followed by rear (28%) and side (18.7%). Lower extremities were mostly crushed (17 or 11.3%) followed by head & neck (12 or 8%). 77.3% victims had sustained fractures followed by abrasions 73.3% and contusion 64%. In the majority of the cases, brain 32.7% was injured followed by liver and lungs, 28.7% and 27.3% respectively. Skull was found fractured in 44% of the cases followed by fractures of thoracic bones 40%. Considering areas or parts of body injured far over majority (62%) of the cases sustained head and neck injuries followed by injuries to the lower extremities (54.7%). Spot death occurred in maximum of the victims (53 or 35.5%).

Key words : *Autopsy, Victim, Impact injury, Head Injury*

INTRODUCTION

The first injury crash was supposedly suffered by a cyclist in New York City on 30 May 1896, followed a few months later by the first fatality, a pedestrian in London.[1,2] From these small beginnings a terrible stream of deaths and injuries has followed. There was approximately 999000 road traffic deaths in 1990.[3] In 2002 nearly 1.2 million people worldwide died as a result of road traffic injuries.[1] The vast majority – 90% – of road traffic deaths were in low-income and middle income countries. This represents an average of 3242 persons dying each day around the world from road traffic injuries. In addition to these deaths, between 20 million and 50 million people globally are estimated to be injured or disabled each year.[4,6] What is worse, without increased efforts and new initiatives, the total number of road traffic deaths worldwide and injuries is forecast to rise by some 65% between 2000 and 2020,[3,7] and in low-income and middle-income countries deaths are expected to increase by as much as 80%.[8]

An accident is a result of defects laying either in the host (victim or road user), agent (vehicle) and environment (road conditions and social atmosphere) or a combination of the defects laying in all three factors. The human factor has been reported in the literature as being the most prevalent contributing factor of road traffic crashes.[9-11] The number of vehicle registered in the area is increasing drastically along with the population with each passing year which has caused an increase in fatalities in RTA.[12]

The primary role of autopsy surgeon is to find out the cause of automobile deaths may it be accident,

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shear ill luck, rash or negligent driving, suicide or homicide. Recording of injuries at post-mortem may facilitate not only in the award of compensation by the court but also in apprehending the defaulting drivers.

The present study has been carried out to study the distribution, nature and types of injuries received during fatal RTAs, and to suggest possible preventive measures.

MATERIAL AND METHODS

Materials for the present study comprises of 150 fatal cases due to RTAs brought to the mortuary of the Department of Forensic Medicine and Toxicology, B.P. Koirala Institute of Health Sciences, Dharan, Nepal for medico-legal post mortem examination. These cases were from various police stations of Sunsari district and also adjoining districts. The duration of the study was from 2010 to 2011.

The inclusion criteria were all the cases the history of fatal road traffic accident will be taken for study.

Cases with no specific history of road traffic accident were excluded from this study.

The detailed information about the cases was collected from different sources as under:

1. The inquest report and other relevant papers brought by the police along with the dead body in fatal cases.
2. Interviewing the police personal accompanying the dead body.
3. Interviewing the relatives, neighbors, friends or other persons accompanying the dead body.
4. Autopsy examination findings.

All collected data were compiled tabulated in a master table. Finally the computation of data was done with the help of *Microsoft Excel and SPSS* program in tabular forms and observations were recorded, analyzed and discussed.

RESULT

DISCUSSION

Every day as many as 140,000 people are injured on the world's roads. More than 3,000 die and some 15,000 are disabled for life. Each of those people has a network of family, friends, neighbors, colleagues or classmates who are also affected, emotionally and otherwise. Families' struggle with poverty when they lose a breadwinner or have the added expense of caring for disabled family members.[13]

Out of 360 medico-legal autopsies conducted during 2008-2009, 150 cases (41.7%) were of RTAs. This has reflected major public health problem.

Figure 1 shown, highest number (29.3%) of fatalities occurred in 21- 30 yrs age group followed by the age group 31-40 yrs (21.3%), 11-20 yrs (14%), > 60 yrs (10.7%) and 51-60 yrs (8%) victims respectively. Singh YN et al (2005) found almost 1/3rd of total RTA victims were of age group 21-30 years followed by 31-40 years age group (24.78%).[14] BR Sharma et al (2007) found majority of the victims were young adults of the age group 21-30 years, 437(39%); of which the 21-25 years age group accounted for 249(57%) victims.[15] Over 3/4th of the victims were males (76.7%) while the females constituted only 23.3% cases resulting in male to female ratio of 3.28:1. This finding is in accordance with studies done by Eke et al (2000)[16], Kochar et al (2002)[17], Singh and Dhattarwal (2004)[18] and Kaul et al (2005)[19].

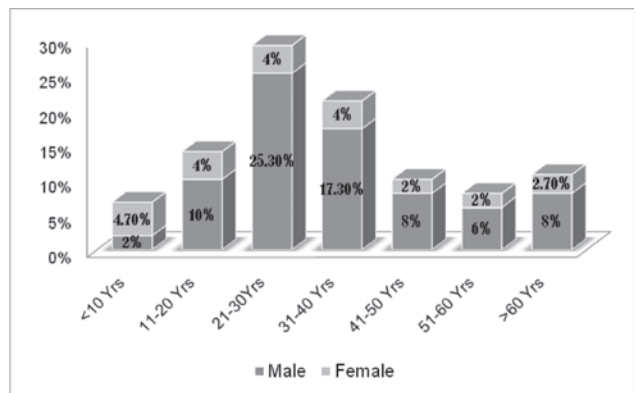

Fig. 1. Age and Sex of The Victims

Figure 2 shown, about 3/4th of the fatal accidents took place on highways (74.7%) followed by city roads (12%), village roads (8%) and other places (5.3%). In the study conducted by Kaul A et al (2005) concluded that largest number (83.05%) fatal RTA have occurred on highways and very few have occurred in lanes and other places.[19] In present study (Figure 3), 45.3% of victims came from rural areas followed by those belonging to the semi urban and urban communities, 28.7% and 26% respectively.

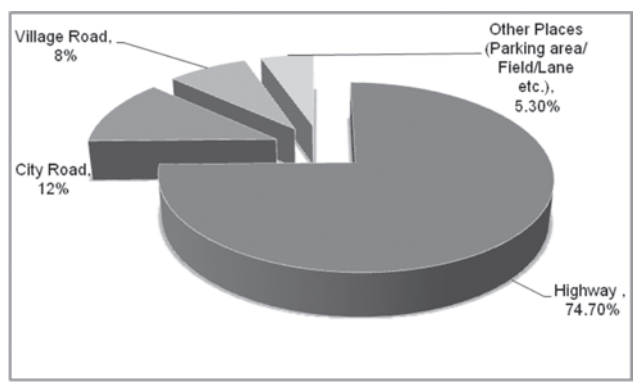

Fig. 2. Distribution of Accidents in Relation to Types of Roads

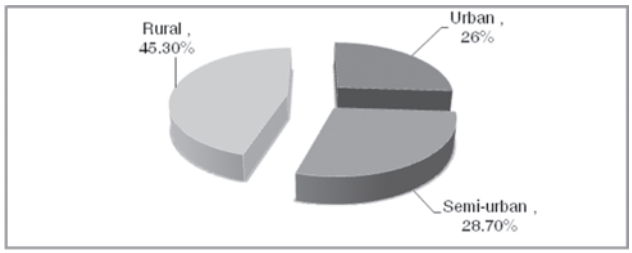

Fig. 3. Community Character of The Victims

In our study (Figure 4), majority of victims (39.3%) were pedestrians followed by motorcyclists (26.7%). In most of the series of fatal cases of RTAs studied by different workers, it has been observed that pedestrians were the most common victims of such accidents, Agrawal and Agarwal (1967)[20], Chandra et al (1979)[21], Maheshwari and Mohan (1989)[22] and Srivastav et al (1989)[23].

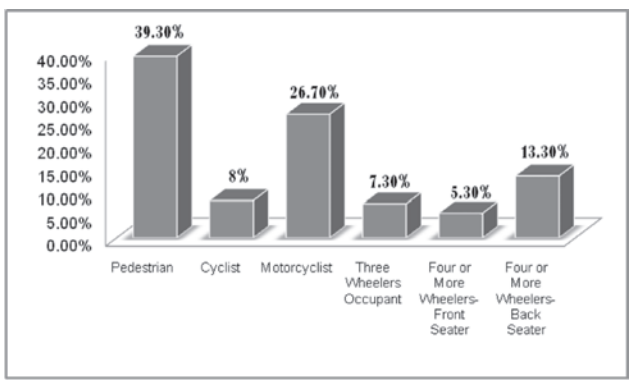

Fig. 4. Type of Road Users/Victims

In the present study (Figure 5-7), majority (61.3%) of the cases sustained secondary injuries followed by secondary impact injuries (58.7%) and primary impact injuries (45.3%) out of those cases where the impact injuries were identified. Since a single crash may lead to multiple primary impacts in a victim, a total of 89 primary impact injuries have been recorded on the different parts of the body of which majority were found distributed in the lower extremities (17.3%) followed by pelvis (11.3%), thorax and upper extremities (8% each). A total of 110 secondary impact injuries have been recorded on the different parts of the body of which majority were noted in the head & neck (20.7%) followed by lower extremities (15.3%) and upper extremities (14%). Lower extremities sustained the maximum (22%) of secondary injuries, followed by injuries in head & neck (18%) and upper extremities (14%). An analysis of the direction of the collision, front to front collision were more frequently involved (30.7%), closely followed by impact from behind (29.8%) and right or left side (16.7%).

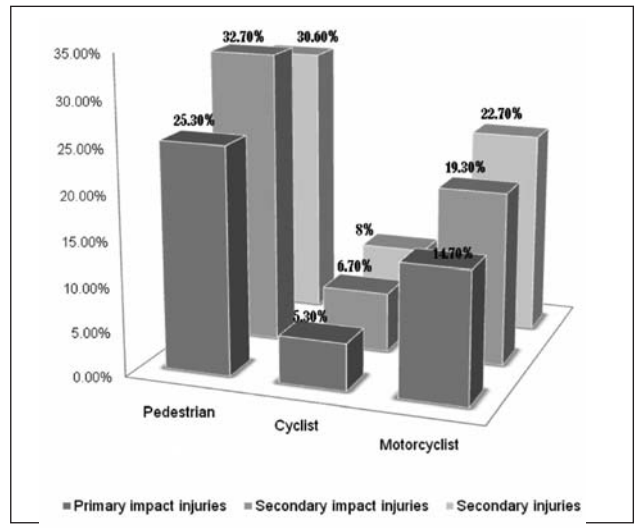

Fig. 5. Distribution of Impacts* Amongst Different Types of Road Users

*Only those cases have been recorded, where the impact injuries were identified.

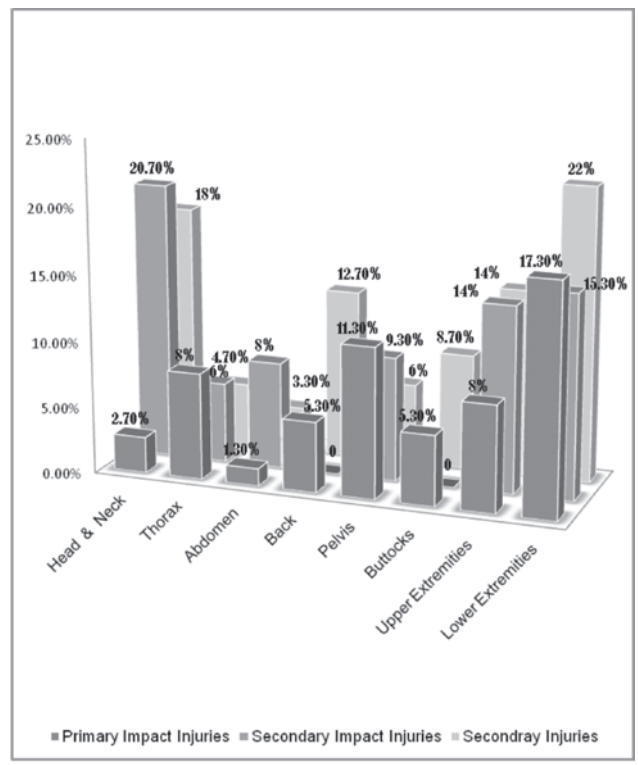

Fig. 6. Distribution of Primary Impact, Secondary Impact and Secondary Injuries on Different Parts of the Body

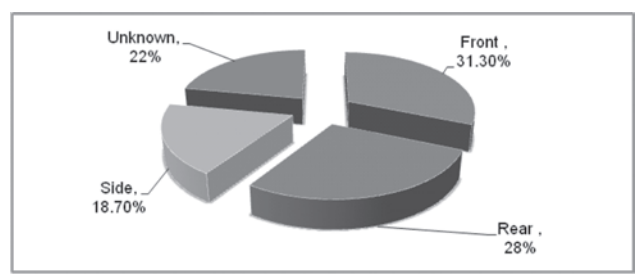

Fig. 7. Cases in Relation to Sites of Initial or Primary Impacts

Present study (Figure 8) shown, lower extremities were mostly crushed (11.3%) followed by head & neck (8%), pelvis (6%), thorax (4.7%) abdomen (3.3%) and back, buttocks and upper extremities (1.3% each). Kaul A et al (2005) noted lower extremities, head & neck and pelvis in descending order.[19]

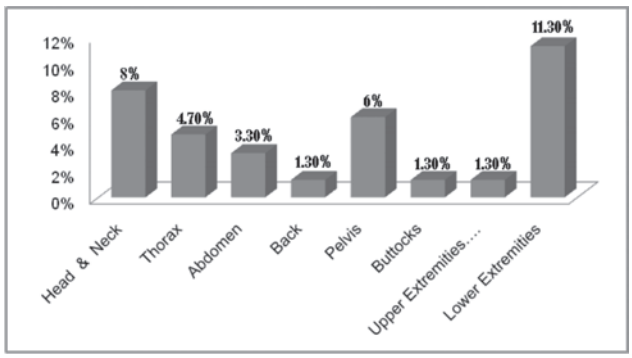

Fig. 8. Crush Injuries on Different Parts of the Body

Figure 9 shown, in a majority of the cases, brain (32.7%) was injured followed by liver and lungs, (28.7%) and (27.3%) respectively. Most of the cases of pedestrians sustained visceral injury to brain (13.3%) followed by liver (12%) and lungs (9.3%). Cyclist had equal number of brain, liver and lungs injuries (3.3% each) while brain (11.2%) injury turn out to be maximum in motorcyclists. Liver (4%) was injured in majority of three wheelers occupant followed by lungs (2.7%). Brain and lungs (2%) each was injured in majority of four or more wheelers-front seater, while in back seater, injuries followed as lungs (5.3%), liver (4.7%) and heart and vessels (2%). Gissane (1963)[24] and Kaul A (2005)[19] in their study, also found largest number of brain injuries followed by injuries to the lungs and liver.

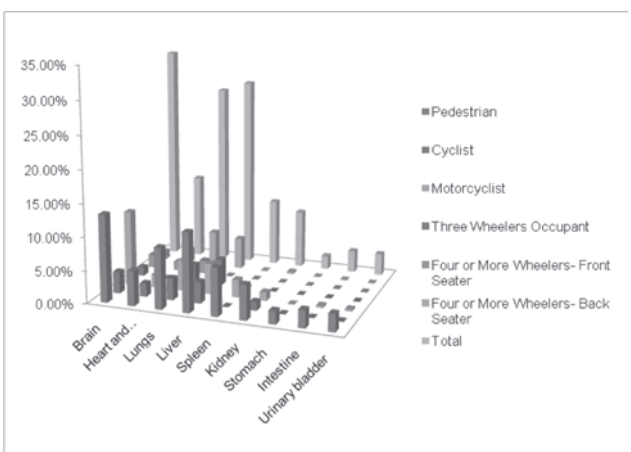

Fig. 9. Visceral Injuries Amongst Different Types of Road Users

Figure 10 shown, skull was found fractured in a majority (44%) of the cases followed by fractures of thoracic bones (ribs, sternum, clavicle and spines) (40%) and long bones (33.3%). The same pattern i.e. fractures of skull, thoracic cage and long bones was observed in the pedestrians (16%, 12.7% and 12% respectively). Cyclist and motorcyclist mostly sustained fractures of skull (6% and 16% respectively) followed by fractures of long bones (6% each). Vehicles occupants i.e. three wheelers, four or more wheelers-front and back seater received the maximum of thoracic bones fractures (4.7%), (5.3%) and (8%) respectively. Dhattarwal et al (2004) observed head injuries in 77.6% out of 349 cases and in most of the cases (51.6%), skull was fractured.[25]

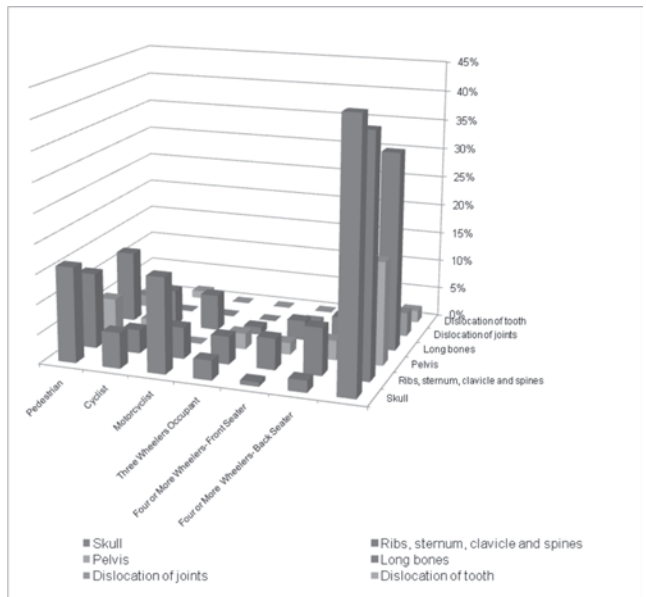

Fig. 10. Different Types of Fractures Amongst Different Types of Road Users

In the present study (Figure 11), spot death occurred in maximum of the victims (35.5%) Only 5 (3.3%) victims were survived for more than 7 days. Sathyiyasekaran (1991) said that twenty were brought dead.[26] Wick (1998) stated that average stay of accident victim in hospital was 35.4 days.[27]

It may be concluded that there is an urgent need to address the epidemic carnage on the roads. In many cases fatal RTAs are caused by human errors and are therefore preventable. With the help of mass media

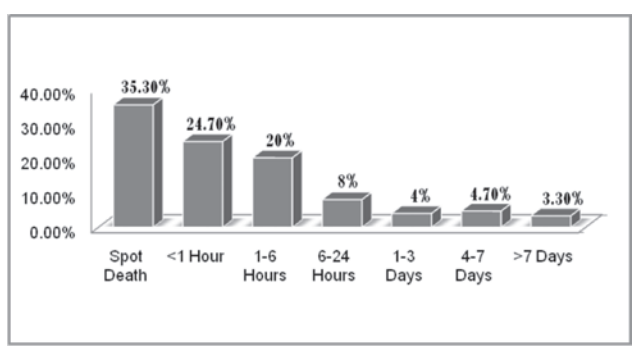

Fig. 11. Period of Survival

they should be make aware regarding risk of traffic accidents and consequences of that along with proper training by authorized centers regarding traffic rules and regulations, issuing licenses after strict testing of driving skills to the medically fit persons only and periodic review of driving skills. Moreover, the recommendation from the world report on Road Traffic Injury Prevention should be considered and promptly implemented.

REFERENCES

1. Seay A, Downing A, Khayesi M, McGee K, Peden M. The global impact. In: Peden M, Scurfield R, Sleet D, Mohan D, Hyder AA, Jarawan E, et al, editors. World report on road traffic injury prevention. Geneva (Switzerland): World Health Organization; 2004.
2. World's first road death. London: Road Peace; 2003 [Online]. 2003 [cited 2008 Nov 21]; Available from: URL: http://www.roadpeace.org/articles/WorldFirst-Death.html
3. Murray CJL, Lopez AD, editorials. The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Boston (MA): Harvard School of Public Health; 1996.
4. Jacobs G, Aeron-Thomas A, Astrop A. Estimating global road fatalities. Crowthorne, Transport Research Laboratory; 2000 (TRL Report 445).
5. Murray CJL, Lopez AD. Global health statistics: a compendium of incidence, prevalence and mortality estimates for 200 conditions. Boston (MA): Harvard School of Public Health; 1996.
6. McGee K et al. Injury surveillance. *Injury Control and Safety Promotion* 2003; 10: 105–108.
7. Kopits E, Cropper M. Traffic fatalities and economic growth. Washington, DC, The World Bank; 2003 (Policy Research Working Paper No. 3035).
8. Breen J, Seay A. The fundamentals. In: Peden M, Scurfield R, Sleet D, Mohan D, Hyder AA, Jarawan E, et al, editors. World report on road traffic injury prevention. Jeneva (Switzerland): World Health Organization; 2004.
9. Lewin. I. Driver training: A perpetual-motor skill approach. *Ergonomics* 1982; 25: 917-924.
10. Farland RA and Moore RC. Human factors in highway safety: A review of evaluation. *New Eng J Med* 1957; 256: 792-798.
11. Evan L. Anew traffic safety a public vision for the United States. *Am J Public Health* 2003; 9: 1384-1386.
12. Traffic fatalities in Nepal [editorial]. *JAMA* 2004; 291:25.
13. World Health Organization, 2004. Road safety is no accident: A brochure for World Health Day 7 April 2004. Geneva [Online]. 2004 [cited 2009 Jul 25]; Available from: URL: http://www.who.int/mediacentre/news/release/2004/pr24/en/.
14. Singh YN, Bairagi KK, Das KC. An epidemiological study of road traffic accident victims in medico legal autopsies. *Journal of Indian Academy of Forensic Medicine* 2005; 27(3): 166-169.
15. Sharma BR, Sharma AK, Sharma S, Singh H. Fatal road traffic injuries in Northen India: can they be prevented? *Trends in Medical Research* 2007; 2(3): 142-148.
16. EKe N, Etebu En and Nwosu SO. Road traffic accident mortalities in Port Harcourt. Nigeria. *Anil Agarwal's Internet J Foren Med Toxicol* 2000; 1: 1-5.
17. Kochar A, Sharma GK, Murari A. Road traffic accidents and alcohol: A prospective study. *International Journal of Medical Toxicology & Legal Medicine* 2002; 5: 22-24.
18. Singh H, Dhattarwal SK. Pattern and distribution of injuries in fatal road traffic accidents in Rohtak (Haryana). *Journal of Indian Academy of Forensic Medicine* 2004; 26(1): 20-23.
19. Kaul A, Sinha US, Pathak YK, Singh A, Kapoor AK, Sharma S, et al. Fatal road traffic accidents, study of distribution, nature and type of injury. *Journal of Indian Academy of Forensic Medicine* 2005; 27(2): 71-76.
20. Agrawal S, Agrawal SN. Fatal road accidents, an analysis of sixty four autopsied body. *Journal of Indian Academy of Forensic Sciences* 1967; 6: 26-32.
21. Chandra J, Dogra TD, Dikshit PC. Pattern of Cranio-Intracranial injuries in Fatal Vehicular Accidents in Delhi, 1966-76. *Med Sci Law* 1979; 19(3): 186-194.
22. Maheshwari J, Mohan D. Road traffic injuries in Delhi: A hospital based study. *J Traffic Medicine* 1989; 17(3-4): 23-27.
23. Srivastav AK, Gupta RK. A study of fatal road accidents in Kanpur. *J Ind Acad Foren Med* 1989; 11(1): 24-28.
24. Gissane W. The nature and causation of road injuries. *Lancet* 1963; 2: 695-698.
25. Dhattarwal SK, Rathee SK, Singh H. Pattern and distribution of fatal injuries in road traffic accidents in Haryana. *Medico-Legal Update* 2004; 4(4): 111-114.
26. Sathiyasekharan BWC. Study of the injured and injury pattern in Road traffic accident. *Indian Journal of Forensic Sciences* 1991;5:63-8.
27. Wick M, Muller EJ, Ekkernkamp A, Muhr G. The motorcyclist: easy rider or easy victim? An analysis of motorcycle accidents in Germany. *Am J Emerg Med* 1998; 16(3): 320-323.

Cheiloscopy: Detection and Development of Latent Lip Prints

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ABSTRACT

The last few decades have seen the development of the exaggerated importance of lip prints as another skin impression. Cheiloscopy deals with the examination of system of furrows on the red part of human lips and is an important tool in forensic science. Traditional lip stick produces a print that is easily identifiable. However, lip prints made without lip stick are invisible known as latent lip prints, requiring reagents that are more sensitive than conventional materials to locate and develop the invisible prints. In this paper the authors describe about the unusual techniques of cheiloscopy used in detection and development of latent lip prints.

Key words : *Cheiloscopy, Latent Print, Detection, Development, Lip prints.*

INTRODUCTION

The professional obligation of the dental surgeon to mankind is not only to serve in examination, investigation, diagnosis and treatment of oral and orofacial lesions of local origin and oral manifestations of systemic diseases, but to also serve in other community services and legal matters as well through forensic science.[1] Keiser-Neilson described forensic odontology as that branch of dentistry which, in interest of justice, deals with the proper handling and examination of dental evidence and with proper evaluation and presentation of dental findings allowing fast and secure identification process. Non-destructive methods of forensic identification like lip prints can be instrumental in successful human identification, sex determination and can be used to verify the presence or absence of a person at crime scene or in mass disasters and accidents.[2] Cheiloscopy is a forensic investigation technique that deals with identification of humans based on lip traces as they are unique in nature in every individual.[3] and unchanged from birth identifiable from sixth week of intrauterine life.[4]

In recent years the cosmetics industry has been producing new lip sticks characterized by their permanent, rather 'persistent', type of stain, that is, when they come into contact with a surface, no color prints are left as in the case of conventional lip sticks. Latent lip prints are produced by these permanent lip sticks[5] and these prints cannot be seen by the naked eye.[6] Because of the different composition of both type of prints that is the visible and the latent lip prints, different reagents have been tried out on their developing.[7] This makes it possible to detect prints, even those invisible, by sophisticated methods.[8]

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HISTORICAL REVIEW

The biologic phenomenon was first described by an anthropologist Fisher in 1902,(5) but it was only in the 1930s, the pioneering criminologist Edmund Locard – "The French Sherlock Holmes" – famously declared, "Every contact leaves a trace" and acknowledged the importance of cheiloscopy.[9] In 1950, Le Moyne Snyder in his book "Homicide Investigation", mentioned the possibility of using lip prints in the matter of human identification.[10] Santos in 1960, suggested that the fissures and the criss-cross lines in the lips could be divided into different groups (simple and compound) and each group further can be divided into further eight subtypes. Renaud in 1972 studied 4000 lip prints and confirmed the singularity of each one. Two years later Suzuki and Tsuchihashi developed another study which resulted in a new classification for lip prints.[11] In 1990, Kaspazak conducted research for 5 years on 1500 persons to elaborate practical use of cheiloscopy.[1] Recently Vahanwala in 2000 conducted a study of lip patterns to promote the important niques.[3] Also in 2005, a

cadaveric study done by Utsuno et al concluded that, even after fixation, a satisfactory identification rate could be achieved from examination of the lip prints. Recently in 2010, El Domiaty et al achieved a detailed cheiloscopic study of the lip-print patterns in Saudi males and females and proved the individuality of lip prints as no identically similar lip-print patterns appeared in two subjects.[4] Alvarez and associates have drawn attention to these prints, which are characterized by their permanence and, are therefore reffered to as 'persistent lip prints'. Although invisible/latent, these prints can be detected and lifted using materials such as aluminium powder and magnetic powder. It is noted that the use of lip sticks is not indispensable for leaving lip prints.[5]

ANALYSING AND DETECTION OF LATENT LIP PRINTS:

Once the experts classify the lip patterns, the recording of the lip prints are done by noting the combination of groove types found in each print. For the examination of lip prints, Hassan and Famy divided lip print into six topographic areas (each lip was divided into three areas- upper right, upper middle, upper left, lower left, lower middle and lower right) and each area was studied alone to determine the type of the grooves.[4]

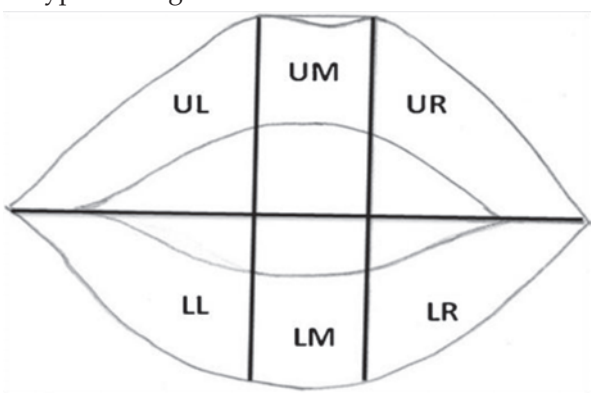

Fig 1: Six topographic areas of lips to be recorded.

RECORDING OF LATENT LIP PRINTS:

The word 'latent' means 'hidden' because the prints are nearly invisible.[6] Latent lip prints can be obtained on a rough surface in suspects/individuals without applying any lipstick after cleaning the lips with the gauze soaked in saline water and drying with sterile cotton. The lip prints are made with sustained pressure for 3 seconds. These prints usually cannot be seen through the naked eyes. Then the reagent powder is sprinkled over the area determined on respective smears of the same individual. The excess powder is removed to visualize the hidden print.[12] These dusted lip prints are later lifted with the help of a latent print tape.[6]

Fig 2: A latent lip print with a reagent powder

DEVELOPMENT OF LATENT LIP PRINTS

Ball states that the vermillion border has minor salivary glands, and the edges of the lip have sebaceous glands and hence, the secretions of oil and moisture from these enable development of latent lip prints, analogous to latent finger prints. Then the reagent powder is sprinkled over the area determined on respective smears of the same individual. The excess powder is removed to visualize the hidden print. Observation should be the 1st step when processing lip prints, using day light. Then, prints are covered with transparent overlays and, when using a magnifying lens, a trace can be successfully done.[12]

Fig 3: developing latent lip prints.

DIFFERENT MATERIALS USED FOR DEVELOPING LATENT LIP PRINTS

The most common and the oldest method to make latent prints visible was to dust them with talcum powder or graphite. The type and the color of the surface determine if talcum or graphite is to be used. Development of lip prints can be made using several substances, such as aluminium powder, silver metallic powder, plumb carbonate powder, fat black aniline dye, or cobalt oxide.[13,14] Luminiscence is specially a useful property for search of invisible evidences at crime scene which can be used for localizing both biological and non-biological fluid stains by use of fluorescent reagents.[15,16]

Fig. 4: Materials used for developing a latent lip print.

a) Aluminium powder:

Mercedes Alvarez Segui et al. in 2000 found aluminium and magnetic powders to be effective for developing latent lip prints.[15] Silver metallic powder and cobalt oxide powder are used in a similar ways. This method is similar to that used in development of latent fingerprints, that involves the application of a finely divided formulation to the impression, with a glass-fibre or a camel hair brush. The powder gets mechanically adhered to the sweat residue defining the ridge pattern, but can easily be blown off the furrows and the ridge becomes visible and the latent print is said to have developed.[17]

b) Plumb carbonate powder:

Plumb carbonate is a white powder which can be used as a developer with a brush, over smooth, polished, metallic or plastic surfaces. Its only limitation is its use over white surfaces. In such circumstances, marphil black powder or fat black aniline dyer are better choices since they both have a darkcolour.[18]

c) Lysochrome dyes:

All lip prints contain lipids which make their development possible by using lysochrome dyes, such as sudan III, oil red O, and sudan black.[14] Lysochromes should then be used since they have the ability to dye fatty acids and are very effective when used on long-lasting lipstick prints, even on porous surfaces.

Ana Castello et al. in 2002 concluded that lysochromes are a highly useful group of compounds for locating and developing recent as well as older latent lip prints on porous surfaces.[16] Ana Castello et al. in 2005 and later in 2006 showed that Nile Red was a very effective reagent to develop old latent lip prints on porous surfaces and when the print was deposited on multicoloured or dark surfaces.[16,19] Esperanza Navarro et al in 2006 showed that sudan III, oil red O, and sudan black are effective for obtaining recent invisible lipstick-contaminated lip mark on corpse's skin.[20] Development process with Sudan Black is done using a brush, small quantity of powder applied on the surface being investigated to locate latent lip print after few minutes the development becomes clearer to present a better quality image.

d) Fluorescent dyes.

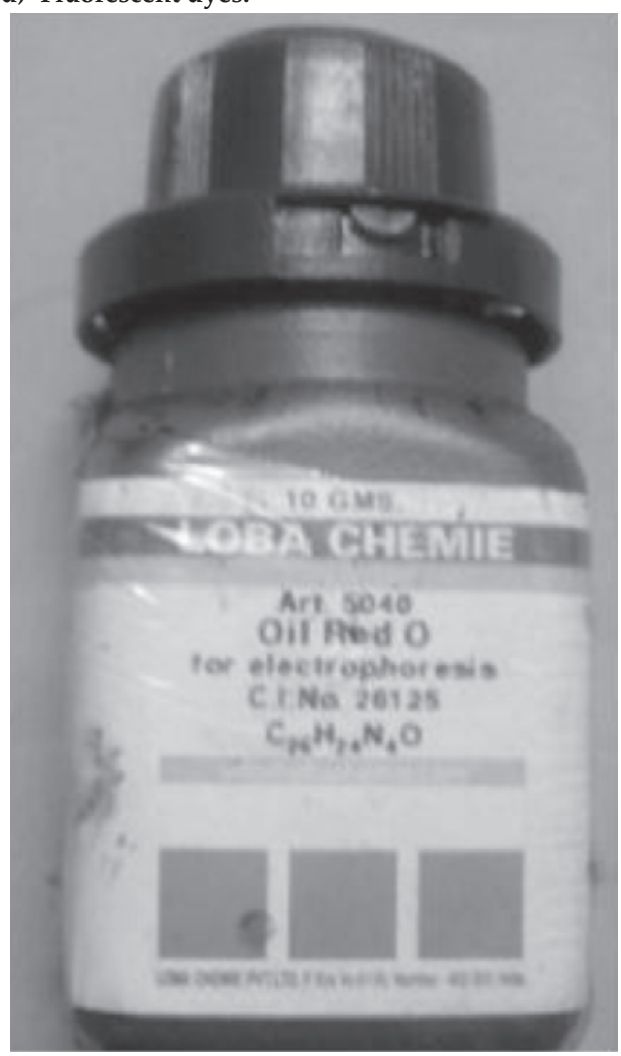

Fig 5: Lysochrome dye

The use of fluorescent reagents is required when the colour of the developer and the colour of the surface on which the lip print lies are the same, or when the lip print is an old brand.[14] In fact, even when located on ''difficult'' surfaces (such as porous or multicoloured ones), latent prints can be easily seen using fluorescent dyes like the Yellowescent Fluorescent latent print powder.[12]

Nile red is used as a potential developer for latent lip prints. For the reagent preparation of 100ml Nile Red solution, dissolve 0.1mg Nile Red in 100ml Ethanol. Developing procedure is by using a brush or a piece of cotton, apply carefully the reagent on the surface where the attempt is being made to locate the latent lip print. For visualizing the print, UV-light (320-400nm)[21] and an alternate light source such as Bluemaxx can be used thus using the property of luminiscece[16] which can be checked using yellow or red glasses for better visualization.[17]

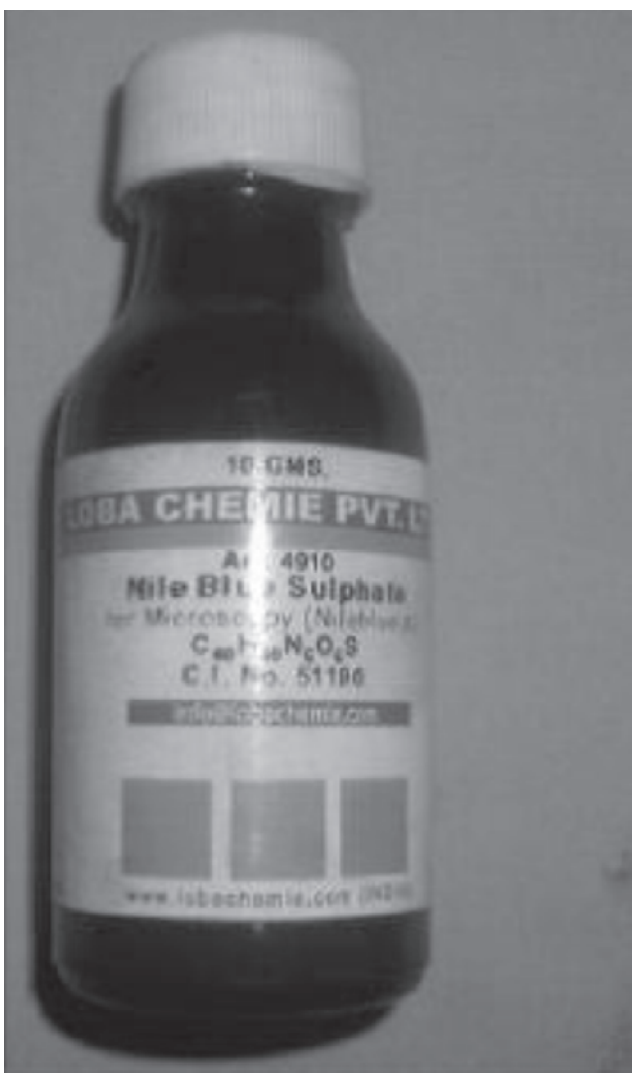

Fig 6: Fluoroscent dye (reagent powder)

e) Chromatography:

The first step in forensic lip stick smear examination is visual comparison but methods used for developing latent lip prints and further detailed examination in chromatography are:

i) Thin-layer chromatography (TLC) of dye stuffs extracted from lip stick smears.
ii) High performance liquid chromatography (HPLC) of extracts using acetonitrite\methanol.
iii) Elemental analysis of inorganic components using an electron probe microanalyzer with an energy-disperse x-ray spectrometer.
iv) Gas chromatography (GC) of waxes.
v) GC/mass spectrometry of trans-esterified derivatives of oils.

The oils present in the lip sticks can be directly analysed using purge-and-trap gas chromatography by gentle heating to 150°C in the equipment without any pretreatment.[22]

Processing lip prints depends on the anatomical, morphological and histological tissue features of lips. Observation should be the first step when processing lip prints, using white and ultraviolet light sources. Using transparent overlays, it is possible to make an overlay tracing. Photographs should be made prior to any processing in order to protect the evidence. Photographing latent prints is a complex process that obeys a strict methodology. In photographs, latent prints can be developed using cyanoacrylate dye or an iodine spray reagent.[12]

PITFALLS IN DEVELOPMENT OF LATENT LIP PRINTS

Lip prints are produced by a substantially mobile portion of the lip. This fact explains the reason why the same person can produce different lip prints, according to the pressure direction and method used in recording the lip print. Manual register of overlay is another problem due to the possibility of some subjectivity. Existence of pathological conditions like lymphangiomas, congenital lip fistulas, lip scleroderma, Merkelson-Rosenthal syndrome, Syphilis, lip cheilitis which invalidate the cheiloscopic study. The only condition which will rule out the cheiloscopic study is burns.[13]

The limitation of aluminium powder is its use over white surfaces. In such circumstances, marphil black powder or fat black aniline dyer are better choices since they both have a darkcolour. Silver nitrate can lead to positive results on non ideal surfaces, such as untreated wood or cardboard. DFO (1, 8-Diazafluoren-9-one) and ninhydrin are chemical developers also used on porous

surfaces. On plastic or waxed surfaces, or on vinyl gloves, using cyanoacrylate dye is a good choice. Cheiloscopy might be the last resort in detecting crimes in certain cases, with advent of latest technologies we might neglect the basic investigation procedures. cheiloscopy might be the last procedure to be used now a days due to its own pitfalls, but it is not the least due to its own importances.[13,23,24]

CONCLUSION

The routine use of forensic dentistry is, however, not yet a reality in India. While it is a forgone conclusion that every medico-legal case will entail a detailed forensic medical investigation, the same, however, is not true of forensic odontology. Latent lip prints should always be considered when processing a crime scene, even if there are no traces of lipstick. However, although lip prints have previously been used in a court of law, its use is not consensual and some authors believe further evidence is needed to confirm their uniqueness.[12,24]

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REFERENCES

1. Preeti S, Saxena Susmita, Rathod Vanita , Cheiloscopy: The study of lip prints in sex identification, January-june 2009; vol 1 issue 1: 24-27.
2. Gondivkar M Shailesh, Indurkar Atul, Cheiloscopy for sex determination, 2010; vol 1 issue 2: 56-60.
3. Saraswathi T.R, Mishra Gauri, Ranganath K. Study of lip prints. Journal of forensic dental sciences, January-june 2009; vol 1 / issue 1: 28-31.
4. Lip Print For Identifcation - Problems and evident solutions Tanta university, Egypt. Taiba University,Saudi Arabia Published on 1 December 2010.
5. Segui Alvarez Mercedes, Feucht Miquel Marcos, Ponce Castello Ana et al. Persistent lip sticks and their lip prints: new hidden evidence at the crime scene. Forensic Science International, 2000; 112: 41-47.
6. Garza. E. Marisol et al. Forensic determination of crime suspects by lip prints and chromatography, 2004-2005.
7. Castello Ana, Verdu Fernando, Alvarez Mercedes- Segui et al. Luminous lip prints as criminal evidence.Forensic. Science. International, 2005; 155: 185-187.
8. Navarro Esperanza, Castello Ana, Lopez .L. Jose, Verdu Fernando et al. Criminalystic: Effectiveness of lysochromes on the developing of invisible lip stick contaminated lipmarks on human skin- A preliminary study. Forensic. Science. International, 2006; 158: 9-13.
9. Nayak Sonal Vahanwala C.D, Pagare S.S. Study of Lip-Prints as Aid for sex Determination. Medico-Legal Update Vol. 5, No. 3 (2005-07 - 2005-2009).
10. L.M. Snyder, Homicide investigation, Springfield, Thomas, 1950, p. 65.
11. Caldas Morais Ines, Magalhaes Teresa, Afonso Americo et al. Establishing identity using cheiloscopy and palatoscopy. Forensic science international, 2007; 165: 1-9.
12. Kumar Prashant, Mastan K.M.K., Patil Shankargouda. Cheiloscopy: Efficacy of flouroscent dye over lysochrome dye in developing invisible lip prints. Int. Journal of Contemporary Dentistry 2010,December; 1(3).
13. Bindal U, Jethani S.L, Arora M. Lip prints as a method of identification in human being. J. Anat. Soc. India, 2009; 58 (2): 152-155.
14. Alvarez Sengui M, Miquel Feucht M, Castello Ponce A, Verdu Pascual F. Persistent lipsticks and their lip prints: New hidden evidence at the crime scene. Forensic Sci Int 2000; 112: 41-7.
15. Castello A, Alvarez M, Miquel M, Verdu F. Long - lasting lipsticks and latent prints. Forensic Sci Commun 2002; 4.
16. Castello A, Segui MA, Verdu F. Luminous lip prints as criminal evidence. Forensic Sci Int, 2005; 155: 185-7.
17. Sodhi G.S, Kaur. J, Powder method for detecting latent finger prints: a review, Forensic Sci Int, 1 september 2001; vol 120 issue 3: 172-176.
18. Caldas IM, Magalhaes T, Afonso A. Establishing identity using cheiloscopy and palatoscopy. Forensic Sci Int, 2007; 165: 1-9.
19. Castello A, Verdu F. Development of latent lip prints on multicoloured surfaces, a problem resolved using fluorescent dyes. Indian Internet Journal of Forensic Medicine and Toxicology, 2006; 4;2- Available from: http://www.indian journals.com/ijor.aspx? target = ijor.iijfmt & volume = 4 & issue = 2 & article = 001.

20 Navarro E, Castello A, Lopez JL, Verdu F. Criminalystic: Effectiveness of lysochromes on the developing of invisible lipstick -contaminated lipmarks on human skin: A preliminary study. Forensic Sci Int, 2006; 158: 9-13.

21. Navarro M.D, Castello .A, Verdu. F, More about the developing of invisible lip-stick contaminated lipmarks on human skin: The usefulness of fluorescent dyes, J Forensic Med & Legal Med, 2007; 14 :340-342.

22. The lip print on the glass found in billiard room, http://www.tsl.state.us/Id/pubs/yareadingclub/forensicevidence.net

23. Acharya. A. B et al. A decade of forensic odontology in india. Journal of Forensic Dental Sciences, 2010; vol 2 issue 1: 1

24. Rajendran Sivapadasundaram B. Shafers, text book of oral pathology. 5th ed. New Delhi: Elsvier publication; 2006.p.1223-4.

A Comprehensive Medico-Legal Analysis of Paediatric Deaths at Osmania General Hospital Hyderabad

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ABSTRACT

Introduction: In the early phase of life, even with the advent of urbanization and civilization, children are not considered relatively as safe as they are vulnerable now. The advent of civilization and urbanization has failed to put children in safe zone. The infancy or childhood is no longer a protected period and subjected to all kinds of maladies of the society. Even childhood (or) infancy is not free from the vagaries like suicide, homicide, kidnapping, neglect and also the sexual assaults. Death from unnatural causes is a black remark on humanity. Study of all the factors causing death unnaturally will be useful in reducing the incidence.

Materials and Methods: The present study has been conducted on the deaths of paediatric age group (i.e., 0 – 15 years) which are of medico-legal significance. This study is based on the Medico-legal autopsies conducted at Osmania General Hospital, Hyderabad. A total number of 500 cases occurred during the period of this study from July, 2007 to June, 2009.

Conclusion: The incidence of death in paediatric age group was more between 13–15 years, least between 4–6 years age group, maximum deaths were due to accidents (56.20%) followed by suicides (27.80%) and homicides (7.8%). Total 500 death were studied of which (48.4%) were males and (51.6%) were females. Maximum incidence of paediatric deaths was from middle and lower socio-economic groups.

Key words : *Pediatric Deaths, Manner of Deaths, Uulnerability.*

INTRODUCTION

Childhood is the development process which starts with birth and continues till adolescence. A major proportion of childhood mortality is associated with trauma due to accidents. Childhood injuries are a major public health problem worldwide[1–5] with injuries being by far the leading cause of death for children from early childhood through adolescence.[z] However, child homicide are also standing out with its causes and out come. Homicide rates may be attributed to factors such as socio-economic inequalities, availability of lethal weapons and cultural beliefs and attitudes[9].Suicide is the third leading cause of death in adolescents around the world. *(WHO,* 2002) There is a consensus that children less than 10 yrs has lower tendency to commit suicide. The incidence of child suicides increases during adolescence and young adulthood.[10, 11] Children, who die unnaturally and (prematurely) without passing through the course of nature, are nothing but innocent victims, and by standards every such death needs the perspective of the society. The rising number of deaths in unnatural manner necessitates to study them in depth. The present study was undertaken with an aim to make complete analysis of pediatric deaths with reference to social, cultural and economic factors and detect the factors of vulnerability.

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MATERIALS AND METHODS:

The present study has been conducted on the deaths of paediatric age group (i.e., 0 – 15 years) which are of medico-legal significance. This study is based on the Medico-legal autopsies conducted at Osmania

General Hospital. A total number of 500 cases occurred during the period of this study from July, 2007 to June, 2009. The post mortem examination reports, inquests, panchanama of scene of offence, personal enquiry reports collected from the relatives.

RESULTS

Table1 Paediatric Deaths

Year	Total No of cases	Paediatric cases	Percentage of cases
July 2007	4421	106	4.6%
2008	4476	251	5.6%
June2009	2639	143	6.1%
	11536	**500**	4.3%

Table 2 Manner of Death

	0-3 years			4-6 years			7-9 years			10-12 years			13-15 years			Total
	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T	
Suicides							1	0	1	18	36	54	28	56	84	139
Accidents	5	8	13	17	28	45	29	34	63	42	22	64	70	26	96	281
Homicides	9	12	21	2	4	6	3	6	9	1	0	1	1	1	2	39
Natural	13	18	31	2	1	3	3	2	5	1	0	1	0	1	1	41
Total	27	38	65	21	33	54	36	42	78	62	58	120	99	84	183	500

Table 3 Socio Economic Status (Homicides, Suicides & Accidents)

	Homicides	Suicides	Accidents	Total
Lower	18	69	58	145
Middle	13	21	142	176
Upper	6	49	80	135
Unknown	2	0	1	3
Total	39	139	281	459

Table 4 Motive behind Homicides

	Male	**Female**	**Total**
Unwanted child	4	6	10
Mother committed suicide after killing the child	3	5	8
Sexual assaults	2	4	6
Battery	4	3	7
Rivalry	2	2	4
Non parental battery	2	2	4
Total	16	23	39

Table 5 Motive behind Suicides

	Male	**Female**	**Total**
Neglect	14	49	63
Parent disharmony	4	4	8
Failure in Studies	24	25	49
Punishment by parents	5	14	19
Illness	19	22	41
Total	47	92	139

DISCUSSION

Table 1: paediatric post mortems accounted 4.3% of total post mortems done during the period. The findings seen in our study are more or less similar to the findings of the study done by Agarwal et al[12]

Table 2: Manner of death: out of 500 cases maximum deaths were due to accidents (281, 56.20%) followed by suicides (139,27.80%) and homicides (39,7.8%).These findings coincide with the studies of Canturk et al 2006, WerenkoDD et. al 2000,Romi et al 1998 Verma et. al Roberts et al 1998 and David et al 2004[10,11,15,16,17,18].

Age: Maximum deaths (183,36.4%) were seen in 13-15 yrs age groups least number of deaths (54,10.8%) were between 4-5 yrs age group, which is similar to studies by WerenkoDD et. al 2000, Parkkari J et. al2000 [11,13]

Sex: Female predominance was seen before 0-9 yrs.Male:Female was 1.2:1.This was found reversed in 10-15 yrs age group i.e, 1: 1.2.

Table 3 The exposure to accidents is seen irrespective of socio economic status, where as homicide and suicides were seen in lower and middle economic groups.

Table 4 The motives for homicidal deaths were unwanted babies particularly female child, battering, sexual assault and rivalry. The maximum incidence is seen in 0-3 yrs age group. Similar findings were shown by David et al[17].

Table 6 Modes of Accidental Deaths

	0-3 years			4-6 years			7-9 years			10-12 years			13-15 years			Total
	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T	
R.T.A	3	3	6	2	4	6	13	12	25	22	10	32	42	15	57	126
Wall collapse	1	0	1	0	2	2	0	0	0	1	0	1	0	1	1	5
Fall from height	0	0	0	3	1	4	3	1	4	1	1	2	1	1	2	12
Drowning	0	0	0	1	1	2	2	0	2	3	3	6	4	1	5	15
Burns	0	3	3	8	14	22	6	18	24	7	12	18	7	12	18	85
Electrocution	0	0	0	1	0	1	3	1	4	4	2	6	3	1	4	15
Snake bite	0	0	0	0	0	0	0	0	0	1	1	2	1	1	2	4
Blast injuries	0	0	0	0	0	0	0	0	0	1	0	1	1	1	2	3
Kerosene poisoning	1	0	1	1	0	1	0	0	0	0	0	0	0	0	0	2
Organo phosphate	0	0	0	2	5	7	0	0	0	1	2	3	1	3	4	14
Total	5	6	11	18	27	45	27	32	59	41	31	71	60	36	95	281

Sex murders and sex crimes are another angle where children are increasingly used for sexual abuse. The recent advance is non parental battering where school children are subjected to the cruelty by the teachers.

Table 5 Most suicidal deaths occurred in the domestic environment and these result from fall from height, parents disharmony, and parent's negligence were leading factors, These findings are similar to Canturk et al 2006, Werenko et al 2000 [10,11].

In suicidal deaths the children are mostly confined to their homes and carry the act under seclusion. The precipitation factors are entirely different from adults like failure in exams Even the accidental mortality is out of hands and unpreventable. Same is not the case with suicides and homicides as they are connected with emotions which in turn are influenced by environment.

Every criminal has a motive that generates intention to accomplish the Act of Crime when there is no other visible motive of ill health or illness to fill the void, leading to a vacant and vague motive. Other commonly known motives are outcome of emotional disturbances or educational troubles which are trivial from elderly view point.

Regardless pathology of suicidal trauma a thorough review of parental attitude is the need of hour as their tendency waxes with waning of moral values of in the present day children.

Table 6: Most deaths were accidental in nature In accidental deaths, the violent trauma is contributed by mechanical, thermal and chemical means in that order. Among accidental deaths 90% of the cases were due to road traffic accidents [12, 19,20,21,22,23,24,25.26].

CONCLUSION

The conclusions drawn from the study are:

- The incidence of death in paediatric age group is more between 13 – 15 years[11, 12], least between 4 – 6 years age.
- Out of 500 cases maximum deaths were due to accidents (281, 56.20%) followed by suicides (139, 27.80%) and homicides (39, 7.8%).
- Total 500 deaths were studied of which 242 (48.4%) were males and 258 (51.6%) were females[13].
- Major contribution to the paediatric mortality was from urban areas. Majority of them were due to Road traffic accidents[2, 18, 22].
- Maximum incidence of paediatric deaths were from middle followed by lower and higher income group respectively.
- Parental negligence was leading factor in suicidal deaths[10, 11].

SUGGESTIONS

By following these simple precautions pediatric deaths can be reduced

- Children should not be allowed to cross roads without supervision.
- All children from the beginning need to be educated about road safety.
- Swimming pools, wells, all the water stagnant areas must be well protected.
- Medications, chemical substances like floor sterilizers, mosquito repellents, rodenticides must be kept out off reach of children.

- All children need to be protected from abuse and exploitation. Law in relation to child abuse must be clearly defined, and the Government should also take necessary care of such children.
- There should not be over burden on children at schools and also at home in relation to academic achievements.
- Limiting the family size will attract more care towards the children.
- Good family relations among the members will not allow children to be distracted easily, there by reduces suicides

REFERENCES

1. World Health Organization. World Health Statistics Annual. Geneva: WHO, 1988.
2. Kingma J. Age and gender distributions of pedestrian accidents across the life- span. Percept Mot Skills 1994; 79: 1680–1682.
3. Mazurek AJ. Epidemiology of paediatric injury. Journal of Accidents Emergency Medicine 1994; 11: 9–16.
4. Hamza J, Berg A. Polytraumatised children. Cah Anesthesiol 1994; 42**:** 505–516.
5. Scheidt PC, Harel Y, Trumble AC, Jones DH, Overpeck MD, Bijur PE. The epidemiology of nonfatal injuries among US children and youth. Am J Public Health 1995;85**:** 932–938.
6. Elmer H. Death rates and causes of death among children and youth in Göteborg, Sweden 1971–85. Indicators for public health work in a city. Scand J Soc Med 1994; 22: 249–255.
7. Wesson D, Hu X. The real incidence of pediatric trauma. Semin Pediatr Surg 1995; 4: 83–87.
8. *Crawley T. Childhood injury: significance and prevention strategies. J Pediatr Nurs* 1996;11**:** 225–*232.*
9. Fingerhut L, Christoffel K. Firearm-related death and injury among children and adolescents. Future Child. 2002;12(2): 24-37.
10. Canturk, G.; Canturk, N.; Teke, HY.; Erkol, Z. & Yavuz, MS. Suicidal Deaths Among Children and Adolescents in Ankara Between 2001 and 2006, Journal of Medical Sciences,Vol. 30, No. 2, pp. 474-481, ISSN 1300-0292.
11. WerenkoDD et. al. Child and Adolescent suicide deaths in New Mexico, 1990-1994. Crisis 2000:21(1): 490-497.
12. AgarwalSS et al, Cranio- cerebral trauma death a postmortem study on 0-15 yrs age group JIAFM, 2005 ; 27 (3).
13. Parkkari J et. Al. Childhood deaths and injuries in Finland in 1971 – 1995 – Int. J.Epidemialogy 2000 Jan (3): 516 -523.
14. Wyatt J P et. al. – Hanging deaths in children – Am. J. Forensic Med. Pathol 1998 Dec: 20(4): 463-466.
15. Romi A. Webster, MD, MPH, Patricia G. Schnitzer, Carole Jenny,Bernard G. Ewigman, Anthony J. Alario, Child Death ReviewThe State of the Nation1998 .
16. Verma SK. Pediatric and adolescent strangulation deaths. JForensic Leg Med. 2007 Feb;14(2): 61-64. (sex).
17. Roberts I, Li L, Barker M. Trends in intentional injury deaths in children and teenagers (1980-1995). J Public Health Med**.** 1998 Dec; 20(4): 463-466 (manner).
18. David J Yarwood CHILD HOMICIDE Review of Statistics and Studies June 2004
19. Busuttil, A. (2009). a) Accidental injuries in children, In: Paediatric Forenic Medicine&Pahology, Busuttil, A.; Keeling, JW. pp. (336-344), Edward Arnold Publishers Ltd, 0 340 731575, London.
20. Busuttil, A. (2009). b) Asphyxial Deaths In Children, In: Pediatric Forenic Medicine & Pathology, Busuttil, A.; Keeling, JW. pp. (329-335).
21. Busuttil, A. (2009). c) Heat-Induced Injury or Death, In: Paediatric Forenic Medicine & Pathology, Busuttil, A.; Keeling, JW. pp. 318-328,
22. Brüning, C.; Siekmeyer, W.; Siekmeyer, M.; Merkenschlager, A. & Kiess, W. (2010).Retrospective analysis of 44 childhood drowning accidents, Wiener klinischeWochenschrif, Vol. 122, No. (13-14), (Jul 2010), pp. 405-412.
23. Strang, Heather. Children as Victims of Homicide. Australian Institute of Criminology. Trends and issues in crime and criminal justice, No.53. Canberra March 1996.
24. Bose A, George K, Joseph A. Drowning in childhood: A population based study. *Indian Pediatrics* 2000; 1: 80–84.
25. G R Istre, M A McCoy, M Stowe, K Davies, D Zane, R J Anderson, R Wiebe; Childhood injuries due to falls from apartment balconies and windows Injury Prevention 2003;9:349–352.
26. Bronstein, AC.; Spyker, DA.; Cantilena, LR. Green, JL.; Rumack, BH. & Giffin, SL. (2008).Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 26th Annual Report. *Clinical Toxicology*, Vol. 47, pp. 911–1084.

Autopsy Study of Cut Throat Cases Brought to Morgue of Sion Hospital, Mumbai- A three Year Study

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ABSTRACT

The present retrospective study was carried out in the Department of Forensic Medicine & Toxicology at Lokmanya Tilak Municipal Medical College & Lokmanya Tilak Municipal General Hospital, Sion Mumbai. Total 32 cases collected were collected for this study purpose. The preponderance of Male 26 (81.25%) victims over the female 6 (18.75%) victims with the age group of 21 to 30 years 11 (34.37%) was most commonly affected. Out of total 32 cases 9 (28.13%) cases were of suicidal cut throat and remaining 23 (71.87%) cases were of homicidal cut throat and most common victims of suicidal cut throat were male 8 (88.88%) followed by female 1 (11.11%) victim. In 9 suicidal cut throat cases hesitations cuts over neck found in 8 (88.88%) cases and hesitations cuts over anterior aspect of wrist joints found in 5 (55.55%) cases and victim's suicidal note was found in 3 (33.33%) cases. Previous attempt of suicide was noted in 4 (44.44%) cases, psychiatric illness found in 4 (44.4%) case and most common motives for suicide were chronic illness 6 (66.67%). In 23 homicidal cut throat cases most common motives was breakdown of relationship 7 (30.43%). Maximum size of cut throat injury was 18cmX10cmX8cm and minimum size of cut throat injury was 4cmX3cmX3cm. Jugular vein 32 (100%) was most commonly clean cut structure of neck followed by Carotid artery 21(65.62%) and least commonly involved structure was hyoid bone 1(3.12%) cases. The direction of cut throat wound from Left side of neck to Right side of neck were observed in 25 (78.12%) cases while the direction of cut throat wound from Right side of neck to Left side of neck were noted in 7 (21.88%) cases. In 23 cases of homicidal cut throat, other associated injuries like stab wound, incised wound, chop wound etc were noted in 18 (78.26%) cases.

Key words : *Cut throat, Hesitation cuts, Jugular vein, Carotid artery.*

INTRODUCTION

Cut throat wounds were often found in cases of homicides where sharp edged weapon was used for homicide purpose. In suicidal cut throat cases two or more superficial, parallel, horizontal cuts were often found at the commencement of wound called as hesitation (tentative) cuts. Suicidal cut throat wounds inflicted by a right-handed person are usually high up in the neck and directed obliquely from a higher to a lower level and from below the left ear to the right side under the chin and tailing of wound towards the right side of neck. A homicidal cut throat wounds was invariably very deep and hesitations cuts were absent and wound was often situated at the lower part of the neck and slopes away from the floor of the mouth. In suicidal cut throat cases carotid artery escapes injury as it was drawn beneath sternomastoid muscle and against the spine when head was extended. While in homicidal cut throat cases carotid arteries were often severed.

AIMS AND OBJECTIVES

The present was undertaken in the Department of Forensic Medicine & Toxicology at Lokmanya Tilak Municipal Medical College & General Hospital, Sion Mumbai with aims and objectives to study the pattern of cut throat injuries with special reference to age, sex, internal neck structures involved in cut throat and whether it was suicidal cut throat or homicidal cut throat etc.

MATERIAL AND METHODS

The present retrospective study was carried out in the Department of Forensic Medicine & Toxicology at Lokmanya Tilak Municipal Medical College & General Hospital, Sion Mumbai. Total 32 cases collected from the calendar years of 1st Jan 2008 to 31st Dec 2010 from post mortem reports and police panchnama. The cases which have cut throat injuries and which were declared dead before admission were selected for this

study purpose. The cases where history was incomplete were discarded from the perview of the present study.

OBSERVATIONS AND RESULTS

The present study demonstrated the preponderance of Male 26 (81.25%) victims over the female 6 (18.75%) victims with male: female ratio of 4.33:1. The age group of 21 to 30 years 11 (34.37%) was most commonly affected.

Table 1. Age group wise and sex wise distribution of cases

Age Group (Yrs)	Male	Female	Total
1-10	0	0	0
11-20	4	0	4
21-30	8	3	11
31-40	5	1	6
41-50	5	0	5
51-60	1	0	1
61-70	3	2	5
Total	26	6	32

Out of total 32 cases 9 (28.13%) cases were of suicidal cut throat and remaining 23 (71.87%) cases were of homicidal cut throat. In 9 suicidal cut throat cases most common victims were male 8 (88.88%) followed by female 1 (11.11%) victim. The age group of 21-30 years 4 (44.44%) was most commonly affected in suicidal throat cases followed by age group of 41-50 years 3 (33.33%).

In 9 suicidal cut throat cases hesitations cuts over neck found in 8 (88.88%) cases and hesitations cuts over anterior aspect of wrist joints found in 5 (55.55%) cases and victim's suicidal note was found in 3 (33.33%) cases. Previous attempt of suicide was noted in 4 (44.44%) cases and psychiatric illness found in 4 (44.4%) cases. Most common motives for suicidal cut throat cases were chronic illness 6 (66.67%), in 1 (11.11%) case person had committed suicide as he was seropositive & in 2 (22.22%) cases motives for suicide was not known.

In 23 homicidal cut throat cases age group of 21-30 years 7(30.43%) was most commonly affected followed by age group of 31-40 years 6 (26.08%).

In 23 homicidal cut throat cases most common motives was breakdown of relationship 7 (30.43%) cases followed by revenge 6 (26.08%) and monetary affairs 6 (26.08%), in 3 (13.04%) cases motives for homicide was not known and least observed motives was jealousy 1(4.34%).

The maximum size of cut throat injury in the present study was 18cm × 10cm × 8cm and minimum size of cut throat injury was 4cm × 3cm × 3cm.

Table 2. Distribution of cut throat cases according clean cutting of neck structures

Sr. No.	Internal structures of neck clean cut	No of cases	Percentage (%)
1.	Jugular vein	32	100
2.	Carotid Artery	21	65.62
3.	Thyroid cartilage	14	43.75
4.	Hyoid bone	01	3.12
5.	Trachea	17	53.12

In the present study jugular vein 32 (100%) was most commonly clean cut structure of neck followed by carotid artery 21(65.62%) and least commonly involved structure was hyoid bone 1 (3.12%) cases. The direction of cut throat wound from Left side of neck to Right side of neck were observed in 25 (78.12%) cases while the direction of cut throat wound from Right side of neck to Left side of neck were noted in 7 (21.88%) cases. The tailings of wounds were noted in all 32(100%) cases.

In 23 cases of homicidal cut throat, other associated injuries like stab wound, incised wound, chop wound etc were noted in 18 (78.26%) cases. Total 8 (25%) victims were found under influence of alcohol after receipt of chemical analysis reports.

DISCUSSION

The present study demonstrated the preponderance of Male 26 (81.25%) victims over the female 6 (18.75%) victims with male: female ratio of 4.33:1 which was consistent with studies of Aggarwal N K and Bansal A K[1]; Ghangale A L, Dhawane S G and Mukherjee A A[4]; Ashok Kumar Shetty[2] and Pranav Prajapati and M I Sheikh[8].

In 9 suicidal cut throat cases most common victims were male 8 (88.88%) followed by female 1 (11.11%) victim and age group of 21-30 years 4 (44.44%) was most commonly affected followed by age group of 41-50 years 3 (33.33%). This was consistent with the study of Ashok Kumar Shetty[2], Behera A, Balabantray J K and Nayak S R[3] and Kh. Pradipkumar Singh et al[5].

In 23 homicidal cut throat cases age group of 21-30 years 7(30.43%) was most commonly affected followed by age group of 31-40 years 6 (26.08%) which was consistent with studies of Pranav Prajapati and M I Sheikh[8], Aggarwal N K and Bansal A K[1] and Ghangale A L, Dhawane S G and Mukherjee A A[4].

In 9 suicidal cut throat cases hesitations cuts over neck was found in 8 (88.88%) cases and hesitations cuts over anterior aspect of wrist joints found in 5 (55.55%) cases. Similar finding in suicidal cut throat cases were also reported by Reddy KSN[9], Pillay VV[7] and Mathiharan K and Amrit K Patnaik[6].

REFERENCES

1. Aggarwal N K and Bansal A K: Trends of homicides in capital city of India. Medico-Legal Update. April-June, 2004, Vol.4, No. 2, pp 41-45.
2. Ashok Kumar Shetty: Incidences of suicides in Belgaum, Karnataka. Medico-Legal Update. January-June, 2010, Vol. 10, No.1, pp 19-20.
3. Behera A, Balabantray J K and Nayak S R: Review of suicidal cases, a retrospective study. J Indian Acad Forensic Med, 2005 : 27 (2), pp 100-102.
4. Ghangale A L, Dhawane S G and Mukherjee A A: Study of homicidal deaths at Indira Gandhi Medical College, Nagpur. J For Med and Tox, 2003, 20:1, pp 47-51.
5. Kh. Pradipkumar Singh, Fremingston K. Marak, Kikameren Longkumer and A. Momonchand: Suicides in Imphal. J Indian Acad Forensic Med, 2005, 27 (2), pp 85-86.
6. Mathiharan K and Amrit K Patnaik: Modi's Medical Jurisprudence and Toxicology, 23rd edition Lexis Nexis Butterworths New Delhi 2006, pp 768-769.
7. Pillay VV: Textbook of Forensic Medicine and Toxicology. 14th edition, Paras Publications hydrabad, pp 183-184.
8. Pranav Prajapati and M I Sheikh: A study of homicidal deaths by mechanical injuries in Surat, Gujarat. J Indian Acad Forensic Med, 2010, 32(2), pp 134-138.
9. Reddy KSN : The essentials of Forensic Medicine and Toxicology. 29th edition, K.Suguna Devi, Hydrabad, India, pp. 174-176.

A Study of Thoraco-abdominal Trauma in Fatal Road Traffic Accidents in Aurangabad

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ABSTRACT

Background: Road Traffic Accidents are the fourth leading cause of death following heart disease, cancer and stroke. Hence accidents pose a major medico-legal problem for the population of India. The present study is done to understand the pattern of thoraco-abdominal trauma in fatal road traffic accidents in Aurangabad District of Maharashtra.

Methods: This hospital based retrospective research work was conducted in above Medical College Hospital during May 2003 to 2005 wherein 150 cases were included.

Results: Majority of the victims were males in 30 to 39 years age group, most of them died within 0-12 hours of accident. Maximum victims had thoraco-abdominal trauma and were brought dead.

Key words : *Male, Fourth Decade, Thoraco-abdominal Trauma, Lungs, Liver, Aurangabad.*

INTRODUCTION

Since prehistoric times the Thoraco-abdominal cavity has been looked upon as one of the most vulnerable regions of the body, and injuries involving it have always been considered very serious. As early as 460 BC, Hippocrates was aware of the danger of life caused by injury to liver and spleen. Penetrating and non-penetrating injuries of the abdominothorax accounts for the largest percentage of cases. Thoraco-abdominal injuries was the main cause of death in the past.

Accidents represent a major epidemic of non-communicable disease throughout the world. There are very few countries where accidents do not figure among the five leading causes of death.

Globally, more than a million people die each year in road crashes, slaughter roughly equivalent to nine fully loaded Boeing 747s falling from the sky every day. Among young adults age 15 through 44, road-traffic injuries are the second leading cause of death after AIDS. Twenty million people around the world are injured or disabled each year by road-traffic injuries. These now rank ninth on the grisly list of leading causes of disability, but are projected to rise to third place by 2020 as the number of motor vehicles grows.

According to the World Health Organization, about 85% of all deaths globally due to traffic crashes and 90% of the disabilities caused by them happen in low and middle-income countries. The poorest people in those countries are the hardest hit[1].

According to WHO/World Bank report "The Global Burden of Disease", deaths from non-communicable diseases are expected to climb from 28.1 million a year in 1990 to 49.7 million by 2020 - an increase in absolute numbers of 77%. Traffic accidents are the main cause of this rise. Road traffic injuries are expected to take third place in the rank order of disease burden by the year 2020[2].

As per WHO estimate 26, 65, 000 people were killed due to accident in 1980(5.2% of all deaths) of which 10% were children. The vehicular accidents usually involve the young people between the age group of 20-40 years. With the advent of modern automobile era, the rapidly increasing faster speed transport, road traffic accidents are taking an epidemic form worldwide. In India one person becomes the victim of this man made epidemic in less than five minutes. In Delhi six persons die every day in vehicle mishap, hence Delhi tops in India for the deaths due to RTA.

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Almost all of the deaths of road traffic accident victim have medico-legal implications. It is therefore necessary to establish the cause of death to get compensation from the state or from insurance companies.

MATERIAL AND METHODS

A post mortem study of 150 cases of fatal thoraco-abdominal trauma in road traffic accidents was undertaken at Government Medical College and Hospital Aurangabad, during May 2003 to December 2004.

Being medico-legal cases, details are available in the Department of FMT. The information in separate proforma was collected from the medico-legal documents, discussing with IO and interrogation with relatives and friends.

Biological profile of the victims were noted. The inquest report, case papers, X-rays, USG, C.T. Scans were carefully studied to collect relevant information before post-mortem examination.

OBSERVATIONS AND RESULTS

The present study demonstrated the preponderance of Male 26 (81.25%) victims over the female 6 (18.75%) victims with male: female ratio of 4.33:1. The age group of 21 to 30 years 11 (34.37%) was most commonly affected.

Table 1. Sexwise distribution and marital status of the cases

Sex	No of cases (%out of 150)	Married (%)	Unmarried (%)
Male	127 (84.67%)	93 (73.23%)	34 (26.77%)
Female	23 (15.33%)	18 (78.26%)	05 (21.74%)

Above table shows that Males are at high risk (84.67%),while females (15.33%). Percentage is more in both married sexes than unmarried.

Table 2. Age wise distribution

Age in years	Male (%out of 127)	Female (%out of 23)	Total (%out of 150)
0-9	06(4.73%)	02(8.7%)	08(5.33%)
10-19	09(07.09%)	03(13.04%)	12(8%)
20-29	31(24.40%)	05(21.74%)	36(24%)
30-39	44(34.64%)	03(13.04%)	47(31.33%)
40-49	25(19.69%)	04(17.4%)	29(19.33%)
50-59	05(03.39%)	01(4.4%)	06(4%)
60-69	04(03.15%)	03(13.04%)	07(4.66%)
70-above	03(02.37%)	02(8.7%)	05(3.33%)
Total	**127 (100%)**	**23(100%)**	**150(100%)**

Above table shows that male victims from age group 30-39 years are maximum (34.64%) as compared to the rest of the age groups. The age group 20-29 years ranks second with (24.4%) and 40-49 years ranks third (19.69%).

In comparison with male victims, the female victims shows slight changing trend with maximum fatalities in age group 20-29 years (21.74%).the age group 40-49 years ranks second (17.4%).

Table 3. Distribution of the cases of r.t.a. as per time

Time Range	No. of cases	%	Time of injury	No of cases	%
12Midnight -6AM	13	8.67	12Midnight-3AM	08	5.33
			3AM-6AM	05	3.33
6AM -12Noon	37	24.67	6AM-9AM	14	9.33
			9AM-12Noon	23	15.33
12Noon -6PM	45	30.00	12Noon-3PM	16	10.67
			3PM-6PM	29	19.33
6PM- 12Midnight	55	36.67	6PM-9PM	31	20.67
			9PM-12Midnight	24	16.00
Total	**150**	**100.0**	**24 hours**	**150**	**100.0**

Above table shows that maximum number of cases occurred in night hours in between 6-9PM (20.67%), followed by 3-6PM (19.33%).

Table 4. Distribution of time interval between injury and hospitalisation

Hospitalization In hours	No of cases	Percentage
Directly brought to mortuary.	22	14.67
0-1Hours	65	43.33
1-12Hours	60	40.00
12-24Hours	02	01.33
unknown	01	00.67
Total	**150**	**100.00**

Above table shows that, the majority of injured victims (43.33%) availed medical treatment within 0-1 hours, while 40% of victims availed treatment within in 1-12 hours. The 14.6% of victims were brought dead. Remaining 2% victims hospitalized after 12 hours of injury.

Table 5. Time lapse between injury and death

Time of death In hours	No of cases	Percentage
Died on spot	22	14.67
0-12Hours	110	73.33
12-24Hours	05	03.33
1-7Days	10	06.67
7-above	03	02.00
Total	**150**	**100.00**

The above table reveals that 110 victims died within 0-12 hours after sustaining injuries (73.33%). The 22 victims died on spot (14.67%).

Table 6. Distribution of thoraco-abdominal injuries

Site of injuries	No of cases	Percentage
Chest only	21	14.00
Abdomen only	29	19.33
Combined chest and abdomen	83	55.33
Chest and pelvis fracture	03	02.00
Abdomen and pelvis fracture	05	03.33
Combined chest and abdomen and pelvis fracture	09	06.00
Total	**150**	**100.00**

Above table shows that the highest (55.33%) victims had combination of chest and abdominal injuries followed by abdomen only (19.33%), chest only is 14.0%, combined chest, abdomen and pelvis is 6%, abdomen and pelvis fracture is 3.33%.

Table 7. Thoracic organ injured with or without fracture ribs

Organs	No of cases	% out of 150	with fracture ribs	%	Without fracture rib	%
Lungs	93	62.00	86	92.47	7	7.5
Heart	23	15.34	20	87.0	3	13.0

Above table indicates that lung was the commonly injured thoracic organ in chest 62%. The 92.47% of cases are associated with ribs fracture and 7.5% of cases without rib fracture. The heart was involved in 15.33% victims.

Table 8. Involvement of heart in relation to lungs

Nature of injury	No of cases	% out of 23
Heart with lung injury	20	87.00
Heart without lung injury	03	13.00
Total	**23**	**100.0**

The above table indicates that in 87% victims, the heart injuries were associated with lung injuries. In 13% cases the heart injuries were without lung injuries.

Table 9. Involvement of abdominal organs and type of injury

Organ	Type of injury		No. of cases	Percentage (out of 150)
	Lacerations	Contusions		
Liver	18	87	105	70.00
Spleen	07	43	50	33.33
Kidneys	58	17	75	50.00
	Perforations			
Stomach	18		18	12.00
Intestine	29		29	19.33
Bladder	11		11	07.33

The above table shows that in abdomen, liver injuries were the most common (70%) injuries, the kidney ranked second with 50%, the spleen 33.33%, intestinal perforation 19.33%, stomach perforation 12.00% and bladder rupture 7.33%.

DISCUSSION

Present study shows male predominance in deaths due to thoraco-abdominal injury (male: female; 5.5:1), it is probable due to majority of males being involved in outdoor activities and assignment as compared to females. The study correlates with that of Fimate L. (1980)[3], Banarjee et al (1997)[4] and Jha N (1997)[5].

Sexwise study of marital status of cases shows that the ratio of married male to female is 5:1, married male to unmarried male is 3:1, married female to un married female is 5:1.

The proportion or ratio of married and unmarried cases is the same as observed by Mehta S.P(1968)[6].

Table 2 shows- age group wise percentage of thoraco-abdominal injury, maximum number of cases are (31.33%) in the age group 30-39 years, if a broader age group of 20-49 years is considered then the cases comes to 74.33% . This may be due to the fact that this is the most active period of life and therefore one is most commonly exposed to the outer world for survival. These findings correlate with that of Authors[6,7,3,2].

Table 3 indicates that maximum victims sustained injuries during 9AM-12Midnight (82%). It reveals that active long drive is undertaken during daytime and evening hours. Study reveals that the maximum injuries cases occurred in night hours 6-9PM (20.67%), followed by afternoon 3-6PM (19.33%). Similar observations were made by Author[8].

Table 4 denotes majority of injured victims (43.33%) availed medical treatment within 0-1 hours, while 40% of victims availed treatment within in 1-12 hours. 2% victims hospitalized after 12 hours. A major part of victims (14.6%) brought dead to mortuary. Similar observations were made by Authors[9,10].

Table 5 shows study of time lapse between injury and death. Most of the victims (73.33%) died within 0-12 hours after sustaining injuries. The 22 victims (14.67%) died on spot. Five cases (3.33%) died in 12-24 hours, ten cases (6.67%) died in 1-7 days and three cases (2%) died above 7 days.

The number of patients dying on spot and within one hour of trauma is statistically highly significant; The findings in present study matches, and are in consonance with Banarjee et al (1997)[4].

Table 6 shows highest victims (55.33%) had combination of chest and abdominal injuries followed

by abdomen only (19.33%), chest only (14.0%), Similar observations were made by Author[4].

Table 7 shows that amongst chest organs, lung was commonly injured

(62%). Cases associated with ribs fracture and without rib fracture are 92.47%and 7.5%. Heart was involved in 15.34%. Heart injury associated with and without rib fracture was 13%.

Table 8 shows, in 87% victims, heart injuries were associated with lung injuries and in 13% heart was without lung injuries. The study is in accordance with the Author[11].

Table 9, shows in abdomen, the liver injuries were most common (70%), the kidney ranked second with 50%, spleen 33.33%, intestinal perforation 19.33%, stomach perforation 12.00% and bladder rupture 7.33%. The study correlates with the findings of the Author[5-12].

CONCLUSIONS

Majority victims were males between 30-39 years. 49% abdomino-thoracic trauma cases were brought dead to hospital. The maximum victims (55.33%) had combination of chest and abdominal injuries. Lung was the commonly injured thoracic organ (62%). The 92.47% cases are associated with ribs fracture. The heart was involved in 15.33% victims. The heart injuries were associated with lung injuries in 87% victims. In abdomen commonest solid organ involved was liver (70%), the kidney ranked second with 50%, the spleen 33.33%.

SUGGESTIONS

1. The vehicles should be safe with well engineered roads.
2. The driving licenses should be issued after careful scrutiny and stern disciplinary actions against the negligent drivers.
3. Media should impart public education and awareness of traffic rules.
4. Traffic police should be equipped with an ambulance for trauma care of people.
5. The scenario should be managed by Police, Registered Medical Practitioners and Medicolegal Experts.
6. Seat belts should be made compulsory for vehicle users.

BIBLIOGRAPHY

1. Michael Reich; On the Road With Death .Harvard Magazine, Nov-Dec 2002; Vol. 105, No. 2, 48.
2. WHO "The Global Burden of Disease"; Projected change in the ranking of the 15 leading causes of death and disease (DALYs) worldwide, 1990-2020.
3. Fimate L. "Pattern of vehicular accidents in South Delhi." Journal of Indian Academy of Forensic Medicine. (1980).
4. Banerjee K.K. Aggarwal B.B.L; Kohli Anil. "Study of Thoraco-Abdominal Injuries in Fatal Road Traffic Accidents in North East Delhi." Journal of Forensic Medicine and Toxicology. 14.1 (1997): 40-43.
5. Jha N, Road traffic accident cases at BPKIHS, Dharan, Nepal: One year in retrospect, Journal of Nepal Medical Association 1997; 35: 241-244.
6. Mehta SP. An epidemiological study of road traffic accident cases admitted in Safdarjang Hospital, New Delhi. Indian Journal of Medical Research 1968; 56(4): 456-466.
7. Pande C. D. Pant H. C. Yadav S.D. "Incidence of accidental cranio-cerebral trauma." Indian Journal of Forensic Medicine. 1.2 (1979): 166 - 169.
8. Katie Gilbert, Mark McCarthy. "Deaths of cyclists in London 1985 - 93. The hazards of road traffic accidents." BMJ. 308. (1994): 1534 -1537.
9. K. N. Tiwari, Promod Kumar Varma; Epidemiological study of road traffic injuries in Delhi: Result of a survey, Regional Health Forum. 2004; Vol.8, No.1, 6-14.
10. Daniel. K. Love. et .al. Patterns of death, complications, and error in the management of Motor Vehicle Accident Victims: Implications for a regional system of trauma care, Journal of trauma1983; Vol 23, No 6: 503-505.
11. Glaister. Medical Jurisprudence and Toxicology. 9th Edition. (1950): 310.
12. Mohapatra Shrikant et.al; options in management of solid visceral injuries from blunt abdominal trauma; 2003, Vol.65, issue.3: 263-268.

An Analytical Study of Causes of Death in Fall from Height Cases from Gandhi Medical College & Hospital Mortuary, Hyderabad from 2006 to 2008 year

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ABSTRACT

Falling of people from height is not uncommon. But fatal falls are rare to occur. Incidences of falls resulting in death can be accidental, suicidal or homicidal. Many times people fall with minor injuries. When bodies are brought for autopsy it is sometimes unclear whether the injuries are due to a fall from a height or due to blunt trauma from other causes, especially when the bodies are found near buildings with no eyewitnesses available. The aim was to assess the pattern of injuries and identify features helpful in discriminating between these and injuries due to blunt trauma from other causes.

Key words : *Fall, Height, Head Injury, Fracture, Intracranial Hemorrhage*

INTRODUCTION

Falling of Human body from height is not uncommon all over the world. It may result from simple slippage to voluntary effort made by self or others. Most of the falls occurring from height may result in damage to the body in various degrees. Sometimes it may be even fatal. The injuries received may vary from a closed injury like a contusion to dismemberment or mutilation.

The structure of Human body is complex and the nature has made it to provide safety to the important organs like brain, heart, lungs, kidneys and liver by keeping them under bony cages. Sometimes this type of structure proves more fatal as the broken pieces of bones may pierce the important organs.

When a person falls freely from a considerable height, the body rotates in the air, and may come in contact with the surface by any of its body parts. But mostly head comes first, when a person dives from the height. If he falls voluntarily or fully conscious during the fall, he may project the limbs to the oncoming surface.

A healthy and hale person receives more severe injuries against to popular belief. More the weight of the victim, the severity of injuries is more. Males, because of less amount fat receive more severe injuries to the same weight of female, where fat act like a shock absorber for the impact. At the same time, there is more surface area for given weight in females, which, by its buoyancy forces, provides air resistance, results in a slow fall. Hence females of same weight receive less severe injuries. Clothing present on the body makes the density of the person less to the weight. Clothing acts like sponge to receive the impact. Clothing increases the surface area. Clothing sometimes hooks to the projecting objects during the fall, and dissipate some energy there, by tearing. Hence more the clothing less severe the injuries sustained to the body.

So the nature, distribution and severity of the injuries depend on: 1.Height of the fall 2.Trajectory of the fall 3.Nature of the victim (age, sex, weight, clothing, systemic diseases etc) 4.Rolling and spiraling of the body and intervening structures during fall 5.The part of the body coming in contact with the ground 6.Number of bounces the body made 7.The nature of the surface of ground 8.The intention behind the fall 9.Intoxication status of the victim etc.

MATERIAL AND METHODS

Mortuary table, Autopsy Instruments.

Findings

Following are the various types of injuries noted in different types of fall from height cases leading to death –

In most of the cases, head injuries are the commonest cause of death of the person in fall from height cases. Injuries noted are abrasion, contusion,

laceration, intracranial injuries such as intracranial hemorrhages, viz. Extra-dural, Sub-dural, Sub-arachnoid, intra-cerebral hemorrhages; contusion, laceration of meninges and brain substance, apart from different fracture produced in the skull.

The next commonly affected parts are the limbs especially voluntary fall or jumping on the ground landing on their feet from small heights. The injuries seen are in these cases are abrasion, contusion or laceration of the both feet. In cases where the impact is more, fractures of the calcaneum, talus, both malleoli of the ankle, shafts of tibia or fibula, medial or lateral condyles of the tibia, lower end of femur, shaft of femur, head of femur, acetabulum, sacrum, bodies of vertebrae, ring fracture of base of skull with fracture dislocation of different joints including the sacro-iliac joint have been noticed.

In persons falling on outstretched hand, soft tissue injuries on the palmer surface, fractures of small bones of hand, 1st metacarpal, scaphoid, collie's fracture of radius, shaft of radius and ulna, head of the radius, supra-condylar fracture of humerus, shaft of humerus, head of humerus, glenoid cavity, clavicle and manubrium sterni with fracture dislocation of wrist joint, elbow joint, shoulder joint, sterno-clavicular joint are noted.

In cases of falls on side, supine or prone, fractures of ribs and injuries to the inner organs like lungs, heart, liver, spleen, kidneys or stomach are noted.

CONCLUSION

Head injuries are the most common cause of death among all in this study, which took about 35 lives. Of these, most of them fell from less than 10 feet height and very less fell from more than 31 feet of height. People of young age who received head injuries are workers at construction site.

Injury to neck and cervical spine fracture has become cause of death in 14 individual, and most of them occurred due to fall from more than 31 feet. Injury to thoracic part of the body causing multiple fractures of ribs found to be less common among all which carried only 2 lives.

Blunt injuries to abdomen from fall from height resulting in rupture of liver and spleen causing death occurred in 12 cases in this study. Pelvic fractures and injuries to retro-pelvic region have taken 6 lives. All of them occurred in falls from more than 21 feet. Injuries to multiple body parts and cause of death as polytrauma or multiple injuries took 27 lives.

REFERENCES

1. Gurdjian ES 1975.Impact head injury springfield, Illinois: Charles C. Thomas 279
2. Jennet WB 1970. Some current concepts of head injury pathology. Med Sci Law 6:132
3. P.C.Dikshit, 1st edition. Fall from height, Textbook of forensic medicine & toxicology , peepee publishers, New Delhi, 229-232
4. K.S.Narayana Reddy 27th edition. Falls from height, The essential of forensic medicine & toxicology, medical book company, Hyderabad, 241-244
5. Krishnan & Vij, 3rd Edition, Regional Injuries, Textbook of forensic medicine & toxicology, Elsevier, Saurabh Printers, Noida, 412-430
6. Apurba Nandy, 2nd edition. Regional Injuries, Principles of forensic medicine, , New central book agency pvt ltd, 295-309
7. PV Guharaj & M.R.Chandran, 2nd edition, Regional Injuries, Forensic Medicine, , Orient Longman pvt ltd, Hyderabad, 130 -155

A Study of Pattern of Fatal Head Injury at J.N.M.C. Hospital, Aligarh

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ABSTRACT

The incidence of head injuries is growing with increased mechanization in industry, and an increase in high velocity mode of transport. The injuries could also be caused by a penetrating or blunt force either by direct violence or indirectly. The present study was conducted in admitted cases to Emergency section, J.N. Medical College Hospital, A.M.U. Aligarh. All the dead (162) cases (n = 2850) of head injury reported from 01/01/2008 to 31/12/2009 for treatment are included in the study.

Most of the cases of fatal head injury belong to younger age group (15-44yrs) comprising of 56.88% with male preponderance of 74.69%with majority of the cases belonging to road traffic accident (62.97%).Scalp injury in 94.00 %cases where as 65% were cases of skull fractures and subdural hemorrhage(82.09%) was most common intracranial injury.

Key words : *Head injury, Road traffic accident, Transport Injuries*

INTRODUCTION

Head injury is a major public health problem and has already attained epidemic proportions in India. Traumatic injury in which severe head injury plays a major role in over 50% of cases[2], remains the leading cause of death in person below 45 years of age and overall the third leading cause of death responsible for 8% of all deaths, followed by cardio cerebral vascular disease and cancer and it is the chief cause of death among persons aged 15 - 24 years. About 8, 85, 000 lives are lost yearly by road accidents of which two thirds are in developing countries[3]. As a result craniocerebral trauma places a huge financial and psychological burden upon the society. In India the problem has become more acute over the last two decades, mainly due to increased vehicular traffic and poor maintenance of the road. The number of head injury cases is expected to increase further, due to urbanization, increased vehicular load and high speeding. The instance of suicidal injuries has been reported, produced on the scalp by heavy weapon. Mentally deranged persons are known to commit suicide by striking their head against a wall.

THE AIM AND OBJECTIVES OF THE STUDY ARE

1) To study the pattern and mechanism of fatal head injury
2) It's relation to age and sex.
3) To co relate the injury with its etiology.
4) To study the different types of intracranial lesion and their frequency
5) To find out the mortality arising due to head injury.

The study was done and completed in the department of Forensic Medicine in conjunction with Neurosurgery Unit, Dept. of Surgery along with the department of Emergency at J.N.M.C Hospital A.M.U, Aligarh ,involving subjects admitted in two years in the Emergency of the hospital. The present observations are based on systematic study of these cases.

MATERIAL AND METHOD

For the purpose a retrospective study of all the cases of head injury that reported to the Emergency section J.N Medical College Hospital, A.M.U Aligarh for treatment were included in the study. The information about the patients admitted as cases of head injury was obtained from emergency records and then epidemiological statistics incorporated in the objectives were studied. It was not taken into consideration whether they have taken treatment elsewhere before reporting to this hospital. The clinical presentation of the traumatized patient and diagnostic CT scan formed the basis of the study. The CT scan was read and commented upon by qualified

radiologist in conjunction with neurosurgeon, both were of rank of reader.

CRITERIA FOR SELECTION OF CASES:

1. All cases of head injury which registered in emergency.
2. All the cases that died following admission in hospital.

Head injury cases brought dead to the hospital are not included in the study

RESULTS AND DISCUSSION:

Rapid rise in vehicle and deterioting condition of road leads to increase in accident rate and thus head injury.it is also quite common in assault and gunshot injuries. This results in the double loss to the country. Firstly expenditure is incurred in the treatment of these victims and secondly being in the most productive age group, it results in huge productive man-days loss. An increased incidence of head injury has direct repercussion on increased fatalities.. Total of 2850 cases of head injury came to the emergency of the J.N Medical College Hospital, Aligarh for treatment between the periods from 1/1/2008 to 31/12/2009. Out of these admitted patients 162 died after admission in the hospital, constituting 5.68% of deaths following admissions.

Age Distribution in Fatal Head Injury (Table 1): The age of the victims varied from 15 - 70 years. The peak incidence was observed in the age group 15 - 24 years comprising 36.42% of the cases. It was also observed that 23.46% belonged to the age group 25 - 34 years. More than half 59.88% of cases comprised of age group of 15 - 44 years in the study. Older people i.e. 65 years and above are 5.49% of total cases are the least affected .This is in accordance with studies 4,5, 6,7,8,9,10,11,12, and 13.Individuals in the seventh decade were the least affected .This finding is also consistent with the above mentioned studies. The reason for the above is that young adults are the prime bread earners of the family and remain outdoors during most of the day. Also young persons are by nature indulge in more violence activities persons. Persons in extremes of the age usually remain indoors.

Sex Distribution (Table 1): Males comprised a majority and constituted 121 (74.69%) compared to females who were 41(25.30%). The male to female ratio in the study was 3:1. This is in accordance with studies 4, 14 and 15. However there is some dissimilarity with studies like 5,6,9,10,11,12,14,15,16,17,18,19,and 20 .The reason for the male majority is the same as for age. Females mostly keep themselves indoor mostly due to cultural background.

Table 1. Shows age and sex distribution in fatal head injury cases

S. No.	Age group (Years)	Sex		Victims	
		Male	Female	No.	%
1	15-24	49	10	59	36.42
2	25-34	30	8	38	23.46
3	35-44	17	5	22	13.59
4	45-54	12	7	19	11.73
5	55-64	10	5	15	9.26
6	65 &>	3	6	9	5.54
	Total	**121**	**41**	**162**	**100**

Mode of Injury (Table 2): Cases of road traffic accident were the most common mode of fatal injury in over 15 years of age comprising 102 (62.97%) cases. Gunshot injury was second common cause of fatal head injury comprising 31 (19.12%) of cases while fall from height was the least common This is in accordance with the study 19 and 21 This is due to the reason that pedestrians are most commonly involved victims than any other group. Riders of motorized two wheelers comprise of second most commonly involved group. There is careless speed driving, thrill seeking, overtaking and less stability of the vehicle. Besides, these vehicles are mostly used by 15-44 years age group.

Table 2. Mode of Injury in Fatal Head Injury Cas2

Sl. no.	Mode of Injury	No of cases	%
1	Road traffic Accident	102	62.97
2	Assaults (Hit)	18	11.12
3	Fall From Height	11	6.79
4	Gunshots	31	19.12
5	Occupational	00	0.00
	Total	**162**	**100**

Seasonal Variation (RTA) (Table 3): Winter season (Nov. to Feb) recorded maximum number 43 cases (42.15%) followed by rainy season (July. to Oct.) 32 cases (31.38%).The summer season recorded the least 27 (26.47%) cases. This is in accordance with studies 4 and 9. In winter there are longer hours of darkness and poor visibility. This is not in accordance with study 25, which has reported that 70% of the accidents have occurred in rainy season. The reason for this is that in rainy season there are slippery roads and poor visibility leading to accidents.

Table 3. Fatal Head Injury cases in Different Seasons due to Road Traffic Accidents

Season	No.	%
Summer (Mar. to Jun)	27	26.47
Rainy (July to Oct.)	32	31.38
Winter (Nov. to Feb.)	43	42.15
Total	**102**	**100**

The Period of Survival (Table 4): Most of victims 44 (27.17%) died within 1-6 hrs. followed by 30 (18.52%) who died within 6-12 hr. Thus majority of patients 110 (67.92%) died within 24 hours of admission in the hospital. Similar finding was found in studies 7, 9, 5 and 23 .However study 4 shows some dissimilarity.

Table 4. Survival Period of Admitted Fatal Head Injury Victims

Time duration	No.	%
< 1 hr	15	9.26
1 hr – 6 hr	44	27.17
6 hr – 12 hr	30	18.52
12 hr – 24 hr	21	12.97
24 hr – 48 hr	26	16.05
48 hr – 72 hr	14	8.65
> 72 hr 12	7.38	
Total	**162**	**100**

Pattern and Distribution of Injuries (Table 5): Laceration in head region was most common followed by fracture of skull bone. The proportion of injuries in thorax, abdomen and spinal cord was lesser as compared to injuries on head, neck, upper and lower extremities.

Table 5. Cranial-Intracranial Lesions in Fatal Head Injury cases

Lesion	No. of Cases	(%)
Scalp Haematoma& Laceration	153	94
Skull Fracture	106	65.00
Temporal	49	46.02
Parietal	45	42.45
Frontal 32	30.18	
Occipital	25	23.58
sphenoid	13	12.26
base of skull	28	26.41

Scalp Injuries: There were 153 (94.00%) individuals with injuries to the scalp. The commonest type of injury was laceration and hematoma. Studies 5 and 10 are consistent with our study.

Skull Fractures: Skull fracture was seen in 106 (65.00%) individuals out of total 162 cases. This figure is not consistent with the findings of study *5,11,12* and 23.Facture of temporal bone 49 cases (46.02%) was commonest followed by parietal bone fracture 45 cases (42.45%).. It found frontoparietal region was the commonest site to be involved in the vault and among basal fractures; middle cranial fossa was found in 25.9% cases. Anterior and posterior cranial fossa was less commonly involved.

Intracranial Injuries (Table 6): Among the intracranial injuries, subdural hemorrhage was the commonest in 133 (82.09%) cases. Next common was subarachnoid hemorrhage in 112 (69.13%) cases, followed by extradural hemorrhage in 42 (25.93%) cases. Contusions of the brain parenchyma were present in 56 (34.56%) cases. Study 5,7 and 23 are not consistent whereas study 11 is consistent with our study.

Table. 6. Intracranial Hemorrhage in Fatal Head Injury cases

i)	Extradural	42	25.93 (%)
i)	Subdural	133	82.09 (%)
ii)	Subarachnoid	112	69.13 (%)
	Brain Contusion	56	34.56 (%)

CAUSE OF DEATH (Table 7): Head injury associated with other injury accounted for maximum of 145 (89.50%) cases as a cause of death followed by head injury alone 119 (73.45%) cases. Haemorrhagic shock was cause of death in 26 (16.04%) cases. In rest of 17 cases cause of death is due to aspiration pneumonitis, pneumothorax, haemothorax and spinal shock. Study 9,16 and 26 are consistent and 4 and 10 are not consistent with our study.

Table 7. Major Cause of Death in Fatal Head Injury cases

Type of injury	No. of Cases
Head Injury associated with other injury	145
Head Injury	119
Haemorrhagic Shock	26

SUMMARY AND CONCLUSION:

1. 162(n=2850) patients died, constituting 6.7%.
2. Age varied from 3 - 70 years. The peak incidence was observed in the age group 15 - 24 years comprising 36.42% of the cases and male: female ratio of 3:1
3. Majority of victims were of road traffic accident 102 (62.97%) followed by gunshot 31cases (19.12%) in fatal head injury cases.
4. Winter season (Nov. to Feb.) recorded maximum number 43 cases (42.15%) followed by rainy season (July to Oct) 32 cases (31.38%) in fatal group.
5. Majority of patients 110 (67.92%) died within 24 hour of admission in the hospital. Maximum number of patients 44 (27.17%) died within 1-6 hrs. followed by 30 (18.52%) within 6- 12 hr.
6. Head injury alone is the cause of death in 119 (73.45%) cases. Head injuries in association with other injury were cause of death in 145(89.50%) cases. Haemorrhagic shock was the next common cause of death in 26 (16.04%) cases.

REFERENCES

1. Park K. Park's Textbook of Preventive and Social Medicine, 19th Edition; 2007 p. 340.
2. The Government of India, Ministry of Home Affairs, National Crime Record Bureau, Accidental deaths and suicides in India 19.
3. Global Road safety crisis. Report of the Secretary-General (A/RES/57/309). United Nation N.Y. 2003.
4. Sinha U.S, ArchanaKaul, Kapoor A. K. Pathak Y. K , Sharma S, Aparajita Singh, Singh S. An epidemiological study of fatal road traffic accidents in Allahabad region. Indian Internet Journal of Forensic Medicine & Toxicology Vol. 3. No. 1. 2005.
5. Tyagi AK, Sharma GK, Kumar B. Craniocerebral damage by blunt force impact. Journal of Indian Academy of Forensic Medicine 1986; 1:24-39. 99
6. Sengupta SK, Sinha SN. Road traffic accidents fatalities in Port Moresby: a ten years survey. AccidAnnalPrev 1989; 21 (3): 297-301.
7. Chandra J, Dogra TD, Dikshit PC. Pattern of Cranio-Intracranial injuries in Fatal Vehicular Accidents in Delhi, 1966-76, Medicine Science & Law 1979; 19(3): 186-194.
8. NilambarJha, Chandra ShekharAgrawal. Epidemiological study of road traffic accident cases: A study from Eastern Nepal. Regional Health Forum WHO South East Asia Region; 8 (1) 2004.
9. Singh H, Dhattarwal S.K. Pattern and distribution of injuries in fatal road traffic accidents in Rohtak (Haryana). Journal of Indian Academy of Forensic Medicine 2004; 26 (1): 20-23
10. Gupta S, Roychowdhury U.B, Deb P.K, Moitra R, Chhetri D. Demographic study of fatal Cranio-cerebral road traffic injuries in North Bengal region. Indmedica Vol. 7, No. 1 (2007-01 - 2007-03).
11. Tripude BH, Naik RS, Anjankar AJ, Khajuria BK. A study of the pattern of Cranio cerebral injuries in road traffic accidents. Journal of Indian Academy of Forensic Medicine. 1998 Jan-Mar; 20(1): 9-12.
12. Sathiyasekharan BWC. Study of the injured and injury pattern in Road traffic accident; Indian Journal of Forensic Sciences 1991; 5: 63-68.
13. Pillay VV. Indian Academy of Forensic Medicine, Scientific Articles of XIV Annual Conference 1992; Jan 4-11.
14. Sheikh MI, Pranav P, Tailor C, Kumar S. Pattern of head injury in homicidal deaths. Indian Journal of 1. Forensic Medicine Toxicology. July-December, 2009, Vol. 3, No. 3, pp 18-21.
15. Sharma BR, Harish D, Singh G, Vij K. Patterns of fatal head injury in Road Traffic Accidents. Bahrain Medical Bulletin Vol.25, No.1, March 2003.
16. Equabal Z, Rizvi S.J, Husain M,Shrivastava V.K. A study of head injury in district Aligarh U.P India. Journal of Indian Academy of Forensic Medicine 2005; 27: 103-107.
17. Singh Y.N, Kaustav kr. Bairagi, Kanak Ch. Das. An epidemiological study of road traffic accident victims in medicolegal autopsies. Journal of Indian Academy of Forensic Medicine, 2005 ;27 (3): 166-169.
18. Lalwani S, Kumar A, Agrawal D, Rautji R, Dogra TD. Fatal road traffic accidents and their relationship with head injuries: An epidemiological survey of five years. Indian Journal of Neurotrauma 2008, Vol. 5, No. 2, pp. 63-67.
19. Chen CL, Howng SL. The incidence and mortality rates of head injuries in Kaohsiung City, Taiwan (1991-1992) Gaoxiong Yi XueKeXueZaZhi. 1995 Sep; 11(9): 537-545.
20. Thanni L.O.A. Kehinde O.A. Trauma at a Nigerian teaching hospital. African Health Sciences2006; 6(2): 104-107.
21. Kremer C, Sauvageau A. Discrimination of falls and blows in blunt head trauma: assessment of predictability through combined criteria. J Forensic Sci. 2009 Jul; 54(4): 923-926. Epub 2009 May 26
22. Liko O, Chalau P, Rosenfeld JV, Watters DA. Head injuries in Papua New Guinea. PNG Med Journal 1996; 39(2): 100-104.
23. Fimate L, Chandra S, Dikshit PC. Survival time in relation to consciousness and severity of head injury. Journal of Forensic Medicine & Toxicology 1992; 8: 15-19.
24. Malhotra C, Singh MM, Garg S, Malhotra R, Dhaon BK, Mehra M. Pattern And Severity Of Injuries In Victims Of Road Traffic Crashes Attending A Tertiary Care Hospital Of Delhi Anil Aggrawal's Internet Journal of Forensic Medicine and Toxicology, 2005; Vol. 6, No. 2 (July - December 2005).
25. Eke N, Etebu EN, Nwosu SO. Road traffic Accidents mortalities in Port Harcourt, Nigeria. Anil Agarwal's Internet Journal of Forensic Medicine and Toxicology 2000, 1: 1-6.
26. Sahdev P, Lacqua MJ, Singh B, Dogra TD. Road traffic fatalities in Delhi: causes, injury patterns, and incidence of preentable deaths. Accid Anal Prev 1994 Jun; 26(3): 377-384.

Stature Estimation from Skull and Facial Anthropometry in Central Indian Population

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ABSTRACT

Stature is an important parameter in medico legal examination. It occurs many a times when highly decomposed or mutilated bodies or sometime only remains of skull are brought for medico-legal examination and this is most common in our region where victims are attacked by wild animals in deep forests which makes difficult to identify diseased. In such medico legal examination stature estimation is important including age, sex, race, etc. Each race requires its own formula for stature estimation because racial and ethnic variations exist in population of different geographical regions. The climate and dietary habits of the people of different regions of India are variable. Hence opinions based on the result of studies done in one population cannot be entirely applicable to other population. Considering this scenario the aim of the present study is to estimate the stature from skull and facial anthropometry in this region. The study was conducted on 470 young and healthy students' males 260 and females 210 of 18 to 24 years. Head circumference and byzigomatic diameter were taken by following standards anthropometric methods and technique. In both male and female HC had greater correlation with stature and better parameter for stature estimation than bizygomatic diameter. Stature estimation can be possible with the help of these two diameters when only skull or remains of the skull brought for medicolegal examination.

Key words : *Stature, Anthropometry, Medicolegal, Head circumference, Byzygomatic diameter*

INTRODUCTION

Stature is an important parameter in medico-legal & forensic examination. It occurs many a times when highly decomposed or mutilated bodies or fragmentary remains of skull are brought for medico-legal examination. Sometime only facial remains are brought for examination and this is most common in our region where victims are attacked by wild animals in deep forests. In such situation it becomes difficult to identify diseased. Among the common questioner of medico legal examination e.g. age, sex, race, etc, stature estimation becomes equally important in such cases. There is definitive biological correlation of stature with all the body parts such as extremities, head, trunk, vertebral column etc[1,5,22, 24]. Many studies have been conducted on stature from percutaneous measurements of various body parts including arm, leg, feet, etc[6,13]. There are a few studies for stature estimation from skull and face parameter alone[14,25]. It is proved beyond doubt that each race requires its own formula for stature estimation. The climate and dietary habits of the people of different regions of India are variable. Racial and ethnic variations also exist in population of different geographical regions[26,27]. Hence opinions based on the result of studies done in one population cannot be entirely applicable to other population. Considering this scenario there is a need of systematic study for stature estimation from fragmented and dismembered skull remains. Therefore the present study has been undertaken to determine stature from skull and facial anthropometry in this region.

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MATERIAL AND METHODS

The study was prospective, from Dec 2007 to Sep 2008, in the Dept. of Anatomy Government Medical College, Nagpur which is located in the central part of India. In the present study 470 medical students were taken comprising of males 260 and females 210 within

the age group of 18 to 24 years. This age group was selected because generally stature at 18 years accepted as an adult[28]. Healthy students without any obvious congenital or acquired deformity of spine, extremity and head were taken in the study. A prescribed proforma was designed for recording the findings. Measurements were recorded to the nearest millimetre. The following parameters were recorded:

1. Stature: - The stature was measured in standing position to the vertex in Frankfurt plane by using anthropometric rod.
2. Head Circumference (HC) - Taken with the help of measuring tape, from the smooth area between eyebrows (Glabella) around the maximum projection of the occiput (Opisthocranion) to the Glabella.
3. Bizygomatic diameter (BZD) - The distance between the most laterally situated points on the Zygomatic arches-the Zygia, which is not a fixed point but determine with the spreading calliper.

All the above measurements were taken by author at a fix time between 2 to 5 pm only to eliminate the discrepancies due to diurnal variation. The measurements were taken three times and their mean value was taken as a final measurement. The data were analyzed using regression analysis and correlation coefficient.

RESULTS

The study consists of 470 healthy subjects consisting of 260 male and 210 female. The age related data is presented in Table 1. The descriptive statistics for the three measurements recorded in the sample are shown in Table 2. In the sample, the average height of male and female was 170.97 (± 6.80) cm and 156.89 (± 5.89) cm respectively. The results show that the differences between all male and female variables exhibited statistically significant differences ($p < 0.001$). The regression equations were derived and shown in Table 3. There is separate equation for skull and facial parameter. The regression equations have been calculated by regression analysis of the data and the values of constants 'a' and 'b' are calculated; where 'a' is regression coefficient of the stature and 'b' is the regression coefficient of any of the skull and facial dimensions. Therefore stature = a + bx, where x is parameter taken in study. It is observed that in both male and female HC had greater correlation with stature (r = 0.366 & 0.400 resp.) with the standard error of ± 6.34 and±5.40 cm respectively. Whereas BZD had lesser correlation with stature(r = 0.212 & 0.271) and the standard error of ±6.65 and ±5.68 respectively in both subjects.

Table 1. Descriptive age-wise and sex-wise statistics

Sex	n (%)	Mean age (years)	± SD	Median	Minimum	Maximum	Mode
Male	260 (55.31)	19.68	1.39	19	18	24	19
Female	210 (44.68)	19.06	1.06	19	18	24	19
Combined	470 (100)	19.42	1.29	19	18	24	19

SD - standard deviation

Table 2. Descriptive statistics for sex-wise height and parameters (in cm)

Sex	Variable	N	Mean (± SD) (in cm)	Minimum	Maximum	Median	CV (%)
Male	Height	260	170.97(±6.80)	151	189.60	171	3.97
	HC	260	54.80(±1.62)	44.50	58.70	54.65	2.18
	BZD	260	13.50(±0.66)	11.40	17.30	13.50	4.88
Female	Height	210	156.89(±5.89)	142	172.70	156.60	3.75
	HC	210	52.95(±1.42)	49	57.20	53	2.68
	BZD	210	12.62(±0.53)	10.60	15.40	12.60	4.19

SD - standard deviation, SE - standard error of mean, CI - confidence interval, CV - coefficient of variation, HC - head circumference, BZD - bizygomatic diameter.

Table 3. Statistical analysis with derivation of regression equation

Sex	Variable	b	r	P value	a	SEE	Regression equation
Male	HC	1.53	0.366	0.000,S	87.13	±6.34	Stature(cm) = 87.13 + 1.53 × HC
	BZD	2.22	0.212	0.000,S	141	±6.65	Stature(cm) = 141 + 2.22 × BZD
Female	HC	1.65	0.400	0.000,S	70.53	±5.40	Stature(cm) = 70.53+1.65 × HC
	BZD	3.006	0.271	0.000,S	118.99	±5.68	Stature(cm) = 118.99+ 3.006 × BZD

b - regression coefficient, r - correlation coefficient, a - intercept, SEE - standard error of estimate, HC - head circumference, BZD - bizygomatic diameter.

Table 4. Comparative data of different studies

Study	Parameter	Sex	Correlation coefficient	SEE
Chiba (1998)	Height Vs HC	Male	0.38	± 7.12
		Female	0.32	± 6.59
Jibonkumar (2006)	Height Vs BZD	Male	0.18	--
Bardale (2006)	Height Vs HC	Male	0.45	± 5.89
		Female	0.44	± 5.37
Kalia (2008)	Height Vs HC	Male	0.14	-
		Female	0.00	-
Krishan (2008)	Height Vs HC	Male	0.78	± 3.72
Agnihotri (2011)	Height Vs HC	Male	0.49	--
	Height Vs BZD	Male	0.17	--
	Height Vs HC	Female	0.37	--
	Height Vs BZD	Female	0.27	--
Present study	Height Vs HC	Male	0.36	±6.34
	Height Vs BZD	Male	0.21	±6.65
	Height Vs HC	Female	0.40	±5.40
	Height Vs BZD	Female	0.27	±5.68

HC - head circumference, BZD - bizygomatic diameter,

DISCUSSION

Stature estimation is important in medicolegal forensic examination and based on a principle that bones or human body part correlate positively with the stature. Therefore there is need to investigate whether there is any possible significant correlation exists between stature and skull parameter? For this task we had taken HC and BZD to estimate stature. In the past, few studies have been conducted so far[20-25] and the comparative data is presented in Table 4.

In this study the correlation is higher and standard error of estimate is lower for HC is better in both male and female for stature estimation than that of BZD when fragmentary remains of skull were there for medicolegal examination. The similar study was carried by Agnihotri AK et al[25] in Indo-Mauritian population (75 male and 75 female of 20 to 28 age group) also found the HC had greater correlation coefficient in male than that of female. Similar findings were noted by Chiba[20] (1998), Bardale[21] (2006) and Jibonkumar[22] (2006) for HC where they found correlation less than 0.5. When compared to the present study, the study of Kalia et al[23] (2008) in Mysorian population found less correlation and that of Krishan[24] (2008) in male Gujjars of north Indian population found statistically significant correlation ($r > 0.5$). This difference is due to geographic and ethnic variation[26-27].

The stature can be estimated by the regression analysis and multiplication factor but the regression analysis is considered best for stature estimation by most of the researchers[24,29]. Statistically the correlation coefficient is considered statically significant if it is above 0.5[25]. In this study, we have noted that HC and BZD have lower value in both sexes. Therefore, it can be stated that these are not preferable parameters but should be utilized in absence of other better parameters such as long bones or when only skull remain is presented for forensic examination.

CONCLUSION

To conclude HC is better parameter than BZD for stature estimation using regression equation in both male and female only when skull or fragmentary remains of skull was brought for medicolegal examination in the absence of long bone as the correlation coefficient for height with long bones such as femur, tibia etc are statistically significant.

Conflict of Interest : None

REFERENCES

1. Wells LH. Estimation of stature from long bones: a reassessment. J Forensic Med 1959; 6: 171-177.
2. Dupertuis CW, Hadden JA. On the reconstruction of stature from long bones. Am J Phys Anthropol 1951; 9: 15-23.
3. Tibbets GL. Estimation of stature from vertebral column in American Blacks. J Forensic Sci 1981; 26: 715- 723.
4. Jason DR, Taylor K. Estimation of stature from cervical, thoracic, and lumber segments of the spine in American Whites and Blacks. J Forensic Sci 1995; 40: 59-62.
5. Nagesh KR, Pradeep Kumar G. Estimation of stature from vertebral column length in south

Indians. Leg Med (Tokyo) 2006; 8: 279-283.
6. Ozaslan A, Iscan MY, Ozaslan I, Tugch H, Koc S. Estimation of stature from body parts. Forensic Sci Int 2003; 132: 40-45.
7. Ozaslan A, Koc S, Ozaslam I, Tugcu H. Estimation of stature from upper extremity. Mil Med 2006; 171(4): 288-291.
8. Patel MP, Joshi NB. Regression equation of height on ulnar length. Indian J Med Res 1964; 52: 1088-1091.
9. Patel MP, Joshi NB, Dongre AV. Regression equation of height on tibial length. Indian J Med Res 1964; 52: 531-534.
10. Giles E, Vallandigm PH. Height estimation from foot and shoe print length. J Forensic Sci 1991; 36: 1143-1151.
11. Kishan K. Estimation of stature from foot print and foot outline dimensions in Gujjars of North India. Forensic Sci Int 2008; 175: 93-101.
12. Bhatnagar DP, Thapar SP, Batish MK, Identification of personal height from the somatometry of the hand in Punjabi males. Forensic Sci Int 1984; 24: 137-141.
13. Rastogi P, Kanchan T, Menezes R, Yoganarasimha K. Middle finger length - a predictor of stature in the Indian population. Med Sci Law 2009; 49: 123-126.
14. Sarangi SK, Dadhi B, Mishra KK. Estimation of stature from somatometry of skull. J Indian Acad Forensic Med 1981; 182: 24-26.
15. Ryan I, Bidmos MA. Skeletal height reconstruction from measurements of the skull and indigenous South Africans. Forensic Sci Int 2007; 167: 16-21.
16. Hayperuma I. On the prediction of stature from cranial dimensions. Int J Morphol 2010; 28: 1135-1140.
17. Patil KR, Mody RN. Determination of Sex by discriminant function analysis and stature by regression analysis: a lateral cephalometry study. Forensic Sci Int 2005; 147: 175 -180.
18. Jadhav HR, Shah GV. Determination of personal height from the length of head in Gujarat region. J Anat Soc India 2004; 53: 20-21.
19. Kharyal A, Nath S. Estimation of stature from maxillofacial height measurements among Brahmins of Himachal Pradesh. Indian J Forensic Odont 2008; 1: 9-12.
20. Chiba M, Terazawa K. Estimation of stature from somatometry of skull. Forensic Sci Int 1998; 97: 87-92.
21. Jibonkumar, Lilinchandra. Estimation of stature using different facial measurements among Kabui Naga of Imphal vally, Manipur. Anthropologist 2006; 8: 1-3.
22. Bardale R, Dixit PG, Dhawane SG. Estimation of stature from somatometry of skull. J Mah Medicolegal Asso 2006; 18: 22-26.
23. Kalia S, Shetty SK, Karthikrya P, Mahima VG. Stature estimation using odontometry and skull anthropometry. Indian J Dental Res 2008; 19: 150-154.
24. Krishan K. Estimation of stature from cephalo-facial anthropometry in north Indian population. Forensic Sci Int 2008; 181: 52-56.
25. Agnihotri AK, Kachhwaha S, Googoolye K, Allock A. Estimation of stature from cephalo-facial dimensions by regression analysis in Indo-Mauritian population. J Forensic Leg Med 2011; 18: 167-172.
26. Trotter M, Gleser G. Estimation of stature from long bones of American whites and Negroes. Am J Phys Anthropol 1952; 9: 463-514.
27. Siddiqui MA, Shah MA. Estimation of stature from long bones of Punjabis. Indian J Med Res 1944; 32: 104-108.
28. Roche AF, Davila GH. Late adolescent growth in stature. Pediatrics 1992; 50: 874-880.
29. Iscan MY. Forensic anthropology of sex and body size. Forensic Sci Int 2005; 147: 107-112.

Pulmonary Candidiasis: Incidental Cause of Death Made on Autopsy

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ABSTRACT

An autopsy case report of 30 year old female patient having widespread bed sores, dyspnea and high grade fever who died within an hour of admission to the hospital. An autopsy was conducted and viscera consisting of parts of lungs, kidneys, liver, spleen and heart were sent for histopathological examination. Histopathological examination of lungs revealed presence of chronic inflammatory infiltrate along with numerous colonies of round to ovoid fungal spores. Differential diagnosis of Candida and Cryptococcus was kept and using PAS and mucicarmine stains pulmonary candidiasis was confirmed. The present case highlights pulmonary candidiasis comes under category of severe mycoses and is an incidental diagnosis made on autopsy.

Key words: *Pulmonary candidiasis, Cryptococcosis, PAS, Mucicarmine*

INTRODUCTION

Mycoses is defined as infections caused by eumycotic organisms such as Candida, Aspergillus, Cryptococcus, Zygomycetes and other fungal species excluding infections caused by filamentous bacteria such as Actinomycetes, Pneumocystis carnii along with superficial infections such as dermatophytosis.[1,2] Mycoses detailed in autopsy records were defined as severe if they were direct cause of death and caused severe pulmonary infection involving both lobes of lungs.[2] The purpose of this article is to highlight that in presence of fungal spores in both lungs in an immunocompromised patient differential diagnosis of candida and cryptococcus should be made in absence of involvement of other organs. In this case using special stains like PAS (Periodic Acid Schiff) and mucicarmine, pulmonary candidasis was confirmed as cause of death. Present case highlights pulmonary mycoses may be considered as one of the most important cause of death in an immunocompromised patient.

CASE REPORT

A 30-year-old female patient suffering from lower limb paralysis since 10 years was admitted to emergency with high grade fever, dysponea and wide spread bed sores. She died within an hour of admission. Autopsy was conducted and viscera consisting of both lungs, kidneys, liver, spleen and heart were sent for histopathological examination.

The cause of death was ascertained on autopsy and histopathological examination of viscera especially lungs. On gross examination lungs were solid [Fig. 1].

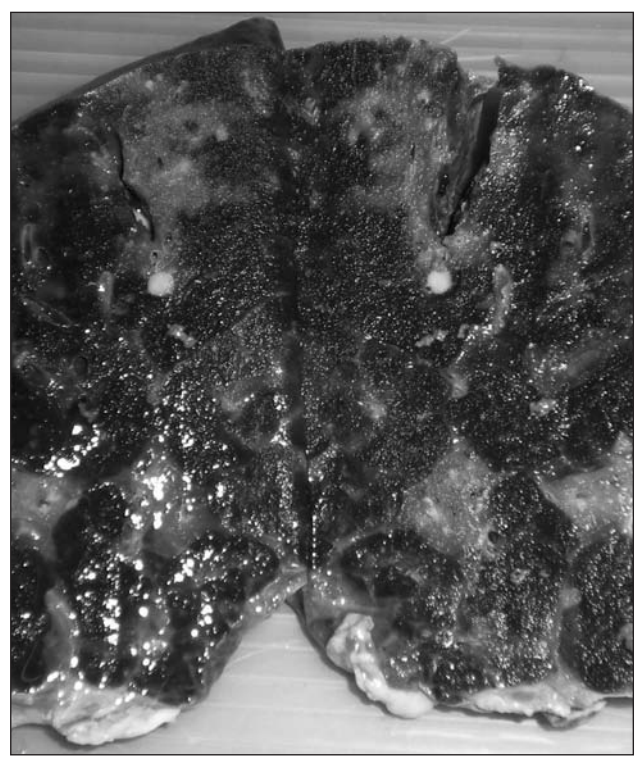

Fig. 1. Gross appearance of lungs

Microscopic examination on haematoxylin and eosin [H&E] stain revealed presence of round to ovoid spores of fungus along with chronic inflammatory infiltrate and thickening of alveolar septa [Fig. 2].

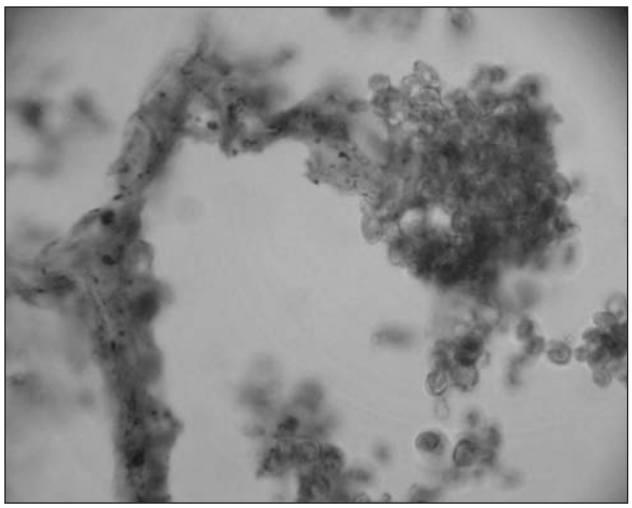

Fig. 2. Sections from lungs show numerous colonies of round to ovoid fungal spores (H& E, x400).

On the basis of H&E stain two differential diagnosis of candida and cryptococcus was kept. Special stains PAS [Periodic acid Schiff] and mucicarmine were done. PAS highlighted budding spores of candida [Fig. 3]. Mucicarmine stain was negative as it did not highlight any capsule ruling out cryptococcus. Microscopic examination of other organs was unremarkable.

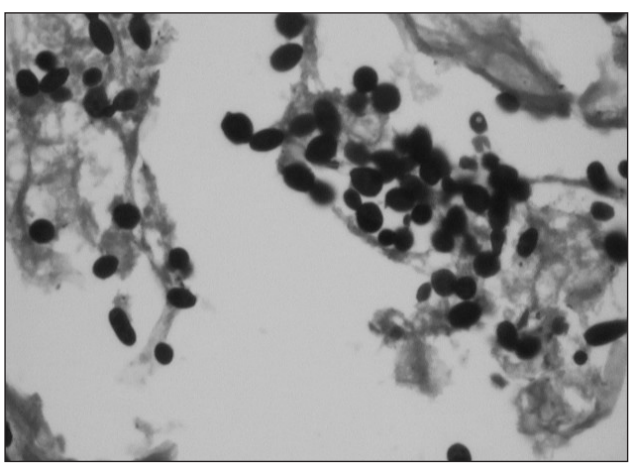

Fig. 3. Sections from lungs shows spores of Candida showing budding (PAS x400)

DISCUSSION

Pulmonary infections are the most common form of documented tissue invasive infections observed in immunocompromised hosts.[3,4,5,6,7] Immunocompromised hosts are defined by susceptibility to infection with organism of little native virulence in normal individual.

Yamazaki T, Kume HS et al defined mycoses as infections caused by eumycotic organisms such as Candida, Aspergillus, Cryptococcus, zygomycetes, trichosporon and other fungal infections excluding infections caused by actinomycetes, Pneumocystitis carnii and superficial infections such as dermatophytoses.[1, 2]

Yamazaki T, Kume HS et al documented that mycoses detailed in autopsy records were defined as severe if they (i) were direct cause of death, (ii) caused severe pulmonary infection involving both lobes of the lung, (iii) caused severe visceral infections of two or more organ systems including those involving the CNS, which were not regarded as the direct cause of death, (iv) caused multi organ system infections of three or more organ system excluding the CNS (v) caused fungemia.[1,2] The present case fulfills the first two enlisted criteria so making it as severe mycoses.

Kume H, Yamazaki T et al documented that among the total number of deaths in Japan, the frequency of mycotic infections particularly candidiasis as the certified cause of death has increased noticeably in recent years.[2] McNeil et al reported similar increase in frequency of invasive mycoses between years 1980 to 1997 in the USA, pointing out that number of cases with invasive mycosis increased from 1557 to 6534.[8]

Banerjee SN, Emori TG, Culver DH et al and Kume H, Yamazaki T et al observed that the lung and bronchial system was involved most frequently, regardless of pathogen species. This suggests that the lungs and bronchi are at the highest risk of being exposed not only to exogenous pathogens such as aspergillus, cryptococcus but also to candida species. Although candida species is commensal of digestive tract, it is possible for them to be a major causative agent of systemic infection in immunocompromised patients.[2, 9] Candida is known to be involved in nosocomial transmission.[9,10] These studies are in concordance with the present case.

Yamazaki T, Kume HS et al highlighted that if patient suffers from mycoses and dies due to it in the presence of underlying disease than it should be certified as major cause of death because of difficulty in making the diagnosis.[1]

The purpose of this case report is to highlight that in an immunocompromised patient pulmonary candidiasis involving both lobes of lungs qualifies as severe mycoses and is direct cause of death.

REFERENCES

1. Yamazaki T, Kume HS, Murase S, Yamashita E, Arisawa M. Epidemiology of Visceral Mycoses:

Analysis of Data in annual of the Pathological autopsy cases in Japan. J Clin Microbiol 1999; 37: 1732-1738.

2. Kume H, Yamazaki T, Abe M, Tanuma H, Okudaira M, Okayasu T. Increase in aspergillosis and severe mycotic infections in patients with leukemia and MDS: Comparison of the data from the Annual of the Pathological Autopsy Cases in Japan in 1989,1993 and 1997: Pathology International 2003; 53 : 744-750.
3. Fishman JA, Rubin RH. Infection in organ-transplant recipients. N Engl J Med 1998; 338:1741
4. Etlinger NA, Trulock EP. Pulmonary considerations of organ transplantation. Part I. Am Rev Respir Dis 1991;143: 1386.
5. Winstow DJ, Emmanouilides C, Busuttil Rw. Infections in liver transplant recipients. Clin Infect Dis 1995; 21: 1077.
6. Ramsey PG, Rubin RH, Tolkoff- Rubin NE et al. The renal transplant patient with fever and pulmonary infiltrates:etiology, clinical manifestations and management. Medicine (Baltimore) 1980; 59: 206.
7. Rosenow EC 3rd. Diffuse pulmonary infiltrates in the immunocompromised host. Clin. Chest Med 1990; 11: 55.
8. Mc Neil MM, Nash SL, Hajjeh RA et al. Trends in mortality due to invasive mycotic diseases in the United States, 1980-1997. Clin Infect Dis 2001; 33: 641-647.
9. Banerjee SN, Emori TG, Culver DH et al. The National Nosocomial Infections Surveillance System 1991. Secular trends in nosocomial primary blood stream infections in the United States.1980-1989. Am J Med 1991; 91: 586-589.
10. Kullberg BJ, Oude Lashof AM. Epidemology of opportunistic invasive mycosis. Eur J Med Res 2002; 7: 183-191.

Analysis of Homicidal patterns in Bangalore City, India

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ABSTRACT

This article describes the homicide pattern in Bangalore city, India. Out of the 528 medico-legal autopsies performed at Dr. B R Ambedkar Medical College, between August 2010 and July 2011, 50 cases were homicidal on the basis of police inquest and autopsy. Results showed that about 28% of victims were between 31-40 years age group, married and belonged to lower socio-economic status. The male: female ratio was 1.94:1.0. A large number (42%) of homicides occurred during night times and 38.88% were related to alcohol consumption. The main motive (83.3%) was vengeance. Head injury was the leading cause of death in 36% and 56 % victims died on spot.

Key words: *Homicide, Autopsy, Bangalore, Vengeance.*

INTRODUCTION

Homicide is the death of one human being as a result of the conduct of another.[1] They are ubiquitous. Many of them may occur in a rage and few with lot of planning. The assailant may be an innocent or a professional with involvement of one man in a killing or many assailants along with multiple precipitating factors. Sometimes a different person may be killed than actual man on whom it was planned. The victims are not only children, old people, females or incapacitated persons but also healthy and hale person may be killed and the circumstances differ in each of these deaths. Most of the times, the victim is caught alone. The Methods adopted, the weapons used, the time of killing, etc., differs from case to case, place of occurrence and time. There has been a global increase in homicide and it causes over 5, 00,000 deaths per year worldwide.[2-5]

Data related on homicides from few cities of India are available.[6-9] There is limited data about homicidal deaths in Bangalore city and analysis of homicidal deaths in a society can be the first step in developing strategies to prevent it. Hence, an analytical study was conducted to know the details of such deaths in regard to the above factors.

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Legal aspects of homicide:

In Indian Penal Code, there are several sections dealing with Homicide. Some of these sections are 299; 300; 301; 302; 303; 304; 304 — A; 307; 308 etc.[10]

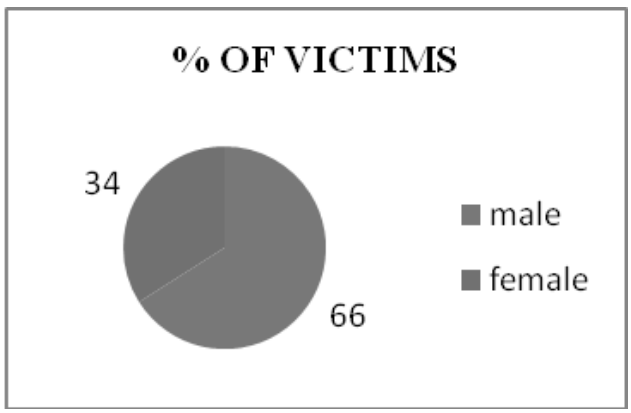
MATERIAL AND METHODS

The study was undertaken in the Department of Forensic Medicine and Toxicology and Physiology at Dr. B R Ambedkar Medical College, Bangalore, from August 1st 2010 to July 31st 2011. Out of 528 medico legal autopsies done in this period, 50 cases were determined to be of homicidal death on the basis of police inquest and autopsy. These cases were examined regarding their socio-demographic characters, part of body involved, time of death, reasons for homicide and manner of death.

The material regard to them, as Inquests, first information reports, statements made by the relatives, hospital records, panchanamas of scene of offence etc., were collected from the Police, apart from the Postmortem examination reports from the Department. Some information was also collected in person from the relatives, who attended the Mortuary at the time of Postmortem examination, where ever it is possible. Visit to the scene of offence were made whenever felt necessary. The data was entered in

master chart and was analyzed using appropriate statistical analysis — measures of central tendency.

Chart 1. Sex determination in homicidal cases



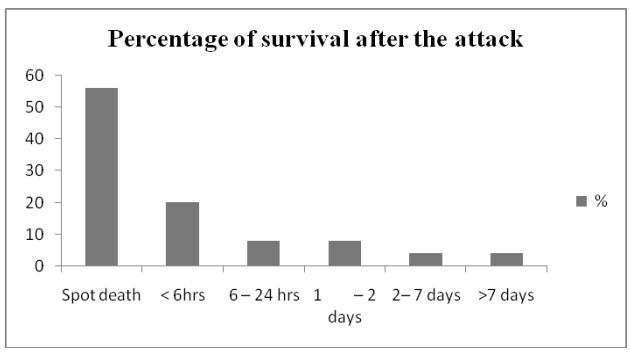


Chart 2. Period of survival after the attack

RESULT AND DISCUSSION

During this analytical study, it was found that out of 50 cases, male preponderance was seen33(66%) cases in comparison to 17(34%) cases of females] in ratio of 1.94:1.0. This indicates males are more exposed to the outside environment and indulge in more violent activities. Similar findings were observed by Mohanty M K[6], Hugar BS [9], Zafar Marri M[11], Bhupinder S[12] and Alan fox J.[13] But in a study conducted by Kominato Y in Japan, male to female ratio of victims was 1:1.[14]

People in 31-40 years age group were more vulnerable to homicides as 14 (28%) deaths occurred in this group. The next age group is 41-50 years, in which 10(20%) deaths occurred. This finding was slightly different from other studies observations where age group of 20- 31 years were more vulnerable for homicides. [8, 9, 11, 12] 30% victims were illiterates and belonged to lower socio-economic status as per modified kuppuswamy's classification. Concordant findings were found by Edward G.[15] This imply that marital disharmony, mental illness/stress illiteracy, financial problems and unemployment may play an important role in homicides.

A large number (42%) of homicides took place in night. Similar findings were observed by Wahlsten P [16] and Gupta A [17] whereas in a study conducted by Vougiousklakis, [18] a large number (26.9 %) occurred in noon. In our study 38.88 % of homicides were alcohol related. Similar finding was noted by Edward G.[15] It could be due to the fact that homicide being heinous crime, it is well planned and the assailant tries to execute it without being witnessed by others and easy escape during night time. Added to this alcohol consumption gives the assailant a sense of false courage by inhibiting the normal functions of frontal lobe of the brain.[19]

The main motive behind homicide was found to be vengeance (83.3%). This was comparable with the observations by Vougiousklakis T,[18] and in a study conducted by Alan Fox J,[13] it was argument.

Head injury was the leading cause of death and 18 people (36%) died as a result of head injury. Similar finding was observed by Mohanty K[8] whereas in a study conducted in Peshawar, Pakistan showed that firearms were targeted to chest and abdomen areas and sharp weapons were targeted to head and neck regions.[11] These differences in the type of trauma may be because of the availability and choice of the weapon used in a different societies and cultures and head injury often being irreversible leads to death. [20]

Maximum number of the deaths (56 %) occurred on the spot which was similar to that observed by Hugar B S. [9] In the present study few peculiar observations were made, that is one mother killing her child before committing suicide by throwing in a well. One father killed his daughter by burns. In fact burns were made on his wife who was not amicable to him and the daughter died accidentally when she came into the fire. Even the children are killing their parents. One son killed his father because he was giving all his property to his younger sister.

CONCLUSION

Homicidal deaths constituted 9.46% of medico-legal autopsies performed during one year. Many victims were in the 31-40 years age group, male, married, illiterates and belonged to lower socio-economic status. The homicides occurred during night with vengeance being the main motive and alcohol related. Head injury was the leading cause of death and majority of victims died on spot.

Limitations

1. Number of homicides analyzed was less.
2. Lack of advanced investigating procedures.
3. The study is not complete because of several hindering factors, like non-cooperation from the

Table 1. Age distribution of homicide victims

Age (years)	Number of victims
0-9	2
10-19	4
20-29	7
30-39	14
40-49	10
50-59	8
60-69	4
□70	1

Table 2. Motives behind homicidal cases

Motive	Number of victims
Vengeance	18(36%)
Defensive attack	2(4%)
Monetary	7(14%)
Monetary rage	6(12%)
Sexual jealous	5(10%)
Removing obstacles	3(6%)
Unknown	9(18%)

Table 3. Time of occurrence of homicides

Time	Number of victims
Early morning	8(16%)
Morning	11(22%)
Midday	6(12%)
Evening	4(8%)
Night	31(62%)

Table 4 Period of survival after attack

Period of survival	Number of victims
Spot death	28(56%)
< 6hrs	10(20%)
6 – 24 hrs	4(8%)
1 – 2 days	4(8%)
2 – 7 days	2(4%)
>7 days	2(4%)

Table 6. Causes of death of victim

Cause	Number of victims
Head injury	18(36%)
Multiple injuries	11(22%)
Stab injuries	7(14%)
Throttling	4(8%)
Ligature strangulation	3(6%)
Drowning	1(2%)
Pressure over neck	2(4%)
Burns	1(2%)
Decapitation	1(2%)
Complications of injuries	2(4%)

Investigating Officer, non compliance of the relatives.

Recommendations

1. In the Indian scenario the investigating officer, the forensic pathologist and the judiciary system work independently and not in tandem as in the western countries where the homicide unit is constituted who share their knowledge in solving a crime. Hence investigating officer should work/co-ordinate with the forensic pathologist in solving homicides.
2. Night time, bar and alcohol are always interrelated. In the present study it was observed that many homicides were related to alcohol consumption. It is well known about the damages related to sale and consumption of alcohol. Government should not give licenses to liquor shops in excess and awareness about the hazards of alcohol to be conveyed to the public. Strict enforcement has to be done by police to make all bars to close by 11.30pm.
3. Strict enforcement of law on possession of dangerous weapons like sharp heavy cutting weapons/firearms.
4. Mobile Medical Clinics and organization of medical psychiatric services Door-to-Door with Forensic Psychiatrist as team leader. Strict implementation of mental health check-ups should be done.

Conflict of Interest : Nil

REFERENCES

1. Mant AK. Taylor's Principles and Practice of Medical Jurisprudence. 13th Ed. New Delhi: B.I. Churchill Livingstone, 1994: 215.
2. Reza A, Mercy JA, Krug E. Epidemiology of Violent Deaths in the World. Injury Prevention. 2001; 7: 104-111.
3. Fornes P, Druilhe L, Lecomte D. Homicide among youth and young adults, 15 through 29 years of age. A report of 138 cases from Paris and its suburbs 1991-1993. J. Forensic Sci. 1996; 41 (5): 837-840.
4. Rosenburg ML, Mercy JA. Rosenberg ML, Finley MA, Assaultive Violence in America, a public health approach. Oxford: Oxford University Press, 1991: 14-50.
5. Hasan Q, Shah MM, Bashir MZ. Homicide in Abbottabad. J Ayub Med Coll Abbottabad, 2005; 17 (1): 78-80.
6. Mohanty MK, Mohanty S, Acharya S. Circumstances of crime in homicidal deaths. Med Sci Law. 2004 Apr; 44(2): 160.
7. Homicide trends in Delhi, https://www.novapublishers.com/catalog/

product_info.php?products_id=20478India pp.129-142. Dated: 29/8/20011.

8. Manoj Kumar Mohanty T.S. Mohan Kumar, Arun Mohanram and Vikram Palimar Journal of Clinical Forensic Medicine Volume 12, Issue 6, December 2005, Pages 302-304.
9. Hugar SB, Chandra G, Harish S, Jayanth SH, Pattern of Homicidal Deaths, J Indian Acad Forensic Med, 32(3).
10. Narayana Reddy K.S., The Essentials of Forensic Medicine and Toxicology Medical Book Company, Hyderabad, 29th edition 2010 page 259.
11. Murad Zafar Marri, M. Zahid Bashir, Anjum Zia Munawar, Zahid Hussain Khalil, Inayat urRehman Khalil, Analysis of homicidal deaths in Peshawar, Pakistan, J Ayub Med Coll Abbottabad 2006; 18(4)
12. Bhupinder S, Kumara TK, and Syed AM, Pattern of homicidal deaths autopsied at Penang Hospital, Malaysia, 2007-2009: A preliminary study. Malaysian J Pathol 2010; 32(2): 81–86.
13. Alan Fox. J., Zawitz M. W., 2000. Homicide Trends in the United States: 2002 Update, Bureau of Justice Statistics Crime Data Brief, 1-4, available on www.OJP.usdoj.gov/bjs/homicide/homtrnd.htm, accessed on 2nd August 2007.
14. Kominato. Y. et al., 1997. Homicide Patterns in the Toyoma Prefecture, Japan, Medicine, Science and Law, 37(4): 316-320.
15. Edward green, Russel P. Wakefield. The Journal of Criminal Law and Criminology, vol.70, No.2. pg 172.
16. Wahlsten. P. et al., 2007. Survey of Medico legal Investigation of Homicides in the City of Turku, Finland, Journal of Clinical Forensic Medicine, 14: 243-252.
17. Gupta Avnesh et al. 2004. A study of Homicidal Deaths in Delhi. Medicine, Science and Law, 44 (2): 127-132.
18. Vougiouklakis. T, Tsiligianni C, 2006. Forensic and Criminologic aspects of murder in North-West (Epirus) Greece, Journal of Clinical Forensic Medicine, 13: 316-320.
19. Shivakumar B. C., Vishwanath D, Srivastava P C, J Indian Acad Forensic Med. April-June 2011, Vol. 33, No. 2. Pages 120-124.
20. Essam M. Ali; Amal A. Elbakry & Mahmoud A. Ali, A study of elderly unnatural deaths in medicolegal autopsies at dakahlia locality. Mansoura J. Forensic Med. Clin. Toxicol. Vol. XV, No. 1, Jan. 2007. Pages 33-42.

A Radiological Study of Epiphyseal Union of Knee Joint for Age Estimation

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ABSTRACT

Age is an important parameter for medico-legal cases. Many times doctors are called upon to give opinion about age of a person. For this objective methods of age determination are required. Age of epiphyseal union is an important objective method of age determination. But these ages varies with racial, geographic, climatic and various other factors. These variations have suggested need of separate standards of ossification for separate regions.

Present work is undertaken to work out ages of epiphyseal union around knee joint for Vidarbha region. Study is carried out in total 160 healthy subjects (88 girls and 72 boys) aging from 13 to 23 years & having length of residence in Vidarbha not less than 10 years. The chronological age upto the day of examination is determined & A-P view of right knee joint is taken in each case. These radiographs are studied to determine age of union of epiphyses of lower end of femur , upper end of tibia & upper end of fibula. These ages are compared with those reported from various states of India & also from other countries & found to vary appreciably. Sexual dimorphism for ages of epiphyseal union is also studied.

Key words: *Epiphyses, Ossification, Femur, Tibia, Fibula*

INTRODUCTION

Determination of age of an individual is a subject mater of great medico-legal and academic interest. Very frequently doctors are required to opine about the age of a person which may be for medicolegal purpose, for juvenile court procedures, or for entry to the government services. Registration of birth is still extremely incomplete in India & many times real age is concealed with various intentions. Also many times mutilated skeletal remains of lower limb present as MLC work & it becomes difficult to comment on age of the bones. So it becomes necessary to use some objective method to find exact age of a person. Among various methods of age determination, ages of appearance & union of epiphyses with diphyses, as observed radiologically is considered to be a reliable guide.

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Though these ages are fairly constant for a particular bone, there are great variations with racial, geographic, climatic & various other factors. Appreciable variations have been recorded not only by workers from different countries (Paterson 1929[15],Flecker1942[7]) but even by the workers from various provinces of Indian subcontinent (Pillai 1936[13], Galstaun 1937[8,9], Gupta et al 1974[5], Mishra B.D. 1966[14], Saksena & Vyas 1969[16]) Due to this, need for separate standards of ossification for separate regions have been suggested (Loder et al 19931[12], Koc et al 2001[11], Crowder et al 2005[4]). Such standard have been investigated in many states of India s.But no such standards have been investigated for Vidarbh region. So the present work is undertaken to investigate the same in Vidarbh.

MATERIALS AND METHODS

The present study was carried out in the Department of Anatomy, Government Medical College and Hospital, Nagpur (Maharashtra).

Study was performed in total 160 subjects (72 boys & 88 girls) having ages from 13 to 23 years. The length of residence of each subject in Vidarbha region was ascertained and those having less than 10 years stay in the Vidarbha region were excluded from the present

investigation. All the subjects belonged to middle socio-economic status. Freedom from musculo-skeletal, nutritional and endocrine disorders and also from any debilitating ailments in childhood was taken into account. Height, weight and general physical development were recorded in all cases and the menstrual history of girls was also accounted for. Dietetic history was also taken for all subjects.

Accurate age, as for as possible, was determined in each case based on the statements of the subjects, supported by their school leaving certificates. The subjects were divided into ten groups as 13-14, 14-15, 15-16, 16-17, 17-18, 18-19, 19-20, 20-21, 21-22, 22-23 years according to their ages. The distribution of boys and girls in each age group is shown in Table 1.

All these subjects were examined clinically and radiologically. Antero-posterior views of right knee joint were taken in each case.

Criteria of union:-The union was considered as complete when the space between diaphysis (shaft) and epiphysis was fully obliterated and bony in architecture and density, indistinguishable from the epiphysis and diaphysis in its neighbour-hood. Periosteum between the epiphysis and diaphysis should be in continuity without any notching at the periphery of epiphyseal line. Cases of recent union, where a white transverse line was still seen in place of the epiphyseal cartilage, was also taken as complete union and the so called epiphyseal scar was disregarded. The youngest age group showing complete union in 100% subjects was taken as criteria for generalization.

A-P view of right knee joint showing unfused epiphyses around knee joint

A-P view of right knee joint showing complete union of epiphyses around knee joint

Observation & results

Data is tabulated and statistically analysed———

Table 1. Distribution of subjects according to age & sex

Age (years)	No. of cases		Total	%	
	Boys	Girls			
13-14	0	6	6	3.75	
14-15	0	6	6	3.75	
15-16	10	10	20	12.5	
16-17	8	10	18	11.25	
17-18	10	8	18	11.25	8.5
18-19	10	12	22	13.75	Non
19-20	10	8	18	11.25	Significant
20-21	8	10	18	11.25	
21-22	8	10	18	11.25	
22-23	8	8	16	10	
Total	**72**	**88**	**160**	**100%**	

Table 2. Age of union of lower end of femur with shaft

Age group (years)	No. of cases examined		Number of cases showing complete union		%	
	Boys	Girls	Boys	Girls	Boys	Girls
13-14	0	6	-	0	-	0
14-15	0	6	-	4	-	66.66
15-16	10	10	4	8	40	80
16-17	8	10	4	10	50	100
17-18	10	8	8	8	80	100
18-19	10	12	10	12	100	100
19-20	10	8	10	8	100	100
20-21	8	10	8	10	100	100
21-22	8	10	8	10	100	100
22-23	8	8	8	8	100	100

Table 3. Age of union of the upper end of tibia with shaft

Age group (years)	No. of cases examined		Number of cases showing complete union		%	
	Boys	Girls	Boys	Girls	Boys	Girls
13-14	0	6	-	0	-	0
14-15	0	6	-	0	-	0
15-16	10	10	0	4	0	40
16-17	8	10	2	6	25	60
17-18	10	8	6	8	60	100
18-19	10	12	10	12	100	100
19-20	10	8	10	8	100	100
20-21	8	10	8	10	100	100
21-22	8	10	8	10	100	100
22-23	8	8	8	8	100	100

Table 4. Age of union of upper end of fibula with shaft

Age group (years)	No. of cases examined		Number of cases showing complete union		%	
	Boys	Girls	Boys	Girls	Boys	Girls
13-14	0	6	-	0	-	0
14-15	0	6	-	0	-	0
15-16	10	10	0	2	0	20
16-17	8	10	2	4	25	40
17-18	10	8	4	6	40	75
18-19	10	12	6	12	60	100
19-20	10	8	10	8	100	100
20-21	8	10	8	10	100	100
21-22	8	10	8	10	100	100
22-23	8	8	8	8	100	100

RESULTS

From Table 2, complete union of lower end of femur is first seen in 100% boys at 18-19 years & in 100% girls at 16-17 years. So the age of union of lower end of femur is 18-19 years for boys & 16-17 years for girls.

From table 3, complete union of upper end of tibia is observed at 18-19 years in 100% boys & at 17-18 years in 100% girls. So, the age of union of upper end of tibia is 18-19 years for boys & 17-18 years for girls.

For upper end of fibula, complete union is first seen in 100% cases at 19-20 years in boys & at 18-19 years in girls. So the age of union of upper end of fibula is 19-20 years for boys & 18-19 years for girls.

In case of all the epiphyses studied, union if found tobe earlier in girls than boys.

Table 5. Comparison of ages of union of epiphyses around knee joint given by Various workers in India with findings of present study

Authors	Lower end of femur		Upper end of tibia		Upper end of fibula	
	Male	Female	Male	Female	Male	Female
Hepworth (1929) (Punjab)[10]	$16^{1/2}$-$17^{1/2}$yrs	$16^{1/2}$-$17^{1/2}$yrs	$16^{1/2}$-$17^{1/2}$yrs	$16^{1/2}$-$17^{1/2}$yrs	$16^{1/2}$-$17^{1/2}$yrs	$16^{1/2}$-$17^{1/2}$yrs
M.J.S. Pillai (1936) (South India)[13]	17yrs	17yrs	17yrs	17yrs	17yrs	17yrs
Galstaun (1937) (Bengal)[8,9]	17 — 18 yrs	14 — 17yrs	16 — 17 yrs	14 — 15 yrs	14 — 16 yrs	14 — 16 yrs
Misra B.D. (1966) (Gujarat)[14]	18yrs	17yrs	19 ½ yrs	18 yrs	20 yrs	19 ½ yrs
Saksena and Vyas (1969)[16] (Madhya Pradesh)	18 — 19yrs	16 —17yrs	18 —19 yrs	16 — 17 yrs	18 — 19 yrs	16 — 17 yrs
Gupta et al (1974) (U.P.)[5]	18 — 19yrs	17 — 18yrs	18 —19 yrs	17 — 18 yrs	20 —21 yrs	20 — 21 yrs
Present Study (2007) (Vidarbha)	18 — 19yrs	16 — 17yrs	18 — 19 yrs	17 — 18 yrs	19 — 20 yrs	18 — 19 yrs

Table 6. Comparison of ages of epiphyseal union around Knee joint given by various workers from other countries with findings of present study

Authors	Lower end of femur		Upper end of tibia		Upper end of fibula	
	Male	Female	Male	Female	Male	Female
Davies and parsons (1927) (English)[6]	19yrs	19yrs	19-20 yrs	19-20 yrs	Above 20 yrs	Above 20 yrs
Paterson (1929) (English)[15]	18 yrs	16 yrs	18-19 yrs	16-17 yrs	18yrs	16 yrs
Flecker (1942) (Australian)[7]	19 yrs	17 yrs	18 yrs	15 yrs	19 yrs	17 yrs
Cunningham (1953) (European)[2]	18 yrs	16-17 yrs	19-20 yrs	17-18 yrs	20 yrs	18-19 yrs
Frazer (1958) (European)[3]	18-19 yrs	17 yrs	19-20 yrs	17-18 yrs	19-20 yrs	17-19 yrs
Gray (1995) (European)[1]	18 yrs	16 yrs	18 yrs	17 yrs	19 yrs	17 yrs
Present study (2007) (Vidarbha India)	18-19 yrs	16-17 yrs	18-19 yrs	17-18 yrs	19-20 yrs	18-19 yrs

DISCUSSION

Ages of epiphyseal union around knee joint has been worked out in present study. These are 18-19 years for males & 16-17 years for females in case of lower end of femur; 18-19 years for males & 17-18 years for females in case of upper end of tibia; 19-20 years for males & 18-19 years in case upper end of fibula.

From table 5, the age of union of lower end of femur corresponds with Saksena & Vyas (1969-M.P.)[16],with Gupta et al (1974-U.P.)[5] in case of boys, with Misra B.D,(1966- Gujarat)[14], Galstaun (1937-Bengal)[8,9], M.J.S. Pillai(1936-south India)[13], Hepworth (1929-Punjab)[10] in case of girls, but not in case of boys.

The age of union of upper end of tibia corresponds with Gupta et al (1974-U.P.)[5] in case of both sex, with Saksena & Vyas(1969-M.P.)[16] in case of boys & with Misra B.D.(1966-Gujarat)[14] in case of girls. However, the same age reported by Hepworth (1929)[10] for Punjabees, by M.J.S. Pillai (1936)[13] for South-Indians, by Galstaun (1937)[8,9] for Bengalees, by Misra B.D.[14] for gujrati boys & by Saksena & Vyas[16] for girls in M.P. does not match with present study.

The age of union of upper end of fibula correspond with that reported by Misra B. D.[14] for Gujaraties but differ in case of all other authors.

From table no. 6, age of union of lower end of femur corresponds with Frazer (1958-Europe)[3]. The age given by Davies & Parson (1927-English)[6], Flecker (1942-Australians)[7] matches with the upper limit of age for boys found in present study whereas the age given by Paterson (1927-English)[15], Cunningham (1953-European)[2] & Gray (1995-European)[1] matches with the lower limit of age found for girls in present study.

Age of union of upper end of tibia for boys found in present study corresponds with Paterson(1927-English)[15], Flecker (1942-Australian)[7] & Gray(1995-American)[1] ; that for girls corresponds with Cunningham(1953)[2], Frazer(1959)[3] & Gray(1995)[1] in case of Americans.

Age of union of upper end of fibula found in present study matches with Flecker (1942-Australians)[7], Cunningham (1953-Americans)[2], Frazer (1959-Americans)[3], Gray (1995-Americans)[1] but differ in other cases.

SEXUAL DIMORPHISM:

As far as sexual dimorphism is concerned, present study support the commonly accepted view that the epiphyseal union occurs earlier in girls than boys.

CONCLUSION

Thus, ages of union of epiphyses around Knee joint have been attempted in present study. For lower end of femur, it is found tobe 18-19 years for boys & 16-17 years for girls, for lower end of tibia, it is found to be 18-19 years for boys & 17-18 girls & 19-20 years & 17-18 years in case of upper end of fibula .

On comparison with other authors, these ages are found to vary greatly not only all over the world but also in different states of India which may be due to the geographical variations. So, this suggests need of separate standard of ossification for separate regions.

From comparison with authors from western countries, it may be concluded that greater height of white races than eastern is not due to the time lag in the epiphyseal union but it may be due greater growth per year which may be contributed to genetic factors.

REFERENCES

1. Bannister L.H. :Editor, of Gray's Anatomy Descriptive and applied 38th Ed.: Page no. 626,636, 639,669,684,697,711, 1995.
2. Brash J.C.: Editor, Cunningham's textbook of Anatomy, 9th Ed. London: Oxford Univ. Press pp. 297, 305 and 309, 1953.
3. Breathnach A.S.: Editor, of Frazer's Anatomy of human 5th ed. London: J.A. Churchill Ltd: page no. 77, 87, 118, 127, 142, 1958.
4. Crowder C. and Austin D. Age ranges of epiphyseal fusion in the distal tibia and fibula of contemporary males and females.J. Forensic Sci. 2005; 50 (5): 1001-1007.
5. Gupta et. al. A roentgenologic study of epiphyseal union around elbow, wrist and knee joints and pelvis in Boys and Girls of Uttar Pradesh.J. Indian M.A.1974; 62: 10-12.
6. Davies D.A. and Parsons P.G. Age order of appearance and union of the normal epiphyses as seen by X-rays. J. Anat. 1927; 62: 58- 7.
7. Flecker H. Time of appearance and fusion of ossification centers as observed by Roentgenographic methods. Am. J. Roentg. 1942; 47: 97-159.
8. Galstaun G. A study of ossification as observed in Indian subjects. Indians J. Med. Res.1937; 25: 267-324.
9. Galstaun G. Some notes on the union of epiphyses in Indian girls. Indian Med. Gaz. 1937; 65: 191-192.
10. Hepworth S.M. On the determination of age in Indians from a study of the ossification of the epiphyses of the long bones. Ind. Med. Gaz. 1929 March:

11. Koc A. et al. Assessment of bone ages: is the Greulich-Pyle method sufficient for Turkish boys? Pediatr. Int. 2001; 43 (6): 662-665.
12. Loder R.T. et. al. Applicability of the Greulich and Pyle skeletal age standards to black and white children of today. Am. J Dis. Child 1993; 147(12): 1329-1333.
13. M. J. S. Pillai. The study of epiphyseal union for determining the age of South Indians. Indian J. Med. Res. 1936; 23: 1015-1017.
14. Misra B.D. Fusion of ossification centers as observed by roentgengraphic method in Gujarat – Distal epiphysis of femur and proximal epiphyses of tibia and fibula. A preliminary report. J. Anat. Soc. India 15: 132-137, 1966.
15. Paterson R.S. Radiological investigation of the epiphyses of the long bones. J. Anat.1929; 64: 28 --46.
16. Saksena J.S. and Vyas S.K. Epiphyseal union at the wrist, knee and iliac crest in Residents of Madhya Pradesh. J. Indian M.A.1969; 53: 67-68.

Cutaneous Manifestations of Spinal Dysraphism in an Adult Male - A Case Report

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ABSTRACT

Spina bifida occulta is the mildest form of neural tube defect and often it is also called as hidden spina bifida. In other terms it is also known as Occult Spinal Dysraphisim (Occult means Hidden, Dysraphisim means Failure of embryological fusion). It is caused by incomplete closure of the embryonic neural tube. Vertebrae overlying the spinal cord are not fully formed and remain unfused and open during the embryonic life. Failure of the fusion leads to Occult Spinal dysraphisim. The present study was done on a male cadaver, who was died as the result of poisoning and had a disorder of Spina bifida occulta with cutaneous manifestations. Having a tuft of hair over lumbo sacral region is an unique form of spinal dysraphism with innocuous external appearance. Incidentally the case was diagnosed. As the case was uncommon, so it was taken for the study. All the external and internal findings were correlated with the findings of spina bifida occulta without tethered cord. The study gives the results that the impotency will not be associated in spina bifida occulta without tethered cord and having the tuft of hair is an unique feature of identification of an individual.

Key words: *Spina Bifida Occulta, Congenital Dermal Sinus, Folate Deficiency, Occult Spinal Dysraphisim (OSD), Neural Tube Defects.*

INTRODUCTION

Spina bifida malformations fall in to four categories.

1) Spina Bifida Occulta 2) Meningocele
3) Spina Bifida Cystica (Myelomeningocele)
4) Myelocoele.

The most common location of the malformations is the lumbar and sacral areas.

1) **Spina Bifida Occulta**(Latin "Split spine") :- Spina Bifida Occulta is the mildest malformation of neural tube defect. Posterior vertebral arch shows the defect, without herniation of the dural sac, or its contents. In most of the cases, it is asymptomatic. But in some children, abnormalities are seen. Quite often, localized lipoma, dermal sinus or excessive hair is seen on the skin surface at the site of the defect.

   Spina Bifida Occulta is of two types.

A) Spina Bifida Occulta without tethered cord- It is a closed form type, in which one or two vertebrae are involved and defect will be invisible with no neurological, musculoskeletal, bladder and bowel disorders.

B) Spina Bifida occulta with tethered cord it's another type of closed form. The Cord will be restricted within the canal. The characteristic features are thickened filum terminale, fibrous bands of tissue, dermal sinus tracts; Longitudinal bony spurs with in spinal canal. Lipoma may be seen within spinal canal (Lipomyelomeningocele.)

2) **Meningocoele :** It is a **o**pen type of neural tube defect, causing protrusion of meninges through the bony defect, results in a cystic swelling filled with cerebrospinal fluid. Nerve elements remain intact. Minimal trauma may lead to CSF leakage & severe infection.

3) **Myelomenigocele:-** It is an open type of neural tube defect known as Spina Bifida Cystica. Spinal cord and nerve roots protrude through an abnormal opening in the spinal arch. The opening may be covered by a thin membrane.

4) **Myelocoele**:-Open type of neural tube defect, and spinal cord opens posteriorly.

MATERIAL AND METHODS

During the post mortem examination, a 32-years-old male cadaver had a tuft of hair over lumbo sacral

region. As the findings were abnormal, the case was taken for the study and examined thoroughly and the findings were noted, photographs were taken.

Case report- A 32-year's-old male was admitted in Govt General Hospital, Anantapur, Andhra Pradesh, on 07.12.2008 at 6.50 P.M. with a history of consumption of poison. Inspite of timely intervention & treatment and the condition was deteriorated and he died on 08.12.2008 at 10.30 P.M. On requisition of the Station House Officer, of concerned police station, the Post Mortem Examination was conducted with P.M No 654/10 on 09.12.2008 at 2.30 P.M. As per the inquest, the deceased was normal prior to the death and had three daughters &was having a desire to get a male child, but at the same time, he wished to arrange the marriage of his daughters, but he could not succeed in all these aspects, hence committed suicide. During the post mortem examination, the body was fresh and few external findings like a tuft of hair over lumbo sacral region were noticed. The Hair was present in two branches like plaits and a hairy patch was seen around the plaits and the other important external and internal findings were noted. In the present case, incidentally the diagnosis was made as Spina Bifida Occulta without tethered cord. So, the case was taken for the study. The important post mortem findings were noticed as below.

On external examination :-

1. A deep dimple in the lumbo sacral area covered with a layer of skin with hyper trichosis (Abnormal hairy patch on the skin of the back.) and associated with Lumbo sacral Dermal sinus and Rudimentary hairy tail protruding from the lower back in two branches, measuring 30 cms and another one is 20 cms, like a twisted ropes (Plaits).

Fig. 1. Cutanious manifestations of Spinal dysraphism with Excess hair.

2. Cigarette burn type of skin marking was noticed.

On internal examination :-

1. All the internal organs were congested. 2. Stomach contained 50 ml. of brownish colored liquid with abnormal smell & mucosa was congested 3. Dissection over lumbo sacral region over lower back of the trunk , at the site of the defect, there was a split spine with defect in the Posterior lumbar 4 and 5 vertebral arches without herniation of the Dural sac and case was diagnosed. The blood & viscera were preserved and forwarded to the Regional Forensic science laboratory for the chemical analysis.

Results of the investigations:- The External and internal findings were correlated with the findings of spina bifida occulta without tethered cord. Cause of death was due to Phosphide poisoning (RFSL REPORT).

DISCUSSION

The etiology of neural tube defect is unknown, however with the available theories, the combination of genetic, environmental, and nutritional factors are believed to be the important factors and few of the known factors are folic acid deficiency, maternal insulin dependent diabetes and maternal use of certain anti convulsant drugs. Ex: Divalprox sodium and Sodium Valproate.

During normal embryonic development, the greater part of CNS, including spinal cord arises from the medullary plate cells along the back of embryo. During the early development, the edges of the plate begin to curl up towards each other, creating the neural tube, which is a narrow sheath that closes to form the brain and spinal cord of the embryo. As the development progresses, the top of the neural tube becomes brain and the remainder of the tube becomes the spinal cord. This process is usually complete by the 28th day's pregnancy. If the process fails, it results in neural tube defects. Spina bifida is a type of neural tube defect results, the failure of the fetus spine to close properly during the first month of pregnancy. Failure of the fusion leads to occult spinal dysraphisim and causes spinal deformities, gait changes, bowel problems and orthopedic complications. This often presents at the child hood with abnormal skin findings, neurological deficit or infection. Even with presence of this disorder few cases progress in to the adult hood.

Folic acid and vitamin B12 are important substances, which reduce the occurrence of the neural tube defects. Folate is an important substance that is required for the production and maintenance of the new cells and for DNA and RNA synthesis. Neural tube defects can be detected during pregnancy by prenatal tests. Elevated Maternal Serum Alpha Fetoprotein (MSAFB), detection of neural tube defects with High Resolution Ultrasound and testing with amniocentesis for the elevated levels of alpha fetoprotein (AFAFB) & acetylcholinesterase (AFAChE), are helpful investigations during 16 to18 weeks of the gestation.

Similar adult post mortem studies are not available for the comparison. Based on the findings, the Spina Bifida Occulta without tethered cord was diagnosed and all the external and internal findings were correlated with the findings of occult spinal dysraphism. The other tests like X ray, MRI Scan could not be done due to the non availability of the facilities in the mortuary.

CONCLUSION

Spina bifida occulta is a neural tube defect and is a rare condition in the community, can be diagnosed during the post mortem examination. Quite often, this condition is associated with impotency, due to the tethered cord. The present study gives the result that with spinabifida occulta without tethered cord, will not affect the normal sexual life of an individual and the individual can lead the normal life. Having a tuft of hair over lumbo sacral region like a plait, is an unique feature of identification of an individual. It may be taken as a sample study for further research work.

RECOMMENDATIONS:

1. All women of childbearing age should eat a diet(whole grains, cereals, dried beans, leaf vegetables and fruits) high in folic acid or take 0.4 mg of folic acid each day, especially one month prior to conception.
2. Spina bifida can be corrected surgically both intra uterine and after the birth.
3. Early evaluation in new born who are born with hair on their back.

REFERENCES

1. Robert C. Tasker, Robert J. McClure, Carlo L.acerin,Oxford Handbook of Pediatrics, Ist publish, reprint 2011,176.
2. Swarna Rekha Bhat, Achar's Text book of pediatrics, 2009, 4th edi, 498.
3. Andrew H. Kaye-Essential Neurosurgery, 2005, III rd edi, 165.
4. G.D.Clark.Neuro, clin N Am 20 , 2002, 917-939.
5. John B. Bodenstener, James F. Bale Jr, Brain and Development, Pediatric Neurology, Neurol clin N Am20 (2002) 917-939.
6. Raymond D.Adams, Maurice Victor, Allan H. Ropper, Principles of Neurology, 1998, 6th edi, 405-413.
7. R.C.G. Russell, N.S.Willams, C.J.K, Buistrode, Bailey & love's Short practice of surgery, 2000, 23 edi, 457.
8. Vinay Kumar, ABUL K. ABBAS, NELSON FAUSTO,Robbin's and cotran, Pathologic Basis of Disease, 2004, 7 th edi, 1354.
9. Edited by R.K. Karmakar, J.B.Mukherjee's Forensic Medicine and Toxicology 2007, 3rd edi, 657.
10. Edited by B Subrahmanyam, Modi's Medical Jurisprudence and Toxicology- 2001, 22nd edi, 461.
11. Goh SL, TAN JVK, K wek kyc, Yeo Gsh, Recurrent Neural tube defects, case report, Singapore Med J 2006, 47(8), 728-729.
12. Nina Johns, Wasim Ai-Salti, Phillip cox[2], Mark D.Kil by-A comparative study of Prenatal Ultra sound findings and Post mortem examination in a tertiary centre, May 2004, Vol 24, 339-346.
13. CC. Michael James and L.P.Lassman,The Journal of Bone and Joint Surgery, Spinal Dysraphism, The Diagnosis and Treatment of Progressive lesions in Spina Bifida Occulta , Nov 1962, Vol 44B, No.4, 828-840.
14. Gordon Brockle hurst,Developmental Medicine &Child Neurology, April 1971, Vol 13, issue 2, 147-163.
15. SIR WILLIAM GOWERS,A case of cervical Myelopathic Trichosis, 1907, vol xc, 455-467.
16. Other link -http://www.chg.duke.edu/diseases/ntd.html.
17. Other link - http://en.wikipedia.org/wiki/neural_tube_defect.

Expression of Dengue Antibodies in Hemolysed Blood Sample in Autopsy - A Case Report

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ABSTRACT

Dengue Fever is the serious health problem in tropical countries and caused by the Dengue virus of flaviviridae family. Dengue fever is characterized by, high grade fever, frontal headache, retro- ocular pain, myalgia, arthritis and rash. Dengue virus is primarily transmitted by vector Aedes aegypti. The virus is sensitive to heat and is susceptible to many common disinfectants including ethanol, sodium hypochlorite and glutaraldehyde. It is stable in dried blood and exudates for several days at room temperature.

The present study reveals that the presence of dengue viral antibodies, in hemolysed blood of exhumed body after 10 days of death.

Key words: - Dengue fever, Dengue Hemorrhagic fever, Dengue shock Syndrome.

INTRODUCTION

Dengue fever (Break—Bone disease) is an infectious disease caused by dengue virus; there are four serotypes of Dengue virus DENV 1- 4, that can cause the full spectrum of the disease. Humans are the reservoirs and the vector is female Aedes aegypti mosquito, rarely by Aedes albopictus mosquitoes. Mosquito will become infective for its life time, after biting a primary host(Human),virus spread to the all the tissues including salivary gland of mosquito, subsequently the virus is released in to saliva. Replication of virus in human occurs in WBC and interferon's are released. These are responsible for clinical features like fever, myalgia & arthralgia. Initial infection result in mild illness, reinfection with a different strain, results in severe form of disease, sometimes hemorrhage & shock. In severe form of the disease there is increased vascular permeability of smaller vessels, result in fluid leakage and collection of fluid in the cavities. There may be hypotension & severe thrombocytopenia. Dengue can be life threatening in people with chronic diseases such as, diabetes and asthma. Vertical transmission from mother to the unborn is possible. Dengue can also be transmitted via infected blood products and through organ transplantation.

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CLINICAL FEATURES

Children are commonly affected, followed by young adults & older people. Three types of presentations are seen- Dengue fever, dengue hemorrhagic fever and dengue shock syndrome. The incubation period is 3 — 15 days.

1) Dengue fever (DF): - Fever with nonspecific symptoms including headache, retro-ocular pain, myalgia & occasional hemorrhage.
2) Dengue Hemorrhagic Fever (DHF):- severe form result in loss of intravascular volume and circulatory insufficiency. Severe bleeding, particularly bleeding into internal organs are observed, patient shows altered liver function tests and severe thrombocytopenia.
3) Dengue Shock Syndrome (DSS):- Severe form of DHF plus hepatic failure & encephalopathy. The previously infected patients with other serotypes of Dengue virus are at an increased risk. The recovery phase occurs next, with the resorption of the leaked fluid in to the vascular compartment of the body.

Grading of the Severity

Grade I: positive tourniquet test.

Grade II: spontaneous bleeding

Grade III: Circulatory failure.

Grade IV: Profound shock and Hypotension

Few cases of myocardial dysfunction, Spleenic rupture & hemorrhagic Encephalopathy, myocarditis are reported from different parts of the world.

DIAGNOSIS

The diagnosis of dengue is typically made on clinical history with poly serositis.

LABORATORY DIAGNOSIS BY:-

- Hematological parameters — Increased PCV%, severe thrombocytopenia, leucopenia, prolonged PT, APTT.
- Biochemical — Altered Liver Function tests, raised ALT & AST, decreased Albumin.
- Serological & Others -— Demonstration of IgG and IgM antibodies. IgM and IgG are produced by 2 to 5 days of the infection; IgM peaks by 2 weeks & disappears by 90 days after the primary infection. IgG detectable at low titre at the end of the first week & slowly increase and persists after several months of the infection.
- Viral isolation by culture.

INTERPRETATION OF DENGUE SEROLOGY

IgM	IgG	Interpretation
Negative	Negative	Too early sample or not dengue fever
Positive	Negative	Early stage of primary dengue infection.
Positive	Positive (low titre)	Recent or ongoing primary dengue infection.
Negative	Positive (low titre)	Past dengue infection
Positive	Positive (High titre)	Secondary dengue infection
Negative	Positive (High titre)	Secondary dengue infection

TREATMENT OF DENGUE FEVER WHO guidelines

1) Prompt supportive treatment, intra venous fluids & crystalloids.
2) Close observation for signs of bleeding & circulatory compromise.
3) Platelet transfusion if count falls < 50000/ cumm.
4) Oxygen if necessary.

CASE REPORT

9 months old female child treated for the fever by different pediatricians, finally succumb to death.

THE SEQUENCE OF THE EVENTS

On 3.9.10 - A female child of 9 months age, taken to a pediatrician for c/o fever & cold & treated accordingly.

On 5.9.10 patient was again seen by the pediatrician, symptomatic treatment was given.

On 16.9.10 again patient was seen by the same pediatrician and symptomatic treatment was given.

On 17.09.10 as there was no relief even after two subsequent visits to the first pediatrician, the child was taken to another pediatrician. On the same day few hematology investigations were done on advice of the second pediatrician. The important results were PCV 29%, Platelets < 90000/cmm, the child developed seizures and child was referred to higher centre, Bangalore on the same day.

On 18.09.10 the child was admitted with H/o of hematemesis, seizures, and investigations were done, important reports were Hb 9.2%,TC 18,300/cmm, PCV 29.6% , platelet count < 20,000/cmm, PT 12.7sec, INR -10, APTT-50.1 sec, Serology — No Data Available.

On 19.9.10 the child became hypotensive and treatment was continued.

On 20.9.10 patient was not responded to the treatment and condition was deteriorated and died. Case was diagnosed as ARDS and DIC with Dengue shock syndrome.

On 21.9.10 the body was buried.

On 25.9.10 the Father of the deceased lodged complaint on the first pediatrician, for the medical negligence, so the case turned Medico Legal.

On 26.9.10 the Mandal Executive Magistrate sent requisition to the professor, Department of Forensic Medicine, Govt Medical College, Anantapur to conduct Exhumation.

On 27.9.10 exhumation and spot postmortem examination was conducted I.e. 8th day after death. Body was buried in the wet soil and large amount of salt crystals were used lay-in the grave and the signs of decomposition were delayed significantly. Findings at Autopsy were multiple hemorrhagic spots over back of the trunk, over the surface of the cranium and stomach, approximately 1000 ml of haemolysed blood in the Chest and peritoneal cavities.

MATERIAL AND METHODS

1) The Haemolysed blood sample for the serology.
2) Tissue bits from lungs, liver, kidneys and heart for histopathology.
3) Viscera for the chemical analysis were sent.

Fig. 1. A Case of Dengue Shock Syndrome- Exhumed On 8th day.

Fig. 2. Shows - Hemolysed blood in the thoracic cavity & Hemorrhagic spots over the cranium.

Fig. 3. Rapid Card Test —Positive for IgM &IgG Dengue Anti bodies.

Results of the investigations

1. Serology of hemolysed blood revealed Anti Dengue Viral Anti bodies IgG & IgM by rapid card test. The purple bands of IgM &IgG antibodies along with control line were clearly visible on the card.
2. Histopathology report correlated with clinical diagnosis.
3. RFSL Report -No poisonous substance was found.

DISCUSSION

With First exposure of dengue infection, antibodies are produced in the body and they neutralize the virus [2.] With Secondary infection with different serotype, it causes DHF or DSS.

Halsted proposed the theory that with sequential infection heterotypic antibodies will be produced and are responsible for DHF/ DSS, and it is due to antibody Depended enhancement (ADE). The poor binding non— neutralizing antibodies delivers in to the white blood cells and cross-react with the virus and forms antigen antibody complexes and they are taken up by the macrophages and increase the virus load by their multiplication. The IL-1and TNF are liberated and act on the vascular endothelium, in turn releases nitric oxide, C3, C5 and histamine. It activates CD4 and CD8 cells and releases cytokines which causes increasing vascular permeability; thrombocytopenia, defective platelet function, and decreased coagulation factor and complement activation.

In the present case the autopsy was conducted on the 8th day of the death and post mortem changes were sluggish and findings were consistent with that of the Dengue shock syndrome. Hemolysed sample was collected and tested with immunochromatography rapid card test; after 2 days after collection I.e. 29.9.10 (on the 10th day after the death).

The positive results of dengue viral antibodies were traced on the rapid card in form of purple lines, even on 10 day after the death. The other supplementary tests like ELISA were not done due to the non availability of the facilities.

The present study correlating with the clinical diagnosis and the statements of the standard text books, that the viral antibodies can be detected from the autopsy samples, cause of death can be ascertained with autopsy samples. The similar studies are not available for the comparison.

CONCLUSION

Detection of dengue antibodies IgM and IgG are useful in diagnosis and can be detected from the autopsy samples, even on the 10th day after the death and tracing the antibodies give the evidence about the cause of death. Increased awareness, early diagnosis and case management according to WHO guidelines are needed to reduce the further mortality of DHF/ DSS cases. Mosquito control and personal protection against mosquito bites are to be followed. It may be taken as a sample study for further research work.

REFERENCES

1. Swarna Rekha Bhat, Achar's Text book of pediatrics, 2009, 4th ed, 298- 300.
2. A Santhosh Kumar, Manual of Pediatrics, 2008, 2nd ed, 228-232.
3. Dr Prakash Mahajan, Management of symptoms in General practice, 2011, 1St ed,140-141.
4. Aditi Aikat, A Sarkar, N Tudu, S Moitra, P P Pal, T K Sarkar, Diagnostic Dilemma of Dengue fever: how much reliability are there in rapidity, Journal of the Indian Medical Association, Aug 2011, Vol 109, No 08, 543-545.
5. I.Gordon, H.A.Shapiro, S.D. Berson, Forensic medicine — A guide to principles, 1988, III rd ed, 173.
6. Stephen J. McPhee, MaxineA.Papadakis, Lawrence M.Tierney, Current Medical Diagnosis and Treatment, 2008, 47th ed, 1204-1205.
7. Anantha Narayayana and panikar, Text book of Microbiology, 2009, 8th ed, 519.
8. D.R. Arora, Text book of Microbiology, 2008, 3rd ed, 592.
9. Dr. K.S.Narayanareddy, The Essentials of Forensic Medicine and Toxicology, 2006, 25 th ed, 124.
10. S.K.Kabra, R.Juneja, Madhulika, Y. Jain, T. Singhal, L. Dar, S.S.Kothari, S.Broor, Myocardial dysfunction in children with dengue haemorrhagic fever, The National Medical journal of India, 1998, Vol.11, No.2, 59-61.
11. Apatcha Pungjitprapai and Terapong Tantawichien,A Fatal case of spontaneous Rupture of the spleen due to Dengue Virus Infection: Case Report and Review, Southeast Asian J Trop Med Public Health journal, May 2008, Vol 39, No 3, 383-386.
12. Luiz Jose de Souza, Andre Luiz de, Oliveira Martins, Paulo Cesar Leitao, Paravidini, Rita Maria Ribeiro Nogueira, Carlos Gicovate Neto, Diogo Assed astos, Edno Wallace da Silva Siqueira and Rodrigo da costa carneiro, Hemorrhagic Encephalopathy in Dengue Shock Syndrome, BJID, 2005; 9 (June) 257-261.
13. Anon srikiatkhachorn, Plasma leakage in dengue hemorrhagic fever, Theme Issue Article, 2009, 1042-1049.
14. Public Health Notifiable Disease Management Guidelines, dengue fever, December 2005, 1-6.
15. Alan L Rothman,Clinical presentation and diagnosis of dengue virus infections, 2008. http:/ / www.utdol.com/online/content/topic.
16. Kosala GAD Weerakoon,Sena nayake AM Kularatne, Deepthika H Edussuriya, Sarach Chandra KA Kodikara, Laxman PG Gunatilake, Vasanti G Pinto, Ashoka B Seneviratne and Sunethra Gunasena, Histopathology diagnosis of myocarditis in a dengue outbreak in Srilanka, 2009. BMC Res Notes, 2011.vol 4, 268.
17. Scott B Halstead, Antibody, Macrophages, Dengue Virus Infection, Shock, and Hemorrhage: A Pathogenic Cascade, Reviews of Infectious Diseases, May-june 1989,Vol 11, supliment 4, 830-831.

Estimation of the Time of Death by Evaluating Histological Changes in the Pulp

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ABSTRACT

Dental pulp is enclosed in hard tissue and hence it is well protected and preserved for long time. Preservation of dental pulp is seen long after damage to other tissues is evident. Thus it is an ideal tissue for analysis of post-mortem changes. In this study we have made an attempt to quantify the amount of degenerative changes in pulp at increasing time interval. We have analyzed the changes in staining characteristics and structural details, in Haematoxylin and Eosin stained pulp tissue harvested at varying time intervals.

Key words: *Pulp Analysis, Degenerative Changes, Staining Characteristics.*

INTRODUCTION

With the death of an organism post-mortem changes occur which modifies the composition, causes mutilation & skeletalization that makes identification progressively more difficult almost to the point of impossibility.

The dental pulp occupies the centre of each tooth and consists of soft connective tissue. The pulp is housed in the pulp chamber of the crown and in the root canal of the root. The pulp present in the crown is called coronal pulp and the pulp present in the root is called radicular pulp. The coronal pulp is protected by enamel and dentin where as the radicular pulp is protected by cementum and dentin. All the three tissues, enamel, dentin and cementum are hard calcified tissues, with enamel being the hardest calcified tissue in the human body. So dental pulp is enclosed and well protected by hard dental tissues. It has been rightly described as the cosseted tissue of the body.

Determining the time of death of a person is an important aspect in Forensic Medicine as well as Forensic Odontology .There are a variety of methods for determining the time of death of a person. Some are for the investigators at the crime scene while some are for the pathologists at the autopsy.

Various methods includes-

- Rigor mortis.
- Liver mortis
- Alger mortis
- Eyes
- Food in the stomach
- Body lice .

All the methods mentioned above have some limitations and the body needs to be well preserved[1,2,3]. Extensive mutilation or increased time lapse make these methods unavailable for post-mortem examination.

In such cases, the pulp tissue has an additional advantage of being better preserved than the rest of the soft tissue in the body, inspite of the lack of use of preservatives and fixatives .

In this study we have attempted to record the histological changes in the pulp tissue, stained by Haematoxylin and Eosin, under light microscope at increasing time intervals. We have attempted to draw conclusions regarding the approximate time of death of an individual on the basis of histological changes in the pulp at increasing time interval.

MATERIALS AND METHOD

This study was carried out after obtaining approval from Institutional Ethical Committee.

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The study was comprised of 40 extracted teeth collected from the Department of Oral And Maxillofacial Surgery of Sharad Pawar Dental College,Wardha. The teeth were not preserved in any preservative so that exact change in the pulp could be evaluated. Further evaluation of the extracted teeth was carried out in the Department of Oral Pathology And Microbiology of Sharad Pawar Dental College, Wardha. The collected teeth were longitudinally sectioned at different intervals of time like 6, 24, 48, 72 & 96 hours and pulp was extirpated from them.

The extirpated pulp was then subjected to routine processing of soft tissue for Haematoxylin and Eosin staining procedures.

The processed slide was examined histopathologically under microscope. The changes were evaluated and described .

RESULTS

We have seen the following histopathological changes of pulp tissue after 6,24,48,72,96 hours.

6 HOURS CHANGES

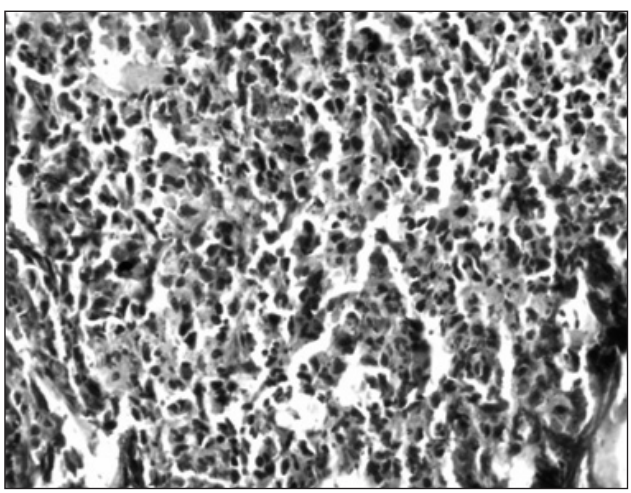

Fig. 1. Photomicrograph shows the following changes in pulp after 6 hours.

1. Haemoatoxylin and Eosin staining characteristic is well preserved.
2. Tissue architecture is maintained.
3. Cells are adherent.
4. Inflammatory cells can also be appreciated.
5. Collagen fibers are well demarcated.
6. Fat cells in tissue is seen.

24 HOURS CHANGES

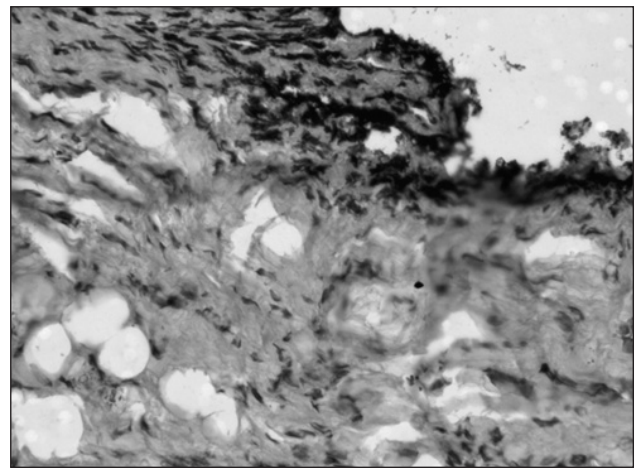

Fig. 2. Photomicrograph shows following changes in pulp after 24 hours.

1. Haematoxylin and Eosin staining characteristic is good.
2. Connective tissue cells shows separation at places.
3. Few inflammatory cells can be appreciated.

48 HOURS CHANGES

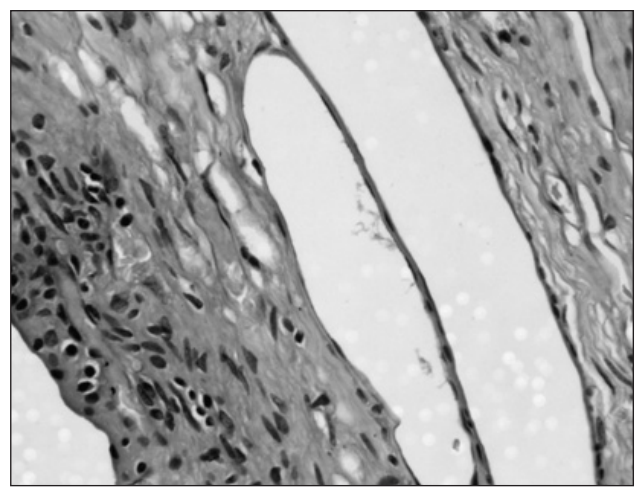

Fig. 3. Photomicrograph shows following changes in pulp after 48 hours.

1. Haematoxylin stain is well preserved but eosin has fainted.
2. Blood capillaries are dilated.
3. Connective tissue shows further separation.

72 HOURS CHANGES

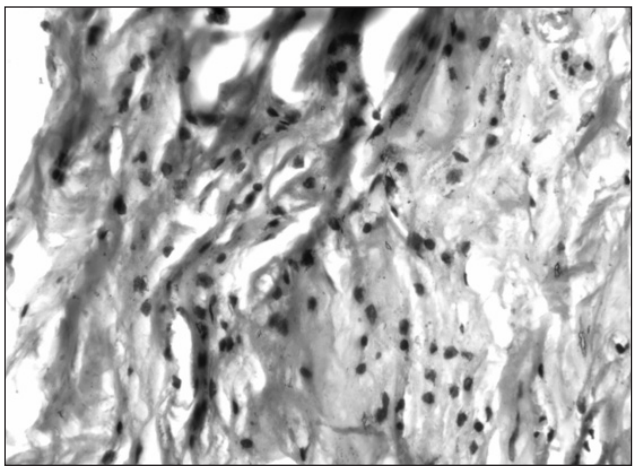

Fig. 4. Photomicrograph shows following changes in pulp after 72 hours

1. Haematoxylin stain becomes a little fainter.
2. Smudging of collagen fibers seen .
3. Individual fibres can not be clearly appreciated.

96 HOURS CHANGES

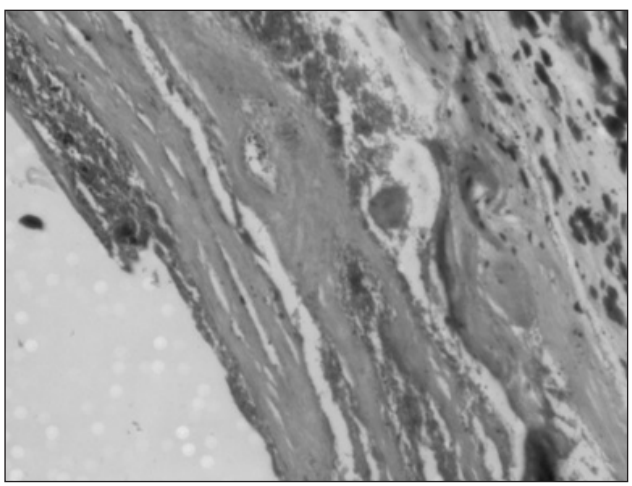

Fig. 5. Photomicrograph shows following changes in pulp after 96 hours

1. Haematoxylin stain is lost more in the central area than in the periphery.
2. Structural characteristic of collagen fibres lost.
3. Homogenization of collagen fibres seen.

DISCUSSION

Due to its location in the innermost position in the teeth and being surrounded by hard dental tissues, the pulp is inaccessible to exterior factors which are the usual culprits in tissue degeneration. Histology of pulp is found to be preserved for a much longer period than other tissues of the body. The well preserved morphology was reflected in the fact that the cellular and nuclear outline was clear and cell organelles were distinct upto 72 hours. Upto 72 hours, the staining characteristic of pulp was retained and sharp, indicative of the maintenance of structural integrity of nuclear material. There was no smudging or blurring of boundaries between different tissues. The well preserved nature of the tissue is maintained for 72 hours.This is in contrast with the destruction and degeneration seen in other tissues of the body as early as 24 hours.[1,2,3] This indirectly is indicative of the fact that DNA preserved in the pulp would also be of good quality for a longer period of duration. This can be of good help in identification of victim as well as time of death, even if the body is discovered after a much later.

CONCLUSION

From the above study we came to a conclusion that after death, loss of integrity of pulp occur much later compared than the other parts of the body. Hence it can be of use for DNA extraction and thereby identification of victim.

Due to the lack of loss of integrity of the pulp tissue for a longer period of time, even after death and due to the sequential degenerative changes that occur in the pulp, as evaluated from the histopatholological study mentioned above, it could be also of use to arrive at an approximate time of death of an individual.

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Rupesh Maladhari, LaoratoryTechnician ,Sharad Pawar Dental College, Sawangi(Meghe), Wardha.

Conflict of Interest : None

REFERENCES

1. Dikshit P.C. Changes after death. In Text Book of Forensic Medicine And Toxicology. Dikshit P.C. 1st Edn. Peepee Publishers and Distributors (Pvt)Ltd. Pg-89-111.
2. Nandy Apurva. Death and postmortem changes. In Principles of forensic Medicine. Nandy Apurva. 2nd Edn. New Central Book Agency Ltd. Pg-133-173.
3. Reddy Narayan KS. Post mortem changes. In The Essentials of Forensic Medicine And Toxicology. 27th Edn. Medical Book Company. Pg-157-212.

Case report - Unusual Cases of Late Deaths in Hanging

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ABSTRACT

Hanging is that form of asphyxia which is caused by suspension of the body by a ligature which encircles the neck, the constricting force being the weight of the body. Hanging is the method commonly used to commit suicide. In Latur district it is one of the common causes among the suicidal manner of death. In hanging death occurs immediately if the cervical vertebrae are fractured or rapidly in case of asphyxia, but usually death occurs in five to eight or ten minutes if blocking of air passages is only partial. Death is, as rule, slow in case of various congestions 1. In present cases the two victims survived for 3 and 32 days after hanging respectively.

Key words: *Hanging, Fatal period, Hypoxic brain death, Late death*

INTRODUCTION

Hanging can be defined as the ligature compression of the neck by weight of one's own body due to suspension. When the point of suspension is over the center of occiput, there is a maximum possibility of occlusion of the arteries and this is known as typical hanging, while all other points of suspension are called atypical hanging. In most of cases of hanging, Asphyxia is the most common cause of death. Asphyxia , venous congestion , combination of asphyxia and venous congestion , cerebral ischaemia (Anoxia), shock, fracture or dislocation of cervical vertebrae are the causes of death in hanging[1,2,5]. Death is delayed for several days is rare in hanging. The usual fatal period is 3 to 5 minutes[1, 2]. In present case (1) a married female of 19 year old hanged herself in her own house and her relatives removed the ligature from the neck and admitted her in the Government Medical College Latur for further treatment. She was in unconscious state in hospital for 3 days and died in the hospital. In case (2), A married male of age 86 year old attempted suicide by hanging with 'rope', point of suspension was an iron rod of his room. Relatives removed the ligature from the neck and immediately admitted him in Hospital and he died in hospital after 32 days.

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Case report 1

A married female of age 19 year attempted suicide by hanging with 'saree', point of suspension was an iron rod of the roof. Relatives removed the ligature from the neck and immediately admitted her in Government Hospital, Latur. At the time of admission patient was unconscious complaining of convulsions, bleeding through the nose, crepitations present on chest examinations (? aspiration pneumonia), tachycardia, irritable, pupils were dilated and sluggishly reacting to the light, O_2 saturation was 99%. Patient was intubeted and sedated and taken on ventilator. Instead of all hospital measures the patient was died after 3 days. The postmortem was done in the hospital mortuary. On external examination a single ligature mark present on the neck of length 19 cm and maximum width of 2 cm. Right end if ligature was 5 cm below the right ear and left end was 8 cm below left ear .Ligature mark was going upwards, backwards and laterally and deficient on posterior surface of neck. Mark was slightly above the thyroid cartilage. On dissection of neck, there was no contusion to strap muscles, no tear to large vessels of the neck. Thyroid cartilage and hyoid bone were intact. Lungs were congested and edematous, shows patechie on the surfaces. Brain was preserved for pathological examination, on microscopic examination cerebrum reveals vacuolar change with parenchymal congestion and cerebellar parenchymal congestion. Meninges were congested. Other internal organs were intact & congested.

Photograph of case report (2) - A : Ligature mark seen on neck

Photograph of case report (2) - B : Ligature mark seen on neck.

Case report 2

A married male of age 86 year old attempted suicide by hanging with 'rope, point of suspension was an iron rod of his room. Relatives removed the ligature from the neck and immediately admitted him in Hospital. At the time of admission patient was in disoriented state and left sided hemi paresis. The deceased was k/c/o myocardial infarction since 15 years. Police inquest suggested that due to chronic disease, he attempted suicide. In hospital he was treated with oxygenation and medical management. During hospitalization his M.R.I. revealed large acute infarct in right cerebral cortex. E.C.G. revealed old anterolateral infarct. Instead of all hospital measures the patient was died after 32 days. The postmortem was done in the hospital mortuary. On external examination a single ligature mark present on the neck of length 21 cm, maximum part present on right surface of neck going to left surface of neck , left end present at mandible line between chin and angle of mandible and maximum width of 1.4 cm. Right end of ligature was 8.5 cm below the right mastoid. Ligature mark was going obliquely upwards, backwards and laterally and deficient on posterior surface of neck. Mark was 1.8 cm above the thyroid cartilage. On dissection of neck, under the ligature mark subcutaneous surface is white, glistening. There was no contusion to strap muscles, no tear to large vessels of the neck. Thyroid cartilage and hyoid bone were intact. Lungs were congested and edematous, large infarct found in right cerebral cortex. Liver, Spleen, Kidneys were intact, kidneys shows cyst containing fluid in the parenchyma of organs.

DISCUSSION

Usual fatal period in hanging is 3 to 5 minutes[2]. Death is delayed very rarely in hanging. Person can not be survived from unconsciousness in hanging. Fremibgston k. morak & R. Balaraman reported a case of 18 year girl who survived for 28 days after hanging, the girl attempted suicide in her house by shawl[3]. Verma SK & Agrawal BBL reported a case of an adult male who survived for 39 days after he was accidently hanged while helping passengers trapped in the lift of an O.P.D. in teaching hospital[4]. In case the victim is released from the suspension then death may be delayed even for few days depending on the site of damage in the brain having residual signs like amnesia, mental confusion and neurogenic disturbances[5]. Ambade V.N., Dongare A.P. & Dixit P.G. reported four cases in which two victims survived for seven days and another two victims survived for six days after ligature compression of neck and then died[6]. Hausmann R & Benz P reported a case of delayed death 4 days after an attempted suicide by hanging where the individual was conscious and showed no neurological abnormalities[7].Vaghela DR , Patel PR reported a case in which victim committed suicidal attempt by hanging himself at home , who was saved and admitted in general hospital and he died after 36 days due to irreversible brain damage and respiratory arrest[8]. Aggrawal NK, Kishore U, Agarwal BB reported a female victim she was conscious throughout in the hospital and died after nine days due to cerebral damage due to cerebral anoxia [9]. In present study two victims survived for 3 and 32 days after hanging respectively.

CONCLUSION

Death may be delayed in hanging due to various causes like degree of the constricting force applied, time required to release of the compression of around the neck. It is difficult to regain the consciousness after hanging due to the irreversible cerebral damage.

REFERENCES

1. K Mathiharan and Amrit K Patnaik: Modi's Medical Jurisprudence and Toxicology 23 rd edition; pg. 566-570.
2. Dr. K.S.N. Reddy : the essentials of forensic medicine and toxicology 2010 , 29th edition; pg. 302-303.

3. Fremington K. Marak & R. Balaraman : ' Delayed death in hanging', J Indian Academy Forensic Medicine, 30 (3); pg. 149-150.
4. Verma SK & Agarwal BBL : Accidental hanging with delayed death in lift. Med Sci Law.1999; 39 (4); pg. 342-344.
5. Apurba Nandy : Principles of forensic Medicine including Toxicology; pg. 517-522.
6. Ambade V.N., Dongare A.P., Dixit P.G. : 'Delayed death in ligature compression around neck', Journal of Forensic Medicine and Toxicology, Year 2004, Vol: 21 (2) : pg 34-36.
7. Hausmann R, Benz P. : 'Delayed death after attempted suicide by hanging', Int J Legal Med 1997 : 110 (3) : pg 164-166.
8. Vaghela DR , Patel PR : 'late death in a case of hanging': Anil Aggrawal's Internet Journal of Forensic Medicine and Toxicology, 2009; Vol. 10, No. 1.
9. Aggrawal NK, Kishore U, Agarwal BB: 'Hanging delayed death (rare phenomenon)' Med Sci Law. 2000; July 40 (3); pg. 270-272.

Variations in Length of Facial Nerve in the Segments of Temporal Bone of Male Cadavers in South Indian Region

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ABSTRACT

In the present study facial nerve was dissected in 25 male temporal bones. The length of facial nerve in different segments of temporal bone was measured. In petrosal segment the mean length of facial nerve was 7.82 mm ± 0.27(SD=1.32). In labyrinthine segment mean length was 3.4mm ± 0.9(SD=0.45). In tympanic segment mean length was 12.13mm ± 0.7(SD=0.35). In mastoid segment it was 12.24mm ± 0.38(SD=1.90). The mean length of nerve to stapedius was 15.71mm ± 0.13 (SD=0.63) and mean length of chordatympani nerve was 19.01mm ± 0.17(SD=0.82). (These both nerves had measured from geniculate ganglia).

The present values varied with previous workers in some segments and agreed in some segments of temporal bone. Hence these values had regional or racial significance could be due to biological variation or outcome of hybridized race but important for clinician, radiologist and mainly for Otosurgoen to anticipate the position of facial nerve to avoid its injury.

Key words: *Petrous, Tympanic, Labyrinthine, Mastoid, Otosurgery*

INTRODUCTION

Perhaps facial nerve is the only nerve which has long course in bony canal. From brain stem to terminal branches it changes its direction five times to innervate muscles of 2nd arch[1] Facial expressions are controlled by facial nerve, is an important means of communication because facial nerve is composed of approximately 10,000 neurons, out of them 7,000 are mylienated and innervate the nerves of facial expression. Due to its torturous course and variations in its length in different segments, its more prone for injuries in many otological approaches. Hence second most common cause for facial paralysis is reason for malpractice during surgery[2]. Although it looks safer in bony canal but facial canal may be thin or nerve may be uncovered by erosion of bones due to disease. If the nerve is injured at tympanic and vertical course of mastoid part homo lateral stapedial reflex, sub mandibular salivary gland secretion on that side, and taste function of anterior 2/3rd if tongue is affected[3].

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MATERIALS AND METHODS

The material for this study was 15 male temporal bones were selected from dissection theatre of JJM medical college and 10 male temporal bones from SSIMS and RC Davangere. Temporal bones were taken out by removing the soft parts attached to it. The facial nerve was exposed as per the method given in cunningham manual vol.III[4]. The length of the nerve was measured by thread and scale (in mm).

RESULTS

Table No 1 – Shown the length of facial nerve at petrosal segment of temporal bone (length was measured at the entry into internal auditory Meatus to the superior semicircular canal). The mean length of facial nerve was 7.82mm ± 0.27(SD=1.32).

Table No 2 – Shown the length of facial nerve at labyrinthine segment of temporal bone (measured from superior semicircular canal to the geniculate ganglia). The mean length of the nerve was 3.4mm ± 0.9(SD=0.45).

Table No 3 – Shown the mean length of facial nerve at tympanic segment of temporal bone (The length was measured from geniculate ganglia to processus cochlearformis). The mean length was 12.13mm ± 0.7SD=0.35).

Table 1. Length of facial nerve at petrous part of temporal bone (length was taken at the entry into internal auditory Meatus to the superior semi circular canal)

No of cadavers (25)	Length mm	Mean mm	SD	95% C.I.
3	5.2			
13	7.4	7.82	1.32	7.55±8.08
6	9.0			
3	9.9			

Table 2. Length of facial nerve at labyrinthine part of temporal bone (length was measured from superior semicircular canal to the geniculate ganglion)

No of cadavers (25)	Length mm	Mean mm	SD	95% C.I.
11	3.0			
8	3.4	3.4	0.45	3.31±3.49
4	4.0			
2	4.4			

Table 3. Length of facial nerve at tympanic part of temporal bone (length was measured from geniculate ganglion to process us cochlearformis)

No of cadavers (25)	Length mm	Mean mm	SD	95% C.I.
2	11.74			
17	12.00	12.13	0.35	12.06±13.2
4	12.40			
2	13.2			

Table No 4 – Shown the length of facial nerve at mastoid segment of temporal bone. (The length was measured from medial wall of tympanic cavity where the nerve runs vertically downwards on the posterior wall of tympanic cavity to stylomastoid foramen). The mean length was 12.24mm ± 0.38(SD=1.90).

Table 4. Length of facial nerve at mastoid part of temporal bone (length was measured from the medial wall of tympanic cavity where the nerve runs vertically downwards in the posterior wall of tympanic cavity to stylo mastoid foramina)

No of cadavers (25)	Length mm	Mean mm	SD	95% C.I.
2	8.4			
12	11.2			
1	12.0			
4	13.4	12.24	1.90	11.86±12.62
2	14.5			
3	15.0			
1	15.4			

Table No 5 – Shown that, length of nerve stapedius. (The distance was measured from geniculate ganglia). The mean length of the nerve was 15.71mm ± 0.13(SD=0.63).

Table No 6 – Shown the length of the level of branching of chorda tympani nerve (The distance was measured from geniculate ganglia) the mean length of the nerve was 19.01mm ± 0.17 (SD=0.82)

Length of branches in the mastoid part of temporal bone

1) Nerve to stapedius – It arises from upper part of mastoid part of facial nerve.
2) Chorda tympani - It arises from mastoid part of facial nerve 2 mm below the nerve to stapedius.

Table 5. Length of level of branching of nerve to stapedius (distance measured form geniculate ganglion)

No of cadavers (25)	Length branching of nerve to stapedius	Mean mm	SD	95% C.I.
17	15.3 mm			
6	16.4 mm	15.71	0.63	15.58±15.84
2	17.2 mm			

Table 6. Length of level of branching of chorda tympani (Distance measured from geniculate ganglion)

No of cadavers (25)	Length branching of chorda tympani (mm)	Mean	SD mm	95% C.I.
15	18.4			
4	19.3	19.01	0.82	18.84±19.18
3	20.2			
3	20.5			

DISCUSSION

In the present study mean length of facial nerve at petrous part of temporal bone was 7.82mm± 0.27(SD=1.32). In labyrinthine part mean length was 3.4mm ± 0.9(SD=0.45) and tympanic part was 12.13mm ±0.7(SD=0.35) and in mastoid part was 12.24mm± 0.38(SD=1.90). These values of present study differs with previous workers of north India they observed the length of facial nerve at tympanic segment was 11.1mm±0.8 and in mastoid segment was 15.4mm± 2.14[5] and European studies had mean length of facial nerve at labyrinthine segment 3.4mm, and in tympanic segment 8-11mm and in mastoid segment 10-14mm[6] which partially was in agreement with present study. Similarly Romania cadaveric study also had the length of facial nerve in tympanic segment was 9.15 to

Table No 7 – Shown the comparison of the present results with the pervious workers of India and abroad.

Table 7. Length of facial nerve at different segment of the temporal bone in different regions (Places)

Name of the worker	Name of the place	Length in Petrous part mm	Length in Labyrinthin part mm	Length in Tympanic part mm	Length in Mastoid part mm
Nager C T Protracter etal 1982	European	-	3.4	8-11	10.14
Yadhav S P Ranga R 2006	North India	-	-	11.1 ± 0.8	15.4 ±2.34
Boemae R L Navarette 2007	Spain	-	-	-	13.34(SD = 1.43)
Waheed E etal 2009	Iraq	5 – 10	3.5 – 4	12.47± 0.43	-
Kharat R D Golharetal 2009	Maharastra	-	-	9.28	13.7
Nicoleta Maru etal 2010	Roman	-	3.14 – 5.27	9.15 – 12.03	11.23 – 15.07
Present Study 2011	South IndiaKarnataka	7.82 ±0.27	3.4 ±0.9	12.33 ±0.7	12.24 ±0.38

12.03mm, in labyrinthine segment was 3.14 to 5.27mm and in mastoid segment varied between 11.23 to 15.07mm[7] which also partially in agreement with present study. In Iraqi temporal bones had mean length of facial nerve at pertous part was 5 to 10mm, and in labyrinthine part varied between 3.45 to 4mm in tympanic part it was 12.47mm ± 0.43[8]. While mean length of facial nerve in mastoid segment was 13.34mm (SD=1.43) in temporal bones of Spain[9]. In Maharashtra, Cadaveric study mean length of facial nerve in tympanic segment was 9.28mm and in mastoid segment was 13.7mm[10].

Mean length of the nerve to stapedius and chordatympani was also found variation in length was also found variation in length because temporal bone region is common place for variation, in the length of facial nerve and its branches[11].

The probable reason for these variation could be the different morphological elements of temporal bone viz, petro mastoid, tympanic, squamous, styloid process. Facial nerve being the nerve of IInd arch runs behind the cartilaginous bars of Ist and IInd arch (which are typically arranged) to innervate muscles of facial expression[12].

Moreover during development, motor axons of the arches undergo an intricate feat of path findings to reach their target muscles. These pathways are regulated by same chemo attractants and chemo repulsants produced by mesenchyme. If there is altered expression of these agents may lead to variations in the length of facial nerve[13].

Developmentally chondrified otic capsule stops its ossification and forms a canal to give space for facial nerve and vestibule cochlear nerve which will later ossified into petrosal part on temporal bane vestibule cochlear nerve has limited distribution but facial nerve has to innervate muscles of facial expression by torturous journey. Moreover this variation of length of facial nerve in temporal bone is due to dual ossification of temporal bone i.e. partly cartilaginous and partly membranous, Rate of the bone growth and maturation is influenced not only by age and sex but by the economic status, individual body weight possibly by function. Regional and racial differences also required to be taken differences also required to be taken into account. In addition to this morphometrical values of membranous bones are uncertain.

Mastoid process is observed only in gorilla and man. In gorilla it was rudimentary and medially placed but in human it was shifted laterally with prominent process which is filled with more amount of pnematization to act against antigravity movements of skull and adopt erect posture[14].

Regarding these partial variations and partial agreement values (Table No 7) it can be hypothesized that, it was due to environmental, genetic and different dietary habits because in the present study dissected temporal bones belonged to dark skinned, short statured, long headed cadavers, presumably hybridized Dravidian race. Hence these obtained values will also have regional and racial significance apart from clinical, surgical and radiological importance because most of the Indian anthropologist believes that all the races of mankind have migrated to India in olden days at different centuries. Hence these values of partial variation and partial agreement might be rooted back to their ancestors[15].

CONCLUSION

The variations in the length of facial nerve at petrous part was 7.82mm ± 0.27(SD=1.32) at labyrinthine part 3.4mm ± 0.9 (SD=0.45) at tympanic part 12.13mm ± 0.7 (SD=0.35) at mastoid part 12.24mm ± 0.38(SD=1.90) length of nerve to stapedius was 15.71mm ± 0.13 (SD=0.63) and length of chordotympani was 19.01mm ± 0.17(SD=0.82). These values will be useful to the Otosurgoen and radiologist, clinician because the segments of temporal bone is known for variations and anamolies which often brings surprise for Otosurgoen to face less frequent or even unseen anamoly and the operative result is disaster. More over these variations represent hybridization of race and regional significant.

REFERENCES

1. Dutta AK, Facial nerve. Essential of human anatomy 1989, vol.III 1st edition III, 308-315 current Books International Publication
2. Weit.R.J, Iatrogenic facial paralysis. otolaryngol. clin. north. Am. 1982, 15(4), 773-780.
3. Decker. GA, Lee MC Gregor's synopsis of surgical anatomy 1986, XIIth edition. 176,184, 363-368. John Wright and Sons Ltd. Techno house Redcliffe way Bristol publication
4. Romanes G.J, Cunningham's Manual of practical Anatomy Vol.IIIrd. Head Neck and Brain 2002 (18th edition). ELBS and Oxford University press publication.
5. Yadhav. SP, Ranga A, Sirohiwal BL, Chanda R – Intra-temporal facial nerve Indian journal of Otolaryngology and Head and Neck Surgeon 2006. 58(1), 27-30.
6. Nager. G.T, Protractor B. Facial canal normal anatomy, variation and anamolies Ann. Otol. Rhinol. larangeal Suppl. 1982 97, 45-61.
7. Nicoleta Maru, Cheija AC, Mogoante C.A, Prejuoi anu B. Intra temporal course of facial nerve morphological, topographical and morphometic features. Roman Journal of Morphology and embryology 2010 51(2), 243-248.
8. Waheed E Saad M. Kamal I, Yousuf I, Fatwaz EJENT "Egyptian Journal of Ear Nose, Throat and Allied Sciences 2009, 10, 18-21.
9. Boenac R.L, Navaretle ML. Morpho metric study of mastoid segment of facial nerve. Acta Otorrinolarangal. ESP 2007, 58 (5), 178-181.
10. Kharat R.D. Golhar and Pati Cy - Study of intra temporal course of facial nerve and its variations 25 bones dissection. Indian Journal of Otolaryngology and Head and Neck Surgery 2009, 64 39-42.
11. Boemo R.L, Navarret ML, Lareas etal. Anatomical relationship between the position of sigmoid sinus, tympanic membrane and diagastric, ridge with mastoid segment of the facial nerve Ur. Arch Otorhinolaryngology 2008 265, 389-392.
12. Ernest Frazer J, Development of temporal bone. Anatomy of human skeleton 1933, IIIrd edition, 212-217. A Churchill, 40 Gloucester place Portman square London publication.
13. Larcen W J, Human embryology. Innervation of developing limb bud and arches 4th edition. Churchill living stone, Philadelphia 1993; 640-643
14. Santa luca. AP, A re-examination of presumed Neanderthals-like fossils. Journal of Human evolution 1979, 7, 619–636.
15. Raghavan P, Invasion of Aryans presumption expert chitlee. K Sethi: Tribunae news service. http://www.tribunaeindia.com/2004-1103/chd.html. Sci Law. 2004 Apr; 44(2): 160.

Morphometrical Study of Mitral Valve

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ABSTRACT

25 Male Cadveric hearts preserved in formalin in the dept of Anatomy dissection theatre of Dr V.M. Govt Medical College Solapur were studied metrically and morphologically.

1) The mean value of the mitral valve Circumference was 8.97 cm with (SD ± 0.7). 2) The mean value of length of anterior leaf let was 1.8 cm and that of posterior leaf let was 1.4 cm. 3) Thickness of both ant and posterior leaflets was having same mean value of O.2 cm(SD ± 0.44cm). 4) Length of papillary muscle i.e. mean length of anterio- lateral Papillary muscle was 1.8 cm (SD±0.3) and post. Med. Papillary muscle was 1.6 cm(SD±0.3). There all observations were more or less in agreement with previous workers[1,2] but Incidence of heads of papillary muscle differed with previous workers[1].

Morphologically the leaf lets were smooth, Crescent shaped and shape of annulus was Saddle shaped or 'D' Shaped and non-planar. Both papillary muscles with intact chorda tendane were observed which indicates normalcy of "Mitral Valve"

Key words: *M.V. = mitral valve, A.L. = antero lateral, P.M. = postero-medial, C.M. = centimeter, M.A.C. = mitral annular calcification A.V. = atrioventricular.*

INTRODUCTION

"Mitral Valve" is named after its appearance like mitre of Bishop" It is Situated between left atrium and left ventricle forming inlet of ventricle and outlet of atrium. It is also called as bicuspid valve due to possession of two cusps anterior and posterior.

As tomographic techniques are increasingly used for diagnosis of cardiac diseases. Hence Morphometric study of mitral valve is important for clinicians, Surgeons and echo cardiologists. More over, a tiny instrument inserted in to MV enhance or regulate the Contraction of M.V. during systole and diastole.

"Mitral Valve" consists of four major components. Annulus, leaflets chordae tendanae and papillary muscles. If anyone among these is abnormal congenitally or acquired, leads to dysfunction of mitral valve[3] causing varied number of diseases because regurgitation of mitral valve is quite common clinical entity[4] which is likely to increase in future due to predicted demographic changes, It is also associated with poor long term survival. Moreover mitral annular calcification (MAC) is quite common in females, diabetes mellitus, and increasing body mass Index in adults[5].

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MATERIALS AND METHODS

A total No of 25 male formaline fixed heart were obtained from Department of Anatomy. The circumference was measured and then heart was carefully dissected and left atrium and ventricles were opened without disturbing the position of M.V. Length was measured from base to summit with the help of scale and thread. The circumference was measured by Vernier calipers.

OBSERVATIONS AND RESULTS

Circumference of the M.V was measured in Cms. The maximum circumference was 9.7cms and minimum circumference was 8.2cms and mean value was 8.97cms (±0.7) (Table 1). The length of the anterior leaflets varied Between 1.5cm to 2.2 cms, and mean value was 1.8cms (SD±0.3) and length of posterior leaflet varied between 1.2cm to 1.7cms and the mean value was 1.4cm (SD±0.2cm). The anterior leaflets were larger than posterior leaflet. The thickness of

anterior and posterior leaflets were almost same, which varied between 0.1cm to 0.3cms and the mean value was 0.2cm (SD±0.44cm) (Table 2), Number of heads of papillary muscles The AL papillary the incidence of single head was 60%, double head was 20%, triple head was 12% and more than triple head was 8%. The P.M papillary muscle incidence of single head was 48% double head was 32% and triple head was 12% and more than triple head was 8%. (Table 4). The length of papillary muscle, the length of AL Papillary muscle varied between 1.3cms to 2.2cms with mean value of 1.8cms to (SD±0.3cm) and the length of P.M Papillary muscle varied between 1.2cm to 2cms with the mean value was 1.6cm (SD±0.3cm).

Morphologically the Mitral Valve was like crescent shaped and the shape of annulus was saddle shaped and not-plane.

DISCUSSION

The mean value of mitral valve circumference was 8.97cm (SD±0.7) (Table 1) This Value is more or less in agreement with Chie chi and Lees (1956), Moreover the diameter or circumference of mitral valve increases along with body mass Index of the individual[6]. The length and thickness of the anterior and posterior leaflets (Table 2) The mean value was 1.8cm (±0.3cm) and 1.4cm (±0.2) respectively. Thickness mean value of anterior and posterior leaflet was 0.2 cm.This observed value was also more or less in agreement with findings of Rusteds Chiebly and Edward (1952)[7], Chie chi and Lees (1956), Ranganathan etal (1970) (Table 3). The anterior leaflet was larger than posterior leaflet but the thickness of the both leaflets was same 0.2cm (SD±0.44). It shows that the presently studied mitral valve was normal (Physiological). Incidence of heads papillary muscles (Table 4). The incidence of AL papillary muscle had single head was 60% double heads 20% triple head 12% and more than triple heads was 8%. In PM papillary muscles, The incidence of single head was 48% double head was 32s% triple heads was 12% and more than triple heads was 8% but present study does not agree with previous studies chei chi and Lees 1960 (Table 5), Length of AL and PM Papillary muscles (Table 6), The mean value of AL papillary muscle was 1.8cms (SD±0.3) while length of PM papillary muscle had mean value of 1.6cm (SD±0.3), These findings are more or less in agreement with chei chi and Lees (1956) whose values were 3cm maximum and 1.8 cm minimum in AL Papillary muscle. While 2.5 cm Max. and 1.4 cm Min. (Table 7)

Thickness of anterior leaflets and posterior leaflet showed same mean value of 0.2cms (SD±0.44) which indicates that mitral valve was normal (Physiological) because anyone valve can also become thicker or both valves becomes thicker in moderate to severe regurgitation[8], The Annulus of mitral valve is saddle shaped, which indicates the normalcy of mitral valve, because annulus dilates during calcification and becomes disjunction in prolapse of mitral valve[9], The presently studied leaflets were smooth, delicate and devoid of calcification, because in calcification, and rheumatism the leaf lets become rigid and coarse. The leaflets or valves of orifice were crescent shaped. This shape confirmed the non-prolapse of mitral valve,

Table 1. Comparative study of circumference of Mitral Valve orifice in formalin preserved hearts (in cms)

	Maximum	Minimum	Mean Value	SD
Chie chi and Lees1956	11.5	8.5	10	± 0.9
Present Study	9.7	8.2	8.97	± 0.7

The present study of circumference of mitral valve was more or less in agreement with previous studies.

Table 2. Length and thickness of anterior and posterior leaflets (in cms)

Particular	Length cms		Thickness (cms)	
	Ant leaflet	Post leaflet	Ant leaflet	Post leaflet
Maximum length	2.2	1.7	0.3	0.3
Minimum length	1.5	1.2	0.1	0.1
Mean valve	1.8	1.4	0.2	0.2
Standard deviation	0.3	0.2	0.44	0.44

Mean Value of ant. leaflet 1.8 cm. SD± 0.3,
Mean Value of post. leaflet 1.4 cm. SD ± 0.2,
Mean Value of thickness of ant leaflet and post leaflet was same 0.2cm SD ± 0.44.

Table 3. Comparative study of length and thickness of anterior and posterior leaflets (in cms)

Particulars	Maximum length of leaflets			Maximum length of leaflets			Maximum thickness of leaflets			Maximum thickness of leaflets		
	Ant. Leaflet	Post. Leaflet	Mean & SD	Ant. Leaflet	Post. Leaflet	Mean & SD	Ant. Leaflet	Post. Leaflet	Mean & SD	Ant. Leaflet	Post. Leaflet	Mean & SD
Ranganathan	2.4	1.4	-	1.6	1.1	-	0.4	0.4	-	0.1	0.1	-
Present Study	2.2	1.7	1.8±0.3	1.5	1.2	1.4±0.2	0.3	0.3	0.2±0.4	0.1	0.1	0.2±0.44

The present study was more or less in agreement with previous studies.

Table 4. Number of heads of papillary muscles observed in Mitral Valve

Anterolateral papillary muscle			PosteroMedial papillary Muscle		
No of Heads	No of Specimen	%	No of Heads	No of Specimen	%
Single	15	60%	Single	12	48%
Double	5	20%	Double	8	32%
Triple	3	12%	Triple	3	12%
More than 3	2	8%	More than 3	2	8%

60% single head in AL muscle and 50% in PL muscle.

Table 5. Number of heads of papillary muscles of "Mitral Valve" (in cms) observed by Che Chi and Lees 1956

Types of Papillary muscle	Ant. Lat. Muscle		Post. Med. Muscle	
	No of Cases	Percentage	No of Cases	Percentage
Single Head	87	82.8	31	29.5
Double Head	15	14.3	57	54.3
Triple Head	03	2.9	12	11.4
More thanThree	-	-	05	4.8

These findings does not agree with present study

Table 6. Length of papillary muscles of "Mitral Valve" (in cms)

Particular	Length Antero lateral papillary muscle muscle	Length of Postero Medial papillary
Maximum length	2.2	2.0
Minimum length	1.3	1.2
Mean valve	1.8	1.6
Standard deviation	0.3	0.3

Mean value of length of AL papillary muscle was found to be 1.8 ± 0.3. While mean length of PM (Papillary Muscle) was found to be 1.6 ± 0.3.

Table 7. Comparative study of length of papillary muscles of "Mitral Valve" (in cms)

Particulars	Anterio- lateral			Posterio-Medial		
	Max	Min	Mean & SD	Max	Min	Mean & SD
By Chei Chi & Lee 1956	3	1.8	2.41± 0.09	2.5	1.4	1.98± 0.37
Present Study	2.2	1.3	1.8±0.3	2.0	1.2	1.6±0.3

The mean values of the present study was more or less in agreement with Chei Chi & Lee 1956

because in prolapse of mitral valve the cresent shape changes to redundant as the leaflets tethered to tensor apparatus[10]. Occasionally leaflets assume hooded appearance with pocket like doming towards atrium. These pockets occur between chordal attachment during the prolapse of mitral valve[11]. Rapture of chordae tendane, rapture of papillary muscle and abnormal motion of Mitral.Valve. wall is secondary to myocardial ischemia[12,13].

SUMMARY AND CONCLUSION

The present study was carried out in formaline fixed hearts of 25 adult male cadavers. The mitral valve was exposed and length breadth and thickness of mitral valve was studied. The circumference was mitral valve orifice was measured. Similarly anteriolateral posterior-medial Papillary muscles were measured and single double and multiple number of papillary muscles was noted. The mean length of anterior leaflet was found to be 1.8cm and thickness was 0.2cm while mean length of posterior leaflet was 1.4cm and thickness was 0.2cm. the mean circumference of mitral orifice was 8.97cms, mean length anterio-lateral papillary muscle was found to be 1.8 cms, while posterior medial papillary muscle was 1.6 cms. incidence of single head in AL Papillary muscle was 60% double head was 20% triple head was 12% and more than triple head was 8%. Incidence of posterior-medial papillary muscle the single head was 48% and double head was 32% and triple head was 12% and more than three heads was 8%. All the values were compared with previous studies which were more or less in agreement with previous studies.

The arrangement of mitral valve and its contents are important for clinician, surgeon and echo-cardiologists because mitral valve is approached in orthogonal, cross section, and short axial planes. Moreover AV (Atrio Ventricular) bundle is more or less directly related to Mitral valve. Hence morphological and metrical study is important for surgeon to avoid damage to AV bundle during surgery.

REFERENCES

1. Chie chi MA, Lees. WM. And Thompson R: Functional Anatomy of Normal valve. Journal of thoracic surgery, 32, 378, (1956).
2. Ranganathan, Lam JHC and Wigle ED: Morphology of human Mitral valve II The valve leaflets. Circulation vol.XLI (1970). 372-380PP
3. H.O. Sy. Anatomy of mitral valve. Heart (2002) 88, (IV), 5-10.
4. Schmitto JD. Lees LS. Mokashi SA. Bolman RM 3rd, cohm LH, Chen Fy : Functinal mitral regurgitation Multi Media Cardiothoracic Surgery (2010).18(6), 285-291.
5. Kanjamauthai S, Nasir. K, Kat ZR, Rivera JJ, Takasu J, Blumenthal R S, Eng J, Bud off M J : Relationship of Mitral annular calcification to cardiovascular risk factors; The multi-ethnic study of Atherosclerosis. (MESA). Multimedia manual of cardio thoracic surgery, (2010). 214, 96-112.
6. Rajila Rajendam. HS, Seshayyans, Victor.A, Murugesan. N, Sundara Murthy : The Study of

Mitral Valve annular dimension in relation to the body surface area in The Indian Population European.
Journal of Cardiothoracic surgery, (Abstract) (2010) PMID- 20943411 (Pub Med- as Supplied by publisher).

7. Rusted Schefly and Edward: Studies of Mitral Valve 1, Anatomic features of Mitral Valve and associated structures circulation 6, 825-831 (1956).
8. Malkawsk, MJ. Boudalas.H. Wooley CF: Spectrum structural abnormalities in the floppy Mitral Valve echo Cardio graphic evaluation. American Heart Journal (1996). 132, 145-151.
9. Angelimi A, Hosy Anderson R.H: Histological Study of atrioventricular junction in hearts with normal and prolapsed leaflets of the Mitrel Valve. British Heart Journal (1988) 59, 712-716.
10. Cheng TO, Xie MX, Wang X.F: Evaluation of Mitral Valve prolapse by four dimensional echo cardiography American Heart Journal (1997) 133, 120-129.
11. Becker A.E, dewit APM : The Mitral Valve apparatus; a spectrum of normality relevant to mitral valve prolapse. British Heart Journal (1980) 42, 680-689.
12. Becker.AE Davis M.J : Pathomorphology of Mitral Valve prolapse. In Valve prolapse mitral Valvular regurgitation 2nd edition Armonk Futura Publishing Company (2000) 91-114.
13. Davies MJ, Moore BP. Braimbridge M.V: The floppy Mitral valve Study of Incidence pathology and complications in surgical necropsy and forensic Material. British Heart Journal(1978), 40, 468-481.

A Comparative Study of Injuries of Lower Extremity in Junior Foot Ball Players in Both Sexes

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ABSTRACT

37 injured males and 28 females of junior foot ball players, injured to lower extremity were studied. The age group of players was 12 to 17 years. More stretching was observed in calf muscle of male 19 (54%) and female 11 (42%) Quadriceps stretching in males 10 (27%) and females 9 (34%), stretching of hamstrings in males 5 (13.5%) and females 4 (15.4%) and least stretching was observed in gluteal region males 3 (8.1%) and females 2 (7.7%). Injures at ankle joint was highest in males 11 (29.8%), females 7 (26.9%) and knee joint injurywas second highest males 9 (24.3%), females 7 (26.9%) and third highest was Foot and toes in males 5 (13.5%), females 3 (11.6%) and least was hip joint, males 1 (2.7%), females 1 (3.8%) but statistically insignificant ($P > 0.05$).In personal position of players, a) Highest injuries of male players i.e. defender had injuries at leg bones 11(30%), Middle fielder had injuries at knee joint 11 (30%), and Strikers had injuries at ankle joint 11(30%). b) In females highest injuries i.e. defenders and middle fielders had injuries at ankle joint 9(30%), 8(30%) and strikers had injuries at knee joint 6(25%) but goal keeper of both sexes had least injuries and no injuries at hip and ankle joints. Overall highest injuries were observed in ankle joint followed by knee joint and hamstrings but least injuries were observed in gluteal region in both sexes. The acute injuries were 90% and sustained injuries were 10%. It is interesting to note that lower age groups had more injuries as compared with higher age group. This anatomical study will help the radiologist and orthopedic surgeons to treat the injured and sports authorities to take preventive measures.

Key words: *Ankle joint, Knee joint, Quadriceps, Hamstrings, Calf muscles.*

INTRODUCTION

Although our country players of foot ball participate in international sports also but due to phobia of injuries most of the players lag behind hence it was rightly said that "aching moments are longer than the years". Moreover most of the players come from rural areas where they are not trained properly and succumb to injuries.

Junior players had more vascularity, biomechanical actions are more active and potential. But the skeleton of the particular individual is able to adapt to its owners way of life[1] and rapidly changing forces are generated in the body by gravity muscle actions and impacts. These are passed to the skeleton, inspite of being light in weight and slender in build bones keep their shape under load and rarely break. The reason for this efficiency of skeleton are (a) bone is stiff, rigid, elastic, strong, tough yet light material. (b) The bones display this material in an economic manner. (c) The intensity and suddenness of the forces developed in the body are modified by the soft tissues, so that their effects on the bones are minimized in particular some contracting muscles tend to compress the underlying bone in its long axis and this counteracts the development of dangerous tensile stresses.

Apart from this the more important is the muscles and ligaments control by the nervous system are able to protect the skeleton form fracture by controlling the intensity rate and direction of forces which are applied to the bones. By the same taken muscle contracting at the wrong time and in the wrong place

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can break the bone or at least stretch the ligaments. A drunken man with relaxed muscles can often escapes injury in circumstances where a man with tensed muscle would suffer a fracture[2]. Actual stresses and strained develop in a skeleton when it is loaded are very difficult to determine.

Hence injuries are due to lack of confidence and confusion apart from architecture of skeleton and nutritional status of individual because the brain control is in use of "external limbs" which are unnecessary in cases where the tools are unchangeable because they are part of man's own body and are linked to brain by nervous system and learn their jobs so well that they never require to be prompted.[3]

MATERIALS AND METHODS

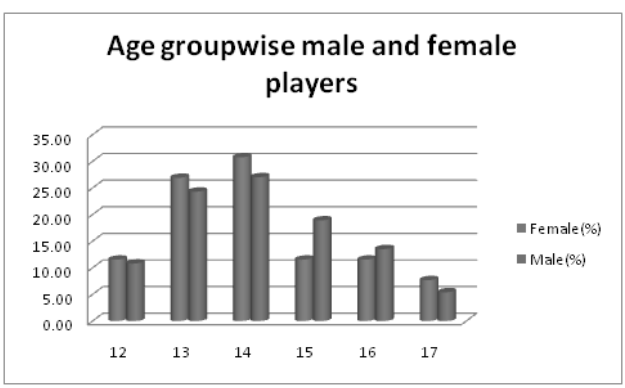
During the district level sports of junior foot ball tournament of Davangere which was held for fifteen days in which players from different places were participated. The injured 37 males 26 females of age between 12 to 17 years were studied and their injures were compared in both sexes.

RESULTS (FINDINGS)

Table 1. Shown that, 13 to 14 years of age players were more in both sexes but it was statistically insignificant (P >0.05).

Table 1. Comparative study of injuries of lower extremity in both sexes of junior foot ball players

Age (years)	12	13	14	15	16	17	Total	Mean ± SD	P value
Male	4	9	10	7	5	2	37	14.16 ± 1.36	p>0.05 Not significant
Female	3	7	8	3	3	2	26	14.07± 1.41	



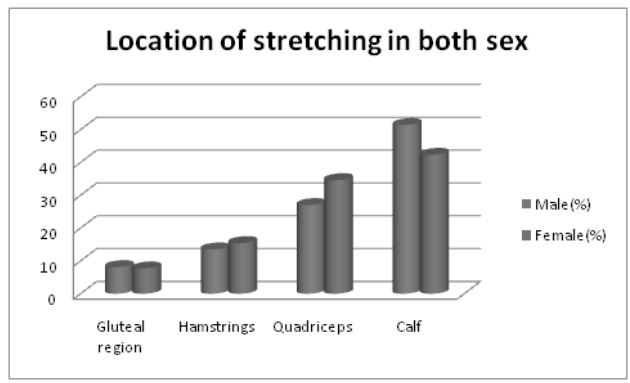
There is statistically no significant difference observed in both sexes of junior foot ball players

Table 2 Shown that highest location of stretching was in calf muscles in males 19 (15.4%) and in females 11(42.3%) and second highest location of stretching was at quadriceps males 10(27%), females 9 (34.6%) and least stretching was at gluetal region 3(8.1%) in males and 2(7.7%) in females but all values were statistically insignificant (P >0.05).

Table 2. Location of stretching at different ages in both sexes

Location	Male	Female	Z test	P value
Gluteal region	3(8.1%)	2 (7.7%)	0.60	p>0.05*
Hamstrings	5(13.5%)	4 (15.4%)	0.20	p>0.05*
Quadriceps	10(27%)	9 (34.6%)	0.64	p>0.05*
Calf	19(51.4%)	11(42.3%)	0.70	p>0.05*

*indicate non significant.



There is statistically no significant difference observed in Location of stretching at different ages in both sexes of junior foot ball players

Table 3 Highest locations of injuries were in ankle joint of both sexes. Males 11(29.8%) and female 7(26.9%) and second highest injuries location was at knee joint in both sexes. Males 9(24.3%) and females 7 (26.9%) and third highest injuries at foot and toes in males 5 (13.5%) and in females 3(11.6%) and least location of injuries at hip joint , in males 1(2.7%) and in females 1(3.8%)but all the values were statistically insignificatnt (P >0.05).

Table 3. Location of injuries in both sexes at different age groups

Location	Male N=37	Female N=26	Male (%)	Female (%)	P value
Hip joint	1	1	2.7%	3.8%	N.S.
Quadriceps	2	2	5.4%	7.7%	N.S.
Hamstrings	3	2	8.1%	7.7%	N.S.
Knee joint	9	7	24.3%	26.9%	N.S.
Leg bones	3	2	8.1%	7.7%	N.S.
Calf muscle	3	2	8.1%	7.7%	N.S.
Ankle joint	11	7	29.8%	26.9%	N.S.
Foot and toes	5	3	13.5%	11.6%	N.S.

N.S. indicates non significant

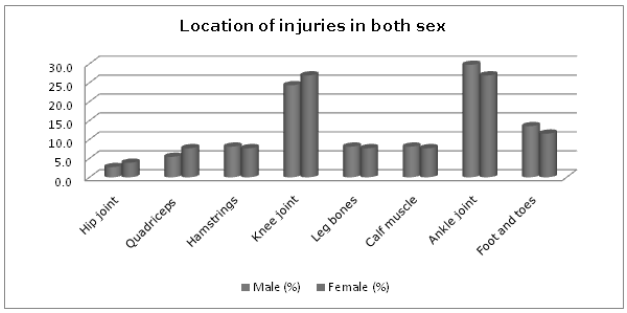


Table 4 Had shown highest stretching at calf muscles followed by quadriceps and hamstrings at 12, 13, and 14th years of age and least stretching was in 16th and 17th years of age in both sexes.

Table 4. Location of stretching in males and females at different ages

Location	12 years (%)	13 years (%)	14 years (%)	15 years (%)	16 years (%)	17 years (%)
Gluteal region Male	0 (0%)	1 (11.1%)	1 (10%)	1 (14.3%)	0 (0%)	0 (0%)
Gluteal region Female	0 (0%)	1 (14.3%)	1 (12.5%)	0 (0%)	0 (0%)	0 (0%)
Hamstring Male	1 (25%)	2 (22%)	2 (20%)	1 (14.3%)	1 (20%)	0 (0%)
Hamstring Female	0 (0%)	1 (14.3%)	1 (12.5%)	1 (33%)	0 (0%)	0 (0%)
Quadriceps Male	125%	333%	330%	228.5%	240%	1 50%
Quadriceps Female	1 (30%)	2 (28.5%)	3 (37.5%)	1 (33%)	1 (30%)	1 (50%)
Calf Male	250%	333%	440%	342.8	240	1 50%
Calf Female	2 (70%)	3 (42.8%)	3 (37.5%)	1 (33%)	2 (70%)	1 (50%)

DISCUSSION

In the present study stretching and injuries of both sexes was compared (Table No.2 & 3). Injuries or stretching were least in gluteal region. As gluteal muscles are antigravity muscles and chief extensor of thigh and Ilio-femural ligament is a strongest ligament of the body. Stretching of hamstring muscles in males 5(13.5%) and females 4(15.4%) and injuries in males 3(8.1%) and in females was 2(7.7%). Hamstrings are flexors of knee and extensor of hip hence in the injury of knee joint of contracture of hamstring muscle is common[4].

Stretching of quadriceps in males 10(27%) and in females 9(34.6%) and injuries of the same muscles in males 2 (5.4%) and in females 2(7.7%) it was more in females[5]. Quadriceps are extensors of leg, rectus femoris flexes the hip with illiopsoas and prevents dislocation of patella along with vastus medialis.

In the present study stretching or injuries of calf-muscles was observed. Stretching in males 19(51.4%) in females 11(42.3%). Injuries in males 3(8.1%) and in

Table 5 Had shown highest injuries at ankle joint followed by knee joint, hamstrings and foot and toes. More injuries in the ages of 12, 13, 14th and 15th, and less injuries in the age of 16th and 17th years in both sexes.

Table 5. Location of injuries in males and females at different ages

Location	12 years (%)	13 years (%)	14 years (%)	15 years (%)	16 years (%)	17 years (%)
Hip joint	—	—	—	—	—	—
(Male)						
Hip joint	—	—	—	—	—	—
(Female)						
Quadriceps	—	—	—	1(14%)	1(20%)	—
Male						
Quadriceps	—	—	—	—	—	—
Female						
Hamstring	1(25%)	2(22%)	2(20%)	1(14%)	1(20%)	—
Male						
Hamstring	1(33%)	1(14.2%)	1(12.5%)	1(33%)	—	—
Female						
Knee joint	1(25%)	3(33%)	2(20%)	2(28.6%)	1(20%)	1(50%)
Male						
Knee joint	1(33%)	2(28.5%)	3(37.5%)	1(33%)	1(33%)	1(50%)
Female						
Leg bones	—	—	—	—	—	—
Male						
Leg bones	—	—	—	—	—	—
Female						
Calf muscle	—	—	—	—	1(20%)	—
Male						
Calf muscle	—	—	—	—	—	—
Female						
Ankle joint	2(50%)	4(44%)	5(50%)	2(28.6%)	1(20%)	1(50%)
Male						
Ankle joint	1(33%)	3(42.8%)	3(37.5%)	1(33%)	1(33%)	1(50%)
Female						
Foot & toes	—	—	1(10%)	1(14%)	—	—
Male						
Foot & toes	—	1(14.2%)	1(12.5%)	—	1(33%)	—
Female						

Table 6 Had shown the injuries in the personal position of players, the middle fielder and strikers had highest injuries as compared goalkeeper and defender. However strikers of both sexes had most injuries.

Table 6. Injuries and players position

Location Injury	Goal keeper		Defender		Middle fielder		Striker	
	Male	Female	Male	Female	Male	Female	Male	Female
Hip joint	0	0	2(5%)	1(5%)	7(20%)	1(5%)	2(5%)	3(10%)
Quadriceps	7(20%)	5(20%)	7(20%)	5(20%)	3(10%)	5(20%)	—	—
muscles								
Hamstring	7(20%)	5(20%)	4(10%)	3(10%)	—	—	4(10%)	5(20%)
Knee joint	4(10%)	4(15%)	4(10%)	1(5%)	11(30%)	5(20%)	9(25%)	6(25%)
Leg bones	7(20%)	4(15%)	11(30%)	4(15%	4(10%)	5(10%)	4(10%)	1(5%)
Calf muscles	6(15%)	4(15%)	2(5%)	1(5%)	4(10%)	3(10%)	4(10%)	2(8%)
Ankle joint	0	0	5(15%)	9(30%)	7(20%)	8(30%)	11(30%)	5(20%)
Foot & toes	6(15%)	4(15%)	2(5%)	2(7%)	2(5%)	3(10%)	4(10%)	4(15%)

females 2(7.7%). These values were quite lesser than previous workers[6]. Stretching or injury was due to abduction which may lead to spiral fracture of lateral malleolus, than evulsions of tibial collateral ligament with or without evulsion of flake of medial malleolus and finally posterior margin of lower end of tibia shears off against talus bone. It occurs in potts fracture[7], often seen in foot ball players.

Moreover stretching of calf-muscles, hamstrings, quadricepses were observed in lower age group like 12, 13 and 14th (Table. 4).

Knee joint injuries in males 9 (24.3%) and in females 7(26.9%) (Table 3). It was observed in all age groups of both sexes (Table 5). It was second highest injury because medial menisci is more vulnerable to injury than lateral menesci because of its fixity to collateral ligament and greater excursion during rotator movements, moreover injury to anterior cruciate ligament is quite common than posterior[8]. It may be due to violent hyperextension of knee joint in football player. Tear of this ligament leads to antero-posterior mobility because rotating movement are caused by menisci and popliteus muscle.

Ankle joint injuries of present study, in males 11(29.8%) and in females 7(26.9%), foot and toes in males 5(13.5%) and in females 3(11.6%). It was more or less in agreement with previous workers[9-11]. The probable reason could be inversion and eversion greatly helps the foot in adjusting it to uneven and slippery ground. When feet are supporting body weight these, movements occur in a modified form called supination and pronation which are forced on foot by the body weight.

When injuries of particular player was studied (Table. 6), middle fielder and striker had more injuries as compared to goalkeeper and defender. However strikers of both sexes had more injuries.

The acute injuries were 90% and sustained were 10% which was more or less in agreement with previous workers[12]. Hence care has to be taken to avoid serious complications[13], especially knee and ankle joints[14].

Nerves involved in injuries of lower extremity are (1) sephenous nerve, the nerve can be entrapped in the sub-sartorial (Hunters) canal, injury to this causes pain over medial knee and anterior tibial area. (2) sciatic nerve compressed by piriformis and pains in sacral and gluteal area (3) Injury to common peroneal nerve causes at head of fibula and pains at lateral knee and antero lateral tibial area (4) deep peroneal nerve get compressed in the anterior tarsal tunnel (inferior extensor retinaculum) which causes pain and tenderness in dorsum of foot which is quite common in foot ball players (5) posterior tibial nerve – It is compressed in the medial tarsal tunnel causes burning pain and numbness over bottom of foot[15]. In present study injuries were quite common in smaller age group than higher age hence if injures are neglected it may cause undue complication because most of the players bones have not yet ossified. Hence they should be trained for prevention method of injures also[16] and cleaned, neat play ground is also necessary.

CONCLUSION

The highest injuries were observed in ankle joint followed by knee joint and hamstrings and least injuries were in gluteal region in junior football players' especially lower age groups than higher age groups (irrespective of sexes). It could be due to phobia and confusion because of less experience. It clearly indicates that as players grow older there will be less injures. Hence player must be trained to rescue from the injuries apart from teaching the tactics of foot ball games. This anatomical study will help the radiologist, orthopedic surgeon to treat the injuries and sport authorities to take the preventive measures.

ACKNOWLEDGEMENT

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Conflict of Interest: Nil

REFERENCES

1. Pritchard J.J, Growth and repair of the bone Oxford University press publication Ely house. London (1974) ISBN 0199141460.
2. Bourne G.H., Biochemistry and physiology of bone IInd edition 3 volumes(1972) Academic press publications. New York and London.
3 Crown ford. OGS Man and his past. (1921) Page 1-19. Oxford university publication.
4. Elliot M.C., Hamstring muscle strains in professional foot ball players a review. American journal of sports medicine 2011, 39(4), 843-850.
5. Holschem J.C., The female athlete Southern Medical Journal 2004, 97(9), 852-858.
6. Erk Strand J., Hug gland M. Epidemiology of muscle injuries in professional foot ball (soccer) players. American Journal of Sports Medicine 2011.18, (E pub med print).
7. Chourasia B.D., Anatomy of regional and applied lower limb and abdomen (1986) 36-39 (re-print)

C.B.S. publishers and distributors-Dehli-110032.
8. Ristic V. Ninkovics, Harhaji V. Causes of anterior cruciate ligament injuries. Med Pregl. 2011, 63 (7-8), 541-545.
9. Ishimaru D., Ogawa H. Sumi H. Sumiy Lower extremity injuries in snowboarding, (2010) Japan (H.O.) Gifu university publication.
10. Kordi R., Comparison of the incidence of nature and cause of injuries sustained on dirt field and artificial turf field by amateur foot ball players. Sports Med. Arthrose. Rehabil. Ther. Techno. 2011, 9, 3-6.
11. Emery C.A Meeu wisse. W.H. Etal Survey of sports participation and sports injury in category and area high school. Clinical Journal of Sports Medical 2006, 16(1): 20-26.
12. Fuller C.W. Molly M.G. Epidemiological study of injuries in men's international under 20 Rugby union tournaments. Clinical Journal of Sports Medicine 2011 E pub med. Print (25).
13. Ray W.Z., Mackinnan S.E. Nerve problems in lower extremity. Foot Ankle Clinic 2011, 16(2), 243-254.
14. Walden M., Hag gland M. Prospective study of injuries in professional foot ball during 2001-2002 seasons. British Journal of Sports Medicine. 2005, 39, 542-546.
15. Patil P.G., James N.C. Peripheral and central nerves system surgery for pain, in wall and Melzacks Text Book of pain Edstefen B.M. Martin K. (2006), 5th edition 591-601, Elsevier Ltd. Company Publication.
16. Taylor C.J., Pizzar T. Ames N. Orchard J.W., Gabbe B.J. Groin pain and hip range of motions is different in indigenous young Austrian foot ball players, a Journal of Science Medicine of Sports 2011 March 26 E pub med of print.

Incidence and Patterns of Skull Fractures in Accidental Deaths at Davanagere, Karnataka

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ABSTRACT

Fatalities as a result of accident claim a substantial number of lives in developing countries like India. Deaths due to road traffic accidents (RTA), may call upon entire spectrum of medico legal expertise. Injuries to head are common in road traffic accidents (RTA) and in cases of domestic accidents like fall from height, sports accident, industrial accidents etc., This necessitates us to widen the spectrum of study with respect to head injury involving skull fractures in fatal accidents. Hence the present study was conducted to know the incidence and patterns of skull fractures in fatal accidents with special reference to RTA.

Skull fractures were observed in 41.8% of the total accidental deaths. Amongst these skull fractures, RTA was commonest cause (86.3%). Most victims were male (81.6%), motor cyclists were the commonest victims. Linear fracture (38.8%) was commonest pattern of fracture in RTA and depressed fracture (48.1%) was in cases of fall from height. Sub-arachnoid was the commonest type of intracranial hemorrhage.

Key words: *Road Traffic Accidents; Fall from Height: Skull Fracture, Intracranial Hemorrhage.*

INTRODUCTION

Head is the vital organ and the most vulnerable part to receive injury. Head injuries associated with fracture skull are common in road traffic accidents (RTA) as well as in cases of domestic accidents like, fall from height, industrial and sports. This necessitates in widening the spectrum of study with respect to head injury involving skull fractures in fatal accidents. "Head injury" as defined by the National advisory for Neurological diseases and Stroke council "is a morbid state resulting from gross or subtle structural changes in the scalp, skull and/or contents of the skull, produced by mechanical forces"[1].

In this study we have concentrated upon the pattern of fatal skull fractures as a result of accidents i.e., road traffic accident, fall from height and other accidents. We have also endeavored to know the number of cases survived to reach the hospital. Here we have tried to ascertain the incidence and patterns of skull fracture, along with the vehicles involved, the time of occurrence, safety criteria observed and simultaneously comparative studies within city limits.

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OBJECTIVES

- To know the
  - Incidence and patterns of skull fractures in fatal cases of accidents with special reference to Road Traffic Accidents.
  - Co-relation of skull fractures with other intracranial lesions.
- To suggest preventive measures to avoid these fatalities.

METHODOLOGY

The present cross-sectional study was carried out on the victims of fatal accidents autopsied at Chigateri general hospital mortuary, Davangere from July 2005 to June 2007. Detailed information regarding deceased and various factors regarding the circumstances of accident like, type of victim, time of accident and other relevant information were gathered from all possible sources like police records, hospital records and also by direct interrogation with investigating officer, eye witnesses, relatives and friends of the deceased. In

addition to these X-ray, CT scan, MRI reports were examined for the presence or absence of fracture before commencing the autopsy. In each case, a thorough external and internal examination was conducted using standard autopsy protocols, so as to identify and locate the skull fracture and other injuries. The data thus obtained was recorded in the predesigned and pretested proforma, which comprised relevant data that is concerned with the objectives of the present study and was analyzed later.

All the victims of accidents i.e. Road Traffic Accident, fall from height, and other non vehicular accidents, with skull fractures were included in the present study. Age of the deceased was calculated as to the nearest completed years. The age groups of victims were categorized in an interval of 10 years. The cases were divided into 4 time slots as to the time of accident i.e. morning (6.00 am to 12 noon), afternoon (12.00 pm to 6.00pm), evening (6.00 pm to 12 midnight) and night (12.00 am to 6.00 am). The victims were categorized into pedestrian, pedal cyclist, motorcyclist, occupant of motor vehicle and others. The offending vehicles were divided into pedal cycle, motorcycle, motor vehicle, animal drawn vehicle, others (stationary objects like wall, tree etc.).

RESULTS

Table 1. Cases Showing Skull Fractures in Accidental Deaths

Type of accident	No. of victims	Cases of skull fracture
RTA	408 (89.9%)	164 (40.1%)
Fall from height	35 (7.7%)	22 (62.8%)
Others	11 (2.4%)	4 (36.4%)
Total	454 (28.3%)	190

Table 2. Age Wise Distribution of Victims

Age group	RTA	FFH	Others	Total
0 — 10	7 (4.2%)	0	0	7 (3.7%)
11 — 20	15 (9.1%)	6 (27.2%)	0	21 (11.1%)
21 — 30	52 (31.7%)	6 (27.2%)	1 (25%)	59 (31.1%)
31 — 40	37 (22.5%)	3 (13.6%)	0	40 (21.1%)
41 — 50	29 (17.6%)	1 (4.5%)	1 (25%)	18 (9.5%)
51 — 60	13 (7.9%)	4 (18.1%)	1 (25%)	18 (9.5%)
61 — 70	10 (6.0%)	1 (4.5%)	1 (25%)	12 (6.2%)
71 — 80	1 (0.6%)	1(4.5%)	0	2 (1.11%)

Table 3. Pattern of Skull Fractures in Accident Victims

Type of accidents	Linear	Depressed	Comminuted	Base	Sutural
RTA	77 (38.8%)	22 (11.1%)	55 (27.7%)	30 (15.1%)	14 (7.0%)
FFH	6 (22.2%)	13 (48.1%)	5 (18.5%)	4 (11.1%)	-
Others	0	1 (25%)	3 (75%)	1 (25%)	-
Total	83 (36.2%)	36 (15.7%)	63 (27.5%)	34 (14.8%)	14 (6.1%)

Table 4. Skull Bones Involved in Accidents Victims

Skull bones	Temporal			Parietal			Frontal	Occipital	Base of skull		
	Lt	Rt	Bl	Lt	Rt	Bl			A	M	P
RTA	48	31	10	35	29	15	38	34	7	18	5
FFH	3	4	1	3	9	0	2	5	0	4	0
Others	2	0	0	0	0	0	1	0	0	1	0
Total	53	45	11	38	37	15	41	39	7	23	5

Table 5. Intracranial Hemorrhage in Various Skull Fracture

Type of skull fracture	Intracranial hemorrhage		
	EDH	SDH	SAH
Linear	35 (42.1%)	28 (33.7%)	12 (14.4%)
Depressed	1 (25%)	20 (55.5%)	10 (27.7%)
Comminuted	0	8 (12.6%)	45 (71.42%)
Base of skull	4 (11.7%)	15 (44.1%)	17 (50%)
Sutural	6 (42.8%)	3 (21.4%)	5 (35.8%)

DISCUSSION

The results of our study on the pattern of skull fracture due to accidental deaths were analyzed and compared with other studies conducted at various places in India and abroad. Total 454 cases of death due to accidents were autopsied at Chigateri hospital mortuary during the period, July 2005 to June 2007, out of which the present study included 190 cases of skull fractures due to accidental deaths.

In the present study skull fracture were seen in majority of the victims belonged to the male population in the age group of (21-40yrs) [3,9]. The reason being that the young male (81.6%) adults are the prime bread earners for the family involved in outdoor activities where as females (18.4%) usually are home makers and stay in the house[2,4]. Maximum number of accidents occurring during morning 6 AM to 12 PM (38.4%) probably due to high rash hour traffic urgency to reach the work place and inadequate traffic control, followed by accidents in the evening 6 PM to 12 AM may be due to exhaustion, driver's fatigue, and poor vision, lack of street lights[45]. Motor cyclists (46.3%) formed because of recent increase in the availability of over designed motor vehicles. with rash and negligent driving by the younger population[10].

The maximum victims of skull fractures succumbed to death on spot and within 6 hours (24.3%). Period of survival (14%) has not shown any improvement despite advanced of medical facilities. This also attributes to delay in bringing the victim for Hospitalization[5]. In Heavy Vehicles, truck (47.5%) and buses (28.6%) being the major offenders owing to their high speed driving, congested roads, fatigue, intoxication etc [8,10].

Scalp lacerations (46.8%), followed by contusion (32.1%) are commonly associated with skull

fractures[10, 11]. Fissure/Linear fracture (38.8%) was commonest followed by comminuted fracture (27.7%)[9]. Sub arachnoid hemorrhage (39.6%) was commonly associated intracranial hemorrhage followed by subdural hemorrhage (29.3%) and extradural hemorrhage (21.1%) in RTA[3, 9].

The Linear fracture was commonly associated with extradural hemorrhage (42.1%)[9]. Depressed skull fracture was associated with SDH (50.5%) commonly found in cases of fall from height (11.7%)[9,10, 11]. In vault fractures temporal bone (48.1%) was the commonest involved. In fracture base of the skull middle cranial fossa (34%) was the commonest observed[3,12]. The patterns of other injuries associated with skull fracture are mainly thoracic injuries (31.6%) and injury to the limb (28.4%)[6, 7].

SUMMARY AND CONCLUSION

The following conclusions were drawn after a detailed study of 190 cases of skull fractures due to accidental deaths at Chigateri general hospital mortuary, Davangere. Skull fractures due to accidental deaths accounted for 41.8% of the total accidental deaths autopsied during July 2005 to June 2007. In the victims of skull fracture due to accident 86.3% were due to RTA, 11.7% due to fall from height and 2% due to other accidents. Majority of the victims were aged between 21-40 years (52.2%). Males comprised the majority of victims as compared to females in the ratio 4.2:1. Majority of the accidents occurred in the morning between 06.00 am to 12.00 pm (38.4%). Most of the victims died on spot or within 6 hours of the incident (47.4%). Motor cyclists (46.6%) were the commonest victims involved in the study cases of RTA. The commonest offending vehicles were heavy vehicles (76.1%). Lacerations (46.8%) were the commonest injury over the scalp. Linear fracture (36.2%) skull was the commonest pattern among the skull fracture. Linear fracture was commonest fracture in cases of RTA, depressed fracture was commonest fracture in fall from height. The vault fractures were common than the base fracture of the skull.

In vault fractures, temporal bone was the commonest bone involved. In fracture base of skull, middle cranial fossa fracture was common than the anterior cranial fossa and posterior cranial fossa. The commonest intracranial hemorrhage was subarachnoid hemorrhage followed by extradural hemorrhage. Extradural hemorrhage was commonly associated with linear fracture (42.1%), subdural hemorrhage in depressed fracture (55.5%) and subarachnoid hemorrhage in comminuted fracture (27.7%). Thoracic injuries were the commonest associated injuries with skull fractures.

SUGGESTIONS

- Plan communities so that living places, working places, schools and shops are within reach so that they do not have to travel long distances every day.
- Provide safe crossings and side foot paths and lanes for pedestrians and cyclists.
- Design or improve roads to separate road users going at different speeds and in different directions.
- Improve the visibility of roads, road signs, and vehicles during day and night.
- Implement graduated driver licensing system whereby drivers are permitted to drive with experienced drivers initially, until they are fully competent.
- Helmets should be made compulsory on all riders of bicycles, motorcycles and mopeds.
- Detect and respond to RTA in a timely manner with good network systems that should include emergency care, transportation, early treatment and recovery.
- Ensure resident and employees safety due to fall from height by safe boundary of roof & balcony and hand railing of stairs[7].

REFERENCES

1. Tedeschi CG. The Wound: Assessment of organs system. In : Tedeschi CG, Eckert WG, Tedeschi LG, ed., Forensic Medicine, Vol.1, Philadelphia USA, 1977: p. 29-75.
2. Tonge JI, O'Reilly MJJ, Davison A, Johnson GN, Wilkey IS. Traffic-crash fatalities (1968-73): Injury patterns and other factors. Med Sci, Law 1977; 17(1): 9-24.
3. Chandra J, Dogra TD, Dikshit PC. Pattern of Cranio-intracranial injuries in fatal vehicular accidents in Delhi, 1966-76. Med Sci Law 1979; 19(3): 186-194.
4. Shkrum MJ et al. Skull fractures in fatalities due to motor vehicle collisions. JFSCA 1994; 39 (1): 107-122.
5. Konrad CJ, Fieber TS, SCuepfer GK, Gerber HR. Are fractures of the base of the skull influenced by the mass of the protective helmet? A retrospective study in fatally injured motorcyclists. J Trauma 1996; 41(5): 854-858.
6. Hartshorne NJ, Harruff RC, Alvord EC. Fatal head injuries in ground-level falls. Am J Forensic Med Pathol 1997; 18 (3): 258-264. Hartshorne NJ, Harruff RC, Alvord EC. Fatal head injuries in ground-level falls. Am J Forensic Med Pathol 1997; 18 (3): 258-264.
7. Sanjeev L, Agnihotri AK, Ashok T, Murthy OP. Patterns of injuries in fatal falls from height-A retrospective review. JFMT 1999; 16(2): 38-45.

8. Sharma BR, Harish D, Sharma V, Vij K. Dynamics of road-traffic fatalities in Chandigarh a surprise. JFMT 2002; 19(1): 25-29.
9. Yavuz S, Asirdizer M, Cetin G, Balci YG, Altinkok M. The correlation between skull fractures and intracranial lesions due to traffic accidents. Am J Forensic Med Pathol 2003; 24: 339-345.
10. Singh H, Dhattarwal SK. Pattern and distribution of injuries in fatal road traffic accidents in Rohtak (Haryana). JIAFM 2004; 26(1): 20-24.
11. Gupta S, Roychowdhary UB, Deb PK, Moitra R, Chettri D. Demographic study of fatal craniocerebral road traffic injuries in North Bengal region. Medicolegal Update 2007; 7(1): 1-3.
12. Chavali KH, Sharma BR, Harish D, Sharma A. Head injury: The principal killer in road traffic accidents. JIAFM 2006; 28(4): 0971-0973.

Study of Morphological Changes in Endometrium in Cases of Abnormal Uterine Bleeding and its Correlation with Age, Clinical Diagnosis and Bleeding Pattern

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ABSTRACT

Excessive and irregular uterine bleeding is the most frequent problem in gynecology. Any bleeding excessive in duration, frequency, or amount for a patient is abnormal. This study aimed at studying the morphological changes in cases of pre, peri and post menopausal bleeding in cases of abnormal uterine bleeding. The strength of association between the age of the patient, clinical diagnosis and bleeding pattern with the histopathology were also studied.

Endometrium from patients complaining of abnormal uterine bleeding was studied in 141 cases. The histopathology was correlated with the age groups, clinical diagnosis and bleeding patterns. Age was found to correlate significantly with histopathology in pre and postmenopausal groups. Metrorrhagia was reported in maximum number of cases (71%). DUB was the largest clinical diagnosis group(54.6%). Overall, the most common histopathological diagnosis was normal endometrium(46.8%). Histopathology was found to correlate significantly with clinical groups, clinical diagnosis and bleeding patterns.

Key words : *Abnormal Uterine Bleeding, Endometrium, Histopathology.*

INTRODUCTION

Excessive and irregular uterine bleeding is the most frequently encountered and perplexing problem in gynecological practice. It has been stated that any bleeding that is excessive in duration, frequency, or amount for a particular patient should be considered abnormal.1 Majority of diagnostic curettages are due to this. Uterine bleeding which is not associated with any organic cause is called dysfunctional uterine bleeding (DUB). It is a diagnosis of exclusion.[2,3] Therefore DUB is generally ascribed to poorly understood derangement in the functional effect of hormones on the endometrium.4 Anovulatory cycles are probably the most common cause of DUB in women of reproductive age group followed by early decline or persistence of corpus luteum causing abnormal progesterone stimulation of endometrium. DUB in the older age group requires thorough screening for malignancy. The known organic causes of abnormal uterine bleeding include endometrial polyp, adenomyosis, leiomyomas, endometritis, atrophy, intrauterine devices, oral contraceptive use, abortion, ectopic pregnancy, hyperplasia, malignancies, gestational trophoblastic diseases, blood dyscrasias, anticoagulants, severe renal or liver diseases and hypothyroidism.

This study was undertaken with the objective of studying the morphological changes in cases of pre, peri and post menopausal bleeding in endometrial curettings and hysterectomy specimens in cases of abnormal uterine bleeding. It was further attempted to study the strength of association between the age of the patient, clinical diagnosis and bleeding pattern with the histopathology.

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MATERIAL AND METHODS

Endometrium from patients complaining of abnormal uterine bleeding was studied in 141 cases

as dilatation and curettage (D & C) material or as endometrial biopsy. In cases where hysterectomies were done as a therapeutic measure (56 cases), the endometrium was collected from the gross specimens. Detailed clinical history and relevant investigations were recorded. Paraffin blocks from study material were made following standard procedure. Routine hematoxylin and eosin stain was done in each case and special stains like PAS and reticulin were done where required. Histopathological diagnoses were duly recorded. The histopathology was correlated with the age groups, clinical diagnosis and bleeding patterns.

Seven groups were formed depending on histopathological diagnoses. These included proliferative and secretory phase endometrium, atrophic endometrium, endometritis, endometrial hyperplasia, carcinoma and endometrial polyp. Proliferative and secretory phase endometria were grouped together as normal endometrium for statistical purposes. Similarly, cases of hyperlastic endometrium and endometrial malignancies were grouped together for statistical analysis.

The cases were divided into clinical groups viz premenopausal, perimenopausal and postmenopausal groups depending on their age and menstrual history.

Seven groups were formed based on the clinical diagnoses which included carcinomas , DUB, endometritis, fibroid uterus and endometrial polyp. Cases of fibroid uterus and endometrial polyp were grouped together and all cases of malignancies were kept together.

Six groups were formed depending on the bleeding pattern and included menorrhagia, metrorrhagia, polymenorrhoea, polymenorrhagia, continuous bleeding and postmenopausal bleeding. Three major groups were formed out of these which included menorrhagia(n=58), metrorrhagia(n=71)and post menopausal bleeding(n=12). The metrorrhagia group included clubbed cases of metrorrhagia(n=26) along with polymenorrhea(n=18), polymenorrhagia(n=16) and continuous bleeding(n=11), grouped together for statistical purposes.

We intended to study the prevalence and distribution of the histopathological diagnoses among the various groups of bleeding pattern, clinical diagnoses and clinical groups. Also, it was attempted to study the strength of association of the interrelationships amongst these groups.

RESULTS

Four hundred and twenty six specimens were screened which included endometrial biopsies(n=288) and 138 hysterectomy specimens. Of these, 149(35%) presented with abnormal uterine bleeding. Amongst these, 141 were selected for detailed morphological and histopathological study. Cases of amenorrhea/ histologically confirmed pregnancy were excluded from the study group.

Of 141 cases, the endometrial tissue was obtained by curettage in 85 cases (60.2%) and from hysterectomy specimens in the rest (n=56, 39.8%).

The mean age of the patients was 29.5 ± 13.5 years with that being 56± 2.5 years in the atrophic group, 38.6± 13.3 years in endometritis group, 39.9± 7.9 years in hyperplasia group and 37.5± 9.6 years in normal endometrium group.

Age was found significantly different with respect to histopathology (p<0.001) on post hoc comparison on application of one way Anova followed by Bonferroni. It was found that age in endometritis, hyperplasia and normal endometrium group was not statistically significant and that in atrophic endometrium group was significant with p<0.001. Most of the cases (n=79, 56%) belonged to the premenopausal age group followed by the perimenopausal group (n=45, 31.9%). Only 17 cases (12.1%) were in the postmenopausal group. (Table 1)

Table 1. Comparison of clinical groups with histopathological diagnosis

		Histopathological diagnosis				
		Atrophic Endometrium	Endometritis	Hyperplasia	Normal endometrium	Total
Clinical Groups	Perimenopausal n(%)	7(43.8%)	0(0%)	17(35.4%)	21(31.8%)	45 (31.9%)
	Perimenopausal n(%)	8(50.0%)	2(18.2%)	3(6.3%)	4(6.1%)	17 (12.1%)
	Perimenopausal n(%)	1(6.3%)	9(81.8%)	28(58.3%)	41(62.1%)	79 (56.0%)
Total		16(100%)	11(100%)	48(100%)	66(100%)	141 (100%)

Out of various bleeding patterns, metrorrhagia was reported in maximum number of patients (n= 71, 50.4%); followed by menorrhagia (n= 58, 41.1%) and postmenopausal bleeding (n= 12,8.5%). (Table 2)

Table 2. Comparison of clinical diagnosis with histopathological diagnosis

		Histopathological diagnosis				
		Atrophic Endometritis	endometrium	Hyperplasia	Normal Endometrium	Total
Clinical diagnosis	DUB	11(68.8%)	0(0%)	32(66.7%)	34(51.5%)	77 (54.6%)
	Endometritis	0(0%)	11(100%)	0(0%)	0(0%)	11 (7.8%)
	Fibroid	2(12.5%)	0(0%)	15(31.3%)	29(43.9%)	46 (32.6%)
	Malignancy	3(18.8%)	0(0%)	1(2.1%)	3(4.5%)	7 (5.0%)
Total		16(100.%)	11(100.%)	48(100.%)	66(100.%)	141 (100%)

Table 3: Comparison of type of bleeding with histopathological diagnosis

	Histopathological diagnosis					
		Atrophic Endometritis	endometrium	Hyperplasia	Normal Endometrium	Total
Type of bleeding	menorrhagia	3(18.8%)	3(27.3%)	19(39.6%)	33(50.0%)	58 (41.1%)
	metrorrhagia	6(37.5%)	8(72.7%)	27(56.3%)	30(45.5%)	71 (50.4%)
	Postmenopausal bleeding	7(43.8%)	0(0%)	2(4.2%)	3(4.5%)	12 (8.5%)
Total		16(100%)	11(100%)	48(100%)	66(100%)	141 (100%)

Histopathological examination of the endometrium revealed normal endometrium in 46.8% (n=66) including proliferative endometrium in 29.7% (n= 42) , secretory endometrium in 16.9% cases(n= 24). Among the cases showing proliferative endometrium - early, mid and late proliferative phase were seen in 71.4%, 19.1% and 9.5% cases respectively. Early and late secretory endometrium were seen in 19.1% (n=4) and 80.9%(n=17) of all the cases with secretory activity.

Hyperplastic endometrium was seen in 32.6% (n=46) cases. According to the criteria described by the 'International Society of Gynaecological Pathologists', hyperplastic endometrium was further subsidivided into simple (n=28, 62.2%), compelx (n=15, 33.3%) and atypical hyperplasia(n=02, 4.4%).

The incidence of endometritis in the present study was found to be in 11 cases (7.8%). Endometrial polyp was diagnosed microscopically in 1.4% cases(n=2). Atrophic endometrium was found in 11.3% cases of the 141 cases studied.

On comparison of clinical groups with histopathology, it was found to be statistically significant overall on application of test of proportion. Pierson chi square test was applied thereafter and there was a statistically significant correlation between pre and postmenopausal groups with histopathological diagnosis(p<0.001). However the perimenopausal group did not show significant correlation.

Comparison of clinical diagnosis groups with histopathology was stastically significant with p<0.001. On further study, DUB, endometritis, fibroid groups were found to correlate with histopathology with a highly significant p<0.001. There was marginally significant correlation in the malignancy group (p 0.048).

Similarily, on comparison of bleeding pattern groups with histopathology, statistically significant correlation was seen in entirety. On intragroup comparison, the correlation of postmenopausal patients(n=12)was only group to be statistically significant(p<0.001).

DISCUSSION

Of all the patients presenting with abnormal uterine bleeding 56.0% were less than 40 years of age. Age was found significantly different with respect to histopathology(p<0.001) in this study. However, correlation of age with endometritis, hyperplasia and normal endometrium group was not statistically significant. On the other hand, comparison of age versus atrophic endometrium group was significant with p<0.001.

Solapurkar in her series of menstrual abnormalities (1084 cases) reported an incidence of 56.2% of patients under 35 years (610 cases)5 and Sanaullah Gazozai 6 observed an incidence of 56.1 below 40 years of age.

In the present study, 12.1% of cases were above 50 years of age. On comparing the clinical groups against histopathology, we found a statistically significant correlation between pre and postmenopausal groups with histopathological diagnosis(p<0.001). However the correlation of perimenopausal group was statistically insignificant.

In the data published by Sanaullah Gazozai 6, an incidence of 8% was observed. This variance may be due to the fact that she included pathological secondary amenorrhoea also in her study.

Of different varieties of bleeding patterns, metrorrhagia inclusive of metrorrhagia itself, polymenorrhoea and polymenorrhagia put together were the most prominent (n=71; 50.4%) presenting symptom, followed by menorrhagia(n=58; 41.1%). The comparison of bleeding pattern groups with histopathology was statistically significant (p<0.001).Other workers have found menorrhagia to be the most common pattern of beeding7. However, a low incidence of menorrhagia ranging from 26.3% to 25.9% has been found in other studies.[5,8]

Cases where no obvious gross pathology was noted were labelled as DUB by the gynecologists and this was found to be the largest group (54.6%) as regards the clinical diagnosis is concerned. Comparison of clinical diagnosis groups with histopathology was highly significant in the present study in cases of DUB, endometritis and fibroid groups . The malignancy group showed a marginally significant correlation too. The incidence of DUB reported by other authors ranges from 24.1% to 44.1%.[5,8,9]

Fibroid was found in 32.6% of cases in the present study. Reported incidence of fibroid in the literature ranges from 46%- 48% in in perimenopausal women and cases of menorrhagia respectively.[10,11]

In 42.4% cases, normal endometrium was reported with proliferative endometrium in 29.7% and secretory in 12.7% cases. The same has been reported between 49.6% to 74% in the published literature.[6,8,9,12]

Hyperplastic endometrium was observed in 32.6% of cases in our study, which is in close agreement with that reported in the literature.5,8,10 However, Gazozai reported the incidence of hyperplastic endometrium in 11% cases only.[6] Among all the cases of hyperplasia, simple hyperplasia was found in 62.2% cases. Tariq (2004) also showed similar incidence of simple hyperplasia as 62.9%.[13]

We found endometritis in 11 cases(5.6%). Out of these, granulomatous endometritis was seen in four cases. Granulomatous endometritis has been found to be tubercular in most cases in the published literature.[14] Though abnormal uterine bleeding is not the common mode of presentation of TB endometritis, its incidence has ranged from 0.6% to 1.2%.[5,9,15,16]

We had three cases of endometrial polyp(2.1%). The reported incidence is from 1.2% to 8%.[5,9,13]

Atrophic endometrium was reported in 16 cases (11.3%) in the present study, out of which, 41.7% cases were in postmenopausal women. Tariq (2007) found 27% of cases of atrophic endometrium in postmenopausal bleeding patients.[13]

CONCLUSIONS

In this study, patients presenting with abnormal uterine bleeding were less than 40 years of age(premenopausal) in 56.0% cases. Age was found to correlate significantly with histopathology in pre and postmenopausal groups. Metrorrhagia was reported in maximum number of cases (71%). DUB comprised the largest group with respect to clinical diagnosis(54.6%). Overall, the most common histopathological diagnosis was normal endometrium(46.8%). Histopathology was found to correlate significantly with clinical groups, clinical diagnosis and bleeding patterns.

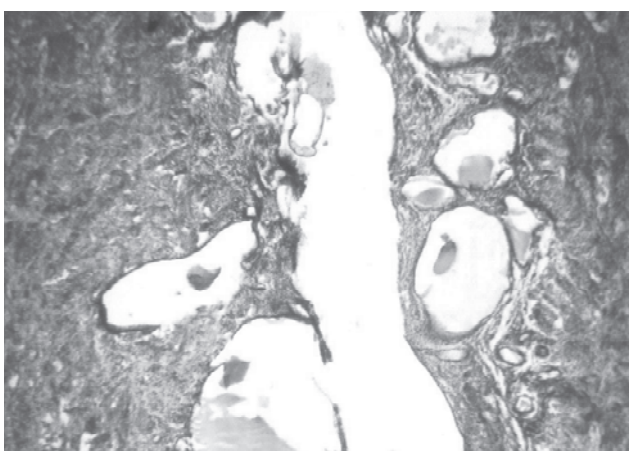

Fig. 1. Atrophic endometrium, H&E, X100

REFERENCES

1. Wentz A.C. Abnormal uterine bleeding. In: Jones, H.W., Wentz, A.C., and Burnett, L.S. (Eds.)

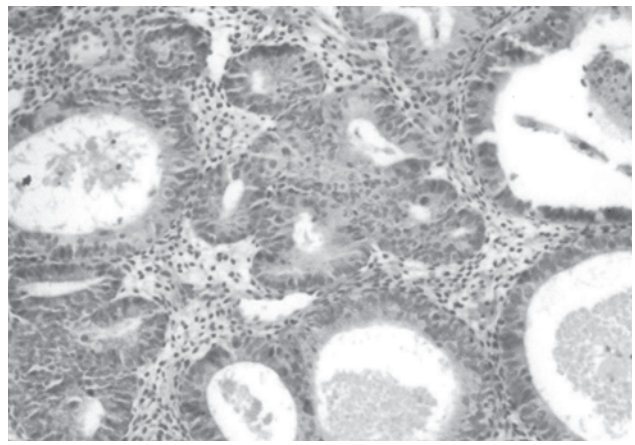

Fig. 2. Endometrium carcinoma, H&E, X400

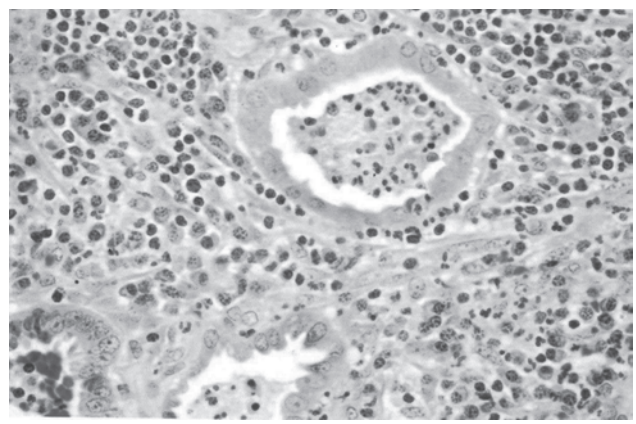

Fig. 3. Chronic endometritis, H&E, X400

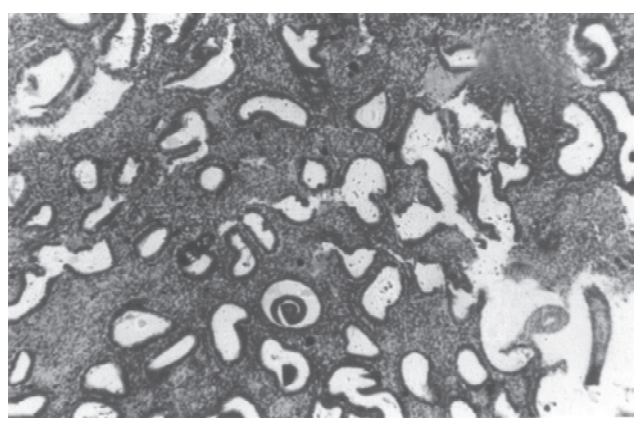

Fig. 4. Complex hyperplasia, H&E, X100

Novak's textbook of Gynecology, 11 ed. Baltimore, Williams and Wilkins, 378.
2. Aksel S,Jones GS. Etiology and treatment of dysfunctional uterine bleeding.Obstet Gynecol 44: 1.
3. Beer AE. Differential diagnosis and clinical analysis of dysfunctional uterine bleeding.Clin Obstet Gynecol 13: 434.
4. Speroff L, Glass RH, Kase NG. Clinical gynecological endocrinology and infertility, 3rd Ed .Williams & wilkins,Baltimore, 467-492.
5. Solapurkar, M.U. Endometrial spectra in women at different ages. J. Obstet. Gynaec. India 36 (1): 139.
6. Gazozai S, Bugti Q A, Siddiqua A and Ehsan N. Excessive uterine haemorrhage - A histopathological study. Gomal Journal of Medical Sciences. 2004; 2: 13-15.
7. Moghal M. Diagnostic value of endometrial currettage in abnormal uterine bleeding. A histopathological Study. JPMA, 1997; 47 (12): 295-299.
8. Pinto Rosario, Y. Dysfunctional uterine bleeding in the post menopausal age (A review of 137 cases). J. Obstet. Gynaec. India 19 (5) : 606.
9. Patra, D.C. and Giri, A.Kf . Clinicopathological studied of menorrhagia. Journal of Indian Medical Association 81 (10): 344.
10. Sarfraz T, Tariq H. Histopahological finding in menorrhogia a study of 100 hysterectomy specimens, Pal J Pathol 2005; 16 (3): 83-85.
11. Bhosle A, Fonseca M. Evaluation and histopathological correlation of abnormal uterine bleeding in perimenopausal women. Bombay Hospital Journal. 2010; 52: 69-72.
12. Nayak, S.R., Vaitjya, R.R. and Thakur, S.S. Geriatric problem : Abnormal bleeding per vaginam after the age of 40. J. Obstet, Qynaecol India 2 6: 585.
13. Sarfraz T, Tariq H. Histopahological finding in menorrhogia a study of 100 hysterectomy specimens, Pal J Pathol 2005; 16 (3): 83-85.
14. Novak, E.R, and Woodruff, J,D. Histology of the endometrium. In: Novak's Gynecologic and Obstetric Pathology 8th ed,, Philadelphia, W.B. Saunder's Co., p. 171, 250.
15. Sagar S. Clinicopathological correlation of abnormal uterine bleeding at the age of 45 years and above. J. Obstet. Gynaecol India. 1980; 30: 165.
16. Tyagi S.P., Ashraf, N.I., Abbasi, N., Prasad, M., and 'Mohsln, S. Endometrial tuberculosis (A histopathological study of 100 cases) J. Obstet. Gynaecol India. 1977; 27: 935.

Sewer Gas Poisoning: A Case Report

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ABSTRACT

Two cases of toxicity from exposure to sewer gas are presented here. Both the patient had exposed suddenly to the sewer gas with effect of loss of consciousness and respiratory paralysis leading to asphyxia and death.

The literature and those two case reports support various modalities of precautionary measures that have to be implemented to avoid deadly outcome from the exposure of these gases to the employees and workers involved in sewage work.

Key words : *Sewer Gas, Loss of Consciousness, Asphyxia.*

INTRODUCTION

Sewer gas is a complex mixture of toxic and non-toxic gases that can be present at varying levels depending upon the source. It is formed during the decay of household and industrial waste.[1] The principal constituents are nitrogen, methane, ammonia, carbon dioxide, carbon monoxide and hydrogen sulphide. The most dangerous of all these is hydrogen sulphide which acts by reversible inhibition of the respiratory enzyme cytochrome oxidase similar to that of cyanide poisoning. Concentrations above 200 parts per million (ppm) produce direct irritant effects on exposed mucosal surfaces, and pulmonary oedema on prolonged inhalation. Higher levels (above 500 ppm) paralyze the respiratory centre, and almost instantaneous loss of consciousness. The familiar smell of 'rotten eggs' is not a reliable warning sign as paralysis of the olfactory nerve makes the gas odorless at lethal levels.[1-5]. Carbon dioxide and methane have a saturated gas density approximately 1.5 and 0.6 times that of air, respectively with little or no characteristics odor. Methane is extremely flammable, has a wide explosive range, and a low flash point which may result in a substantial fire and explosion hazard. It is also possible to have other flammable gases in sewers that originate from spills and leaks of flammable liquids. Ammonia has a distinct, strong odor with good warning characteristics which are present well before attaining toxic levels. It is unlikely that acutely toxic levels of this material would be present from common sewage reactions.[1] Most reported cases have been associated with agitation of slurry prior to, or during sewer cleaning procedures.[2] All of the above gases are colorless at the concentrations commonly encountered in sewage systems.[1]

Sanitary and farm workers can be exposed to sewer gas during the cleaning and maintenance of municipal sewers, manure storage tanks, and home septic tanks. If unnoticed while working in such conditions deadly levels of sewer gas can cause instantaneous loss of consciousness and death of that worker.[4,6] Early recognition and detection is crucial to protect employees from deadly exposures. Employees working in areas that contain or have the potential to contain sewer gas should learn to recognize the signs and symptoms, how to monitor for its presence above dangerous level, and know how to take measures to protect themselves. Availability of efficient and functioning Instrumentation is mandatory to continuously monitor the atmosphere in confined spaces for sewer gas, its components and other gases as well as oxygen deficiencies.

MATERIAL AND METHODS

We report two cases of the death by asphyxia due to sewer gas poisoning which were brought for post-mortem examination at Department of Forensic Medicine and Toxicology at Indira Gandhi Government Medical College, Nagpur.

Case No 1: A 32 year old male was brought dead to Casualty IGGMC, Nagpur with history of

unconsciousness while cleaning manhole. As per the history he was apparently healthy before incidence. The autopsy was conducted in Dept of Forensic Medicine at this institute. On external examination clothes were wet, stained with mud and weed seeds of brinjal. Rigor mortis was well marked in the whole body; post mortem lividity was present over back and buttocks, fixed and dark. There was oozing of reddish fluid from mouth and nostrils, with bluish discoloration of fingertips and nails. On internal examination there was mud mixed with brinjal seeds in larynx, trachea and bronchi. Both lungs showed signs of drowning. Petechial hemorrhages were present on the visceral pleura. Heart contained dark red, fluid blood. All organs were congested. Viscera were preserved for chemical analysis which did not revealed any poison while qualitative analysis of blood test results detected carbon monoxide. The opinion as to the cause of death was given as Asphyxia due inhalation of sewer gases associated with drowning.

Case No 2: A 40 year old male was brought dead with history of sudden loss of consciousness and fall in to the septic tank. He along with his younger brother was removing the sewage from the septic tank with the help of electrically driven water pump at his home. In half way of his work he went near the opened part of the tank to observe how much of the tank was empted. All of sudden he lost his consciousness and fell into the tank. His brother rushed to help him near the tank but he felt strong odour of rotten eggs and dizziness, because of which he ran away from the site and was admitted to hospital where he recovered completely.

The autopsy was conducted at Department of Forensic Medicine, at this institute. On external examination clothes and skin were moist and soiled with fecal matter. There was smell of rotten eggs nearby the body. The rigor mortis was well marked in the whole body; post mortem lividity was present over back and buttocks, fixed. Brownish frothy mucous was oozing out from nostrils. Bluish discoloration was present on the fingers and fingernails of both the hands. There were 4 small abrasions on the body and no other injury or fracture. On internal examination brain was congested and oedematous. Mud and fecal matter was present in the larynx and trachea extending up to the terminal bronchioles. Both lungs were showing signs of drowning. Heart was congested with dark red fluid blood. All other organs were congested.

Viscera were preserved for chemical analysis which did not revealed any poison while blood test results were negative for carbon monoxide.

The histopathology examination of various organ revealed oedema and congestion in the brain, congestion in the heart, congestion; oedema and vegetable matter in the alveoli of the lungs, congestion in the spleen, liver and pancreas while congestion and hemorrhage was present in the kidneys. The opinion as to the cause of death was given as Asphyxia due to sewer gas poisoning associated with drowning.

DISCUSSION

In the first case narrated above the person was working for municipal corporation sewage cleaning department. As a routine he went for the sewage cleaning at evening hour on that eventful day along with his coworker. He had checked the toxic gases level by burning candle. As the candle was burning in the manhole and it was kept open for considerable time according to his satisfaction, he was confident of safety. His coworker was standing by the manhole. But as the sufferer entered in the manhole at first he was not having any problem and his coworker was also satisfied. But after sometime as the person was removing the block in the sewage the gases trapped under the crust might have get liberated in the vicinity. The workers neglected the warning smell. After some time as the level of sewer gas went on increasing the person felt suddenly unconscious and get drowned in the sewage water. The water level of the sewer pipe at that time was very less so that it was impossible for any conscious person to get drowned in it. Even if the victim was alive when he succumbed to toxic level of the gases he could not save himself due to loss of consciousness. The fellow coworker tried to the best of his level to rescue the victim but the victim was very heavily built and he could not be rescued to the surface. Lack of preplan, work without self breathing apparatus, misadventure of the worker and absence of emergency equipment ended in failure of rescue operation.

In the second case same thing did occurred. The elder brother ignorant and overconfident of the complications of working in sewage started the electric pump and lifted some of the sewage from the pit. Due to overconfidence of working he did not take the help of trained personnel. In between the sewer gases get accumulated in the vacant space of the tank due to disruption of the crust. When the person leaned over the edge of the tank to watch how much of the sewage is removed he suddenly exposed himself to very high level of poisonous gases and lost his consciousness. Once he was in the pit he got drowned in the sewage. His brother tried to rescue him but due to good fortune he could rush himself to safe place once he noticed rotten smell and dizziness.

Circumstances in both cases are same. The history of constant presence of smell suggests that both the workers were playing with danger without proper precautionary and safety measures. Although quantitative and qualitative analysis of the sewer gases at the scene was not done in these cases the clinical picture and history are consistent with the inhalation of high concentrations of sewer gases, particularly hydrogen sulfide leading to loss of consciousness in both the cases.

Moreover Micheal A et. al.[5] states that the diagnosis of hydrogen sulfide which is one of the dangerous components of sewer gas is primarily clinical. Eve though the measurements of thiosulfate levels in the blood, tissues and urine can be used to confirm the hydrogen sulfide exposure while the environmental monitoring is the best likely source for real time analytical confirmation.

Due to insufficient electron accepting ions (anaerobic conditions) the biochemical reaction taking place in the sediment of sewer various gases are produced[3]. Highly toxic components of sewer gas include hydrogen sulfide, ammonia, methane and carbon dioxide. The most dangerous of these is hydrogen sulfide which acts by reversible inhibition of respiratory enzyme cytochrome oxidase[1, 2, 5, 7].

Hydrogen sulfide is a colourless gas with a characteristic odour of 'rotten eggs'. Acute massive exposure (above 1000ppm) of this gas causes sudden unconsciousness and immediate death. It is primarily cellular toxin. It interacts with cytochrome c oxidase, disrupting mitochondrial metabolism and slows oxidative phosphorylation producing cellular hypoxia and ultimately leading to central neurological and respiratory failure. Micheal A et. al.[5] quotes that in acute poisoning, several case reports examine the clinical syndrome of immediate "knockdown," sometimes also termed the "slaughterhouse sledgehammer". Death occurs primarily as a result of central respiratory arrest[2, 5, 7, 8, 9]. Heikki E et. al.[7] quotes an Interesting phenomenon known as "olfactory extinction" which describes the loss of perception of the odor as the level increases. This is thought to be caused by paralysis of the olfactory nerve[5,7,10] so that the individual can not perceive the smell even if he is working in environment having very dangerous levels of the gas. This explains the sudden loss of consciousness of the person working near sewer pit in our case 2.

The use of the traditional methods for accessing the level of toxic gases in the sewage system can cause much dreaded complication or death. The reliability on the smell, burning candle, keeping the manhole open for some time before actual work, working without primary aid or breathing apparatus, working without preplan are few dangerous deeds that the sewage workers do in day to day life. But sometimes this leads to loss of valuable life[11].

CONCLUSION

1) Sewer gas poisoning is one of the important occupational health related hazard that is usually neglected.
2) Without any warning signs the person can loose the consciousness and fatal accidents can occur.
3) Working without respiratory apparatus is call to death in confined sewer.
4) Advanced method of air monitoring is necessity of sewage work.
5) Trained staff must always be availed whenever necessary.
6) Proper preplan of the work should be formulated before starting any work in sewage system.
7) Blood, tissues and urine should be preserved for detection of thiosulfate in suspected cases of sewer gas poisoning.

From the above two cases and discussion we recommend the following

- Be prepared with the working plan before actual work starts.
- Be aware of the hazardous gases likely to be present in the drainage system.
- Ensure that all the necessary safety precautions recommended have been properly implemented and all the machinery are in are in working condition.
- Do not rely on the traditional methods for detection of level of gases in sewage system.
- Always use respiratory & or self breathing apparatus whenever necessary.
- Do not work singly whenever entry into a manhole or drainage system is required.
- Assign a suitable standby person outside the manhole to maintain constant communication with the workers inside and provide necessary assistance.
- Always take assistance of the trained working staff whenever necessary to clean the domestic sewage tank.
- Keep monitoring the workspace and surrounding areas and be alert to dangerous conditions.
- Immediately evacuate from the drainage system if there are signs indicating that the safety and health of workers may come under threat.
- Call for emergency assistance immediately and implement the emergency plan in case of an accident.

- Do not enter the manhole to rescue the collapsed victims without any self breathing respirator and support from other rescuers.
- Do not give mouth to mouth respiration to the victim as gas poisoning can occur through breath to the rescuer.

Conflict of Interest : Nil

REFERENCES

1. Gary M Hutter, Reference data sheet on sewer gases, Meridian Engineering and Techanology, November 1993, http://www.ehs.iastate.edu/forms/sewergases.pdf (accessed May 15, 2011)
2. M Spires, O C Finnegan , Near death due to inhalation of slurry tank gases, The Ulster Medical Journal, October 1986, Vol 55 (2), pp. 181-183.
3. Jihyoung Kim, Jung Soo Lim, Jonathan Friedman, et al, Sewer Snort: A Drifting Sensor for In-situ Sewer Gas Monitoring, Proceedings of the 6th Annual IEEE communications society conference on Sensor, Mesh and Ad Hoc Communications and Networks 2009, Rome, Italy, June 22-26, 2009. http://mslab.kaist.ac.kr/wikipages/files/adhoc2011. pdf (accessed May 15, 2011).
4. U.S. Department of Labor, Occupational Safety and Health Administration Fact Sheet, October 2005, http://www.osha.gov/OshDoc/data_Hurricane_Facts/hydrogen_sulfide_fact.pdf (accessed May 15, 2011).
5. Micheal A. Policastro, Edwad J. Otten, Case Files of University of Cincinnati Fellowship in Medical Toxicology: Two Patients with Acute Lethal Occupational Exposure to Hydrogen Sulfide , Journal of Medical Toxicology, June 2007, Volume 3, Number 2.
6. Rajnarayan R. Tiwari, Occupational health hazards in sewage and sanitary workers, Indian Journal of Occupational and Environmental Medicine, December 2008, volume 12, issue 3, 112-115.
7. Heikki E. Nikkanen, Michele M. Burns, Severe Hydrogen Sulfide Exposure in a Working Adolescent, Pediatrics, 2004, volume 113, pp 927-929.
8. F. Dean Kemper, A Near Fatal Case of Hydrogen Sulfide Poisoning, Cannad. Med. Ass. J, May 21, 1966, Volume 94, pp 1130-1131.
9. Stephen B. Hooset, Villiam Van Alstine, Matti Kiupel, et al, Acute pit gas (hydrogen sulfide) poisoning in confinement cattle, J Vet Dragn Invest, 2000, volume 12, pp 272- 275.
10. Bassam Doujaiji, Jaffar A Al-Tawfiq, Hydrogen sulfide exposure in adult male, Annals of Saudi Medicine, 2010, Volume 30, Issue 1, pp 76-80.
11. Labour department, The Government of Hong Kong Special Administration Region Booklet on Prevention of Gas Poisoning in Drainage Work, December 2007. http://www.labour.gov.hk/eng/public/oh/Drainage.pdf (accessed May 15, 2011).
12. Times News Network, NMC cleaner enters manhole, dies, The Times of India, Nagpur, Tuesday, August 19, 2008 pg- 5.

Estimation of Stature from Head Height in Hyderabad

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ABSTRACT

The present study is mainly meant at estimation of stature from Head Height in a cross section of population in Hyderabad city. The height of head was measured between vertex and tragion. Rod compass with head-height needle was used to measure head height. Height of the subject was measured with standard Anthropometer with subject in anatomical position. Measurements were taken at fixed time to avoid diurnal variation. The results obtained were analysed and attempt was made to derive a formula between head height and total height of an individual. The study shows that there is a definite correlation between head height and height of an individual. In spite of the racial and ethnical variation, this formula may be applicable to other regions and races, more or less effectively. Looking at the scarcity of work done, between head height and height, attempt by workers will be more useful in anthropometry and in forensic medicine.

Key words : *Anthropometry, Forensic Anthroplogy, Correlation, Estimation of Stature, Height, Head Height.*

INTRODUCTION

Forensic Anthropology is the application of anthropology's specialized knowledge of human sex, race, age, stature and individual variations to medico-legal cases. It has a varied array of means and methods to identify the culprit as well as the victims in criminal cases. The stature or body height is one of the most important and useful anthropometric parameters which determines the physical identity of an individual[1,2].

Therefore, determination of stature from different body parts and skeletal remains has obvious importance in the identification of the criminals and victims. In cases where the evidences are skeletal remains, forensic anthropology has put forward means to estimate the stature from these skeletal and even fragmentary bones.

Many studies have proven beyond doubt that there is positive correlation between stature and length of long bones. In a similar approach, many workers have demonstrated positive correlation between stature and other body dimensions. However, no data is available in the literature regarding the estimation of stature from the different measurements of head, except Saxena et al (1981), who derived a regression equation between head-length and height in Agra population (UP)[3] and Jadav and Shah who did a study on 727 medical students in Gujrat (2004)[4].

Therefore, the present work attempts to find out a correlation and to derive a regression formula between head Height and body height in a cross section of the population of Hyderabad city.

MATERIALS AND METHODS

A total of hundred volunteers, gathered from all walks of life, were selected for the project and the ages of subjects ranged from 20 to 60 years. This range of age was chosen as it nullifies the changes caused by growth in younger age group as well as the loss of stature seen in older age due to bony changes. The measurements were taken during a fixed time i.e. between 2 to 5 p.m. to avoid effects of diurnal variations. Although the subjects were selected from different socio-economic backgrounds, care was taken not to include obviously malnourished or obese individuals and persons with physical abnormalities.

The measurements for stature were taken using a standard Anthropometer with the subject in the anatomical position. Head height is taken as the projective distance between the vertex and the tragion. A Rod compass with a Head Height needle was used to measure the head height[1].

RESULTS

Table I. Different parameters studied

PARAMETERS	AGE (years)	HEIGHT (cms)	HEAD HEIGHT (cms)
RANGE	20-60	141.5-177.1	9.7-13.8
MEAN	32.5	162.5	12.02
Standard Deviation	11.96	7.67	0.81

Table I gives the details about the different parameters gathered in the present study. The age ranged between 20-60 years in the group. The height varied from 141.5 to 177.1 cms having a mean of 162.5 cms and standard deviation of 7.67 cms. The head height was in the range of 9.7-13.8 with a standard deviation of 0.81.

Table II. Coefficient of Correlation between height and head height

PARAMETER	COEFFICIENT OF CORRELATION
General population	0.63
Males	0.56
Females	0.688

Using linear regression analysis, a constant (a) and a regression coefficient (b) were estimated with height as the dependent variable and head height as explanatory or independent variable.

Stature can be estimated using the formula[5]:

Ht = (a) + (b) x Hh

Where

Ht = total height or stature

Hh= head height

(a)= constant

(b)= regression coefficient

The coefficient of correlation, denoted by r, is defined as a measure of the degree of relationship between two variables x and y. the value of r lies between -1 and +1.[5]

r = +1 shows a perfect positive correlation.

r = -1 shows a perfect negative correlation.

r = 0 shows an absence of correlation.

DISCUSSION

It can be observed from the tables and statistics that the independent or explanatory variables are strongly related to the dependent variable.

In the present study both male and female subjects were considered separately. The relationship is similar in both sexes as can be observed from the tables. The confidence level taken is 95% with standard error of up-to ± 5 cms.

Significant correlation between height and different parts of the body has been shown by various workers like Singh and Sohal (1951) on height and length of clavicle[6], Charnalia (1961) on height and foot-length[7], Athawale (1963) on height and forearm bones[8]. Shroff and Vare (1979) derived the height from the length of superior extremity and its segments[9].

Regarding estimation of stature from various measurements of head, only two references are available. One of Saxena et al (1981) who derived a regression equation between head length and height in Agra population (3) and the other of Jadav HR and Shah GV (2004) who derived a correlation between head length and height among medical students in Gujarat [(4)].

No study had been done on relationship between height and head height, hence the present study.

Using the regression equation:

Height in general population= 88.37+6.17x head height

In males, Height= 106.64+4.9x head height

In females, Height= 95.32+5.22x head height

The percentage of head height to stature in general population is 7.4%

In males percentage is 7.3%

In females percentage is 7.5%

The percentage method is useful for rough estimation of height but the formulae outlined above are simpler, practical and give accurate results and therefore can be used in day-to-day forensic investigations.

Table II shows the coefficient of correlation between height and head height in general population as 0.63, where as in males it is 0.56. It is highest in females at 0.688.

The present study is a non-invasive, non-time-consuming and non-expensive method, which does not need any specialized training, hence can be used by anyone.

Out of the various parameters available for stature estimation, this appears to be equally accurate, less

tedious and less cumbersome. The availability of the head or part of the head or even dry skull is enough to assess the stature of the individual from the formula derived in this study.

However, as these formulae have been derived from a limited number of individuals, a more extensive survey with a larger sample size is essential to prove their accuracy.

Conflict of Interest : Nil

REFERENCES

1. Prof. B. M. Das and Dr. Rajan Deka. PHYSICAL ANTHROPOLOGY PRACTICAL, 2003; 15-26.
2. M. F. Ashley Montagu. AN INTRODUCTION TO PHYSICAL ANTHROPOLOGY, 3rd edition, 1960; 555-572.
3. Saxena SK, Jeyasingh P, Gupta AK and Gupta CD. The estimation of stature from head - length. Journal of anatomical society of India 1981; 30: 78-79.
4. Jadav HR and Shah GV. Estimation of stature from head length. Journal of anatomical society of India 2004; 54: 20-21.
5. Dr J. V. Dixit. Principles and practice of Biostatistics. Fourth edition 2009. Bhanot Publishers, Jabalpur.
6. Singh B and Sohal HS. Estimation of stature form Clavicle in Punjabis; A preliminary report. Indian Journal of Medical Research 1951; 40: 67-71.
7. Charnalia VM. Anthropological study of foot and its relationship to stature in different castes and tribes of Pondicherry state. Journal of Anatomical Society of India 1961; 10: 26-30.
8. Athawale MC. Anthropological study of height from length of forearm bones. A study of one hundred Maharashrtrian male adults of ages between twenty five and thirty. American Journal of Physical Anthropology 1963; 21: 105-112.
9. Shroff AG and Vare AM. Determinatin of height from length of superior extremity and its segments. Journal of Anatomical Society of India 1979; 28: 53.
10. Colin Wilson. Bertillon discovers anthropometry. CLUES! A history of Forensic Detection, Warner Books Edition 1989; 97-118.
11. Brian Lane. The Encyclopaedia of Forensic Science 2004.

Credibility of Pubic Length, Ischium Length and Ischio-pubic Index in Identification of Gender

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ABSTRACT

Identification of the sex from the skeletal remains is the prime work of forensic experts. The present study is carried out to determine the sex of the hip bone from ischium length, pubic length and Ischio-Pubic Index. One hundred adult human hip bones of known sex (50 male and 50 female) available in the Department of Anatomy and Forensic Medicine & Toxicology, M.R. Medical College & K.B.N. Institute of Medical Sciences, Gulbarga are used for the present study. Among all the three variables statistically analyzed, the most reliable parameter, is ischio-pubic index, 86% of male hip bones and 82% female hip bones, i.e., overall 84% of hip bones can be sorted out by this parameter alone. This parameter is found to be statistically significant. With respect to the length of Ischium, the percentage of hip bones identified by demarking point alone is 16% of male and 38% of female. This parameter also found to be statistically significant.

Key words : *Pubic Length, Ischium Length, Ischio-Pubic Index and Gender.*

INTRODUCTION

Identification of sex of the deceased from skeletal remains is in practice since long ago, as it becomes important expert evidence in the court of law. In medicolegal cases forensic experts are often asked for their expert opinion regarding species, age, sex, race, stature and probable cause of death from skeletal remains. In Medicolegal cases bones form important evidence in establishing identity of deceased. The determination of deceased sex is prime step in skeletal analysis.

The hip bone alone can show sexual differentiating criteria since fetal life. Though the primary function of hip bone is locomotor and withstanding the compressive and other stresses due to weight, it also shows characteristic sexual differences. The innominate or hip bone or pelvic bone is formed from three bones which are fused in an Yshaped epiphysis involving the acetabulum. These bones include: Pubic, Ischium and Ilium[1]. Measurements of these bones and determination of related indices and angles have been very useful in forensic science, clinical practice, (most especially Obstetrics and Gynaecology) and physical Anthropology[2,5].

Different workers have done sex differentiation studies of hip bone in different population, some of these are: Washburn (1948) in American Whites and Negroes, and in Bantu and Bushman (1949), Hann and Washburn (1953) in Eskimos, Davivong (1963) in Australian Aborigines, Day and Pitches Willmot (1975) in English population, Shamersingh and P.B.Raju (1977; 78; 79) in Varanasi zone, Scidier (1980) in Australians, Jankiram S and Thomas J.M. (1991) in population from Karnataka.

Results of these studies have shown wide range of variations in standards of morphological and morphometric sex differences. These variations occur because the skeletal growth is mostly influenced by factors like race, heredity, climate, environment, nutrition, etc., which are likely to change from region to region, hence, it is essential to carry out such studies in different regions.

MATERIALS AND METHODS

One hundred adult human hip bones of known sex available in the Department of Anatomy and Forensic Medicine & Toxicology, M.R. Medical College & K.B.N.

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Institute of Medical Sciences, Gulbarga are used for the present study.

Out of 100 hip bones, 50 are of males and 50 are of females. All the hip bones are dry, free of damage or deformity and were completely ossified.

Following instruments are used for the measurements of various parameters of hip bone:

1) Sliding vernier caliper
2) Compass
3) Triflanged stainless steel caliper.
4) Chalk,
5) Marker pencil

Sufficient care is taken to avoid manual error and all measurements are taken personally.

From each hip bone following parameters are measured and indices are obtained in the manner described below:

1. Length of pubis: It is the maximum distance between the morphological center of acetabulum and upper margin of pubic symphysis measured with sliding caliper (Fig. 2).
2. Length of ischium: It is the maximum distance between morphological center of acetabulum and deepest point on the ischial tuberosity measured with sliding caliper (Fig. 2).

Morphological Center

This is the central point of the acetabulum which lies approximately at the intersection of the inner edge of the articular surface of the acetabulum with a straight line prolonging the lower part of acetabular border of the ilium downward. This is the point where the three main pelvic elements, the ilium, ischium and pubis meet. It is better seen before union of these elements. This point can be identified approximately in the adult because: (1) Frequently there is an irregularity, both in the acetablum and inside the pelvis; (ii) there is a change in thickness which may be seen by holding the hipbone upto a light and (iii) often there is a notch in the border of the articular surface in the acetabulum.

3. Ischio-pubic index:

$$\text{Ischio-pubic index} = \frac{\text{Length of pubis}}{\text{Length of ischium}} \times 100$$

The values of range, mean, standard deviation, calculated range (mean±3SD), Demarking points and Identification points were obtained. For each parameters with male range of a-b and female range of c-d, values 'a' and 'd' were the identification points (I.P) for females and males respectively. Any hip parameter reading less than 'a' was regarded as female hip bone and greater than 'd' was regarded as male hip bone and in case where female range is more than male then 'b' and 'c' were identification points (I.P) for female and male respectively. Similarly Demarking Points (D.P) was calculated from calculated range i.e., Mean ± 3 S.D (a = minimum value in male range, b = maximum value in male range, c= minimum value in female range and d=maximum value in female range). Subsequently 't' test was applied.

Observations

One hundred hip bones (50 males and 50 females) are studied in the present study. In all the hip bones length of Pubis, length of Ischium and Ischio-pubic index is measured. As the first part of study parameter is analyzed statistically and mean, standard deviation, range, calculated range (mean±3 SD), demarking points (DP) and identification point (IP) are obtained. Percentage of the hip bones identified by demarking point, percentage of hip bones identified by identification point is calculated. Then't' test is applied.

Comparative bar diagram of male and female values are drawn.

Explanation of Table-1

Table 1. Statistical analysis of the length of pubis

Sl. No.	Particular	Male (mm)	Female (mm)
1.	Number of bones	50	50
2.	Range	68-78	67-87
3.	Mean	73.96	75.4
4.	Standard deviation (SD)	2.36	5.71
5.	Statistical Significance	InSig	InSig
6.	Identification Point	<67	>78
7.	Percentage of identified bones	0	30%
8.	Calculated range (mean±3SD)	66.89-81.03	58.26-92.54
9.	Demarking point	<58.26	>81.03
10.	Percentage beyond Demarking Point	0%	20%
11.	Standard Error	0.33	0.81

't'test=1.65; p>0.05 *statistically Insignificant

The mean length of pubis of male hip bone is 73.96 mm ranging between 68-78 mm. The mean length of pubis of female hip bone is 75.4 mm with the values ranging between 67-87 mm. The S.D. for male and female are 2.36 & 5.71 respectively. The range of mean ± 3 S.D. in males and females is 66.89-81.03 mm & 58.26-92.54 mm respectively. Identification point for male is <67 mm & for female it is less than >78mm. The demarking point for males is <58.26 mm and for

females it is >81.03 mm, and the percentage of hip bones identified by demarking point alone is 0% of male and 20% of female. 't' test is Insignificant with P>0.05.

Table 2. Statistical analysis of the length of ischium

Sl. No.	Particular	Male (mm)	Female (mm)
1.	Number of bones	50	50
2.	Range	74-92	62-80
3.	Mean	82.92	71.54
4.	Standard deviation (SD)	4.06	5.15
5.	Statistical Significance	Sig	Sig
6.	Identification Point	>81	<74
7.	Percentage of identified bones	58%	72%
8.	Calculated range (mean±3SD)	70.74-95.1	56.1-86.98
9.	Demarking point	>86.98	<70.74
10.	Percentage beyond Demarking Point	16%	38%
11.	Standard Error	0.57	0.73

't'test=12.27; p<0.001; *statistically highly significant

Explanation of Table 2

The mean length of ischium of male hip bone is 82.92 mm ranging between 74-92 mm. The mean length of ischium of female hip bone is 71.54 mm with the values ranging between 62 - 80 mm. The S.D. for male and female are 4.06 & 5.15 respectively. The range of mean ± 3 S.D. in males and females is 70.74-95.1 mm & 56.1-86.98 mm respectively. Identification point for male is >81 mm & for female it is less than <74 mm. The demarking point for males is >86.98 mm and for females it is <70.74 mm, and the percentage of hip bones identified by demarking point alone is 16% of male and 38% of female. 't' test is highly significant with P<0.001.

Table 3. Statistical analysis of the Ischio-Pubic index

Sl. No.	Particular	Male	Female
1.	Number of bones	50	50
2.	Range	78.65-97	95.71-111.4
3.	Mean	89.39	105.44
4.	Standard deviation (SD)	4.17	3.94
5.	Statistical Significance	Sig	Sig
6.	Identification Point	<95.71	>97
7.	Percentage of identified bones	98%	98%
8.	Calculated range (mean±3SD)	76.89-101.9	93.62-117.3
9.	Demarking point	<93.62	>101.9
10.	Percentage beyond Demarking Point	86%	82%
11.	Standard Error	0.59	0.56

't'test=19.78; p<0.001; *statistically highly significant

Explanation of Table-3

The mean value of ischio-pubic index of male hip bone is 89.39 ranging between 78.65-97.0. The mean value of ischio-pubic index of female hip bone is 105.44 with the values ranging between 95.71-111.4. The S.D. for male and female are 4.17 & 3.94 respectively. The range of mean ± 3 S.D. in males and females is 76.89-101.9 & 93.62-117.3 respectively. Identification point for male is <95.71 & for female it is >97. The demarking point for males is <93.62 and for females it is >101.90, and the percentage of hip bones identified by demarking point alone is 86% of male and 82% of female. 't' test is highly significant with P<0.001.

DISCUSSION

Length of Pubis:

The mean length of pubis of male hip bone is 73.96 mm ranging between 68-78 mm. The mean length of pubis of female hip bone is 75.4 mm with the values ranging between 67-87 mm. The S.D. for male and female are 2.36 & 5.71 respectively. The range of mean ± 3 S.D. in males and females is 66.89-81.03 mm & 58.26-92.54 mm respectively. Identification point for male is <67 mm & for female it is less than >78mm. The demarking point for males is <58.26 mm and for females it is >81.03 mm, and the percentage of hip bones identified by demarking point alone is 0% of male and 20% of female. 't' test is Insignificant with P>0.05.

Table 4 shows comparison of length of pubis of present study with findings of other workers. It shows that overall length of pubis was greater in case of female than male, which was also seen in present study.

Table 4. Showing comparison of the Length of Pubis[6,10]

Sl. No	Author	No. of Cases		Mean (mm)		Demarking Point (mm)	
		Male	Female	Male	Female	Male	Female
1	Washburn (1948) Whites	100	100	73.8	77.9	<69	>83
2	Washburn (1948) Negroes	50	50	69.2	86.6	<79	>88
3	Washburn (1949) Bantu	82	70	66.2	73.2	<66	>78
4	Washburn (1949) Bushman	26	29	60.40	66.8	<60	>67
5	Davivongs (1963	141	41	195.39	177.40	>208.96	<167.25
6	Singh & Raju (1977)	120	80	72.27	79.65	—	—
7	Bagade (1981)	68	33	67.27	71.28	<63	>76
8	Present study (2009)	50	50	73.96	75.4	<58.26	>51.03

Length of Ischium

The mean length of ischium of male hip bone is 82.92 mm ranging between 74-92 mm. The mean length of ischium of female hip bone is 71.54 mm with the values ranging between 62 - 80 mm. The S.D. for male

and female are 4.06 & 5.15 respectively. The range of mean ± 3 S.D. in males and females is 70.74-95.1 mm & 56.1-86.98 mm respectively. Identification point for male is >81 mm & for female it is less than <74 mm. The demarking point for males is >86.98 mm and for females it is <70.74 mm, and the percentage of hip bones identified by demarking point alone is 16% of male and 38% of female. 't' test is highly significant with P<0.001.

Table 5 shows the comparison of the length of ischium of the present study with other workers. It was found from it that the average length of male ischium was greater than female ischium. The average length and demarking points of Ischium length in present study were comparable with the findings of all other.

Table 5. Showing comparison of the Length of ischium[6-10].

Sl. No	Author	No. of Cases		Mean (mm)		Demarking Point (mm)	
		Male	Female	Male	Female	Male	Female
1	Washburn (1948) Whites	100	100	88.4	78.3	>93	<75
2	Washburn (1948) Negroes	50	50	86.6	77.5	>86	<79
3	Washburn (1949) Bantu	82	70	80.3	74.8	>84	<71
4	Washburn (1949) Bushman	26	29	72.2	66.9	>76	<66
5	Davivongs (1963	89	72	81.18	74.72	>83	<74
6	Singh & Raju (1977)	120	80	83.22	75.87	—	—
7	Bagade (1981)	68	32	84.41	76	>85	<76
8	Present study (2009)	50	50	82.92	71.54	>86.98	<70.74

Ischio-Pubic index

The mean value of ischio-pubic index of male hip bone is 89.39 ranging between 78.65-97.0. The mean value of ischio-pubic index of female hip bone is 105.44 with the values ranging between 95.71-111.4. The S.D. for male and female are 4.17 & 3.94 respectively. The range of mean ± 3 S.D. in males and females is 76.89-101.9 & 93.62-117.3 respectively. Identification point for male is <95.71 & for female it is less than >97. The demarking point for males is <93.62 and for females it is >101.90, and the percentage of hip bones identified by demarking point alone is 86% of male and 82% of female. 't' test is highly significant with P<0.001.

Table 6 shows that comparison of Ischio-Pubic index of present study with findings of previous workers, which shows that the value of Ischio-Pubic index was greater in female than in case of male. Our findings were found the same and comparable with findings of previous workers. Ischio-pubic index was found to be the best indicator for sex determination.

CONCLUSION

Among all the three variables statistically analysed, the most reliable parameter, is ischio-pubic index, 86% of male hip bones and 82% female hip bones, i.e., overall 84% of hip bones can be sorted out by this parameters alone. This parameter is found to be statistically significant. With respect to the length of Ischium, the percentage of hip bones identified by demarking point alone is 16% of male and 38% of female. This parameter also found to be statistically significant.

Table 6. Showing comparison of the Ischio-Pubic index[6,10]

Sl. No	Author	No. of Cases		Mean (mm)		Demarking Point (mm)	
		Male	Female	Male	Female	Male	Female
1	Washburn (1948) Whites	100	100	83.6	99.5	<91	>94
2	Washburn (1948) Negroes	50	50	79.9	95	<84	>88
3	Washburn (1949) Bantu	82	70	82.5	98.1	<87	>91
4	Washburn (1949) Bushman	26	29	83.7	100	<93	>91
5	Davivongs (1963	89	72	77.98	92.72	<81	>87
6	Singh & Raju (1977)	120	80	87.19	105.13	—	—
7	Bagade (1981)	68	32	79.72	94.16	<81.71	>93.33
8	Present study (2009)	50	50	89.39	105.44	<93.62	>101.9

REFERENCES

1. Chommy, S.S., 2000. Osteology of pekvic bore. Lasts anatomy, Regional and applied 10th edition Churchill Livinstone, Harcourt Publishers, Ltd., pp:158-163.
2. Igbigbi, P.S. and Nanaono A.M. Igbigbi, 2003. Determination of sex and race from sub-pubic angle in Ugandans. American journal of Forensic Medicine and Pathology, 24(2): 168-171.
3. Oladipo, G.S., 2006. The Sub-pubic angle in adult indigenous Nigerians. Tropical Journal of Medical Research, 10(1): 15-19.
4. Rogers, T., S. Sanders, 1994. Accuracy of sex determination using morphological tract of human pelvis. J. Forensic sc., 39: 1047-1058.
5. Kurihara, Y.a., Y.o. Kunhara, K. Ohashi, A. Kitazgawa, M. Miyasaka, E. Okamoto, T. Fshikawa, 1996. Radiologic evidence of sex differences. Is tme patient a woman or a man? Am J. Radiol., 167: 1037-1040.
6. Washburn S. L. Sex differences in pubic bone. Am. J. Phys. Anthrop. 1948; 6:199-207.(s)
7. Washburn S. L .Sex differences in the pubic bone of Bantu and Bushman. Am. J. Phys Anthrop. 1949; 7: 425-432.(s)
8. Davivong V. The pelvic girdle of the AustraIian Aborigines, sex differences, sex determination. AM. J. of Phys. Anthrop. 1963; 21: 443-455. (s)
9. Singh S and Raju PB. Identification of sex from hip bone. Demarking Points. Journal of Anatomical Society of India, 1977; 26(2): 111-117.
10. Bagade KG. Determination of sex from axial skeleton. Dissertation for MS Anatomy Examination, Marathwada University, Aurangabad, 1981.

A Study of Changing Trends and Patterns of Poisoning at Bangalore

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ABSTRACT

A retrospective analysis of poisoning deaths was conducted at Department of forensic medicine, Kempegowda Institute of medical sciences and research centre, Bangalore over a period of five years (January2005-December 2009).Out of the total 2145 autopsies conducted in the mentioned period, deaths due to poisoning comprised 11.7% cases. The data was analyzed with respect to age, sex, marital status, motive and type of poisoning. It was observed that the incidence of poisoning was highest in the age group of 21-30years (33.7%) followed by 31-40years (22.6%). Male (67.8%) preponderance was more over female (32.1%) and married (61.1%) outnumbered the unmarried (38.9%) in both sexes. The most common type of poisons consumed were organophosphorus (53.2%) followed by over the counter medications (17%).The common reasons of consuming poison were financial constraints (21.4%) followed by ill health (15.55%). This study examines the general pattern and multiple factors associated with poisoning deaths. This rising number of deaths due to poisoning should be addressed immediately and efforts should be directed to reduce this catastrophe.

Key words: *Poisoning, Pattern, Motive, Organophosphorus.*

INTRODUCTION

Poisoning is an escalating medico legal, epidemiological and social problem. It is also one of the leading causes of unnatural death in the world. About 25,447 deaths and 4,987 serious injuries due to accidental poisoning have been reported in India in the year 2007. In Karnataka alone, there were 1619 accidental deaths and 3975 suicidal deaths due to poisoning in the same year and 584 reported deaths[1], particularly in the city of Bangalore, but many cases invariably go under reported.

With development in the field of science and technology, cases of poisoning instead of declining have unfortunately shot up considerably. The advances in all fields have opened up the global markets for newer drugs and chemicals, which are being used to counteract emerging problems, diseases and agricultural pests especially in the field of medicine, industry and agriculture. A significant number of the current population affected by diverse problems of life, have resorted to consumption of these emerging, dangerous substances as an absolute solution to end their sufferings. Thus poisoning has resulted in grave loss of human life and suffering.

An in depth knowledge on the general pattern of poisoning and multiple factors influencing poisoning in a particular region is the key instrument in the reduction of morbidity and mortality caused due to poisoning. This study analyzes the changing trends and patterns of poisoning.

MATERIALS AND METHODS

This retrospective study was conducted in the Department of forensic medicine, Kempegowda Institute of medical sciences and research centre, Bangalore for a period of 5 years from January 2005 to December 2009.This study includes alleged poisoning deaths in the jurisdiction of south Bangalore division and surrounding areas and in-house hospital deaths. Inquest reports, postmortem reports, suicide notes if any, hospital treated records and chemical analysis report were scrutinized to study multiple parameters of poisoning deaths. The main parameters include age, sex, socioeconomic status, educational qualification, marital status, treatment status, motive and type of

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poison. Conformation of poisoning in unknown bodies was excluded in this study.

RESULTS

During the study period, out of the total 2145 cases of unnatural deaths autopsied, 252 cases [11.7%] were reported to be poisoning cases.

Age: The highest incidence of poisoning cases, 85 cases (33.7%) was seen in the age group of 21-30 years. This was followed by the age group of 31-40 years, where 57 cases (22.6%) were reported. The least number were noticed in the age group of 61-70 years where only 7 cases (2.7%) were found, (Fig. 1).

Sex: Among 252 cases studied, 171 cases (67.8%) were males and 81 cases (32.1%) were females (Table 1). In both males and female category, poisoning cases were highest in the age group of 21-30 years.

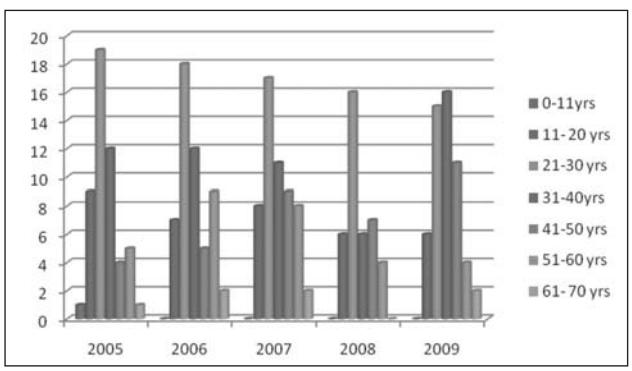

Fig. 1. Age Distribution

Table 1. Sex Distribution

	2005		2006		2007		2008		2009	
Age	M	F	M	F	M	F	M	F	M	F
0-11yrs	0	1	0	0	0	0	0	0	0	0
11- 20 yrs	4	5	3	4	4	4	4	2	1	5
21-30 yrs	14	5	12	6	13	4	9	7	11	4
31-40yrs	8	4	7	5	7	4	4	2	10	6
41-50 yrs	4	0	4	1	6	3	5	2	8	3
51-60 yrs	4	1	9	0	5	3	4	0	4	0
61- 70 yrs	1	0	2	0	2	0	0	0	2	0

Marital status: Among both male and female victims, married 154 (61.1%) outnumbered the unmarried (38.9%) cases.

Socioeconomic status: Higher number of cases being 121 cases (48%) belonged to the lower middle class families. This was followed by 80cases (31.7%) in upper middle families. Least number of cases 12cases (4.8%) was seen in upper class families.

Educational qualification: It was noticed that majority of the victims were matriculates accounting for 102cases (40.5%), followed by graduates accounting for 81 cases (32.1%) and the least were illiterates accounting for 7cases (2.8%), (Fig. 2).

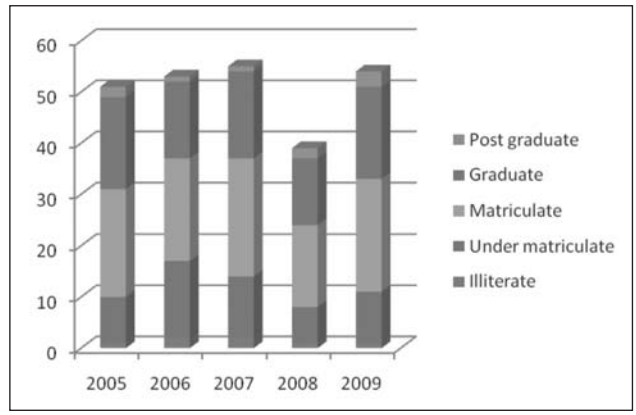

Fig. 2. Educational Qualification

Motive: Financial constraint was the most common reason for the deaths, which was seen in 54 cases (21.4%). This was followed by ill health in 39 cases (15.5%). In 35cases (13.9%) the reason was unknown. Least number of cases being 11 cases (4.4%) was due to unemployment, (Table 2).

Table 2. Motive

	2005	2006	2007	2008	2009
Financial	11	10	13	8	12
Marital	7	5	5	4	5
Family	3	7	4	3	4
Education	4	5	3	2	3
Love failure	5	6	4	2	5
Ill health	7	9	9	6	8
Unemployment	3	3	2	2	1
Unknown	7	5	9	7	7
Depression	4	3	6	5	9

Treatment status: 201 cases (79.8%) received treatment and 51 cases (20.2%) did not receive treatment, (Fig. 3).

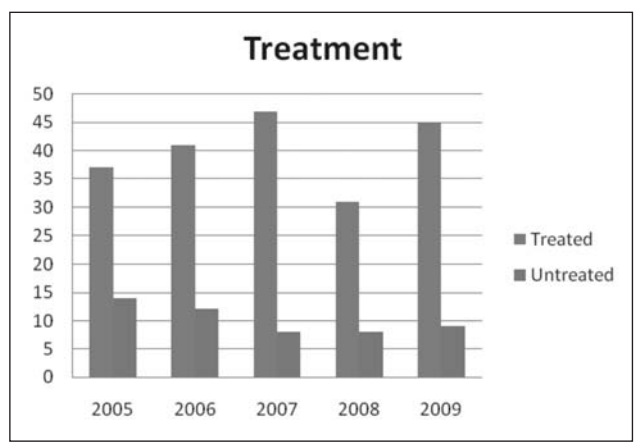

Fig. 3.Treatment

Type of poison: The highest number of cases, 134 cases (53.2%), had consumed organophosphorus compounds. This was followed by 43cases (17%), who had consumed over the counter medications. While 19 cases (7.5%) had consumed a combination of alcohol with organophosphorus compounds and rest 18 cases (7.1%) were due to phosphide ions and 6 cases (2.4%) were due to cyanide, (Fig. 4).

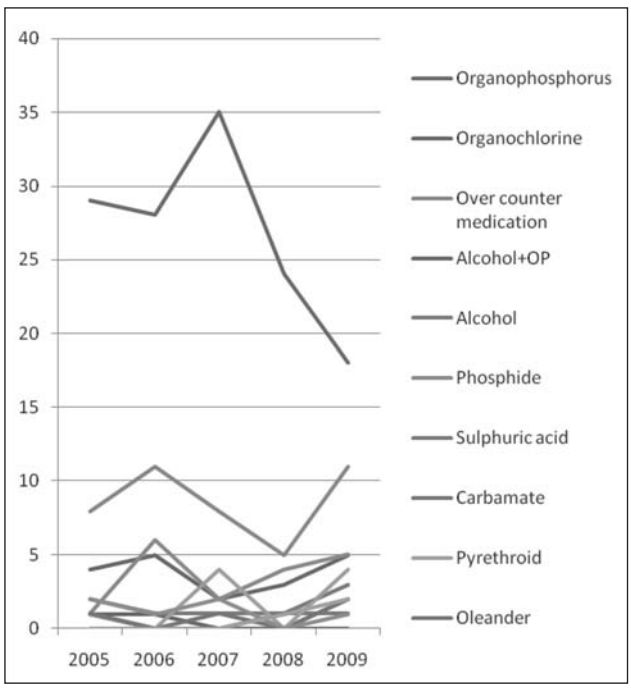

Fig. 4. Type of Poison

DISCUSSION

The maximum incidence of poisoning found in the age group of 21 to 30 years. Similar findings were observed in the studies conducted by S.K Dhattarwal[5], Murari Atul[8] and J.Gargi[7] and disagreement with Taruni Ng[9].This is attributed to factors like failures in academics, romantic failure, family disputes, marital disharmony, unemployment, romantic failures, ill health, and dowry harassment in cases of females. As this age group are at the threshold of building their career and have the utmost zeal and urge to move ahead of others, the ever increasing demands and stress of the modern mechanical lifestyle, contribute to such an act. In children, the common reasons were due to conflict with parents for trivial issues and failure or less percentage in exams.

Males being the main breadwinner in the family bear the burden of earning for livelihood. This was found to be prime reason for the increased incidence of poisoning in males. Since the study involved the subjects residing more in urban setup, the annual income of the lower middle class could not suffice to meet the basic amenities resulting in disillusionment in life amongst them. In an urban setup, though ample opportunities for employment are present, yet there is stiff competition for these jobs by better qualified, which is the main cause for this group to take this extreme step of ending their lives. Similar opinions have been stated by Dalbir Singh[2]and B.R Sharma[3]. But studies conducted by Karamjit Singh[6] and Taruni Ng[9]disagree with this view.

Among financial constraints the reason were excessive debts, poverty, not able to pay the loan, extravagant lifestyle, engaging in activities in an urge to achieve instant richness were the prominent financial causes noticed. Among ill health, in majority of cases evidence of chronic illness like bronchial asthma, tuberculosis, gastrointestinal disorders, diabetes, hypertension and gynecological problems as procured through the history and hospital records, on autopsy corroborated with the findings. It is in disagreement with studies conducted by Dalbir Singh[2].

The reasons for more number of victims culminating to death in spite of treatment may be due to the consumption of highly toxic poisons and also due to difference in individual responses to the type of poison consumed. The treatment not received may be due to the delay in transportation and shifting of the victims to the nearby hospital from the site of incident. Improper and inadequate treatment in hospitals could have also played a key role in victims culminating to death without receiving treatment

The reason for more number of victims to choose organophosphorus were due to low cost, easy availability of highly toxic pesticide, agricultural based economics and to the fact that poisoning by agro-chemicals is practically inevitable because modern farming is unthinkable without the use of these and especially for a developing country like ours. The easy availability and accessibility of over the counter medication like barbiturates, benzodiazepine, antihistaminics, analgesics etc and a belief that it causes less suffering assuring peaceful death, are the other causes for choosing these drugs. It is in agreement with the studies done by Dalbir Singh[2] and Karamjit Singh[6] and is in contrast with the studies of BK sharma[3] and Adarsh Kumar[4].

CONCLUSION

Poisoning deaths are slowly and steadily increasing day by day. There is a huge loss of lives and a devastating impact on the communities. It is vital that emergency resuscitative care services should be made available and strengthened at all health care delivery services. Adequate training and capacity building should be provided in the field of emergency medicine for all health professionals. Strong and strict

regulations, control, restriction and policy implementation on accessibility and availability of over the counter drugs and agricultural pesticides should be exercised. It is also important that psychological services, support and counseling should be provided to the community people specially the students, to handle crisis and problems in their respective lives. Initiating and strengthening their social network is a good option. It is also imperative that the state and central governments address the numerous problems and provide socioeconomic support to the middle class families.

REFERENCES

1. National Crime Records Bureau. Accidental deaths and suicides in India. Ministry of Home Affairs, New Delhi, Government of India, 2007.
2. Dalbir Singh, Jit I and Seema Tyagi.Changing Trends in Acute Poisoning in Chandigarh Zone. The American Journal of Forensic Medicine and Pathology. 1999; 20(2): 203-210.
3. Sharma.B.R, Dasari Harish, Vivek Sharma, Krishan Vij.The Epidemiology of Poisoning: An Indian View Point. Journal of Forensic Medicine and Toxicology. 2002; 19(2): 5-11.
4. Adarsh Kumar, Krishan Vij.Trends of Poisoning in Chandigarh-A six Year Autopsy Study. Journal of Forensic Medicine and Toxicology. 2001; 18(1): 8-11.
5. Dhattarwal S.K, Harnam Singh. Profile of Deaths Due To Poisoning In Rohtak, Haryana. Journal of Forensic Medicine and Toxicology. 2001; 18(2): 28-29.
6. Karamjit Singh, Oberoi.S.S, Bhullar.D.S.Poisoning Trends in the Malwa Region of Punjab. Journal of Punjab Academy of Forensic Medicine and Toxicology, 2003; 3: 26-29.
7. Gargi.J, Hakumat Rai, Ashok Chanana,Gurmanjit Rai,Gaurav Sharma,BaggaI.J.S.Current Trends of Poisoning-A Hospital Profile. Journal of Punjab Academy of Forensic Medicine and Toxicology. 2003; 3: 41-44.
8. Murari Atul, Sharma GK.A Comparative Study of Poisoning Cases Autopsied in LHMC, New Delhi and JIPMER Pondicherry. Journal of Forensic Medicine and Toxicology. 2002; 19(1): 18-20.
9. Taruni Ng, Bijoy Th,Momonchand A.A profile of Poisoning Cases Admitted in RIMS Hospital, IMPHAL.Journal of Forensic Medicine and Toxicology. 2001; 18(1): 31-33.
10. Rahul Jain, Sharikanth Asawa, Shishir Ruia. Status of Poisoning in a Rural Hospital of Maharashtra. Journal of Forensic Medicine and Toxicology. 2001; 18(1): 12-16.

Assessment of Sexual Dimorphism in Permanent Canines among Different Indian Ethnic Groups - A comparative study

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ABSTRACT

Background and objectives: The dentition is considered as a useful adjunct in skeletal sex determination, particularly since teeth are resistant to postmortem destruction and fragmentation. Sex dimorphism in tooth size and the accuracy of odontometric sex prediction, is found to vary in different regions. Therefore this study was aimed at assessing the sexual dimorphism in human permanent mandibular canines and its inter-comparison in different ethnic groups, which will ultimately help in sex determination of human remains using teeth in cases of forensic importance.

Materials and methods: Mandibular casts of dentition of 25 males and 25 females each from Rajasthan, Gujarat and Karnataka population were studied. Right and left buccolingual and mesiodistal canine width and intercanine distance were measured, canine index calculated and the data was statistically analysed.

Results: The mean values of right and left canine index were significantly higher in males compared to females in all the three population groups. Also sex could be predicted correctly by using standard mandibular canine index in 78%, 76% and 82% of Rajasthan, Gujarat and Karnataka populations respectively.

Conclusion: The mandibular canine index is a quick and easy method for determining sex and can prove to be a useful tool in forensic odontology.

Key words: *Canine index; Ethnic Groups; Forensics; Inercanine Distance; Sexual Dimorphism.*

INTRODUCTION

Culture is often defined as coherent, shared patterns of actions or beliefs specific to named groups of people that provide basic life roadmaps or social contexts, defining behavioral norms and interpersonal relationships as well as unwritten rules for proper living.[1] Ethnic origins, religions, and languages are the major sources of cultural diversity. India is a country incredible for its diversity; biological and cultural.[2] India has been declared as the second worst-affected country by terrorist violence after Iraq as per the US State Department's Annual Report 2007.[3] Further, frequent natural calamities and consistently increasing crimes are creating a need for increase in expertise of forensic sciences.

Human teeth are one of the hardest and most chemically stable tissues in the body, even at very high temperatures, making them invaluable tool for personal identification on fragmentary adult skeletal remains.[4, 5] Sexual dimorphism in tooth size has been noted and being explored over the past half-century by odontologists and anthropologists using buccolingual (BL) and mesiodistal (MD) dimensions, termed linear measurements.[6] Various features like tooth morphology and crown size are characteristic for males and females.[7] Usually males possess larger tooth crowns than females in contemporary human

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populations.[5] This may be due to a longer period of amelogenesis for both deciduous and permanent dentition in them.[5]

The identification of sex is of significance in case of major disasters where bodies are often damaged beyond recognition.[4] Of all the teeth in the human dentition, canines are the least frequently extracted teeth, possibly because of the relatively decreased incidence of caries and periodontal disease. [4] Mandibular canines are considered to be the key teeth for sexual dimorphism.[4]

Mandibular canine index calculation for sex determination has been employed in numerous studies on various large populations as it is simple, reliable, inexpensive and easy to perform.[8] The difference in finding in different population is of definite significance, as tooth morphology is known to be influenced by cultural, environmental and racial factors.[8] However, their use in forensic sex identification has not been explored fully specially in Indian populations.[6] The present study endeavors to establish the effectiveness of mandibular canine index and its bucco-lingual measurements in determining gender in three Indian population groups viz. Rajasthan, Gujarat and Karnataka.

SUBJECTS AND METHODS

The study group comprised of a total of 150 subjects, 50 each (25 males and 25 females) from Rajasthan, Gujarat and Karnataka populations in the age group of 14 to 20 years. The methodology was based on the one used by Mughal IA et. al.[8] This particular age group of 14-20 years was selected keeping in mind that all the permanent canines fully erupt by this age and attrition is minimal. Other inclusion criteria for the study were as follows[8]:

- Disease free state of gingiva and periodontium.
- Absence of any infectious or communicable oral mucosal lesion.
- Mandibular anterior teeth being free from developmental anomalies, dental caries and other regressive alterations.
- No increase in overjet and overbite.
- Absence of spacing or crowding in anterior teeth.
- Class I molar and canine relationship.

After obtaining a written informed consent from all the individuals, and a through clinical examination, alginate impressions of mandibular arch were made and were poured immediately in type IV dental stone to minimize any dimensional shrinkage. The following measurements were taken in all casts with the help of a digital vernier caliper (with 0.1 mm resolution [8]:

- The intercanine distance
- Greatest mesiodistal canine width (both on right and left sides)
- Greatest buccolingual canine width (both on right and left sides)

The left and right canine index was calculated as follows based on the formula given by Rao et al: [8,9]

"Mandibular Canine index (MCI) = MD width of mandibular canine/ Intercanine distance."[8,9]

The standard mandibular canine index of the population, used as a cut-off point to differentiate males from females, was obtained from the measurements taken in the sample by applying the following formula:[8,9]

(Mean male MCI± SD)+(Mean female MCI±SD)/2

Measurements were recorded on an excel spreadsheet and subjected to statistical analysis using 't' test and ANOVA. Multiple comparisons and intergroup comparisons were also performed. P value less than 0.05 was considered significant.

RESULTS

The statistical analysis revealed a significant difference in MCI between males and females in all the population groups with both RCI and LCI being significantly higher in males as compared to females (Table 1). The standard MCI for Rajasthan, Gujarat and Karnataka populations was 0.2538, 0.2620 and 0.2492 respectively. The difference in BL canine width between the genders was not significant in all the three population groups (Table 2). The Karnataka population group showed a slightly higher percentage accuracy of 82% in determining gender by using standard mandibular canine index as compared to the other two states, 78% for Rajasthan and 76% for Gujarat, but the difference was non-significant (Table 3).

DISCUSSION

It is a known fact that teeth provide excellent models for the study of relationship between ontogeny and phylogeny. [8] The determination of sex makes identification easier and it is of immense forensic importance.[8] In fact, it has been suggested that the first reported crime in the history of mankind was solved when bite marks were discovered in the remains of

Table 1. Right and left MCI in Different Population Groups Based on Gender

Population group	Right MCI (mean ± standard deviation)			Left MCI (mean± standard deviation)		
	Males	Females	P value	Males	Females	P value
Rajasthan	0.2746±0.1352	0.2420±0.1387	0.0001(S)	0.2641±0.1287	0.2337±0.1364	0.0001(S)
Gujarat	0.2761±0.1643	0.2505±0.1446	0.0001(S)	0.2728 ± 0.1376	0.2485±0.1586	0.0001(S)
Karnataka	0.2764±0.2053	0.2422±0.2050	0.0001(S)	0.2762±0.01778	0.2340±0.2307	0.0001(S)

MCI= Mandibular canine index, Student's 't' test, Significant (S) < 0.05

Table 2. Right and left Bucco-Lingual Canine Width in Different Population Groups Based on Gender

Population group	Right Bucco-lingual canine width (mean ± standard deviation)			Left Bucco-lingual canine width (mean ± standard deviation)		
	Males	Females	P value	Males	Females	P value
Rajasthan	6.3332±0.59573	6.0832±0.32393	0.075 (NS)	6.2940 ±0.60760	6.1036 ±0.36014	0.184 (NS)
Gujarat	6.4908 ± 0.8702	6.5092±0.72458	0.936(NS)	6.1712 ± 0.83132	6.3872 ±0.60550	0.299 (NS)
Karnataka	6.4888±0.60826	6.0548±0.55167	0.11 (NS)	6.4052 ±0.6476	6.1972 ±0.39633	0.178 (NS)

Student's 't' test, Non-Significant (NS) > 0.05

Table 3: Percentage of cases correctly predicted using standard MCI

Population group	No of cases with correct gender prediction using standard MCI	% accuracy	P value
Rajasthan (n=50)	39	78%	0.76 (NS)
Gujarat (n=50)	38	76%	
Karnataka(n=50)	41	82%	

MCI= Mandibular canine index, Chi square test, Non-Significant (NS) > 0.05

forbidden fruits in the Garden of Eden and identified as those of Adam and Eve.[10]

The present study establishes the existence of a definite sexual dimorphism in mandibular canines. Kaushal et al too in their study on 60 subjects in a north Indian population found a statistically significant dimorphism in mandibular canines.[11] Rao et al also found statistically significant sexual dimorphism in mandibular canines in a South Indian population sample.[9] Hashim HA et al in their study on 720 teeth on pretreatment orthodontic casts in a Saudi Arabic population aged 13 to 20 years noticed that the canines were the only teeth to exhibit sexual dimorphism.[12] This sexual dimorphism in canines may be due to the influence of the Y-chromosome which intervenes most in the size of teeth by controlling the thickness of dentine, whereas the X-chromosome, for a long time considered to be the chromosome responsible, only comes into play concerning the thickness of enamel.[8] Canine sexual dimorphism is predominantly a product of rate differences in crown formation, with male canines growing at a faster rate than those of females.[13]

Another reason for this dimorphism may be related to evolution.[14] The canines were functionally not masticatory but were related to threat of aggression and actual aggression.[14] A transfer of this aggressive function occurred from the teeth to the fingers in man and until this transfer was completed, survival was dependent on canines especially in males. [14] Thus in present day humans, sexual dimorphism in mandibular canines is not merely a coincidence but can be expected to be based on functional activity. [14]

Another factor responsible for canine sexual dimorphism may be bimaturism, i.e. process of males and females growing at the same rate, but with males growing for a longer period of time.[13] In another study on Saudi Arabian sample of 503 schoolchildren by Al-Rifaiy et al it was reported that the mean values for mesiodistal maxillary and mandibular canine width was less for females than males, with no statistically significant differences.[15] It may be because it depends on the levels of sexual dimorphism between the absolute dimensions-low sexual dimorphism in one (e.g. MD measurement of canines) and proportionately high sexual differences in the other (e.g. inter-canine arch width), or vice versa, would lead to higher sex estimation accuracy of the MCI; on the other hand, relatively equal levels of sexual dimorphism in both would negate the ability of the MCI to accurately estimate sex.[16]

In the present study, no significance difference was found between the bucco-lingual width of mandibular canines in males and females in all the three population groups. A study done by Acharya AB et al concluded that linear measurements afford better sex discrimination and investigators examining sex differences in the teeth may confine themselves to analyzing BL and MD dimensions per se.[6] Therefore BL and MD dimensions taken together appear to be more valuable for sex discrimination than BL dimensions alone. Another study by Prabhu S et al concluded that overall, the mandibular teeth and BL

tooth dimensions showed greater tendency to be larger in males.[17]

In the present study, when the intergroup comparison was made, the Karnataka population group showed a slightly greater number of cases with correct gender prediction by using standard MCI when compared to the Rajasthan and Gujarat population groups but the difference was not statistically significant. Any measurement of teeth unaccompanied by age, race and sex must be treated with great reserve.[18] Amongst the significant findings that can be obtained from teeth are race, age, sex, habits and racial customs. Molnar (1971) found the existence of a positive correlation between tooth wear and cultural factors .[19] Sex dimorphism in tooth size and the accuracy of odontometric sex prediction, is found to vary in different regions and researchers have advocated the need for population-specific data.[17] The teeth have been studied for sex differences with a view to develop population standards, as well as promote their application in forensic scenarios.[17] Odontometric data and its usefulness in sexing South Asian populations, however, are still largely unexplored.[17]

CONCLUSION

The present study establishes the existence of a definite, statistically significant, sexual dimorphism in mandibular canines in all the three population groups, however no significant difference in its accuracy was observed between these populations. Furthermore, the effect of vast ethnic and cultural diversity on the teeth and jaws needs to be studied in detail to increase the usefulness of forensic odontology. Again the sex identification accuracy using MCI is relatively moderate (nearly 80%), still relegating the dentition to be an adjunct rather than the sole criteria for sex assessment.

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REFERENCES

1. Butani Y, Weintraub JA, Barker JC. Oral health-related cultural beliefs for four racial/ethnic groups: assessment of the literature. BMC Oral Health 2008; 8: 26–39.
2. Das, N. K. (2006). Cultural diversity, religious syncretism and people of India: An anthropological interpretation.Bangladesh E-Journal of Sociology 2006: 3(2). Retrieved from http://www.bangladeshsociology.org/BEJS%203.2%20Das.pdf.
3. Srivastava D. Terrorism and armed violence in India: An analysis of events in 2008. Institute of Peace and Conflict Studies: Special Report 71. May 2009. Retrieved from http://www.ipcs.org/pdf_file/issue/SR71-Final.pdf.
4. Kapila R, Nagesh KS, Iyengar AR, Mehkri S.Sexual Dimorphism in Human Mandibular Canines: A Radiomorphometric Study in South Indian Population. J Dent Res Dent Clin Dent Prospects. 2011; 5(2):51-54.
5. Boaz K, Gupta C. Dimorphism in human maxillary and mandibular canines in establishment of gender. Journal Forensic Dent Sci 2009; 1: 42-45.
6. Acharya AB, Mainali S. Are dental indexes useful in sex assessment? J Forensic Odontostomatol 2008; 27: 2: 53-59.
7. Duraiswamy P, Tibdewal H, Patel K, Kumar S, Dhanni C, Kulkarni S. Sex determination using mandibular canine index in optimal-fluoride and high-fluoride areas. J Forensic Dent Sci 2009; 1: 99-103.
8. Mughal IA, Manzur F, Saqib AS. Mandibular canine index(MCI);its role in determining gender. Professional Med J 2010; 17(3) 459-463.
9. Rao NG, Rao NN, Pai ML, Kotian MS. Mandibular Canine Index - A Clue for Establishing Sex Identity. Forensic Sci Int 1989; 42(1): 249-254.
10. Danielson K. Guest editorial. Int J Forensic Dent, 1978; 1: 1-2.
11. Kaushal S, Patnaik VVG, Sood V, Agnihotri G. Sex determination in north Indians using mandibular canines index. JIAFM 2004; 26(2): 45-49.
12. Hashim MA, Murshid ZA. Mesiodistal tooth width: A comparison between Saudi males and females. Egypt Dent J 1993; 39: 343-346.
13. Schwartz GT, Miller ER, Gunnell GF. Developmental processes and canine dimorphism in primate evolution. Journal of Human Evolution 2005; 48(1): 97-103.
14. Eimerl S, Vore D. The Primates, New York , Times Inc.1965.
15. Al-Rifaiy MQ, Abdullah MA, Ashraf I, Khan N. Dimorphism of mandibular and maxillary canine teeth in establishing sex identity. Saudi Dent J 1997; 9: 17-20.
16. Acharya AB, Angadi PV, Prabhu S, Nagnur S. Validity of the mandibular canine index in sex prediction: reassessment in an Indian sample. Forensic Sci Int 2010; 204(1): 207e 1-4.
17. Prabhu S, Acharya AB. Odontometric sex assessment in Indians. Forensic Sci Int. 2009; 192: 129 e 1-5.
18. Kaushal S, Patnaik VVG, Agnihotri G. Mandibular canines in sex determination. J Anat Soc India 2003; 52 (2): 119-124.
19. Molnar S. Human tooth wear, tooth function and cultural variability. Am J Phys Anthropol. 1971; 34(2): 175-89.

Hospital Waste Management: Indian Perspective

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ABSTRACT

Hospitals' committed to patient care and community healths have been cited to paradoxically defy their own objectives. On one hand they cure patients and on the other, have emerged as a source of several diseases. The Hospital waste is of special kind of waste. By the nature of its origin, hospital waste can become a health hazard and in extreme cases a source of epidemic. In order to provide for better management of hospital waste the Bio-medical (Handling and Management) Rules, 1998 was notified under the Environment Protection Act, 1986. The problem of hospital waste is more of quality as compared to quantity. It has been computed by the WHO that the average waste generated by hospitals in developed countries is in the range of 1-5 kg / bed/day, in comparison, the average for developing countries is between 1 to 2 kg / bed / day. This calls for coordination between all hospital departments that generate waste and agencies employed for the segregation, collection, transportation, treatment and disposal of waste.

Key words : *Hospital Waste, Generation, Segregation, Incineration, Hospital Management, Hazards.*

INTRODUCTION

The second half of last century has witnessed a tremendous change in both disease profiles as well as in drug and technology used to treat them[10]. With rapid advancements in medicinal facilities and ever increasing population, Indian hospitals are producing tones of different kind of waste materials. The modern hospital presents an interface between patient and health care workers on one side, disease and hospital waste on the other side. By the nature of its origin, hospital waste can become a health hazard and in extreme cases a source of epidemic[16]. This calls for coordination between all hospital departments that generate waste and agencies employed for the segregation, collection, transportation, treatment and disposal of waste[10, 2].

Waste generation and disposal has always been the concern of medical professionals, this were being carried out by burning, land filling and burial which conformed to the then existing knowledge of public health. The advent of disposables has brought in its wake attendant ills such as inappropriate recycling, unauthorized and illegal re use and increase in quantum of waste[7].

The world health organisation (WHO) convened a working group on hospital waste management at Bergen in 1983, the group recommended a system approach to the Problem[11].

Legislation in India

The hospital wastes attracted the attention of the Supreme Court as early as in 1994 when a writ petition was filed under Article 32 of the Constitution against the concerned authorities to provide clean and health environment[3]. Issue of improper hospital waste management was the crux of the case in B.L. Wadhera v. Union of India1 Following which the Honourable Supreme Court of India in 1995 directed that all hospitals with minimum of 50 beds should install Incinerators or any other effective alternative, Incinerators should be fitted with a pollution control mechanism conforming to the standards laid down by the central pollution control board, which was asked to regularly monitor the collection, transportation and disposal of waste[4,15].

Consequences of poor hospital waste management

1. Injuries from sharps to hospital personal and waste handlers
2. Nosocomial infection in patients from poor infection control practices.

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3. Risk of infection outside hospital among waste handlers and scavengers.
4. Chemical injury from toxic, corrosive, flammable, reactive and genotoxic waste, Radiation injury due to exposure to vials and accessories contaminated with radioactive spills, radium stores and isolation room in nuclear medicine
5. Disposables being repacked and sold by unscrupulous traders.
6. Discarded drugs being repacked and sold to unsuspecting buyers.
7. Environmental pollution like water, air and soil pollution is because of poor waste management.

Categorisation of hospital waste

Cat no 1: Human anatomical waste, blood and body fluids.
Cat no 2: Animal and slaughter house waste.
Cat no 3: Microbiology and Biotechnology waste.
Cat no 4: Sharp waste.
Cat no 5: Discarded Medicines.
Cat no 6: Solid waste.
Cat no 7: Disposables.
Cat no 8: Liquid waste.
Cat no 9: Incinerator Ash.
Cat no 10: Chemical waste.

Generation of waste:

Waste is generated by all activities in a health care institution that lead to the production of waste. It has been computed by the WHO that the average waste generated by hospitals in developed countries is in the range of 1-5 kg / bed/day, in comparison, the average for developing countries is between 1 to 2 kg/bed/day[11,18].

Quantity Vs. Quality:

The problem of hospital waste is more of quality as compared to quantity e.g. it is estimated that the total amount of hospital waste in Delhi is only 1.5% of the total municipal waste stream[5]. A special obligation to deal with this waste in an effective and safe manner is mandatory due to its composition[8]. However, what is not commonly known that only a small percentage i.e. 10 to 20% of the hospital waste stream is infectious and requires special disposal techniques[13]. 85 % of wastes are non-hazardous, 10% of wastes are infective and hazardous and 5 % of wastes are non-infective and hazardous.

Segregation of waste:

Segregation is the most important pre-requisite in the entire process of waste management; it makes special care to be given to the small quantities of hazardous waste, and is the key to scientific waste management, the minimum segregation that should be done is

a. Non-hazardous / general waste — to be disposed off in the same manner as domestic waste.
b. Infectious and hazardous waste — to be treated with some method before disposal.

Collection of Hospital waste:

It is carried out after segregation by use of separate containers for each type of waste. The use of distinct colour code for each type of waste is a very effective method of ensuring proper segregation, storage, transport, treatment and disposal of waste. Hospitals may use any of the recommended colour codes for containers.

Waste category	Color of container
Infectious waste	Red
Pathological waste	Red
Pharmaceutical waste	Red
Genotoxic waste	Red
Sharps	Yellow
Plastics	Blue
Pressurized containers	Black
General waste	Black

Storage of Hospital waste:

Storage may be taken as holding of hospital waste for a period of time at the end of which the waste is treated and disposed off. Thus storage encompasses the entire time from segregation through transit till treatment and disposal

Precautions in devices used for the storage of waste:

1. Containers should be made of hard plastic.
2. The colour coded containers should be with lids and leak proof.
3. Containers should be washed and cleaned daily after polythene containing waste is removed.
4. Disinfected once a week.
5. Polythene bags should be sturdy, leak proof and 55 micro gauge thick.
6. If polythene bags are incinerated they should be of non-chlorinated plastics.

Waste collection areas should be specifically earmarked, covered and protected from all sides. There should be a clear warning sign on all approaches to the area. It should be located in an area that has minimum patient and staff movement.

Transport of Hospital waste:

a. Intra mural transportation: Waste from its point of generation to the collection area in hospital.
b. Extramural transportation: Transportation from the collection area till its fi**nal disposal.**

Hospital waste treatment technologies:

Treatment is the term used for the processes used to modify the waste with the objective of disinfecting or decontaminating. There are various treatment processes like, Disinfection, Thermal treatment, like Autoclave, Hydroclave, Microwave, Incinerator, Medical waste sterilisation unit (MWSU)[9,12,14].

Final disposal of Hospital waste:

Disposal means Burial, discharge, deposit, dumping, land filling or placing, depending upon type of waste generated measures are taken, For general non hazardous solid waste landfill, pit burial, vermiculture pyrolysis/gasification technology is used. For waste water and liquid waste, it is dispose by discharge into sewers, soaks pits, waste stabilizing pond. Sharp wastes are treated by needle destroyers, disinfection in 1 % savlon or by burial. Human anatomical, blood and body fluids waste by incineration and landfill. Microbiological and biotechnical waste by Hydroclaving, Pharmaceuticals by incineration or landfill, Infectious solid waste by either treatment, Chemical waste chemically treated and diluted/ incinerated[6,7,17].

CONCLUSION

Thus, concluding the topic, health care industries are generating wastes including hazardous and dangerous waste in the form of solid and liquid.

The most vital component of the Hospital waste management plans that have been formulated is to bring about a transformation in the mind sets and develop a system and culture through education, training and persistent motivation of the health care staff. Effective methods should be made for accumulation, segregation through labelled and coloured containers and central storage.

The law relating to Hospital waste management nicely drafted; but the time has come to act seriously and implement the rules effectively. Greater commitment is required on the part of the Government looking into the magnitude of the problem. Since the hospitals have become the source of huge profits. They owe a duty to protect the interest and the safety of its workers and the public.

REFERENCES

1. AIR 1996 SC 2969.
2. Biomedical Waste Management - An Emerging Concern in Indian Hospitals. Author(s): Virendar Pal Singh, Gautam Biswas, Jag Jiv Sharma Vol. 1, No. 1 (2007- 07 - 2007-12).
3. C.M.Jariwala "The Bio-medical Waste: Direction of Law and Justice" J.I.LI. (41) 368 (1999).
4. Draft Biomedical wastes rules, 1997, Gazette of India extraordinary Part II Section 3 dated 16th Oct 1997.
5. Editorial, Havoc Medical Waste Wreaks, Health Action (Environment) 1996: 24
6. Hemangini K. Shah, S.K. Ganguli Hospital Waste Management - A Review Vol. 25, No. 3 (2000-07 - 2000-09).
7. Kela M, Nazareth S, Agarwal R: Implementing hospital waste management; a guide for health care facilities in Shrishti 1997:1.
8. Kerac M: The forgotten patient - discharged and dangerous? a case-study report of seven hospitals. Hospital Waste In India, 1997: 2
9. Krishnan S: Integrated Waste Management Plan, Hospital waste management plan, strategies for implementation, CEE South, 1997.
10. Lyon's Medical Jurisprudence and Toxicology, Delhi Law House, Biomedical Waste Disposal 11th edition 2005 Page no 350-361.
11. Management of waste from hospitals and health care establishments. Euro reports and studies No. 97 WHO, 1985.
12. Park K. Hospital Waste Management. Park's Textbook of Preventive and Social Medicine. M/s Banarasidas Bhanot Publications, New elhi. 18th Edn, 2005: 595-598.
13. Pruthvish S, Gopinath D, Jayachandra Rao M, Girish N, Bineesha P, Shivaram C: Health care waste disposal - an exploration, Department of Community Medicine, M S Ramaiah Medical College 1997; 1-11.
14. Raghunath Patnaik, "Bio-medical Waste Management and the Process of Environmental Governance" 322 Central India Law Quarterly 24 (2001)
15. Raghupathy L, Kathpalia I: Rules on biomedical waste, Indian Journal of clinical Practice. 1995; 6 (4); 84-99.
16. Safe Management Of Wastes From Healthcare Activities Edited by Pruss, E., Giroult and Rushbrook. Geneva, WHO, 1999. Pp 21-29.
17. Singh IB, Sharma RK, 1996. Hospital Waste Disposal System and Technology. Journal of Academy of Hospital Administration, 8 (2): 44-48.
18. Survey of Hospital waste management in south East Asian region, SEA/EH 493 WHO (SEARO) New Delhi September 1995.

Medical Certification of Cause of Death at General Hospital Nalgonda

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ABSTRACT

Medical certification of Cause of death is the most frequently issued certificate by government medical doctors. Cause of death is one of the basic components of a country's health information system. There are many short comings in the routine collection of such information. Analysis of general hospital Nalgonda death certification was done to evaluate how this shortcoming might influence reporting. Our objective was to determine the completeness of death certification by Medical doctors at general hospital Nalgonda. The study was carried out by auditing the data from Birth and Death Registration office at Municipal Commissioner Nalgonda. 156 death notification forms and certificates were analysed at Birth and death Registration Office which was submitted by general hospital Nalgonda, result of the study shows that there is a conflict of opinion related with the meaning of terms 'Cause of Death', and 'Modes of death', amongst the certifying doctors. Study highlights the need to educate and make them aware of importance of correct medical certification of death.

Key words: *Medical Certification of cause of death, Registration of Births and Deaths Act, Continuing Medical Education.*

INTRODUCTION

Death registration and mortality statistics are essential and necessary for planning, implementing, and evaluating public health programmes and policies. It is also helpful with practical issues like hospital reimbursement, life insurance claims, obtaining a probate or succession certificate, settling property claims, releasing gratuity and provident fund claims and deleting the deceased name for the ration card, and voter's list or employer's register[2]. It also helps to evaluate health indicators like infant mortality rate, maternal mortality rate, to know the impact of health services etc and to find out magnitude of emerging and re-emerging diseases. Proper death certification begins with a fundamental understanding of cause and manner of death[6]. A person has legal existence between the recorded timings of birth and death.

Therefore, every medical doctor's has become obligated to issue a cause of death certificate in the unfortunate death of his patient. Incomplete or inaccurate entry in these certificates poses difficulty in obtaining reliable information pertaining to cases of mortality. To overcome this medical certification of cause of death scheme was introduced which is a part of International statistical classification of diseases and health related problems formulated by WHO[10].

Duty of Medical Doctor

As per the provision laid down by the Indian Medical Council (Professional conduct, etiquette and ethic) regulation 2002, certificates are issued by doctors for the purpose of the Acts relating to birth, death or disposal of the dead[8]. When there is death, first medical doctor should diagnose and declare death and second is to decide cause of death and certify. If the death is not a natural death, doctor after declaring death should inform the police of occurrence of the death for further course of action, no need to issue the medical certificate of cause of death. The investigating Police officer proceeds with inquest and the cause of death is decided after medico legal post-mortem is carried out as part of the inquest. The death Certificate should be issued by a registered medical doctor who has been medical attendant of the deceased during life, has attended the patient within past 14 days prior to his death and is satisfied as to the cause of death.

Points to be kept in mind by Medical Doctor while issuing Medical Certificate of cause of death[9]:

1. If doctor is sure of cause of death, there should be no delay for any reason to issue the medical certificate of cause of death.
2. To issue the certificate of cause of death doctor cannot charge any fees.

3. Doctor should not sign cause of death in advance.
4. Doctor should not issue the cause of death certificate without viewing and examining the dead body personally.
5. Doctor should not withhold the issuance of medical certificate of cause of death even if his fees is not been cleared by relatives.

METHODOLOGY

We carried out the study on the cause of death certificate issued by medical doctors of general Hospital Nalgonda to birth and death registration office at Municipal Commissioner Nalgonda. A prior permission to undertake the study was obtained from municipal commissioner to analyse the cause of death certificate issued by medical doctors of general Hospital Nalgonda. 156 death certificates were analysed from the year 2009 after compiling and then analysed manually.

RESULTS

Table 1 Summaries the result of this study. We studied a total of 156 certificates out of which 103 were male deaths and 53 were female death. Terms used to describe modes of death like cardio respiratory arrest, cardiac shock, cardiac failure, respiratory failure, respiratory paralysis etc were mentioned in 59 (37.82%) cases. The cause of death includes any disease or injury responsible to initiate a chain of events incompatible with life resulting in death of a person[5]. It was mentioned properly in 97 cases.

DISCUSSION

Medical doctors are expected to have a complete knowledge of the sequence of events of underlying cause which may lead to death. They should fulfil the requirement of International classification of diseases developed by WHO (10th revision) to comply within Form 4 for hospital deaths in government or private and Form 4 A for non institutional deaths[10]. This study has identified upto 37.82 % of death certificates where medical doctors are confused with the terms 'cause of death' and 'modes of death'. The dilemma is still present amongst medical fraternity even though medical certification of cause of death guideline specifically mentions that the cause of death should not be confused with modes of death.

Table 1. Showing causes of death from General Hospital Nalgonda analysed at Birth & Death registration Municipal office in 2009.

Causes of death	Numbers	Percentage
Cardio respiratory failure	59	37.82%
Congestive cardiac failure	15	9.62%
Tuberculosis	25	16.03%
HIV	06	3.85%
Myocardial Infarction	23	14.74%
Bronchopneumonia	07	4.49%
Septicaemia	13	8.33%
Pyrexia of unknown origin	03	1.92%
Carcinoma Oesophagus	01	0.64%
Unknown	04	2.56%
Total	**156**	**100**

Deaths caused by HIV infection represent a particular difficulty in terms of death certification. 6 cases have been reported, we found in our study. The stigmatization associated with HIV related disease has made doctors reluctant to specify as a cause of death[4]. The code of medical ethics permits overriding the rule of confidentiality and disclosure of the fact that the deceased was suffering from HIV/ AIDS infection[2]. This exception to the code of medical ethics is permitted in public interest and overrides confidentiality[2].

Cardio respiratory failure means that the heart and lungs stopped functioning. Death does not occur without the heart and lungs stop functioning. Cardio respiratory failure is neither a cause of death nor a mechanism of death[5]. In 1999 B. L. Meel showed a highest rate of inaccurate cause of death mentioned as cardio respiratory failure 78.9% in his study at Umata general hospital of South Africa[1]. In 1993 Jordan and bass discovered that 31.9% of a sample of death certificates at a Canadian tertiary care teaching hospital contained major error[3]. In the study of 2008-2009 S Agarwal showed a highest ever recorded rate of inaccurate terms used in cause of death certificate[7].

CONCLUSION

Our study clearly reflects that there is lack of knowledge and ignorance among the medical doctors on the procedure and practice of writing the cause of death certificates. The medical certification of cause of death scheme is an important step in regularizing and maintaining uniformity of issuing cause of death certificates by medical doctors. There is a need for active involvement and co-operation of the medical doctors to make the scheme successful on medical certification of cause of death formulated by the office of the registrar general of India. There is a need to sensitize the medical doctors about the importance of medical certification of cause of death. Periodic auditing of cause of death certificates shall be important to improve the recording of cause of death.

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REFERENCES

1. B. L. Meel "Certificates of death at Umata General Hospital South Africa". Journal of Clinical Forensic Medicine (2003) 10, Page No 13-15.
2. Bokil P.V. "Death Certificate". QPMPA Journal of Medical Sciences 2002; 16(3): 84-6.
3. Jordan JM, Bass MJ. Errors in death certificates completion in a teaching hospital. Clin Invest Med. 1993; 16 (4): 249-255.
4. King M B. AIDS on the death certificate: The final Stigma. BMJ 1989; 298: 734-736.
5. Mathiharan K, Patnaik AK. Modis Medical Jurisprudence and Toxicology. 23rd edition. Lexis Nexis, New Delhi 2006: 241.
6. Office of the Registrar General of India, Vital Statistics Division: Physicians Manual on Medical Certification of Cause of Death, Ministry of Home Affairs, New Delhi, 4th Edition 2000.
7. S.S. Agarwal et.al. "A Study on Appraisal of Effectiveness of the MCCD Scheme". Journal of Indian Academy of Forensic Medicine Vol.34, No.4 Oct- Dec 2010, Page No 318-320.
8. The Indian Medical Council (Professional conduct, Etiquette and Ethics) Regulations. Notification No. MCI 21(2) 2001. The Gazette of India 2002.
9. The registration of Birth and Death Act, 1969 [Act No. 18 of 1969]. Universal Law Publishing Co. Pvt. Ltd, Delhi 2006: 6-8.
10. World Health Organisation: "International Statistical Classification of diseases and related Health problems — Tenth Revision (ICD-10)" volume 2. Instruction Manual. World Health Organization, Geneva 1993. Pages 30-32.

Cheiloscopy: A Tool for Solving Crime and Identification

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ABSTRACT

The Lip prints are unique and distinguishable for every individual like fingerprints. Lip prints has been utilised in court at very few instances, more and more research need to be conducted in this field with regards to use of lip prints in personal identification and criminal investigation, it will certainly help as useful evidence in Identification and a tool to solve crime. The present study was aimed to study lip prints of different individuals in various quadrants of lip. The study group comprised of 50 males and 50 females. The materials used were lipstick, white bond paper and a magnifying lens. Present study shows that lip-prints are unique to an individual and behold the potential to act as a tool to solve crime.

Key words: *Cheiloscopy, Lip prints, Identification, pattern*

INTRODUCTION

Identity means determination of individuality of a person[4]. Like fingerprints, even lip prints can be instrumental in identifying a person positively. Lip prints are normal lines and fissures in the form of wrinkles and grooves present in the zone of transition of human lip, between the inner labial mucosa and outer skin, examination of which is known as Cheiloscopy[4]. The study of lip print is an upcoming tool for identification of persons. The lip print of every person is unique and can be used to fix personal identity[1]. In criminal investigations the material evidence left at the scene of crime frequently provides extremely useful data for investigations and identification materials such as blood, body fluid, hair and prints of fingers, palms and soles have led to the solutions of many difficult cases. Lip prints on wine bottles, glasses or on the love letters have in some occasions helped identification of concerned persons[1,5]. Research studies and information regarding the use of lip prints as evidence in personal identification and criminal investigation. However, studying in depth and establishing further facts and truth in lip prints will certainly help as useful evidence in Identification and a tool to solve crime.

Historical overview

Fischer in 1902 was the first anthropologist to describe the furrows on the red part of the human lips[3]. However, it was only in 1932 that Edmond Locard, one of France's greatest criminologists, recommended the use of lip prints in personal identification and criminalization[1]. In 1950, Synder reported in his book Homicide Investigation that the characteristics of the lips formed by lip grooves are as individually distinctive as the ridge characteristics of finger prints[10]. Suzuki, in 1967, made detailed investigations of the measurement of the lips, the use and collar of rouge, and the method for its extraction to obtain useful data for practical forensic application[9]. Later in 1970, Suzuki and suchihashi, conducted a study on 107 Japanese families and named the grooves on labiorum rurorum as sulci labiorum and the lip prints consisting of these grooves as ' Figura linearum labiorum rubrorum'[8]. Mc Donell in 1972 conducted a study on lip prints between two identical twins and reported that two identical twins seemed to be indistinguishable by every other means but their lip prints were different[1]. Cottone, in 1981, reported in his book Outline of Forensic Dentistry , that Cheiloscopy is one of the special techniques used for personal identification[2]. Recently, Vahanwala in 2000 conducted a study of lip patterns to promote the importance of Cheiloscopy in forensic science identification[12].

Suzuki and Tsuchihashi[8], in 1970, devised a classification method of lip prints, and termed as "Figural linearum labiorum rubrorum" which is as follows:

1. Type I - A clear-cut groove running vertically across the lip.
2. Type I! - Partial-length groove of Type I.(Pronounced as "One Dash")

3. Type II - A branched groove.
4. Type III - An intersected groove.
5. Type IV - A reticular pattern.

MATERIAL & METHOD

Study sample

A sample of 100 individuals comprising 50 males and 50 females were included in the study. All individuals were aged between 18 and 40 years. Lips free from any pathology and injury, having absolutely normal transition zone between the mucosa and skin were included in the study. Consent of all the individuals was obtained for the study.

Study materials

In order to classify the lip prints in this study, the classification scheme proposed by Suzuki and Tsuchihashi [8] was used. Materials used were:

1. Lipstick of bright red colour non glossy
2. White bond paper
3. Magnifying lens

Technique

The lips of the individuals were cleaned and the red colour lipstick was applied on the lips. Over the lipstick, the folded white bond paper was placed and the subject was asked to make a lip impression in the normal rest position of the lips by dabbing it in the centre first and then pressing it uniformly towards the corners of the lips. Then a cello tape was then stuck to the white bond paper over lip print for permanent record purpose and then visualized by magnifying lens. While studying the various types of lip prints, each individual lips were divided into six equal compartments, i.e., Three compartments on each lip and were labelled as UR, upper right; UM, upper middle; UL, upper left; LR, lower right; LM, lower middle; LL, lower left.

Observation

Table 1. Age wise distribution of cases

Age Group(In Years)	Number of cases
18-23	47
24-29	23
30-35	19
36-40	11

Table 2. Pattern of Lip print as observed

Types	Type1	Type1!	Type II	Type III	Type IV	Total
Male	61(10.16%)	62(10.33%)	107(17.83%)	46(7.66%)	24(4.0%)	300
Female	63(10.5%)	70(11.66%)	95(15.83%)	39(6.5%)	33(5.5%)	300
Total	124(20.66%)	132(22.0%)	202(33.66%)	85(14.16%)	57(9.5%)	600

A total of 100 individuals were included in the study, comprising of 50 males and females each, in the age group of 18-40 years. In overall study, no individual had single type of lip print in all the six compartments and no two or more individuals had similar type of lip print pattern, When the overall pattern was evaluated among all the lip compartments of the study subjects, it was found that Type II pattern 202(33.66%) was most common, both among males and females having 107(17.83%) and 95(15.83%), respectively. Followed by type I! i.e 132 (22.0%), Type I having 124 (20.66%) and Type III 85 (14.16%).However, the least common was the Type IV pattern 57(9.5%) seen in 24(4.0%) males and 33(5.5%) females.

DISCUSSION

It was observed in our study that no two lip prints were identical. Each individual has its own lip print pattern it was identical with studies by others[8,9,12].It was observed that type II pattern is the most common(33.66%) followed by type I!(22.0%) which is different from the study by Y Tsuchihashi[8,9] where Type III pattern was most common. It may be because of racial variation in Indian population which is different from Japanese population. Other works on Indian subjects have yielded varying results. Vahanwalla and Parekh in their study in Mumbai found that type I was the most frequent[12] Sivapathasundaram et.al studied the lip prints of Indo-Dravidian population and noted that Type III was predominant[7]. Manipady compared Indian and Chinese individuals and found that the incidence of Type II was the highest among Indians.

At scene of crime Investigating officers looks for finger prints at all possible places, presence or absence of finger prints is conclusive of the fact that the person suspected was either present or absent on the scene of crime, likewise the lip print being uniform throughout the life and individualistic[11,12], can be used to verify the presence or absence of a person from the scene of crime, provided there has been consumption of beverage, drinks usage of cloth, tissue/napkin etc. at the crime scene[1,6,11]. In an investigation, Dr. Anil Aggarwal has proved beyond doubt that lip prints are as good as finger prints in criminal identification and can be definitely used when no other means of traditional methods of identification are available[1].

Heredity and lip print:

2 pairs of twins, both pairs of uniovular type were studied in details in all 06 quadrants. It was similar to their parents in few quadrants, but one or the other give different pattern, it was consistent with the study of Mc donell who reported that two identical twins seemed to be indistinguishable by every other means

but their lip prints were not identical[1]. This was different from study by Tsuchihashi et al[8,9] in which they found similar lip prints in twins and their parents. Though the size of samples for twins in our study was small but even one case of uniovular twins showing different pattern creates doubts for heredity in lip print identification.

CONCLUSION

Type II pattern was most common in this study followed by Type I!. In cases of twins we divided the lips in 6 quadrants which showed different patterns from each other. Hence it is necessary to study lip print in detail for proper identification each lip print pattern is unique and can help in identity of the individual. If police keep record of lip print with them along with fingerprint, it will help to solve the crime and can justify the real sinner. However, as far as the legal matters in Indian judicial system are concerned, this technique needs to be used more frequently in routine civil and criminal litigations.

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REFERENCES

1. Aggarwal A. The importance of lip prints (Forensic Files). Available from: http://lifeloom.com//II2Aggarwal.htm. 24th Oct 2008.
2. Cottone JA, Standish SM. Textbook of outline of forensic dentistry. Special Tech (Cheiloscopy) 1981. p. 135.
3. Kasprazak J. Possibilities of Cheiloscopy. Forensic Sci. Int. 1990; 46: 145-151.
4. Nandy A. Principles of Forensic Medicine. 2nd edition. Calcutta: Central; 2003.p. 100-101.
5. Santos M. Queiloscopy: A supplementary stomatological means of identification. International Microform J. Legal Medicine 1967. p. 2.
6. Segui MA, Feucht MM, Ponce AC, Pascual FA. Persistent lipsticks and their lip prints: A new hidden evidence at the crime scene. Forensic Sci Int. 2000; 112: 41-47.
7. Sivapathasundharam B, Prakash PA, Sivakumar G. Lip prints (Cheiloscopy). Indian J Dent Res 2001; 12: 234-237.
8. Suzuki K, Tsuchihashi Y. A new attempt of personal identification by means of lip print. J Indian Dent Assoc 1970; 42: 8-9.
9. Suzuki K, Tsuchihashi Y. Personal identification by means of lip print. J Forensic Med 1970; 17: 52-57.
10. Synder LM. Textbook of Homocide investigation. Identification of dead bodies. 1950. p. 65.
11. Utsuno H, Kanoh T, Tadokoro O, Inoue K. Preliminary Study of Postmortem Identification Using Lip Prints. Foren-sic Sci Int. 2005; 149: 129-132.
12. Vahanwahal SP, Parekh DK. Study of lip prints as an aid to forensic methodology. J Indian Dent Assoc 2000; 71: 269-271.

Evaluation of Blood Ethanol and Opium Level in Non Survived Drivers Due to Traffic Accidents

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ABSTRACT

Objective: Nowadays motor vehicle accidents are one of the commonest accidents with high mortality and morbidity rates, which due to induced disability and the need of various diagnostic and therapeutic methods inflict great economic loss on the society. Alcohol and illicit drug abuse and their side effects are of the most important underlying reasons for such accidents especially in the western countries. Due to safety precautions (seat belt and helmet) misuse, law disobedience, speeding, and slow driver's reaction in alcohol related accidents the consequences are graver and the mortality rates higher.

Methods: In this trial 105 cases, died in motor vehicle accidents, were tested for vitreous alcohol levels and gall bladder illicit drugs.

Results: 8.6% of which had alcohol positive test results and 42.9%had positive gall bladder illicit drug test results. In these trials a strong relationship between sex and education of the deceased and alcohol and opium usage was not found. But there was such a relationship between age of the deceased and alcohol and opium abuse. Also we found the same relationship between alcohol and opium abuse and the days of the week.

Conclusion: According to rampancy of alcohol and illicit drug abuse in deadly accidents, periodic opium tests and random alcohol tests in roads sounds effective in reducing the accidents and mortality rates.

Key words: *Dead Accident, Alcohol, Opium.*

INTRODUCTION

Vehicle accidents are the most common causes of accidents in modern societies which have been associated with high morbidity and mortality. Therefore the need for multiple diagnostic and therapeutic measures caused financial losses to society. Failure to observe traffic laws and to use safety equipment while driving; and drinking and driving are the most important predisposing factors.

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Alcohol abuse is a major cause of many accidents (road transport, domestic and industrial) all around the world[1].

In a study in Taiwan in 2011, approximately 60% of fatal car drivers and 40% fatal motorcycle drivers were because of alcohol consumption[2]. Alcohol was detected in the blood of 54.4% of traffic accidents victims in the study in turkey between 2005 to 2007[3]. In a study conducted in Spain in 1999, 285 cases of traffic accident were investigated and 50.5% of them had consumed alcohol with other drugs[4]. Another study was performed in Canada in 1993 and 35.5% of them had alcohol consumption[5]. More alcohol-related accidents in young people occur in the second and third decades of life[5-8] which resulted in high mortality of the young and efficient society will be lost.

Traffic accidents in addict people after using opium were higher than normal individuals[9]. Trauma and road accidents has been reported to be higher in people driving and using opium alone or in combination with other drugs in a study in Australia in 2008[10] and in Norwegian in 2007 [11].

Alcohol consumption in the various studies is correlated with several factors such as male sex, older drivers and their cultural [7]. Alcohol consumption is often associated with other drugs which Subsequent the use of safety devices like seat belts, helmets are oblivious. Failure to observe traffic laws, driving high-speed, slow down the reaction cause damages by alcohol-related traffic accident and their mortality rate is higher which This article has been cited in studies as well [8]. Alcohol consumption due decreased attention and concentration, delayed response in the necessary arrangements body, a delay in braking reflexes, impaired vision, drowsiness and impaired decision-making power, will cause traffic accidents[12]. In Iran such as many countries there are laws prohibiting alcohol and opium while driving. However objective studies based on measurement of plasma levels of alcohol and opium has been done in Iran. Given the high rate of traffic accidents in Iran, we decided to investigate serum levels of ethanol and opium in traffic accident patients. In order to identify the prevalence of alcohol and opium in traffic accidents that with better informations and a fuller view, the in order to reduce mortality from traffic accident to take.

MATERIALS AND METHODS

This cross sectional descriptive study was performed on the corpses of the drivers who referred to the Forensic Medicine Center of Tehran with the death due to traffic accident. The total numbers of samples were 105 cases which 102 were males and 3 females. The check list was prepared for data collection that included Epidemiological data such as age, sex, time of accident, type of accident, the culpability of drivers, education, and other demographic and associated injury. Questionnaires completed by two trained and experienced technicians, after corpse were brought to Forensic Medicine Center. This information was obtained from family of deceased. The vitreous fluid samples of corpse taken to measure alcohol level because the vitreous is sterile and during the time do not produce alcohol and bile samples, taken to measure opium by forensic experts. Then in laboratory by experts chromatography (TLC and GC) has been studied. Study included of cases that were driver. Because of Corruption corpse, bodies that have died over the 24 hours were excluded of our study.

RESULTS

This study was obtained on 105 cases of traffic accidents who were driver and were died. In this study, 97.1% were men and 2.9% were women. The average age (SD) was 33.2 ± 12.11. 54.3% were married and 45.7% were single. 77.1% had non-university education and the 22.9% had university education. According to report of police, 97% of those who died were the culprit in a traffic accident. Car accident was reported 37.1% and motorcycle accident was 62.9%. Most of the fatal accident, respectively, was on Thursday, Friday and Saturday. In this study 8.6% of dead drivers had positive test for alcohol and 42. 9% of them had positive test for opium. There was no positive test for both alcohol and opium simultaneously in the samples. There were not significant differences in the frequency of alcohol consumption between female and male drivers. The relationship between consumption of ethanol and age of deceased drivers was statistically significant (P value, 0.03) Despite the higher prevalence of alcohol and opium consumption in those who had lower education levels, this difference was not statistically significant (P value, 0.34 and 0.13 respectively) The prevalence of opium use in male drivers was more than female drivers which was not statistically significant (P value, 0.25).

The statistical analysis revealed that there is relationship between the use of opium and driver's age (P value, 0.02). The elderly drivers had a higher rate of opium use (%). On holidays, traffic accidents were higher (%) and there was significant relationship between opium use and times of the week. (P value < 0.001). Finally, the relationship between alcohol consumption and opium use and Blame for the accident was also significant (P = 0.02).

DISCUSSION

Results of this study showed that fatal cases were mainly young men, in age of 30-40 years. Western studied showed younger age of fatal drivers in road traffic accident[13,15]. A study which was done in Portugal showed that Accidents were preponderant in males, between 21 and 30 years-old[13]. Another study which was conducted in UK revealed that males between the ages of 17-24 were most likely to be involved in a road traffic accident[14].

There is increasing concern throughout the world about incidence of drugs in driving and its proportion to road trauma. In most countries, alcohol and cannabinoids were the most detected drugs in fatal crashes[13,15].

In this study 8.6% of dead drivers had positive test for alcohol and 42.9% of drivers had positive test for

opium. In a study in Australia it was found that almost 10% of cases involved both drugs and alcohol[15]. In addition to this In a study which was conducted in France, cannabis was found alone in 60% of injured driver cases and associated with alcohol in 32%[16], but It is important to note that there is no positive test for alcohol and opium for them simultaneously in this study. Moreover, fatally injured drivers in this survey had lower positive test for alcohol than other studies[13-16]. This is probably due Islamic law and legal prohibition of alcohol in Iran. In this survey there was relatively high positive test for opium in fatally injured drivers. Opium is produced mainly in Afghanistan which makes major social and health problems for countries, particularly Iran as the neighborhood[17]. Iran has a proved transit role in the opium trade and with other drugs from Afghanistan to western countries, makes an easy access to opium in Iran. Prevalence of opium users in fatal crash injury in this study was relatively high; it was much higher than western studies in this regard. In an Australian survey which evaluated opium level in killed drivers it was revealed that just 5% of them were opioids –positive[15]. Moreover this study showed that 65% of opioids-positive drivers were using other drugs such as benzodiazepine and cannabis. In this study, based on existing data, there was significant relationship between ethanol consumption and gender and days of the week. There was also significant relation between opium use and days of week and driver's age .In the most of other studies cannabis and stimulant drugs were the most frequently detected agents that caused traffic accidents. In Longo MC Study in 2000, there were no significant differences between males and females in the culpability of drivers[18] but in our study the culpability of males drivers was higher. Of course in our study numbers of female drivers were very low but the total of them were culpable in traffic accident. In another study by Drummer OH in Australia In 2004, there was no association of opium with culpability[19], but in our study there was significant relation between use of opium and car accident. The prevalence of drugs that associated with accident are different to other studies, because how to access drugs in countries is different and is associated with popular culture and legal regulations. In Ralph Hingson study in Boston, the serum level of alcohol was associated with car accident[6], which was similar with our study.

CONCLUSION

We concluded that consumption of Alcohol and opium and driving is one of the most important factors in creating severe traffic accident cause mortality. This may be due to the influence of alcohol and opium in alter of mental state and decrease of consciousness , attention, concentration, and decrease of reaction speed of body and impaired decision making. Therefore, drivers education and Random tests for opium and alcohol on the road can be an effective role in reducing traffic accidents and severity of accidents.

Limitation of the study: Of course, small sample size was an inevitable limitation of this study and thus performing a multicentre in larger group of cases is recommended. Moreover in this study we could not measure canabinoid and other drugs level, its measurement in fatal drivers cases in another study is recommended

REFERENCES

1. M Haddad, MD. Lester. Edition 3,1998, Saunders
2. Huang WS, Lai CH. Survival risk factors for fatal injured car and motorcycle drivers in single alcohol-related and alcohol-unrelated vehicle crashes. J Safety Res. 2011 Apr; 42(2): 93-9. Epub 2011 Mar 21.
3. Akgür SA, Ertas H, Altntoprak AE, Ozkan M, Kitapcoglu G. Prevalence of alcohol in blood samples from traffic accident cases in Turkey. Am J Forensic Med Pathol. 2011 Jun; 32 (2): 136-139.
4. Del Rio MC & etal. Alcohol use among fatally drivers in spain. Forensic Sci int. 1999; Oct 11: 104 (2-3): 117 -125.
5. Stoduto G, Vingilis E, Kapur BM, Sheu WJ, McLellan BA, Liban CB. Alcohol and drug use among motor vehicle collision victims admitted to regional trauma unit. Accid Anal Prev. 1993; Aug 25(4): 411-420.
6. Hingson R, Winter M. Epidemiology and consequence of drinking and driving. Alcohol Res 7- Margolis LH, Foss RD, Tolbert WG. Alcohol and motor vehicle – related death of children as Health. 2003; 27(1): 63-78.
7. Margolis LH, Foss RD, Tolbert WG. Alcohol and motor vehicle – related death of children as passengers, and bicyclists. JAMA. 2000; may 283 (17): 2245-2280.
8. Brewer RD, Morris PD, Cole TB, Watkins S, Patetta MJ, Popkin C. The risk of dying in alcohol–related automobile crashes among habitual drunk drivers. N Eng J Med. 1994; Aug 25/331 (8): 513-570.
9. Majdzadeh R, Feiz-Zadeh A, Rajabpour Z, Motevalian A, Hosseini M, Abdollahi M, Ghadirian P. Opium consumption and the risk of traffic injuries in regular users: a case-crossover study in an emergency department. Traffic Inj Prev. 2009 Aug; 10(4): 325-329.
10. Reece AS. Experience of road and other trauma by the opiate dependent patient: a survey report. Subst Abuse Treat Prev Policy. 2008 May 3; 3: 10

11. Engeland A, Skurtveit S, Mørland J. Risk of road traffic accidents associated with the prescription of drugs: a registry-based cohort study. Ann Epidemiol. 2007 Aug; 17(8): 597-602. Epub 2007 Jun 18
12. Voas RB, Tippetts AS, Fell J. The relationship of alcohol safety laws to drinking drivers in fetal crashes. Accid anal prev. 2000; Jul 32(4): 483-492.
13. Costa N, Silva R, Mendonça MC, Real FC, Vieira DN, Teixeira HM. Prevalence of ethanol and illicit drugs in road traffic accidents in the centre of Portugal: An eighteen-year update. Forensic Sci Int. 2011 Sep 9. [Epub ahead of print]
14. Elliott S, Woolacott H, Braithwaite R. The prevalence of drugs and alcohol found in road traffic fatalities: a comparative study of victims. Sci Justice. 2009 Mar; 49(1): 19-23.
15. Drummer OH, Gerostamoulos J, Batziris H, Chu M, Caplehorn JR, Robertson MD, Swann P. The incidence of drugs in drivers killed in Australian road traffic crashes. Forensic Sci Int. 2003 Jul 8; 134(2-3): 154-162.
16. Mura P, Kintz P, Ludes B, Gaulier JM, Marquet P, Martin-Dupont S, Vincent F, Kaddour A, Goullé JP, Nouveau J, Moulsma M, Tilhet-Coartet S, Pourrat O. Comparison of the prevalence of alcohol, cannabis and other drugs between 900 injured drivers and 900 control subjects: results of a French collaborative study. Forensic Sci Int. 2003 Apr 23; 133(1-2): 79-85.
17. Karrari P, Mehrpour O, Balali-Mood M. Iranian Crystal—A misunderstanding of the crystal-meth. J Res Med Sci. 2012(In press).
18. Longo MC, Hunter CE, Lokan RJ, White JM, White MA. The prevalence of alcohol, cannabinoids, benzodiazepines and stimulants amongst injured drivers and their role in driver culpability: part ii: the relationship between drug prevalence and drug concentration, and driver culpability. Accid Anal Prev. 2000 Sep; 32(5): 623-632.
19. Drummer OH, Gerostamoulos J, Batziris H, Chu M, Caplehorn J, Robertson MD, Swann P. The involvement of drugs in drivers of motor vehicles killed in Australian road traffic crashes. Accid Anal Prev. 2004 Mar; 36(2): 239-248.

Death Due to Diffuse Axonal Injury: A Case Report

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ABSTRACT

Head injuries due to various causes are commonly encountered by forensic pathologist, and most of the deaths due to head injuries have findings viz, fractures and intracranial hemorrhages. In the absence of either, Diffuse Axonal Injury (DAI) a type of traumatic brain injury, to be considered. DAI is one of the most common and devastating types of traumatic brain injury. A clinical entity which is difficult to demonstrate at routine autopsy, but by using special stains and techniques, a positive diagnosis of DAI can be made. A case of road traffic accident with head injury having died due to DAI is being presented.

Key words: *Road Traffic Accident, Cause of death, DAI.*

INTRODUCTION

Diffuse Axonal Injury (DAI), which refers to extensive lesions in white matter tracts, is one of the major causes of unconsciousness and persistent vegetative state after head trauma. It occurs in about half of all cases of severe head trauma and also occurs in moderate and mild brain injury.[3] The outcome is frequently coma, and over 90% of patients with severe DAI never regaining conscious.[2] Concussion may be a milder type of diffuse axonal injury. The majority of DAI cases documented have been due to traffic accidents and some due to falls from height. DAI is caused by angular or rotational acceleration of the victim's head. DAI represents approximately one half of all intra-axial traumatic lesions. DAI has been considered a primary-type injury, with damage occurring at the time of the accident. Research has shown that another component of the injury comprises the secondary factors (or delayed component). The degree of microscopic injury usually is considered to be greater than that seen on diagnostic imaging and the clinical findings reflect this point.

DAI is suggested in any patient who demonstrates clinical symptoms disproportionate to his or her CT-scan findings. DAI results in instantaneous loss of consciousness, and most patients (>90%) remain in a persistent vegetative state, since brainstem function typically remains unaffected. DAI rarely causes death and injured demonstrate multiple areas of injury on computed tomography (CT) scans.

CASE REPORT

History

On 30 .8.09 at around 11.15 pm a 45 year old female pillion rider on a motor cycle suddenly fell down and sustained head injury when her husband (rider) was trying to maneuver a speed breaker. She was unconscious for a brief period of 10 -15 minutes later regained consciousness at the spot of accident. No history of seizures or ENT bleeds. She was a known diabetic and on regular treatment.

Course in the hospital

Shifted to casualty where she vomited once, vitals were within normal limits, GRBS was found to be 174mg/dl, Cardio vascular system, respiratory system and - clinically NAD; CNS- GCS was 14/15. Conscious but confused, responding to commands with no focal neurological deficit.

Local examination

Contusion over right frontal- parietal region13x6cm, Lacerated wound over right upper eye brow measuring and 6x3cm, Multiple abrasions varying in size 12x5cm to 3x3cm at places over face, upper and lower limbs.

Investigations

Hematological and Urine investigations were normal Chest and Spine XRAYS- Shows normal study CT scan of Brain —Right frontal scalp hematoma, No acute intracranial hemorrhage or mass effect.

Patient was shifted to ICU for observation and treated with antibiotics, analgesics and diabetic treatment. After a short while, patient suddenly deteriorated with cardio respiratory arrest and hence was incubated and put on ventilator, despite all resuscitative measures patient succumbed to death at 4.40 am on 30.8.09.

Post Mortem Findings

External Examination

Dead body of an adult female aged about 45 years, 5 feet and 4inches in length postmortem staining over back, Rigor mortis present all over the body.

External injuries

Surgically sutured wound 4cms over outer aspect of right eyebrow, on dissection it was muscle deep.

Multiple abrasions varying in size 12x5cm to 3x3cm at places over face, upper and lower limbs Right eye contused.

Internal examination

Skull scalp-on reflection extravasations of blood over right fronto-parietal region

Brain and meninges- Intact and congested

C/s white matter–diffuse minute haemorrhages

All other organs were intact and congested

Organs were sent for HPE

Brain —oedematous and congested, white matter shows petechial haemorrhages

Other visceral organs - Intact and congested

Opinion as to cause of death

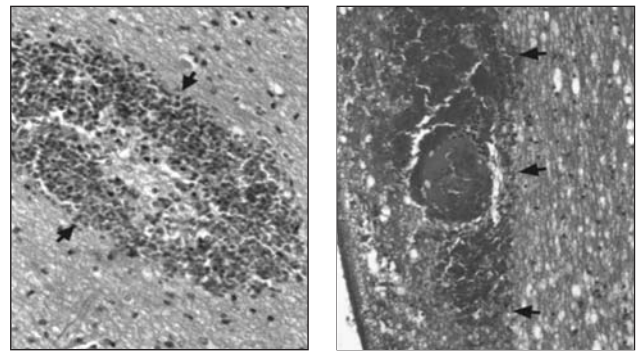
As the clinical findings, autopsy findings and histopathological findings were inconclusive opinion as to cause of death could not be ascertained so specimen of whole brain was sent to department of Neuropathology at NIMHANS Bangalore for further examination.

At department of Neuropathology NIMHANS brain was subjected for further examination by special staining techniques.

Department of Neuropathology, NIMHANS:

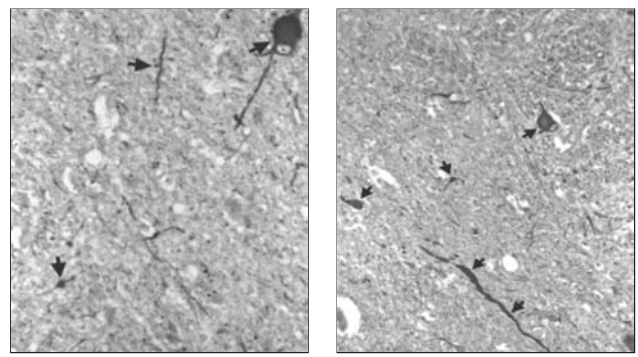
IMPRESSION

The gross and histopathological features are compatible with post traumatic diffuse axonal injury grade-1with multiple white matter hemorrhages.



A B

Fig. 1. A. Subventricular and B. Perivascular fresh hemorrhages secondary to rupture of thin walled veins following traumatic injury and torsion of vessel.



A B

Fig. 2. A Immunostaining with antibody to phosphorylated neurofilament showed labeling of dystrophic neurons. B: Irregularly dilated axons.

Opinion regarding cause of death

On perusal of autopsy findings, hospital case sheet extract and neuropath logical report it was opined as Death is due to Diffuse Axonal Injury consequent upon head injury sustained.

DISCUSSION

Diffuse axonal injury (DAI) that is, widespread damage to axons in the white matter of the brain, was originally defined by Strich' and the concept was expanded by Adams et al.[2]

A major advance in this field has occurred recently since Gennarelli and his group[9] have shown that similar clinical and structural changes can be produced experimentally in subhuman primates using non-impact controlled angular acceleration of the head in the absence of any increase in ICP or hypoxaemia. Adams et al[2] reported a detailed neuro pathological analysis of 45 cases and defined the characteristic features of DAI as (1) focal lesions in the corpus callosum and dorsolateral quadrant of the rostra brainstem adjacent to the superior cerebellar peduncles.

Diffuse axonal injury (DAI) emphasizes features that help to separate focal outer head trauma owing to blows and/or falls from angular acceleration head injuries associated with diffuse inner brain lesions Head injuries sustained in automobile accidents have been associated with diffuse brain damage characterized by axonal injury at the moment of impact. Victim of a motor vehicle accident shows post-mortem findings for both inner cerebral trauma and focal outer cerebral damage. The diffuse degeneration of cerebral white matter is associated with sagittal and lateral acceleration with centroaxial trauma and has a different pathogenesis from outer focal head trauma, typified by subdural hematomas and coup injuries. Unlike outer cerebral injury, over 50 percent of victims with diffuse axonal injury die within two weeks. These individuals characteristically have no lucid interval and remain unconscious, vegetative, or severely disabled until death. Compared to head trauma victims without diffuse axonal injury, there is a lower incidence of skull fractures, subdural hemorrhages, or other intracranial mass effect as well as outer brain contusions. Primary brainstem injuries often demonstrated at autopsy are seen in the reported victim. Diffuse axonal injury is produced by various angles of acceleration with prolonged acceleration/deceleration usually accompanying traffic accidents. Less severe diffuse axonal injury causes concussion[2].

Causes of Diffuse axonal injury

Diffuse axonal injury results from the brain moving back and forth in the skull as a result of acceleration or deceleration. Automobile accidents, sports-related accidents, violence, falls, and child abuse such as Shaken Baby Syndrome are common causes of diffuse axonal injury.

Classification

DAI is classified into grades based on severity of the injury. In Grade I, widespread axonal damage is present but no focal abnormalities are seen. In Grade II, damage found in Grade I is present in addition to focal abnormalities, especially in the corpus callosum. Grade III damage encompasses both Grades I and II plus rostral brain stem injury and often tears in the tissue.

Mechanism

DAI is the result *of traumatic* shearing forces that occur when the head is rapidly accelerated or decelerated, it usually results from rotational forces or severe deceleration.[10] The major cause of damage in DAI is the disruption of axons, the neural processes that allow one neuron to communicate with another. Tracts of axons, which appear white due to myelination, are referred to as white matter. Acceleration causes shearing injury, which refers to damage inflicted as tissue slides over other tissue. When the brain is accelerated, parts of differing densities and distances from the axis of rotation slide over one another, stretching axons that traverse junctions between areas of different density, especially at junctions between white and grey matter[1] Two thirds of DAI lesions occur in areas where grey and white matter meet[1].

Axons seldom rupture at the moment of injury. It is more common that it takes hours or a few days until the axons are detached. Areas most commonly affected are white matter in the hemispheres, corpus callosum and the brain stem. Half of the patients with severe head injury have diffuse axonal injury, but this type of injury also occurs in patients with moderate and mild head injury. The clinical presentation and prognosis will therefore vary. Diffuse axonal injury can present with typical signs revealed by CT, but the CT scan may also be normal, especially when there is no bleeding. New MRI techniques are more sensitive and show that diffuse axonal injury occurs more often than previously assumed. MRI is therefore necessary to give the patients correct diagnoses and adequate rehabilitation and follow-up.[4]

Stretching of axons during injury causes physical disruption to and proteolytic degradation of the cytoskeleton It also opens sodium channels in the axolemma, which causes voltage-gated calcium channels to open and Ca^{2+} to flow into the cell.[1] The intracellular presence of Ca^{2+} unleashes several different pathways, including activating phospholipases and proteolytic enzymes, damaging mitochondria and the cytoskeleton, and activating secondary messengers, which can lead to separation of the axon and death of the cell. Misalignment of cytoskeletal elements after stretch injury can lead to tearing of the axon and death of the neuron. Axonal transport continues up to the point of the break in the cytoskeleton, but no further, leading to a buildup of transport products and local swelling at that point. When it becomes large enough, swelling can tear the axon at the site of the injury Sections of the cerebrum, cerebellum, and brain stem of a rugby player who died 15 hours after being tackled were stained using an immunoperoxidase technique to detect beta-amyloid protein.[6]

IMMONOHISTO CHEMISTRY TECHNIQUES

The conventional histological techniques of (Hand E) and silver impregnation it is not possible to identify with certainty axonal bulbs until about 15 hrs after

injury. The advent of immunohistochemistry has greatly facilitated the diagnosis of DIA on frozen brain tissue and paraffin embedded material.

Transport proteins for axonal damage (anti ubiquitin235 and anti 68, 170 and 200kda neurofilament protein) have suggested.

Immunostaining for Beta —app produced the most sensitive and reliable staining of axonal damage[7].

CONCLUSION

Diffuse axonal injury is a distinct form of head injury, induced by direct external forces at the time of the trauma, and not produced by secondary changes due to a primary injury. This type of lesion may be without conspicuous findings on gross brain examination, or may be found with coexisting conventional types of brain injuries[8]. It is characterized by diffuse retraction balls (axonal swellings), hemorrhage or laceration of the corpus callosum, and hemorrhages in the brain stem. It is of utmost importance for forensic pathologists to be aware of this little-recognized entity[9]. Diffuse Axonal Injury is primarily a non-impact rotational acceleration-deceleration phenomenon, deformation by stretching being the most important factor.

One third of our cases in whom DAI was found at necropsy were not permanently unconscious from the time of injury and hence in such cases where cause of death is obscure at first, subjecting the brain for neuropathology should be considered.

REFERENCES

1. Wasserman J. and Koenigsberg R.A. (2007). Diffuse axonal injury. Emedicine.com Retrieved on 2008-01-26.
2. Parker JR, Parker JC Jr, and JC Overman Intracranial diffuse axonal injury at Autopsy Annals of Clinical and Laboratory Science, Vol 20, Issue 3, 220-224.
3. Peter C Blumbergs, Nigel R Jones, John B North. Diffuse axonal injury in Head trauma Journal ofNeurology, Neurosurgery, and Psychiatry1989; 52: 838-841.
4. Vik A, Kvistad KA, Skandsen T, Ingebrigtsen T. Diffuse axonal injury in traumatic Brain injury]. Nevrokirurgisk avdeling, St. Olavs Hospital, 7006 Trondheim. anne.vik@ntnu.no.
5. Takeshi, Challener, Robert Carroll, Roessmann, Uros.. Diffuse Axonal Injury by Assault American Journal of Forensic Medicine & Pathology: March 1999 - Volume 20 - Issue 1 - pp 70-72.
6. Blunt Head Injury, Greenfield's Neuropathology, Eighth Edition, Oxford University Press, 2008, page854-870.
7. Dolinak David. Forensic Pathology, Elsevier Publications, New York. Pg 444.
8. Knight Bernard, Saukko Pekka. Forensic Pathology, 3rd Edn, Edward Arnold, 2004; 210 -211.
9. Sanders M.J. and McKenna K. 2001. Mosby's Paramedic Textbook, 2nd revised Ed. Chapter 22, "Head and Facial Trauma." Mosby.
10. Shepherd S. 2004. Head Trauma. Emedicine.com. Retrieved on 2008-01-17.

Investigating the Force Relative to Blood Stain Size and Pattern

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ABSTRACT

"Every contact leaves a trace"- Dr. Edmond Locard (1877–1966), all trace evidences represents a characteristic feature in reconstructing the crime scene. Mostly in crime scenes bloodstains can be found these stains can occur when blood falls passively due to force being applied to a body and these tiny droplets are helpful in reconstructing the crime scene. The aim of this research is to investigate the force relative to the bloodstain size and spatter pattern using a force sensor and propose new parameter in categorizing the type of spatter. In order to achieve these aims a set of preliminary experiments, gravity drop and impact spatter were conducted. In gravity drop, the width of the stains under different heights was studied and in impact spatter experiment, a known mass was dropped from different heights and spatter patterns were interpreted. Further sets of experiments were performed with the help of force sensor, which could give value of force in Newton (N). There were variety of weapons used for this project, namely wooden hammer, metal hammer and metal rod. In conclusion of this research, three different ranges of force Mild/Moderate/Severe were defined by interpreting the spatter pattern and stain size for single and multiple blows. Thus, investigating the force of impact based on the stain size, angle of impact and spatter pattern is not conclusive for the judgments. However, we can categorize the type of attack either mild/moderate/severe based on the stain size and spatter pattern that can be helpful in supportive of an argument during trial.

Key words: *Bloodstain Pattern Analysis, BPA, Physics of Blood Pattern Analysis.*

INTRODUCTION

Human Blood is a liquid connective tissue that serves two primary functions; transport and defense. The role of Blood in carrying oxygen to the cells and carbon dioxide to the lungs for disposal through respiration is probably the most well known function. Blood is also responsible for transporting food in form of glucose, lipids, and amino acids from the alimentary canal to the cells. On average, blood accounts for 8 % of total body weight. It varies in both the sexes, 5 to 6 liters of blood for males and 4 to 5 liters of blood for females.

Drop Dynamics

A drop of blood will form a sphere due to attraction of molecules to one another. Blood drops in flight will oscillate, but will quickly damp and form a sphere [1].

Blood Spatter Analysis in Crime Scene

Blood spatter analysis (BPA) focuses on the analysis of the size, shape and distribution of bloodstains resulting from bloodshed events as a means of determining the mechanism of the attack. Conjugating this information with the DNA individualization and wound interpretation from the autopsy examination of the victim by the forensic pathologist provides the basis for the reconstruction of the crime scene involving bloodshed events"[2].

Blood spatter analysis (BPA) is a discipline that uses the fields of biology, physics and mathematics. BPA may be accomplished by direct scene evaluation and /or careful study of scene photographs in conjunction with detailed examination of clothing, weapons and other objects regarded as physical evidence. BPA requires some analytical skills for instance; in a murder scene a forensic blood spatter analysis should reconstruct what could have happened, and how it could have happened, based on the bloodshed. BPA may provide information to the investigators in many areas[2]. Area of convergence, type and direction of impact that produced bloodstains or spatter, mechanisms by which spatter patterns were produced, assistance with the understanding of how bloodstains were deposited onto items of evidence, possible position of victim, assailant, or object at the scene

during bloodshed, possible movement and direction of victim, assailant, or objects at the scene after bloodshed, support or contradiction of statements given by accused and/or witness, correlation with other laboratory and pathology findings relevant to the investigations. Faster drop are generally larger in width (size) and bloodstain with smaller size generally travel lesser distance. The size of the droplet depends upon the speed and in turn speed depends on the amount of force applied[4].

Modern Researches in Blood Spatter Pattern

In 1983, the IABPA (International Association for Bloodstain Pattern Analysis) was formed. The association's stated purpose is to promote the general knowledge, techniques, and understanding of bloodstain pattern evidence[5].

In 2001, Anita Wonder published her book, Bloodstain Dynamics. Although from an overall viewpoint this work is a significant resource, Wonder included issues that were to say the least, controversial. This included the claim that she could differentiate the nature of spatter based on a single stain from any spatter pattern. Despite some of these more controversial issues, the book remains an excellent resource for the discipline[6].

Bloodstain pattern analysis has a nearly 150 year documented history; and as important as the length of that history is, just as important is the fact that it is a consistent history. Long recognized for its ability to support the evaluation of scenes of crime, bloodstain pattern analysis serves the investigator by illuminating "what happened." It cannot tell us in all cases "who." But, BPA helps to reconstruct the crime scene based on the available data. Any useful parameter that can accompany along with the already available data, in reconstructing the crime scene and bringing out a verdict for unsolved blood mysteries is appreciable. From the historical perspective and modern scientific researches can understand that bloodstain pattern analysis unfolded for ages and still it is[7].

Investigating Force

The researches undergone so far are related to angle of impact, velocity of the bloodstain, point of convergence, dryness of bloodstain. But there are no researches undergone to investigate the force of the attack. My aim in this research is to establish a relation between forces with relative to stain size and spatter pattern. In my opinion different forces of impact will have different spatter pattern as well as stain size. So, I defined force ranges based on the spatter patterns as 'Mild/Moderate/Severe' force of impact by practically experimenting with force sensors by using different weapons. It helps in reconstructing the Crime scene with more information. Currently a bloodstain pattern analyst can interprets angle of impact, point of convergence, and number of blows/strikes and mechanism of action. A new parameter 'force' in categorizing the spatter is an added advantage. It can give the type of attack (mild/moderate/severe). As velocities of blood stain are categorized into low/medium/high depending upon the stain size and distance travelled. One can categorize the bloodstain size and spatter pattern based on Force. It can helpful at crime scenes. In solving the crime scene forensic scientists should unfold more mysteries. It can be helpful during the court trial, in supportive of verdict whether the type of attack is mild/moderate/severe.

MATERIALS AND METHODS

ImageJ

ImageJ is a public domain Java image-processing program developed by NIH (National Institute of Health) runs on Windows, Mac OS, Mac OS X and Linux. It can display, edit, analyze, process, save and print 8-bit, 16-bit and 32-bit images. It can read many image formats including TIFF, GIF, JPEG, BMP, DICOM, FITS and "raw". It supports "stacks", a series of images that share a single window. It can measure distances and angles. It can create density histograms and line profile plots. It supports standard image processing functions such as contrast manipulation, sharpening, smoothing, edge detection and median filtering. It does geometric transformations such as scaling, rotation and flips. Image can be zoomed up to 32:1 and down to 1:32. All analysis and processing functions are available at any magnification factor. The program supports any number of windows (images) simultaneously, limited only by available memory. These features make easy for the bloodstain pattern analysts to measure the stain size that are too small or too wet. The snapshot below shows the options available for the ImageJ user.

Force Plate

The Force plate is a force sensor, which is manufactured by "Instruments direct [Service] Ltd, UK. Force Plate and it is a large force sensor tough enough to jump on. About the size and shape of bathroom scale, the Force plate has two ranges one reading up to 3500 N and another up to 800 N. The range varies with -800 to 3500 N and -200 to 800 N. The output is presented in graphical format and

resulted force values are interpreted easily from the graph. Force plate has an external USB output that enables us to connect with a computer. The application which links the force plate and computer is 'Go-link' which was provided along with the force plate kit. After installing the software go-link in the computer the force plate is ready to use. Before beginning the experiment the force plate was calibrated.

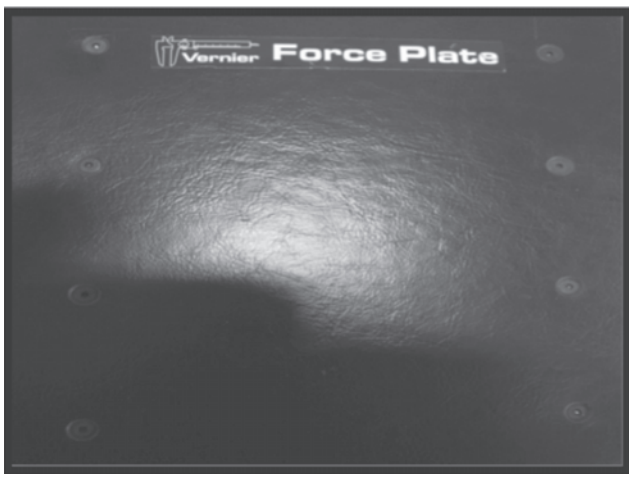

Fig. 1. Fore plate

The force plate is constructed from a base plate and a top plate. The force plate is equipped with two long force sensors on either side. Any force applied to the force plate will distort the sensors, change their resistance and provide the force output value in a graphical format. Force sensors are located between the two plates, when a force is applied to the top plate the sensors are compressed. When strike the force plate the two plates are compressed and can see a positive force reading proportional to the force with which the plate was struck. The force plate then tends to lift up and the two plates will pull, giving a negative reading[8].

WOODEN HAMMER IMPACT PATTERNS

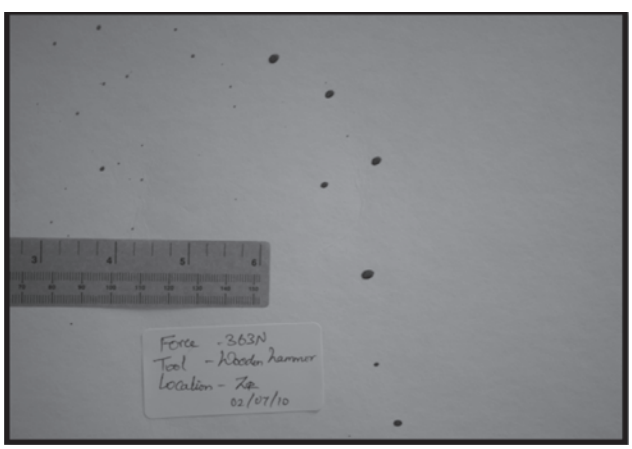

Fig. 2. Mild force

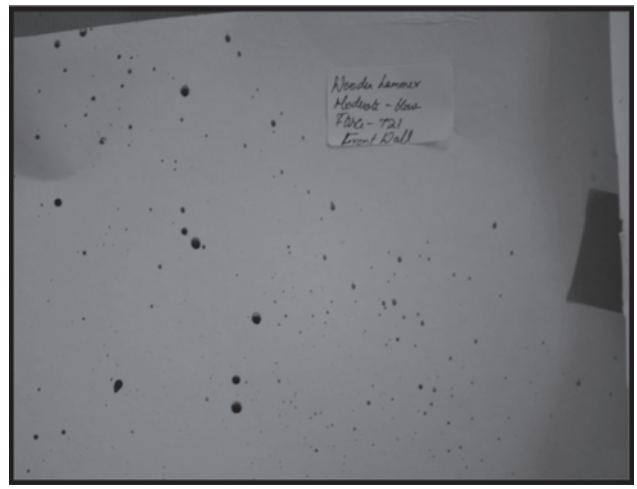

Fig. 3. Moderate force

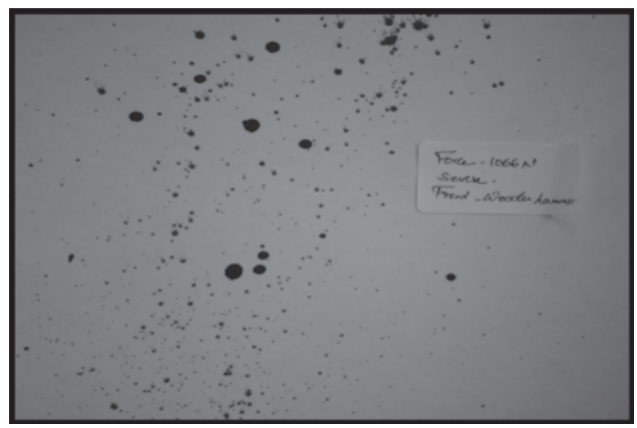

Fig. 4. Severe force

In case of mild attack force, the spatter pattern seems to be clear, in other words there are no mist of blood stains. There are either elliptical shape stains or sphere shaped stains. But no mist of blood spatter pattern found anywhere on the walls. The more elliptical the stain, the more acute is the angle of impact. Round stains indicate the impact was closer to 90 degrees. For a stain to attain a perfect round shape the angle of impact has to be 90 degree. The force output 721N from the wooden hammer is categorized as moderate blow. From the previous pattern for mild force. The difference in pattern from mild to moderate is because the force of impact on the pool of blood overcomes the surface tension and viscosity of the blood. Hence, immediately after the blood receives the force, it spatters widely into tiny droplets. The higher the force, more the droplets are acute, more the acute a stain, more it is elliptical in shape. The force output of 1066N from wooden hammer is categorized to be severe impact. Minimum angle of impact of stains in severe blow is 8 degrees on the walls. The spatter pattern for severe blow is unique, I observed more droplets measuring acute angle. In severe blow, there

were mist of blood stains observed, which has width and length lesser than 1mm.

METAL HAMMER IMPACT PATTERNS

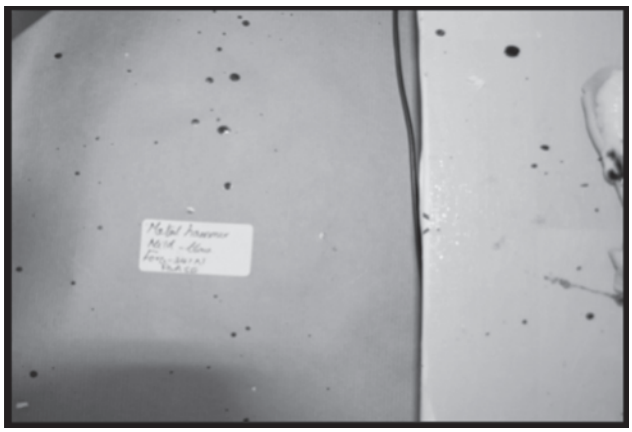

Fig. 5. Mild force

Fig. 6. Moderate force

Fig. 7. Severe force

The mild attack from metal hammer observed to be slightly different from that to wooden hammer mild blow, 341 N. The reason for the divergence is due to the surface area. The surface area of wooden hammer is larger than that of metal hammer. The angle of impact ranges from 20 degrees. The force output of 439N from metal hammer is categorized as moderate blow. In case of mild attack the maximum angle of impact attained was 74 degrees. As mentioned in my earlier interpretation, increase in the force results more acute angle stains. The increase in the amount of force causing the blood droplets to form more acute angle stains on the walls and more obtuse angle stains on the ground.There is a decrease in the angle of impact for increase in force from mild to moderate. For the force value 527 N from metal hammer is categorized as severe blow. In case of severe blow, it has been observed that the blood droplets forms acute angle I observed fine mist of blood spatter patterns on the wall. This shows that the force of impact by the weapon on the target surface is severe. The higher the force received by blood, the lesser the angle of impact made by the droplets. If the angle of impact is obtuse then the force of impact is either mild or moderate. In case of severe blow, the distance travelled by the stains are higher; because of the severe force of impact on the pool of blood.

Fig. 8. Mild force

METAL ROD IMPACT PATTERNS

The force value 375N is categorized as mild attack; the spatter pattern for metal rod mild blow is similar to that of wooden hammer mild attack. The angle of impact of stains of mild force for metal rod mild is above 30 degrees.The force value 490 N from metal rod is categorized as moderate blow.Hence the increase in the force for all causes decrease in the angle of impact more towards acute angle. The range of angle of impact for the stains on the right, left and front walls were between 20 degrees to 55 degrees.The force value

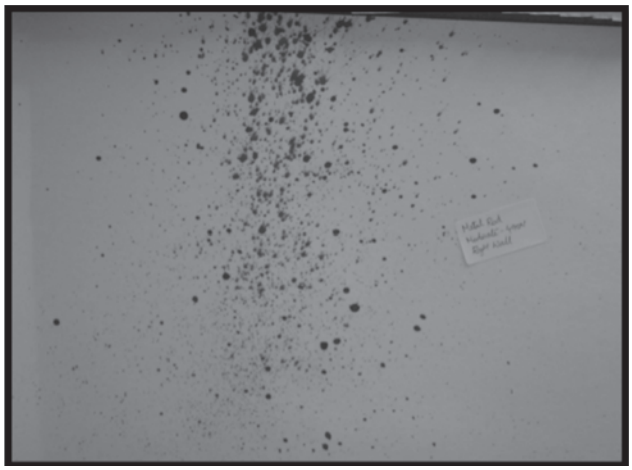

Fig. 9. Moderate force

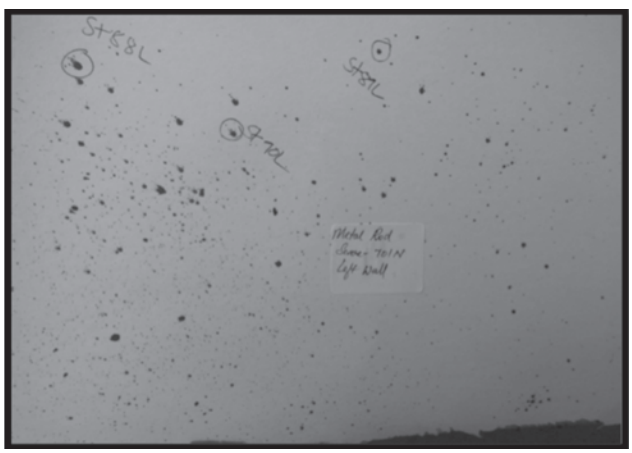

Fig. 10. Severe force

701 N is categorized as severe blow for the metal rod. In the figures above, the spatter pattern for the severe blow is similar to that of gunshot spatter patterns. The spattered area covers the full wall in both right and left walls. In case of moderate blow previously, I observed that the spattered area covers only specific area with more fine mist of blood droplets. This is because when blood receives a large amount of force (say severe), they break into tiny droplets and travel from one place to another when the final velocity is zero they finally come to rest and reach the target surface (obstacles).

FINDINGS

The range of Force for mild/moderate/severe attack are defined as follows, the attack which applies mild force is categorized as mild attack in my research. Then the slightly higher force is categorized as moderate attack, lastly the higher or brutal force, especially causing death of a person/fractures/bone dislocation with large number of blood droplets on the wall is defined as severe attack.

The range of force for the weapons used in the my research are as follows;

Table 1 – Force ranges for wooden hammer, metal hammer and metal rod

TOOL	MILD (Newton)	MODERATE (Newton)	SEVERE (Newton)
Wooden hammer	Upto 533	533-700	800 above
Metal hammer	Upto 350	350-500	500 above
Metal rod	Upto 365	365-550	550 above

The above values are recorded by experimentally with the Force sensor used those values are based on the output of the sensor for the respective blows. The range value may differ for the different sensors. The range may differ but the spatter pattern will be similar. Following are the graphical representation of the weapons used to describe the force range in the above.

CONCLUSION

In concluding my research from preliminary stages of experiments gravity drop and impact spatter, I observed the change in height has an effect on the spatter pattern and spatter area, as the height increases the spatter area increases. When the investigation of force was accompanied by the force sensor, I inferred that for single blow, the increase in force from mild to severe decreased the angle of impact for the droplets or in other words the angle of impact is more towards acute for increase in force. There were more elliptical stains for higher amount of force. For multiple blows, there was no single force value, but I observed the attack was moderate initially, later it reaches the severe or brutal force and ends up with mild force due to the energy dissipating for the assailant. Hence, the multiple blows have ranges of force values as I observed from the force sensor output for multiple blows. The spatter pattern for the multiple blows were unique and I observed fine mists of blood, similar to high velocity impact spatter pattern as in case of gunshot spatter. The variety of weapons used in my research gave specific spatter pattern corresponding to the force. From my research I observed that there is an effect on blood stain size and spatter patter for different forces of impact. Thus, investigating the force of impact based on the stain size, angle of impact and spatter pattern is not conclusive for the judgments. However, it is possible to categorize the type of attack either mild/moderate/severe based on the stain size and spatter pattern, which can be helpful in supportive of an argument during trial.

ACKNOWLEDGEMENT

I sincerely express my gratitude to faculty members Prof Michael Cole, Dr.Trevor Emmet, Anglia Ruskin University for granting permission to carry out my research and for sanction of the sensor, which was pivotal for my research work.

I earnestly thank ALL our department staff members for their valuable guidance provided in completing the work successfully.

Last but not least I thank my parents, family members and friends who have been my moral support in completing this work successfully.

REFERENCES

1. Jim, B., 1990 2nd ed. Understanding Physics for advanced level. Musselburgh: Stanley Thornes (publishers) ltd.
2. Alkin, L., 2005. Blood spatter interpretation at crime scenes [online] Available at: http://findarticles.com/p/articles/mi_go1613/is_2_14/ai_n29186570/ [accessed 10 March 2010].
3. James, H., et. al., 2005. Principles of Bloodstain pattern analysis theory and practice. Boca Raton: CRC press.
4. Tom, et.al 2008. 3rd ed. Bloodstain pattern analysis with an introduction to crime scene reconstruction. USA:CRC press.
5. IABPA, 2008. Recommended BPA Terminologies. [online] Available at: <http://iabpa.org/June% 20% 202008% 20final.pdf> [Accessed June 2010].
6. Wonder, A., 2001. Blood dynamics. London: Academic Press.
7. Louis L., et. al 2004. Directional analyses of blood spatter at crimes scenes and accident scenes for the private investigator.
8. Annette, M., annette@inds.co.uk, 2010. Force plate.[email] Raghuraghavendra Sundarrajan (ragavendrainfoz@gmail.com). Sent Thu, July 22, 2010 at 12:27 PM Available at: <https://mail.google.com/mail/?shva=1#search/Annette%40inds.co.uk/129f9e8be26e746e> [Accessed 22 July 2010].

Analysis of Body Fluids by Head Space-Gas Chromatography (HS-GC) in Illicit Liquor Poisoning

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ABSTRACT

Postmortem, ante mortem blood samples and gastric contents in a case of poisoning by consuming homemade liquor (gudanji) were analyzed to ascertain the volatiles responsible for poisoning. Head Space-Gas Chromatography (HS-GC) highly sensitive technique was used for separation of volatiles and quantitative estimation of ethyl alcohol and methyl alcohol. Gas Chromatograph Clarus 500 coupled with Turbo matrix Head space sampler with gases of highest purity from gas generators Ptl were used in the study. Maximum concentration of ethyl alcohol (689.0871mg %) was observed in the gastric content and methyl alcohol (98.7445mg %) in ante mortem blood of subject-2. Attempt was made to identify the volatiles and to compare the chromatogram of volatiles with chromatogram of water extract of empty container recovered by police from the shed where subjects has consumed home made illicit liquor (gundanji)

Key words: *Illicit liquor, poisoning, methanol, volatiles analysis.*

INTRODUCTION

Alcohol is one of the most widely used psychoactive substances known to man; it has apparently been with us since the dawn of civilization. Breweries flourished in Egypt almost six thousand years ago and there is evidence that Stone Age pre-historic man made alcoholic beverages long before that. The use of alcohol has appeared in varying degrees in most societies throughout recorded history and has traditionally played an important symbolic as well as pharmacological role in many social, religious and medical practices and customs. As the use of alcohol has been almost universal, so has its misuse. What is this drug which has been hailed as the water of life and nectar of the gods by some, and termed by others as second only to war as a source of human problems? Made up of three common elements, carbon. Hydrogen and oxygen, ethyl alcohol (C_2H_5OH) is a colorless, inflammable and volatile liquid. The word alcohol by itself is usually taken to mean ethyl alcohol, although there are a vast number of other substances in the aliphatic alcohol family. Many of which are highly toxic. Methyl alcohol (CH_3OH) is one of the member of this family and also known methyl alcohol, carbinol, wood alcohol, wood naphtha or wood spirit. It is simplest alcohol and is volatile, colorless, flammable, poisonous liquid with a distinctive odor that is some what milder and sweeter than ethanol. Methanol is not intended for human or animal consumption. However it finds its use in products like anti-freeze, canned heating sources like varnish, windshield wiper fluid, paint thinner duplicating fluids gasoline additives, ethanol denaturants, in analytical work and fuel additives [1]. Methanol is a colorless, volatile liquid with the structural formula CH_3OH and a molecular weight of 32. Miscible in water, ethanol and ether and has a specific gravity of 0.81, similar to ethanol (0.79). Methyl alcohol is a cheap and potent adulterant of illicit liquor. Poisoning usually follows ingestion of contaminated alcohol beverages or methylated spirits. Many out break have occurred in different part of country taking heavy tolls of mortality and morbidity. Although trace amount of methyl alcohol is produced by fermentation it does not occurred at a concentration capable of poisoning an individual. In fact such trace amount methyl alcohol is removed from spirits by distillation, but remain in undistilled beverages like beer and wine. Very little methyl alcohol is produced by fermentation, so it poses no threat to consumer of beer and wine. In the event of poor distillation where it may not be completely removed, it still poses no more threat. There are cases and reports that some people went blind or died from drinking homemade liquor. In such circumstances preparations are always made by concocting some kind of a punch using wood alcohol or methylated spirit. Adulteration of cheap liquor by methyl alcohol is common in India. In 1971, 90 deaths were reported from Khopoli due to methanol

poisoning. Subsequently 100 deaths from Hyderabad, 20 from madras and more than 300 deaths from Banglore have been reported [2]. There was a similar outbreak in 1983; when 47 cases were admitted to King Edward memorial hospital, Bombay [3]. Twenty eight cases out of 97 cases of methyl alcohol poisoning admitted to KEM Hospital in August 1988. [4]. Thirteen Cases of fatal poisoning of liquor adulterated with methyl alcohol in Una Distt of Himachal Pradesh were reported by this Laboratory in the year 1997. Recently six cases of Chamba Distt have been reported. Six persons consumed local illicit liquor prepared from gud known as gudanji in local area. The development of symptoms was observed after 12-48 hours such as blurred vision, stomach pain; difficulty in breathing and fatique. As per history three people died after receiving primary treatment and information of other three is not known.

MATERIALS AND METHODS

Subject-1 a male aged about 49 years consumed illicit liquor on 14.6.07 died on same day. Postmortem of deceased was conducted on 17.6.07 and finding indicates congestion of mucous membrane, kidney, lungs and brain and time between death and postmortem was 24 to 36 hours. Subject 2, 3 and 4 consumed three bottle of illicit liquor on 14.6.07 evening and their condition deteriorated on 15.6.07 and were admitted in CHC Bharmour at 9PM on 16.6.07. Subject- 1, 2 and 3 has history of consuming large quantity of illicit liquor.. Subject 2 was a male aged about 30 years; symptoms observed during primary treatment were blurring of vision, and later shifted to Distt Hospital and died on the way. .Post mortem of deceased was conducted on 17.6.07 at 12.45pm finding indicates congestion of lungs and time between death and postmortem was 12-36 hrs. Subject- 3 a male aged about 40 years was admitted in CHC Bharmour on 15.6.07 at 9.30pm in unconscious condition gastric contents and blood samples was preserved. Subject died on 15.6.07 and postmortem was conducted on 17.6.07 at Distt Hospital Chamba autopsy finding indicates congestion of brain and lungs, time between death and postmortem was 24-42 hrs. Subject- 4 a male aged about 36 years was admitted in CHC Bharmour on 16.6.07 at 12.10pm blood sample was preserved. Subject-5 a male aged about 55 years was admitted in CHC Bharmour on 16.6.07 at 1.30pm blood sample was preserved. Subject-6 a male aged about 34 years was admitted in hospital on 16.6.07 at 1.50pm blood sample of subject was preserved. Viscera and body fluids of deceased and subjects under treatment were preserved by doctor for analysis.

Viscera of three subjects and postmortem blood sample of subject-1 and ante mortem blood samples and gastric contents of subject-2 and 3 and ante mortem blood samples of subject- 4, 5 and 6 were sent for analysis. Viscera, blood samples (ante mortem and postmortem), gastric contents of subjects were received in laboratory on 25.6.07 kept under refrigeration and analysis of samples was carried out on 27.6.07.

Chemical used were of AR grade and ethyl alcohol (Merck) and deionized water of Elix-3 Milli-Q system of Millipore was used in analytical preparations. Gas Chromatograph CLARUS 500, TURBOMATRIX Head Space sampler of Perkin Elmer and gases of highest purity from gas generator of Ptl were used in analysis work

Blood and gastric contents selected for standardization was subjected to HS-GC analysis in order to ensure that no ethanol and other volatiles are present in the blood matrix. The effect of small variation in the matrix is eliminates by using internal standard. Different concentrations of methyl and ethyl alcohol (Merck) 100mg, 200mg, 300mg, 400mg, 500mg% w/v were prepared in deionized water and internal standard (TBA) tert-butyl alcohol 4ml (80mg/L) was added to 0.5ml of blood in five vials. Vials were sealed placed in HS auto sampler, thermostated at 70°C for 10 minutes, vapor injected and analyzed. The conditions for GC and HS are given below.

GC Parameters-:

Column	Capillary Elite BAC2 30m x 0.32mm x 1.20 ums
Column temperature	50 °C
Detector	FID
Detector temperature	175 °C
Injector temperature	60 °C
Carrier gas flow	15 ml/minutes.
Zero air flow	450 ml/minutes
Hydrogen gas flow	45 ml/minutes
Attenuation	4
Range	1
HS Parameters	
Oven temperature	70 °C
Needle temperature	75 °C
Transfer line temperature	110 °C
Pressurization time	3.0 minutes
Thermo stating time	10.0 minutes
Withdrawal time	0.5 minutes
Injection time	0.04
Injection volume	1ul

Method development was carried out on Total Chrome software by selecting global parameters, editing components, assigning internal standard to each components, calibrating and assigning different levels of ethyl alcohol in order to get fit type multilevel linear graph (peak area vs. different levels). This constitutes a method for analysis of blood samples. Similarly internal standard (TBA) tert butyl alcohol

was added to blood samples, gastric contents of different subjects and subjected to HS-GC by using above parameters. However head space sensitivity can be increased by adding salts such as ammonium sulphate, sodium chloride, ammonium chloride, potassium carbonate and sodium citrate to the blood samples but this study has been carried out without addition of salt.

RESULTS

Methyl alcohol was detected in viscera of subjects - 1,2 and 3; however quantitative estimation was carried only on body fluids of the subjects. Postmortem blood sample of subject-2 was not supplied and gastric contents and ante mortem blood of subject-2 and 3 were supplied for analysis.

Fig. 1 shows the chromatogram of water extract of container recovered from spot and postmortem samples of subject-1. Peaks at RT (3.50, 4.39 and 5.0) were observed for, methanol, ethanol and isopropanol in water extract of container and peaks at RT (3.50 and 4.39) for methanol and ethanol in postmortem blood sample. There is no peak of isopropanol in postmortem blood sample. Fig. 2 shows the chromatograms of ante mortem blood and gastric contents of subject-2. Peaks for formaldehyde, methanol, ethanol and isopropanol were observed at RT (2.57, 3.48, 4.36 and 4.97) in ante mortem blood samples. Gastric content of subject shows peak at RT (2.57, 3.48, 4.35 and 4.97) for formaldehyde, methanol, ethanol and isopropanol. Fig. 3 shows the chromatogram of subject-3. In postmortem blood no peaks were observed while ante mortem blood indicates peaks at RT (3.10, 3.49, and 4.38) for acetaldehyde, methanol and ethanol. Fig. 4 shows the chromatogram of gastric contents of subject-3, peaks were observed at RT (2.57, 3.48, 4.37, and 4.97) for formaldehyde, methanol, ethanol and isopropanol. Antemorten blood of subject-4 in Fig shows peaks at RT (3.49) for methanol, Fig. 5 shows the chromatogram of ante mortem blood samples of subject-5 and 6 at RT (2.57, 3.48, 4.37 4.97,98 for formaldehyde, methanol, ethanol and isopropanol. Formaldehyde was observed in ante mortem blood and gastric content of subject- 2, gastric contents of subject-3, ante mortem blood of subject-5 and 6. Peaks of isopropanol were observed in container, ante mortem blood of subjects- 2, 5, 6, gastric contents of subject-2 and 3.

Table 1 shows the quantity of ethanol, methanol in postmortem, ante mortem blood samples and gastric contents of the subjects. Variable concentration of ethanol and methanol is shown by different subject. High concentration (689.0871mg%) of ethanol in gastric contents of subject-2 may arise as result of

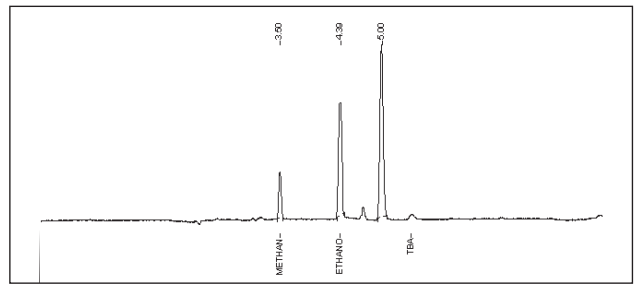
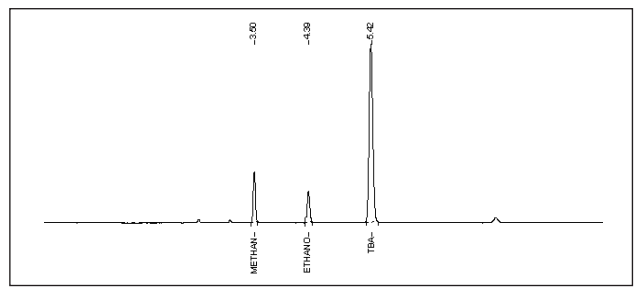


Fig. 1. Chromatograms of water extract of empty container, methanol (RT-3.50), ethanol (RT-4.39) isopropanol (RT-5.0)



Chromatogram of postmortem blood sample of subject-1 Methanol (RT- 3.50) and Ethanol (RT-4.39).

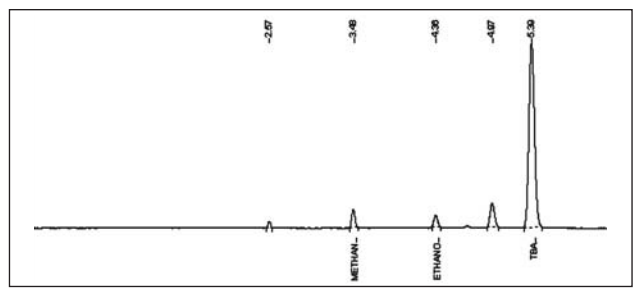
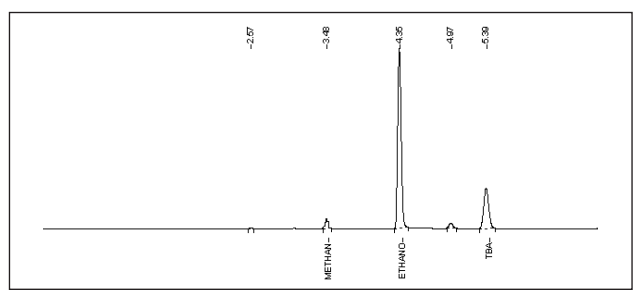


Fig. 2. Chromatogram of ante mortem blood sample of subject-2 formaldehyde (RT-2.57), methanol (RT-3.48), ethanol (RT-4.36), isopropanol (RT-4.97)



Chromatogram of gastric contents of subject- 2, formaldehyde (RT-2.57), methanol (RT-3.48), ethanol (RT-4.35), isopropanol (RT-4, 97)

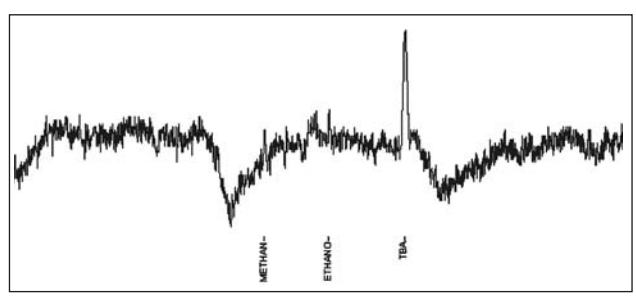
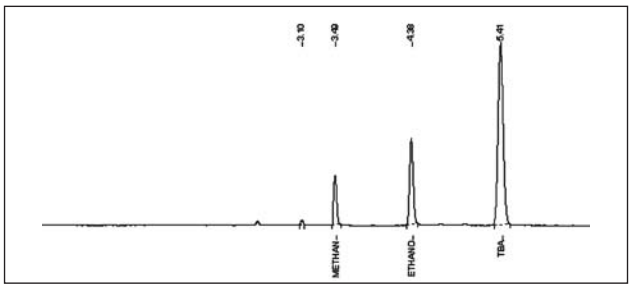


Fig. 3. Chromatogram of postmortem blood sample of subject-3 No defined peak of Methanol and Ethanol.



Chromatogram of ante mortem blood sample of subject-3 Acetaldehyde (RT- 3.10), Methanol (RT- 3.49) Ethanol (Rt-4.38)

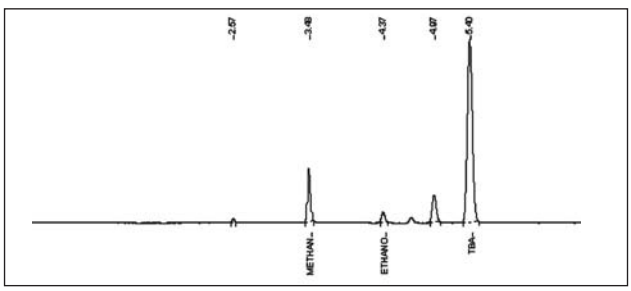
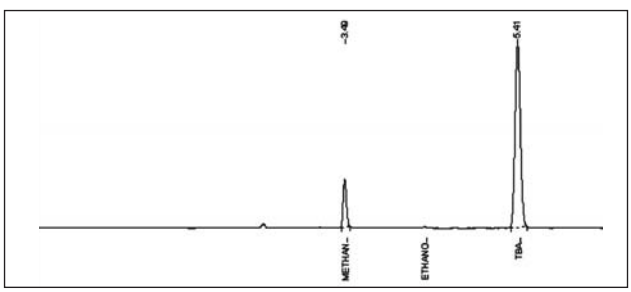


Fig. 4. Chromatogram of gastric content of subject-3, formaldehyde (RT-2.57), methanol (RT-3.48), ethanol (RT-4.37), isopropanol (RT-4.97)



Chromatogram of ante mortem blood samples of subject-4 Methanol (RT- 3.49)

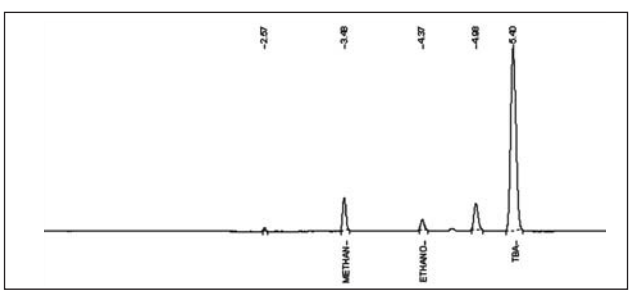
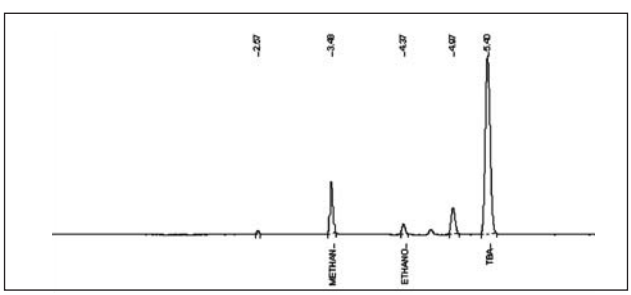


Fig. 5. Chromatogram of ante mortem blood of subject-5 formaldehyde (RT-2.57), Methanol (RT-3.48), ethanol (RT-4.37), isopropanol (RT-4.98)



Chromatogram of ante mortem blood of subject-6, formaldehyde (RT-2.57), methanol (RT-3.48), ethanol (RT-4.37), isopropanol (RT-4.97)

unabsorbed alcohol and negative methanol and ethanol in the post mortem blood of subject-3 and negative ethanol in ante mortem blood of subject-4 may be due to metabolism. However 98.0144mg% ethanol, 86.6506mg% methanol was found in ante mortem blood. 9.4953mg% ethanol and 87.1914mg% methanol in gastric contents of subject-3. 80.1242mg% methanol in ante mortem blood of subject-4. Blood methanol and ethanol shows variation in concentration because of difference in sampling timing, individual variation, and concentration of toxic metabolites and coingestion of ethanol. This indicates that blood level alone is not reliable indicator in case of methanol poisoning, because the toxicity results from the forate[1].

A 44-years-old man suffering from methanol intoxication was found comatose and had blood methanol concentration of (142mg%) died after 40 hrs later[5]. Forty one years old man died after ingesting a large quantity of methanol (284 mg%)) was found in blood and (221mg%) in stomach content[6]. In two fatalities caused by ingestion of methanol concentration of methanol and formic acid were (219mg%), (41mg%) in 1st subject and (196mg%), (38.0mg%) in 2nd subject[7].

Subject (1), (2) and (3) has the history of large amount intake of liquor as compared to subject (4), (5) and (6). All of them consume the same illicit liquor (gudanji) in different quantity and time intervals. The exact amount, time of intake is not known but as per case history symptoms were observed after 12-48 hrs. The time interval between the ingestion of the adulterated drink and the presentation with symptomatology is due to the time taken for the metabolism of methanol. Bonnet et al[8] reported a latent period of 40-72 hrs, while Ravichandran et al [3] reported 8-60 hrs. In study of 28 fatal cases by Mittal [4] the methanol levels in blood as well in viscera were variable with an average methyl alcohol level of 155.87mg% and 420mg% in 7 cases no methyl alcohol was detected.

DISCUSSION

Methanol is intoxicating but not directly poisonous. It is toxic by its breakdown by enzyme alcohol dehydrogenase in the liver by forming formic acid and formaldehyde which causes blindness by destruction of optic nerves and can also be fatal due to its central nervous system depressant properties in the same manner as ethanol. Severe poisoning and death have occurred primarily from ingestion of beverages adulterated with methyl alcohol. Typically within 18 to 48 hours after ingestion the victim develops acidosis, nausea, abdominal pain, headache, shortness of breath

Table 1. Concentration of Ethyl alcohol and Methyl alcohol in the Body fluids of subjects

Subject No.	Postmortem Blood Samples		Antemortem Blood Samples		Gastric Contents	
	EtOH mg%	MeOH mg%	EtOH mg%	MeOH mg%	EtOH mg%	MeOH mg%
1.	35.6477	89.6336	-	-	-	-
2.	-	-	12.9361	98.7445	689.0871	74.2609
3.	nil	nil	98.0144	86.6506	9.4953	87.1914
4.	-	-	Nil	80.1242	-	-
5.	-	-	12.3916	30.3463	-	-
6.	-	-	11.4042	58.6376	-	-

Note: - indicates sample not supplied

and blurred a double vision[9]. Severely poisoned individuals become comatose and may die, and those who recover can be left blind [10]. Ethyl alcohol competes with methyl alcohol for the alcohol metabolism. Due to this reason ethyl alcohol is given in treatment of methyl alcohol poisoning. Like ethanol, methanol is also oxidized in two stages first to aldehyde and then to acid. The specific products of metabolism are formaldehyde and formic acid. Ethanol and methanol when present together, will not be metabolized at the same rate. The rate of ethanol metabolism is seven time faster. Methanol metabolism involves a slow reaction. Formic acid produced is degraded to carbon dioxide and water. However this reaction is very slow formic acid will accumulate in significant amount in severe methanol poisoning. Because hepatic conversion of methanol to formic acid is much slower than ethanol conversion to acetic acid [11].

CONCLUSION

Methanol is a common and potent adulterant of illicit liquors and is responsible for many fatalities. The time interval between ingestion of illicit liquor and appearance of symptoms is due to the slow metabolism of methanol. Absence of ethanol in some samples indicates complete elimination and high concentration of ethanol in the gastric content of subject is due to unabsorbed liquor.

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REFERENCES

1. Matthew J, Ellenhorn, Donald GB, Barceloux, Medical toxicology diagnosis and treatment of human poisoning Elsevier Science Publishing Company 1998.
2. Bade LK, Snore DP, Methyl alcohol poisoning, medical news, Medicine and Law as quoted by Ravichandran et al 980, 1981,
3. Ravichandran R, Dudani RA, alongside AE, Chamber KP, Acharya VN. Methyl alcohol poisoning (experience of outbreak in Bombay); J. post grad med 1974, 30: 69-74.
4. Mittal BV, Desai AP, Khade KR, Methyl alcohol poisoning an autopsy study of 28 cases, J. post grad med 1991; 37: 9-13.
5. N.B. Wu Chem. et al; J. forensic sci; 1985, 30, 213-216.
6. A. Pla et al; Forensic sci int; 1991, 49, 193-196.
7. H. Kinoshita et al; Int. j. legal med; 1998, 111, 334-335.
8. Bonnet Jr, ConyFH, Mitchell GL Jr, Cooper MN, Acute methyl alcohol poisoning a review based on experiences in an outbreak of 323 cases. Medicine 1953, 32: 431-463
9. James C Garriot, Medico legal aspects of alcohol determination on biological specimens PSG publishing 1987.
10. Proctor N.H, and Hughes J.P, Chemical hazard of work place. Lippincott, Phlidelphia, 1978.
11. Fenton John, Ph.D. Toxicology a case —oriented approach CRC PRESS 2002.

Sexual dimorphism of human femur in Western India

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ABSTRACT

Determination of sex is one of the first and important factor in identifying decomposed bodies or skeletal remains.

The correct sex determination of skeleton is a critical requirement in physical anthropology and in medico-legal cases. A study of 100 pairs of adult, fully ossified, normal dried femora (50 males and 50 females) of Western Indian population was done. The various parameters e.g. length, bicondylar width, vertical diameter of head of femur etc. were measured. Univariate statistical tests were applied to access whether the difference between means of each parameter was statistically significant or not. The demarking points for each parameter were determined for both the sexes. The data of the present study was compared with that of the previous studies.

Thus, it will be possible to determine the sex of femur from above parameters and demarking points.

Key words: *Sexing; Femur; Anthropology; Medicolegal.*

INTRODUCTION

Determination of sex from analysis of human skeletal remains has been a well studied field with broad ranging applications extending beyond forensic anthropology and into archeology, paleoanthropology and comparative anatomy.

Postcranial bones other than the pelvis, especially larger limb bones may provide clear evidence of sex. The femur is an important because it gives an additional strong support along with the other bones for determining the age and sex of the deceased.

Aim of study

The aim of the study was to study the sexual dimorphism in human femora of Indian population and its importance in the medico-legal cases.

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MATERIAL AND METHODS

200 adult dried human femora of known sex (100 males and 100 females) were collected from, Government medical college, Miraj, Bharati Vidyapeeth's Medical College, Sangli., Vasantdada Patil ayurvedic Medical College, Sangli. Only adult bones with complete ossification were included in the present study. Bones showing wear and tear, any fracture or pathology were excluded.

Following measurements of the femur were recorded,

1. Trochanteric length of femur

It was measured on the osteometric board. Bone was kept on its dorsal surface on the board. Lateral condyle was kept in contact with the fixed plate of the board and the highest point of the greater trochanter was kept in contact with the sliding plate.

2. Upper end length

The maximum distance between the depression of the head medially and the greater trochanter laterally. It was measured with the help of the Vernier caliper.

3. Mediolateral diameter of the head

With the help of Vernier caliper distance between

most lateral and medial point on the head of femur was recorded. It is also called transverse diameter of the head.

4. Neck shaft angle

The Pearson's method for determining the angle is used. Reference points were determined by sight.

These were as follows,

Head neck axis, the lowest point in the intercondylar fossa and midpoint of the narrowest portion of the shaft. With the dorsal surface of the bone a straight line was projected from the intercondylar low point through the mid point of the shaft. An extension of this line through the projected head neck axis formed the neck shaft angle. The angle of this point is determined by the transparent protractor.

5. Mid-shaft circumference

A tape followed the contour of the mid shaft point of the body of femur. In this way the circumference at the middle of the shaft was measured.

6. Mid-shaft anteroposterior diameter

The anteroposterior diameter at the middle of the shaft of femur was taken up with the help of Vernier caliper.

Observations

The following six different measurements were studied from the femora; viz. Trochanteric length, Upper end length, Mediolateral diameter of the head, Neck shaft angle, Mid-shaft circumference, Mid-shaft anteroposterior diameter.

Range, mean and standard deviation of the measurements of adult, fully ossified femur were calculated. The identification point for each parameter was calculated from the range of each measurement. From this percentage of identified bones was calculated. However, when dealing with normal distributions which is the case in most biological variables, maximum and minimum limits can safely be calculated on the basis of standard deviation[1].

Thus, 'Mean ±1 S.D.' will give the range that covers 68.3% of the area or zone.

'Mean ±2 S.D.' will give the range that covers 95.4% of the area or zone.

'Mean ±3 S.D.' will give the range that covers 99.75% of the area or zone.

But when nearly 100% accuracy of sexing is required e.g. in medico-legal cases, it is advisable to calculate the maximum and minimum limits by adding ±3S.D to the mean value of each measurement. This gives the calculated range. It covers 99.75% of the sample from this zone and will be useful also for any other sample from this zone.

Demarking points were worked out from calculated range[2]. The percentage of bones identified by each demarking point in both sexes was estimated. The demarking points identify sex with 100% accuracy.[3]

The difference observed between means of male and female to know whether it is statistically significant, that is value of 'P' is calculated by applying 'Z' test.

In the present study all the measurements were found to be greater in male femora than in female femora. Sexual difference between the mean of every measurement was highly significant ($p<0.001$). Abbreviations used in following tables are:

1. S.E. = Standard error
2. R.D. = Relative difference
3. P = Probability or The level of significance for difference between two means
4. M = Male
5. F = Female
6. S.D. = Standard deviation
7. S.S.D. = Statistical sexual difference

Table 1. Trochanteric length of femur (in mms)

Sr. No.	Details of measurements	Right		Left	
		Male	Female	Male	Female
1.	No. of bones	50	50	50	50
2.	Range	395-465	360-415	390-470	360-420
3.	Mean	422.1	386.4	423.7	391
4.	Standard deviation	17.73	15.12	17.32	15.49
5.	Statistical significance	P<0.001	P<0.001		
6.	Identification point	>415	<395	>420	<390
7.	Percentage of identified bones	54%	60%	46%	38%
8.	Calculated range	368.91-475.29	341.04-431.76	371.75-475.64	344.53-437.47
9.	Demarking point	>431.76	<368.91	>437.47	<371.75
10.	Percentage beyond demarking point	20%	14%	18%	20%
		S.E.-3.3 R.D.-10.82		S.E.-3.29 R.D.-9.94	

Table 2. Upper end length of femur (in mms)

Sr. No.	Details of measurements	Right		Left	
		Male	Female	Male	Female
1	No. of bones	50	50	50	50
2	Range	78 -96	72 - 85	81 - 97	72 - 83
3	Mean	86.44	76.72	87.64	77.86
4	Standard deviation	4.06	3.37	3.97	2.98
5	Statistical significance	P<0.001	P<0.001		
6	Identification point	>85	<78	>83	<81
7	Percentage of identified bones	54%	62%	84%	74%
8	Calculated range	74.26 - 98.62	66.62- 86.82	75.74- 99.54	68.91- 86.80
9	Demarking point	>86.82	<74.26	>86.80	<75.74
10	Percentage beyond demarking point	42%	22%	48%	22%
		S.E.- 0.75 R.D.-12.96	S.E.-0.70 R.D.-13.97		

Table 3. Mediolateral diameter of head of femur (in mms)

Sr. No.	Details of measurements	Right Male	Left Female	Male	Female
1.	No.of bones	50	50	50	50
2	Range	40 -47	33 - 41	40 - 45	32 - 41
3	Mean	41.58	36.06	41.7	36.5
4	Standard deviation	1.67	2.23	1.58	2.2
5	Statistical significance	P<0.001	P<0.001		
6	Identification point	>41	<40	>41	<40
7	Percentage of identified bones	48%	88%	48%	86%
8	Calculated range	36.60-46.60	29.38 - 42.74	37 -46.44	29.9 -43
9	Demarking point	>42.74	<36.60	>43	<37
10	Percentage beyond demarking point	6%	62%	18%	52%
		S.E.- 0.39	R.D.- 14.15	S.E.- 0.38	R.D.-13.68

Table 4. Neck-shaft angle (in degrees)

Sr. No.	Details of measurements	Right Male	Left Female	Male	Female
1.	No. of bones	50	50	50	50
2	Range	125 - 131	120 - 127	123 - 133	119 -126
3	Mean	127.26	123.44	127.28	122.5
4	Standard deviation	2.02	2.06	2.22	1.96
5	Statistical significance	P<0.001	P<0.001		
6	Identification point	>130	<121	>126	<123
7	Percentage of identified bones	48%	68%	60%	48%
8	Calculated range	121.17- 133.35	117.25- 129.63	120.62- 133.94	116.62- 128.4
9	Demarking point	>129.63	<121.17	>128.4	<120.62
10	Percentage beyond demarking point	2%	14%	28%	18%
		S.E.- 0.41R.	D.-9.32	S.E.-0.42R.	D.-11.38

Table no-5 Mid-shaft circumference of femur (in mms)

Sr. No.	Details of measurements	Right Male	Left Female	Male	Female
1.	No. of bones	50	50	50	50
2	Range	77 - 102	67 - 84	77 - 96	68 - 85
3	Mean	84.54	75.24	84.82	76.36
4	Standard deviation	4.30	4.22	4.64	3.73
5	Statistical significance	P<0.001	P<0.001		
6	Identification point	>84	<77	>85	<77
7	Percentage of identified bones	44%	54%	34%	54%
8	Calculated range	71.65 -97.43	62.6- 87.88	70.89 -98.74	65. 17-87.54
9	Demarking point	>87.88	<71.65	>87.54	<70.89
10	Percentage beyond demarking point	14%	16%	24%	16%
		S.E.- 0.85R.	D.-10.94	S.E.- 0.84R.	D.-10.07

Table 6. Mid-shaft anteroposterior diameter of femur (in mms)

Sr. No.	Details of measurements	Right Male	Left Female	Male	Female
1.	No. of bones	50	50	50	50
2	Range	24 -31	20 - 28	24 - 32	22 - 29
3	Mean	27.26	24.38	27.36	24.44
4	Standard deviation	1.61	1.83	1.78	1.62
5	Statistical significance	(p<0.001)	(p<0.001)		
6	Identification point	>28	<24	>29	<24
7	Percentage of identified bones	20%	34%	10%	30%
8	Calculated range	22.41-32.10	18.89-29.86	20.01- 32.7	19.58 - 29.29
9	Demarking point	>29.86	<22.41	>29.29	<20.01
10	Percentage beyond demarking point	4%	10%	10%	Nil
		S.E.-0.34R.	D.-8.47	S.E.-0.34R.	D.-8.59

DISCUSSION

The trochanteric length and upper end length of femur in the present study is comparable with the study of Leelavathy N[4]. The study of neck shaft angle is compared with the studies by Davivongs V[5], Dennis P. Van Gerven[6] and Sessi DAVS.[7] in that the present study is comparable with only the study of Sessi DAVS. The study of Davivongs and present study show statistically significant sex difference between mean values of mediolateral diameter of head of femur.

The mid-shaft circumference of the femur in present study is compared with the study of Davivongs V, Black TK[8], Dibennardo R[9], Iscan YM[10], Purkait R[11] in that the present study is comparable with the study of Black and Iscan. The mean mid-shaft anteroposterior diameter is comparable with values in all the studies except with the study of Dibennardo R.

CONCLUSION

- The differences in mean values of all parameters in males and in females were statistically significant.
- The upper end length of femur was found to be the ideal parameter for sexual dimorphism of femur.
- Mid shaft anteroposterior diameter of head though proved to be statistically significant, it is less useful in sexing the unknown femur as large percentage of male and female bones lie in the co-extensive range.
- The findings from the present study were comparable with the data available of previous workers

REFERENCES

1. Rao CR. In 'Advanced Statistical Methods in Biometric Research' Biometrica. 1966; 53(3-4): 339-345.
2. Jit I and Singh S. Sexing of the adult clavicle. Ind Jr Med Res. 1966; 54: 557-561.
3. Singh S and Butchi RP. Greater sciatic notch in sex determination. 1978; J Anat. 125(3): 619-624.
4. Leelavathy N, Rajangam S and Janakiram S et al. Sexing of the femora. J Anat Soc India. 2000; 549(1): 17-20.
5. Davivongs V. The femur of the Australian aborigine. Am J Phys Anthrop. 1963; 21: 457-467.
6. Gerven Dennis P Van. The contribution of size and shape variation to patterns of sexual dimorphism of the human femur. Am J Phys Anthrop. 1972; 37: 49-60.
7. Sessi DAVS and Lakshmi CB. Variations of angles and curvatures of femur- male and female- A study. J. Anat. Soc. India (2005); 54(1): 62.
8. Black TK. New method for assessing the sex of fragmentary skeletal remains: Femoral shaft circumference. Am J Phys Anthrop. 1978; 48: 227-232.
9. Dibennardo R and Taylor JV. Classification and misclassification in sexing the black femur by discriminant function analysis. Am J Phys Anthrop. (1982); 58: 145-151.
10. Iscan YM and Ding S. Sexual dimorphism in Chinese femur. Forensic Sci Int. 1995; 74: 79-87.
11. Purkait R and Chandra HA. Study of sexual variation in Indian femur. Forensic Sci Int. 2004; 146(1): 25-33.

Medico Legal Autopsies of Custodial Deaths in Mumbai Region - A two Years Prospective Study

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ABSTRACT

The study comprises of total 108 autopsies of custodial deaths which are referred for post mortem examination by magistrate from various police custodies and prisons in Mumbai region. The study is carried out over period of two year from July 2005 to July 2007. Autopsies were conducted as per NHRC guidelines. The study was mainly aimed to evaluate various factors in relation to custodial deaths like to assess mean age at death, drug addictions in deceased, type of custody and the evidence of injuries, relation of age (in year) with type of offence committed, the common manner and cause of deaths in police and judicial custody. It was observed that of total 108 deaths 16 (14.81%) were in police custody and 92 (85.19%) were in judicial custody. 74% deaths were in age group ranging from 18 years to 45 years; with mean age at death was 34 yrs.75% of deceased were addicted. Alcoholism and smoking was common addiction observed. Maximum number of deceased (59%) was from age group of 26 to 45. Offences more frequently was homicide (31.5%) followed by NDPS related (15.75%). 70.4% of deaths were occurred either before or within 24 hr of admission in hospital. Evidence of injuries is seen in 56% cases in police custody. 93.5% of deaths in judicial custody were from natural causes and tuberculosis is common cause of death (50.5%). 81.2% of deaths in police custody were unnatural and common cause was suicide by hanging.

Key words: *Custodial Deaths, Judicial Custody, Police Custody, NHRC, Natural Deaths, Unnatural Death, Drug Addiction, and Offences Committed.*

INTRODUCTION

Custodial deaths are sensitive issue and autopsy surgeon plays important role in these cases as there is hardly any outside independent evidence in these cases, the fate of the cases would depend entirely on the observations recorded and the opinion given by the doctor in the post-mortem report. Though autopsy performed previously of custodial death is same as routine autopsy but from inception of Protection of Human Rights Act 1993 some additional procedures are included in routine autopsy like postmortem examinations done in respect of deaths in police custody and in jails should be video-filmed. The cassettes are sent to the Commission along with the post-mortem report. In order to make transparency in custodial deaths and to plugs the loop holes in such deaths.

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MATERIAL AND METHODS

The present study is conducted at Department of Forensic Medicine and Toxicology, Grant Medical College Mumbai. The study comprises of total 108 autopsies of custodial deaths which are referred for post mortem examination by magistrate from various police custodies and prisons in Mumbai region. The study is carried out over period of two year from July 2005 to July 2007. Before starting autopsy detailed history of deceased was obtained regarding:

Date of admission in custody, type of offence committed, drug addiction, type of custody.

The hospital record papers are carefully reviewed for clinical history, investigations like X-rays, CT scan, bio-chemical& blood investigations, treatment given,

and probable clinical diagnosis. Inquest papers are carefully viewed particularly about external injuries which are mentioned. In all cases magistrate inquest is conducted and all inquests are videographed. The post-mortem examination is performed by at least two doctors or a panel of doctors with one senior qualified staff member. A detailed and meticulous autopsy with video recording of whole procedure is performed as per guidelines given by National Human Rights Commission[1].

Criterion for selection of cases of custodial deaths is as following:

Incisive criteria:
i) Deaths which occurred in prison custody.
ii) Deaths of which occurred in rout of medical care.
iii) Deaths which occurred in hospital.
iv) Deaths which occurred in police custody

Exclusive criteria:
i) Deaths which occurred from police encounters
ii) Age of custodian less than 18years.

Autopsy is conducted with addition following procedures are done:

i. The post-mortem examination is performed by at least two doctors or a panel of doctors with one senior qualified staff member. A detailed and meticulous autopsy with video recording of whole procedure is performed as per guidelines given by National Human Rights Commission.
ii. No unauthorized persons are to be allowed during autopsy. The time during autopsy when each person is present is included using sign-in sheet.
iii. Videorecording is done in all case. Name and address of person videorecording the autopsy is noted.
iv. Identification of body of deceased is done by relative. If relatives are not presents identification is done by concerned Jailer and videographed.
v. Careful examination is done for the physical findings which indicate evidence of detention or torture .
vi. Multiple parallel Incisions is taken over palms and soles to rule out any blunt injuries.2
vii. In dark skinned person long incision is taken on front and back of each limb, buttocks and on back both side to exclude any deep seated contusion.
viii. Weight of each organ is recorded. Each organ is videographed before and after sectioning.

OBSERVATIONS AND RESULTS

1. Age wise distribution:
The age wise distribution of deceased is calculated which shows that maximum 37.96% of cases are in age group 26-35 years and mean age at death was found 34 years. The incidence of death is more in age group of 26-55 years (59%).

Age(years)	18-25	26-35	36-45	46-55	Above 55	Total
No. of deaths	16	41	23	15	13	108
Percentage	14.81%	37.96%	21.29%	13.88%	12.08%	100%

2. Distribution of type of drug addiction among deceased
Study shows various type of drug addiction among deceased. Smoking and alcoholism is seen together in 52 cases. Out of total 108 cases 81 had various type of addiction. Intravenous drug users are HIV positive. Those of charas smokers had found lung abscesses.

Drug addiction	Alcoholism and smoking	Only Alcoholism	Charas and ganga	Heroin	Cocaine	Total
No. of cases	52	09	11	05	04	81
Percentage	64	11.11	13.58	6	4.9	75

3. Literacy status distribution of deceased

Education	Illiterate smoking	Primary	Secondary ganga	Higher secondary	Graduation	Post Graduation
No. deceased	18	48	21	15	04	02
Percentage	16.66%	44.44%	19.44%	13.88%	03.70%	01.85%

Above table shows the literacy status of 108 deceased, Illiterates comprise 16%, while most of deceased 44% had completed their primary education. 19% secondary.13% higher secondary, 3% graduation, 1.8%posts graduate.

4. Relation of age (in year) with type of offence committed
The deceased under which type of offence they are arrested, under trail or convicted is as shown below:

Type of offence	18-25	26-35	36-45	46-55y	> 55	Total
Homicide and related	4	7	7	8	8	34
Hurt and related	2	3	1	1	1	8
Robbery	7	5	4	-	-	16
Theft	3	5	4	-	-	12
Cheating	-	1	2	-	-	3
Forgery	-	2	1	-	-	3
Rape	-	1	2	1	-	4
NDPS related	3	10	4	1	-	18
Rioting	1	1	-	-	-	2
Extortion	1	2	2	-	-	5
Other	-	2	1	-	-	3
Total	21	39	28	11	9	108

One deceased can be charged with more than one offence at time but out that major offence is mentioned in table no.9.from table it is observed that maximum number of deceased are from age group of 26 to 45 offences more frequently was homicide followed by NDPS related, robbery, theft and hurt related.

5. Evidence of injuries and that suggestive of its nature

The evidence of injuries fresh as well as old which was observed at the time of autopsy and its nature is shown in table below. More than one type of injuries was present in one deceased

Physical finding	Total	Self inflicted	Torture	Homicidal
General beating	02	-	✓	-
Linear contusions on back	01	-	✓	-
Linear contusions on buttocks	03	-	✓	-
Linear contusions on thighs	02	-	✓	-
Contusions on soles	05	-	✓	-
Contusions on palms	04	-	✓	-
Mal united fracture at ankle	02	-	✓	-
Mal united fracture at wrist	01	-	✓	-
Multiple stab injuries	02	-	-	✓
Head injury	01	-	-	✓
Scars on chest & forearms	05	✓	-	-
Abrasions on fore arm/ wrist	01	-	✓	-
Abrasions on knee and leg	02	-	✓	-

In total 20 cases injuries were seen out of which 11were in judicial custody Cases. Three of them are fresh injuries and homicidal in nature. They were due to scuffle between the prisoners in prison. Two presented with multiple stab injuries over chest and abdomen one with head injury. Multiple linear scars on forearms and chest seen in 5 cases those were drug addict. Mal united fracture at ankle seen in two cases and mal united fracture at wrist seen in one case.

9 deceased in police custody shows evidence of injuries and all injuries were fresh and suggest non self inflicted in custody. It is observed that the common method of torture is beating on soles palms, buttocks and thigh.

6. Causes of deaths in judicial and police custody

Custody	Natural	Unnatural	Total
Police	03	13	16
Judicial	86	06	92
Total	89	19	108
Percentage	82.40	17.60	100

It was observed that unnatural deaths are more common in police custody. However natural deaths are more common in judicial custody

7. Age wise distribution of unnatural deaths

Age (years)	suicides	Homicides	Accidental	Total
18-25	6	1	1	8
26-35	4	2	-	6
36-45	2	1	-	3
46-55	1	-	-	1
Above55	-	1	-	1
Total	**13**	**5**	**1**	**19**

It was observed that out of total 19 unnatural deaths. Most of them were suicides.

DISCUSSION

Comparative table showing mean age at deaths and sex distribution of deceased

The present study is comprises of total 108 of custodial deceased with mean age at death was 34 years and out of 108 inmates died only two were of female

Studies	No. of deaths	Period of study	No. of males	No. of females	Mean age at death
Wendy L. wobeser[3]	291	10 yrs	283	8	41yrs
Derrick J. pounder[4]	31	11yrs	31	-	44yrs
Bardale rajesh et. al[5]	70	5yr	70	-	45yrs
Marrissa McCcall[6]	68	1yrs	63	5	39yrs
Okoye M. et. al.[7]	51	6yrs	51	-	42 yrs
Petschel et. al.[8]	96	6yrs	90	6	41yrs
Salive M.E.et.al.[9]	206	9yrs	195	11	39 yrs
Smialek J. E. et. al.[10]	25	1yr	22	3	27yrs
Ghazala sattar[11]	236	2yrs	228	8	41yrs
Present study	108	2yrs	106	2	34yrs

Study of Smialek J. E. et. al.[10] shows that mean age was 27 yr at death. However Bardale rajesh et. al[57] shows that mean age at death is 45 years .in all study very few female inmates are involved.

Alcohol and smoking is predominant in present study followed by charas/ganja smoking Lung abscesses were found in cases of charas smoker's predominance of alcohol and smoking in study is due to easy availability in country. Gore S.M., Bird A.G. et. al.[23] Studied 871 cases of random urine sample of inmate of England and Wales. 32% of them were positive for cannabis and 4% were positive for heroin

Literacy status of prisoners in India[12] as BPRD record shows that 72% of prisoners were completed their primary education in present study 80% of total deceased had completed their primary education and finding were consistent with all India prisoners statistics.

Criminal activity is more common in younger than older most offence committed was homicide (n=34)

and related followed by NDPS related (n=18) and robbery (n=16). maximum numbers of offender were fall in age group 18-35yrs its shows that criminal activity is common in youth population. The present study finding was consistent with Taylor P.J. et.al[13]. Study most vulnerable age group is 18-45, the reason being presence of aggressive trait in this age group. While frequency of crime decreased in old age.

Petschel et. al.[8] Study shows that out of 96 deaths 52 deaths were occurred in police custody and police related operation 46 deaths were occurred in judicial custody and most of deaths in police custody were unnatural and that in judicial custody were natural. Bardale rajesh et. al[5]. Study shows that 10 deaths were occurred in police custody all were unnatural. Present study findings were consistent with above study that unnatural deaths are far much exceeds natural deaths in police custody. However natural deaths were common in prison custody.

Wendy L. wobeser et. al[3] Study shows that there were 56 deaths were accidental which were seen in drug addicts due to excess of drug doses. Which is common in prisoner of Ontario custody? Bardale rajesh et. al[5] Study shows that there were three homicidal deaths of which two were occurred in police custody while one occurred in prison.

CONCLUSION

1. 74% deaths were in age group ranging from 18 years to 45 years; with mean age at death was 34 yrs.
2. Majority of the deceased were male with only 1.86% as females.
3. The literacy status of the deceased showed the prison population to be literate, only 16.66% Of the deceased were illiterate.
4. Evidence of injuries suggestive of torture is seen in 56% cases in police custody.
5. Natural deaths were common in judicial custody. However unnatural deaths were common in police custody.
6. Suicide is common in unnatural deaths; most of suicides were by hanging. Identification of suicidal risk individuals with exploring factor for suicide and initiation of psychological counseling for such cases is recommended.

REFERENCES

1. Souvenir of Medico-Legal Association of Maharashtra 10th Annual Conference 2000 "NHRC guidelines for custodial deaths" (52-58).
2. Dr. Amrit K. Patnaik and k. mathiharan ." Model protocol for Autopsies in Custodial Death" Indian Journal of Medical Ethics June1999, (2) 59-63.
3. Wendy L.Wobeser, Jason Datema, Benoit Bechard, Peter Ford "causes of death among people in custody in Ontario 1990-99 ",Canadian medical Association Journal Nov 2002,167(10): 1109-1113.
4. Derrick J. Ponder "Death behind bars an 11year survey in south Australia " Medicine, science and Law 1986, 26(3)
5. Bardale Rajesh, Shrigiriwar Manish, Vyawahare M S , Dixit P.G., Tayade S. N. "Death behind bars: A five year study of custodial death". Journal of Medico- Legal Update Dec.2005, 5(4)105-109
6. Marissa McCall "Deaths in custody in Australia :2003 National Death in Custody Program " Australia Institute of Criminology ,2004 (Technical &background paper series no.12).
7. Okeye M, Kimmera E H , Reinhard K, "An analysis and report of custodial deaths in Nebraska, USA" J Clin Forensic Med.1999 , Jun;6(2) 77-84.
8. Petschel K,Gall J A. " A profile of deaths in custody in Victoria, 1991-96" J Clin Forensic Med. 2000 Jun; 7(2): 82-87

Aberrations of Human Gender Identification by Amelogenin Test in Libyans (Benghazi City)

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ABSTRACT

Gender identification is essential in forensic science to provide the initial information for criminal investigation. However, gender identification by amplification of amelogenin gene sometimes showed controversial cases, in which male samples was falsely identified as females and female samples was recorded as males . We reported the anomalous amelogenin results of 2 male samples (out of 238 males) represented as females (Y deletions) and another 2 samples with (X deletions) in Benghazi (Libya). The frequency in both was about 0.8 %. Higher than those of the other populations reported.To confirm amelogenin results of the 2 controversial samples, DNA was further used in SRY and Y-STR typing. All samples typed as males but two showed with X chromosomes. From the results, it was highly suggested that for the controversial cases of human gender identification with amelogenin tests, amplification of SRY gene or/and Y-STR markers will be adopted to confirm the gender.

Key words: *Gender Identification, Amelogenin Gene, SRY Gene, Y-STR, Forensic Science, Benghazi, Libya.*

INTRODUCTION

Sex typing is essential in medical diagnosis of sex-linked disease and forensic science. Gender for criminal evidence of offender is usually as the initial information for investigation. For individualization, identification of gender is performed in addition to the STR markers recently. The most popular gene included in the commercial individualization kits for human gender identification is amelogenin gene. However, it was reported that two males were typed as females in 1998 by PCR amplification of amelogenin[1], and later other laboratories also observed the anomalous amelogenin results. Allele dropout of X amelogenin was observed in males, and proved to result from the polymorphism of the primer binding sites[2]. Nevertheless, structural variation is particularly prevalent on the Y chromosome. Higher frequency of deletion for the Y amelogenin region in Indian population group was observed[3,4] and deletion of the fragment Y (p) 11.2 was determined[5]. The male samples would be identified as females in such cases. For Austrian study, the samples which lacked the amplicon of Y amelogenin in males were also identified[6,7]. These samples were confirmed to be from males by amplification of Y-STR and SRY (sex-determining region of Y chromosome) gene. The phylogenetic context, origins and implications for male amelogenin dropouts were reported[8]. We reported the controversial cases, for males of human gender identification by amelogenin test in Benghazi (Libya).

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POPULATION

Libya, a Northern African country, was first inhabited by Berbers, followed by Phoenicians, Greeks, Romans, Arabs and Ottomans. Libya became independent in 1951 after a brief period as an Italian colony; it had been invaded by Italy in 1911.In February 2011 an uprising against the government occurred in the city. Benghazi is the second largest city in Libya and the main city (or capital) of the Cyrenaica region (or ex-Province), located in the North of Africa. Benghazi is located half way between Tripoli in the West (a distance of approximately 1000 Km between these cities) and Cairo in the East (also approximately 1000 Km). Cyrenaica is surrounded by desert on three sides; hence in ancient times the most accessible civilization was to the North, across the Mediterranean, in Crete and Greece, only 400 km away. The population of Benghazi was 500,120 in 1995 (census) and increased to 670,797 in the 2006 census. As with other cities in

Libya, there is a reasonable amount of ethnic diversity in Benghazi. The people of eastern Libya, Benghazi included, have in the past always been of predominantly Arab descent. In recent times, however, there has been an influx of African immigrants into Benghazi. There are also many Egyptian immigrants in Benghazi. A small Greek community also exists in Benghazi; the Greek island of Crete is a short distance from Benghazi and many families in Benghazi today bear Cretian surnames.

In modern times, Benghazi has seen a lot of Libyans from different parts of the country move into the city, especially since the Kingdom era (1951-1969). Many Libyans came to Benghazi from Misrata (About 60% of the population have roots from Misrata, West of Benghazi).

MATERIAL AND METHODS

Informed consent was obtained from 238 unrelated Libyan males (Benghazi region) same sources of studies related to Autosomal and Y chromosomal STRs.

DNA EXTRACTION

DNA was extracted from blood stains collected on FTA® cards (Whatman, Kent, UK) using FTA® Purification Reagent (Whatman) following the manufacturer's protocol and from buccal swabs using QIAamp®DNA Blood Mini Kit (QIAGEN, Hilden, Germany). DNA was quantified using a StepOnePlus™ Real-Time PCR System (Applied Biosystems, Foster City, USA).

PCR AMPLIFICATION OF AMELOGENIN

Amelogenin alleles were determined using a different primer pair from that in the Identifiler Kit. The primer pair used here amplifies a different part of intron 1 of the amelogenin gene (X: p22.1-22.3 and Y: p11.2). The two primer pairs used for capillary electrophoresis [Identifiler] and agarose gel electrophoresis are given in Table1.

Discs of approximately 1.2 mm cut from FTA® cards (>5 ng DNA) or 1 ng of DNA purified from buccal swabs were used to supply the target for amplification with amelogenin primers.

PCR reactions were carried out in 25 µL volumes. Each PCR reaction mixture contained of: 0.1 mg/ml bovine serum albumin (BSA), 12.5 l Taq DNA Polymerase Master Mix 2x (Qiagen), amelogenin forward and reverse primers (Sigma) at 0.5 M, <1 g of template, nuclease-free water to a final volume of 25 µl.

The PCR reaction conditions were the same as those used to amplify part of the amelogenin gene with the Identifiler kit. These were found to give good amplification with this primer pair as well.

PCR was carried out in a Thermocycler 9700 from Applied Biosystems. PCR conditions were as follows: starting temperature was 95°C for 11 min, (denaturing temperature 95°C for 1min, annealing 59°C for 1min, extension 60°C for 1min, 27 cycles), then 60°C for 80 minutes and held at 4°C. PCR amplification blanks (DNA replaced by equal volume of extraction reagent blank control) were used as negative controls. The PCR products were analysed by 1.5% agarose gel electrophoresis at 120V for 1 hr and then stained with ethidium bromide for half an hour and visualized under UV light.

Table. 1 Amelogenin Primer Structures.

Locus	Primers	Product Size
Amelogenin Used with Identifiler kit	-CCCTGGGCTCTGTAAAGAATAGTG-3' 5'-ATCAGAGCTTAAACTGGGAAGCTG-3'	X = 106 bp Y = 112 bp
Amelogenin Used with Agarose Gel	5'-GCTCTGATGGTTGGCC-3' 5'-CCTTGCTCATATTATACTTGA-3'	X = 542 bp Y = 358 bp

```
X   8400 AACTTCAGCTATGAGGTAATTTTTCTCTTTACTAATTTTGACCATTGTTTGCGTTAACAA
         ||||||||||||||||||||||||||||||||||||||||||||| || |||||||| ||| ||
Y   9008 AACTTCAGCTATGAGGTAATTTTTCTCTTTACTAATTTTGATCACTGTTTGCATTAGCAG
                                                Forward Amelogenin Primer (Identifiler)   6 bp deletion on X
X   8460 TGCCCTGGGCTCTGTAAAGAATAGTGTGTTGATTCTTTATCCCAGAT------GTTTCTC
         | |||||||||||||||||||||||||| || ||||||| |||||| ||     |||||||
Y   9068 TCCCCTGGGCTCTGTAAAGAATAGTGGGTGGATTCTTCATCCCAAATAAAGTGGTTTCTC
                                                Reverse Amelogenin Primer site (Identifiler)
X   8514 AAGTGGTCCTGATTTTACAGTTCCTACCACCAGCTTCCCAGTTTAAGCTCTGATGGTTGG
         ||||||||  ||||||||||||||||||  ||||||||||||||||||||||||||||||
Y   9128 AAGTGGTCCCAATTTTACAGTTCCTACCATCAGCTTCCCAGTTTAAGCTCTGATGGTTGG
                                                Forward Amelogenin Primer (Gel Based)
X   8574 CCTCAAGCCTGTGTCGTCCCAGCAGCCTCCCGCCTGGCCACTCTGACTCAGTCTGTCCTC
         |||||||||||||| |  ||||||  |||||  |||||  ||| || || | | ||| |||||
Y   9188 CCTCAAGCCTGTGTTGCTCCAGCACCCTCCTGCCTGACCATTCGGATTGACTCTTTCCTC
                                                -1
X   8634 CTAAATATGGCCGTAAGCTTACCCA-TCATGAACCACTACTCAGGGAGGCTCCATGATAG
         |||||||||||  |||||  |||  || |||||||||||||  ||||||  ||| |||||||  ||
Y   9248 CTAAATATGGCTGTAAGTTTATTCATTCATGAACCACTGCTCAGGAAGGTTCCATGAAAG
                                                -4
X   8693 GGCAAAAAGTAAACT----CTGACCAGCTTGGTTCTAACCCAGCTAGTAAAATGTAAGGA
         ||||||||||  ||||   ||||||||||||||||||  ||||  |  ||||||||||||  ||
Y   9308 GGCAAAAAGTCAACTCTGACTGACCAGCTTGGTTCTATCCCATCCGGTAAAATGTAAAGA
                                192bp insert on X
X   8749 TTAGGTAAGAT+192bp+TTTGGGCAAATCATTTCCTCTTTCTGGAACCCTGGTTTCCTC
         ||||||||  ||     ||||||||||||  ||||||||  |  ||||||||||||||  |||
Y   9368 TTAGGTAAAATTACTAACTTTGGGCAAATAATTTCCTCTCTTTGGAACCCTGGTTTTCTC
                                +7
X   8934 ATCTGGAGAAAGGAAATAATTATAATAACCATATTTCAAAATATTGTTTGGAGAGTAATA
         || ||||  || ||||||  |  |  |||||  || |||||||||||||||   ||||  |||||
Y   9428 ATTTGGACAAGGGAAATTACTGTAATATTCACATTTCAAAATATTG----GAGAATAATA
                                                                -4
X   8994 TAGTTAATGAATATGAAAAGTGCTTTGTCAAGTATAATATGAGCAAGGTTACTGATTATT
         |||||||  | ||| |||| |||||||||||||||||||||||||||||||||||||||  ||
Y   9484 TAGTTAACAATTATAAAAACTGCTTTGTCAAGTATAATATGAGCAAGGTAACTGATTTTT
                                                Reverse Amelogenin Primer site (Gel Based)
X   9054 TTTTGTATCGATTAAATGCCGTATTACTATATGAAGAATCCTCAAACCTAAGGCTAACCA
         |||   || ||||| |||| |||||||| |||| |||||||| ||||||||||| |||| |
Y   9524 TTT---ATTGATTACATGCTGTATTACCATATAAAGAATCCCCAAACCTAAGGTTAACTA
```

Fig. 1. Comparison of X and Y Amelogenin gene sequences using BLASTN (blast.ncbi.nlm.nih.gov). The Identifiler kit distinguishes on the basis of the 6bp deletion using the green primer sites for PCR. The gel based method described here (section) distinguishes on the basis of the 184 bp difference in length between the PCR products amplified using the blue primer sites (the 184 bp difference arises from a combination of the marked inserts and deletions on X and Y (X -1 -4 +192 = 187) & (Y + 7 -4 = 3) giving, difference = 187-3=184bp).

Typing: Amplified products were separated and detected using the ABI Prism 310xl Genetic Analyzer (Applied Biosystems) according to the manufacturer's recommended protocol. The data were analyzed using GeneMapper ID v3.2 (Applied Biosystems). Alleles were assigned according to the International Society of Forensic Genetics (ISFG) guidelines for forensic Y-STR [9].

Quality control: The laboratory has participated in the Y-STR Haplotyping Quality Assurance Exercise (Certified at 2010-5-20). The data were submitted to YHRD (www.yhrd.org) and received the accession number: YA003680.

RESULTS AND DISCUSSION

Samples from the same 238 Libyan males from Benghazi, who had been typed for Y chromosomal STRs and autosomal STRs[10,11], were tested for a different region of the amelogenin gene from that included in the Identifiler kit. This was in order to compare the reliability of an alternative gender specific amelogenin gene amplification with the Identifiler version, which was 100% correct for the sex determination of this population. The test also allows the detection of amelogenin variants that were not revealed by the Identifiler test.

Of 238 unrelated male samples from Benghazi analysed with Amelogenin primers, 2 samples out of 238 (0.8%) were represented as females (Y null) (A9) (Fig. 2).

For example sample A9 (Figs. 2) only generated the X chromosomal amelogenin amplicon, so typed female though the sample was from a male with a full Y STR genotype and a male result for the Identifiler amelogenin test. This suggests that the Y chromosome is largely intact and that the mutation or deletion preventing amplification of the Y amelogenin is confined to a limited region beyond that tested in the Identifiler kit and does not affect any Y STR loci. The two Y null males were unrelated with different surnames.

The frequency of Y null in this Benghazi population (observed with Agarose Gel electrophoresis) is similar to the one detected in Taiwan 0.006% (2.20). And less than Indian, Malay and Austrian populations, they were 3.2-3.6 %, 0.6-0.88 % and 0.02 % for males respectively[4, 6]. The frequency was higher in Indians than other populations, and the possibility was reported that partial Y deletion formed a relatively old and stable haplotype in the Indian ethnic group[4]. Higher frequency of deletion for the Y amelogenin region in Indian population group was due to deletion of the fragment Y (p) 11.2. For Austrian study, the samples which lacked the amplicon of Y amelogenin in males were also identified[6, 7]. These samples were confirmed to be from males by amplification of Y-STR and SRY (sex-determining region of Y chromosome) gene.

the controversial cases, were not only reported for males but also for females, of human gender identification by amelogenin test , two male samples from Benghazi tested with Amelogenin primers for gender estimation, with X deletions (band 542 bp missed) in both samples out of 238 (0.8%) which are already represented as males when typed with Y Filer and Identifiler kits in the current study for same reason where Y deletion occurred in the previous example, X deletions may due same reasons. Both of the samples were bearing different surnames (thin bands some times due to degradation of DNA or defect in Amplifiction process rather than deletion it self and for this reason both samples were repeated twice and gave same results of X deletions). The anomalous amelogenin results of 8 samples in Taiwan. The frequency was about 0.00625 % for males and 0.015 % for females, and lower than those of the other populations reported [11].

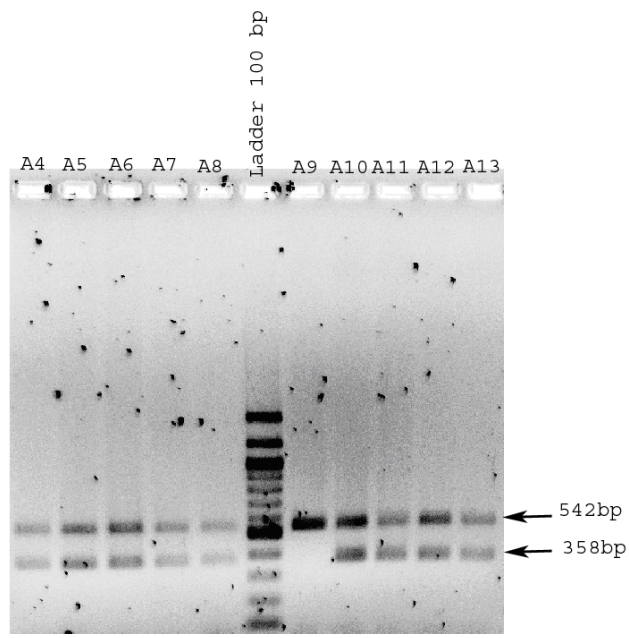

Figure 2. Male Libyan samples from Benghazi Amplified with Amelogenin (A9 known to be male was represented on gel as female).

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REFERENCES

1. Santos FR, Pandya A, Tyler-Smith C. Reliability of DNA-based sex test. Nat Genet 1998; 18: 103.
2. Shadrach B, Commane M, Hren C, Warshawsky I. A rare mutation in the primer binding region of the amelogenin gene can interfere with gender identification. J Mol Diagn 2004; 6: 401-405.
3. Thangaraj K, Reddy A, Singh L. Is the amelogenin gene reliable for gender identification in forensic casework and prenatal diagnosis? Int J Legal Med 2002; 116: 121-123.
4. Chang YM, Burgoyne LA, Both K. Higher failures of amelogenin sex test in an Indian population group. J Forensic Sci 2003; 48: 1309-1313.
5. Chang YM, Perumal R, Keat PY, Yong RY, Kuehn DL, Burgoyne L. A distinct Y-STR haplotype for Amelogenin negative males characterized by a large Y(p)11.2 (DYS548-MSY1-AMEL-Y) deletion. Forensic Sci Int. 2007; 166: 115-120.
6. Steinlechner M, Berger B, Niederstatter H, Parson W. Rare failure in the amelogenin sex test. Int J Legal Med 2002; 116: 117-120.
7. Mitchell RJ, Kreskas M, Baxter E, Buffalino L, Van Oorschot RA. An investigation of sequence deletions of amelogenin (AMELY), a Y-chromosome locus commonly used for gender determination. Ann Hum Biol. 2006; 33: 227-240.
8. Cadenas AM, Regueiro M, Gayden T, Singh N, Zhivotovsky LA, Underhill PA, Herrera RJ. Male amelogenin dropouts: phylogenetic context, origins and implications. Forensic Sci Int 2007; 166: 155-163
9. Gusmão, J.M. Butler, A. Carracedo, P. Gill, Kayser M, W.R. Mayr, N. Morling, M. Prinz, L. Roewer, C. Tyler-Smith, P.M. Schneider; DNA Commission of the International Society of Forensic Genetics, DNA Commission of the International Society of Forensic Genetics (ISFG): an update of the recommendations on the use of Y-STRs in forensic analysis, Forensic Sci. Int. 157 (2006) 187-197.
10. Elmrghni, Y.M. Coulson-Thomas, M. Kaddura, R.A. Dixon, D.R. Williams, Population genetic data for 17 Y STR markers from Benghazi (East Libya), Forensic Sci. Int. Genet. (2011) DOI: 10.1016/j.fsigen. 2011.05.001.
11. Elmrghni, S, Yvette M Coulson-Thomas, Mahmoud Kaddura, Ron A Dixon and D. Ross Williams.. Genetic data provided by 15 autosomal STR loci in the Libyan population living inBenghazi, Forensic Sci. Int. Genet. (2011)doi:10.1016/j.fsigen. 2011.07.006
12. Kao, M.S.; Li-Chin Tsai, James Chun-I Lee; Hsing-Mei Hsieh. Controversial cases of human gender identification by amelogenin Test, Forensic Science Journal; 6 (2): (2007) 69-71.

Effect of Smoking on Plasma Homocysteine Level in Some Libyans as a Risk Factor for Cardiovascular Disease

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ABSTRACT

Back ground: Hyperhomocysteinmia is an independent risk factor for arteriosclerosis, recurrent thromboembolic complications and osteoporosis. Hyperhomocysteinemia may result from deficiency of folate, vitamin B-6 or vitamin B-12. Accordingly homocysteine level is highly influenced by many factors affecting individual life style such as age, sex, smoking and many other factors affected by environmental conditions. Many studies have carried out on homocysteine level in some countries in order to reach an indicative value for future cardiac problems in individuals and to undertake prophylactic measures.

Aim: This study was designed to see the effect of age, sex and smoking on fasting plasma homocystein (F-HCYST) levels in some Libyan Benghazi Citizens.

Methods: of 100 adults volunteers (50 males and 50 females) included active smokers, passive smokers and control groups were chosen as a representative samples for this study. Levels of F- HCYST was measured in relation to age , sex, and smoking by using HPLC Technique.

Results: The F-HCYST levels were higher in males than that in females. According to age the higher F-HCYST levels were found in (30-39 years) and (> 60 years) age groups. As regard to smoking, the levels of F-HCYST were higher in passive smokers of females than in passive smokers of males.

Conclusion: There is increased level of plasma homocysteine in relation to specific age groups, male cases, and the effect of smoking in Libyan population. A preventive measure has to be undertaken.

Key words: *Homocystiene; HPLC, Smoking, Cardiovascular Disease; Libya; Benghazi*

INTRODUCTION

Homocystiene was first identified in patients in 1962 [1]. Is an intermediate sulfur-containing amino acid that is formed during the intracellular metabolism of methionine, which is an essential amino acid supplied by dietary proteins. When homocysteine is formed, it is recycled to methionine after remethylation by two different pathways. The first involves methionine synthetase, which is an enzyme that uses vitamin B12 as an essential co-factor and -methyl-tetrahydrofolate as the methyl donor[2]. The second pathway, which occurs in hepatic tissue, involves the enzyme betaine-homocysteine methyltransferase. Homocysteine may be converted to cystathionine by cystathionine beta-synthetase, which is a vitamin B6-dependent enzyme. Cystathionine is then hydrolyzed to form cysteine[3]. Plasma levels of homocysteine refer to the total pool of homocysteine, since there is very little free homocysteine. Excess homocysteine can damage vascular endothelium by increasing oxidative damage, stimulating smooth muscle cell growth, or activating leukocytes, platelets and plasma clotting factors. Sadly, it is an overlooked marker of cardiovascular health[4] . The Nutrition Committee of the American Heart Association recommends a fasting homocysteine level of <10 umol/l. Serum levels of folate, vitamin B6 and vitamin B12 are inversely correlated with serum homocysteine levels, with folate being probably the most important single nutrient[5]. Elevated levels of plasma homocysteine can cause atherosclerotic vascular disease by increasing arterial endothelial dysfunction. Elevated homocysteine levels may also increase thromboxane-A2 formation in platelet aggregation, proliferation of smooth muscle cells,

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increase activation of factors V and X, increase fibrinogen levels, reduce serum antithrombin activity and increase binding of lipoprotein (a) to fibrin[6].

Homocysteine is highly implicated in vascular diseases and is probably a much more reliable indicator of vascular health than cholesterol is[7]. Homocysteine can be reduced with sufficient vitamin B-12 and folate supplementation with vitamin B-6 sometimes playing a supporting role[8]. The very large Hordaland total plasma homocysteine and cardiovascular risk profile study conducted in Norway identified a positive association between elevated plasma total homocysteine levels and a number of cardiovascular risk factors including smoking[9].

SUBJECTS AND METHODS

Subjects

100 healthy volunteers consist of 50 adult males and 50 adult females from different age groups (20-70 years).

Included 20 active and 20 passive male smokers which divided to (mild, moderate and severe) according to smoking index (the number of cigarettes smoked per day multiply by the duration of smoking by years).

And 40 passive female smokers can measure the toxic effect of smoking here by multiply the hours of exposure per day in the number of the smoking by years, and 20 cases of non smokers of both sexes were chosen for this cross-sectional study.

Those volunteers selected when they presented with their relative patients in hospital after complete informed consent.

5 ml blood samples were withdrawn from every volunteer, centrifuged plasma were separated from each blood sample and kept out of light.

DATA ANALYSIS

Data were analyzed by statistical package of social science (SPSS) Program.

Method

Performance Liquid Chromatographic (HPLC) Measurement of Homocysteine* using CHROMOSYSTEM Gmb- H for measurements of homocysteine level in serum and plasma.

HPLC (High Performance Liquid Chromatography) is a very precise method which has been widely in use for measuring HCY up to now. Homocystine is released from cells in whole blood left at room temperature, so rapid separation of plasma HCY is required for reliability of the test. The plasma should be immediately cooled on ice and then freezed below -20c until time of analysis. Methionine loading test (giving 100mg/kg methionine orally and measuring HCY level 6h later) was established for uncovering hyper-HCY in those suspected to have Hyper-HCY but with normal fasting HCY. But the value of this test has been debated and some authorities believe that measurement of fasting HCY level is enough.

Apparatus: HPLC measurements were performed on Waters instruments equipped with, a model 600 pump, U6K-injector and 996 photodiode array detectors. Separation was performed on -Bondapak C18 (300 × 3.9 mm) column. Flow rate was maintained at 1.5 mL min^{-1}. Chromatographic peaks were electronically integrated and recorded using the powerful Millennium soft ware (version 2.10) of Waters

Materials: Dithiothreitol (DTT) (Sigma), Tetrabutylammonium Hydroxide (TBA) (Sigma) and d,l - homocysteine (Sigma) and Methanol (HPLC grade of Romil). Other chemicals used were of analytical grade.

Procedure: Optimum responses of homocysteine were observed when excitation and emission wave lengths were set at 270 and 474 nm, respectively. Peak areas and concentrations were determined using Hewlett - Packard Chem station chromatographic management system.

Seprations were achieved using an econosphere C18 cartidge column (150 X 4.6mm I.D., particle size 5 um) Obtained from Merck. Prior to the preparation of mobile phase containing TBA, a commercially available 40% aqueous solution of tetrabutylammonium hydroxide was neutralized to pH 6.4 with phosphoric acid. In all cases, the mobile phases were filtered through filters (pore size 0.2 um) and degassed. Binary gradient elution at a flow rate of 1.1 ml / min was used.

RESULTS AND DISCUSSION

We show that from our cross sectional study that the results of homocysteine level in those volunteers increase slightly with males than in females as many studies prove that 10 normal to mild homocysteine

level in females and between intermediate to severe in males , the explanation of that may be due to some other contributing factors as hormonal factors,9. Genetics [11], life styles, as many studies suggested that. Our study prove that the level of homocysteine increased as the age increase especially between age groups (39-49) years , may be some factors effect the homocysteine to be increased as smoking, coffee , alcohol and other physiological influence as hormonal effect.

And the level increased slightly in old age group may be due to some pathological changes in their vessels will affect that especially renal vessels which delay the reabsorption of homocysteine.Most cases of mild and moderate smoking index have mild to moderate homocysteine level and heavy index have intermediate homocysteine level. As duration per years and number of cigarettes per day inactive smokers increase the homocysteine level will be increased and the hazardous on cardiovascular system will be high. Most of those 20 active smokers have intermediate level of homocysteine (30 - 100 µmol/l). As the number of cigarettes per day increased by smokers the level of homocysteine will be increased and most of those 20 cases mild to intermediate level of homocystein in passive male smokers the homocysteine level mainly in between mild to moderate level , this may be due to the duration and time of exposure per hours per day. And this level of homocysteine carries significant risk for cardiovascular diseases.

Most of female passive smokers have homocysteine level in between mild to moderate and intermediate level with relation to their ages. This if compared with passive male smokers the female passive smokers may have the higher level due to the method of exposure in passive females more closely and contact to smoking inside houses that in males, this will go with studies that prove the risk of exposing to exhale of cigarette smoking. As female passive smokers exposed to smoking per year and per hours per day increased the homocysteine level increased this going with that fact which prove that if any passive smoker expose to smoking more than 25 years and 2 hours per day will be excepted as active smoker for 20 years [12]. According to control groups of females and males (nonsmokers) we observe from our study that the female non smokers have homocysteine level higher than that in males, this as we discussed above because the method of exposure in females more close to smoking inside houses than in males, and this will open a door for more studies about this result and to take the non smokers females as a profile for Libyan population to be compared with others.

Table 1. Distribution of cases according to their age groups and results of homocysteine levels µmol/l

20-29 yrs		30-39 yrs		40-49 yrs		50-59 yrs		>60 yrs	
Homocysteine level		Homocysteine level		Homocysteine level		Homocysteine level		Homocysteine level	
male	Female	male	Female	male	Female	male	Female	male	Female
22.6	17.3	26.5	22.3	25.6	29.4	15.3	25.3	36.19	27.4
19.6	32.3	12.88	36.2	16.9	27.9	31.7	26.7	114.1	39.6
15.1	19	31.2	22.2	23.4	17.3	22.01	16.8	61.9	25.6
15.8	12.1	31.91	26.5	27.2	26.8	25.16	17.9	45.9	22.6
22.02	24.34	18.5	23.2	24.9	16.2	21.27	32.1		
21.79	13.11	50.17	35.12	20.9	31.1	32.16	32.2		
24.6	50.1	36.2	23.32	28.3	33.5	23.4	24.1		
15.5	19.3	26.7	27	29.9	14.7	33.8	13.2		
17.2	39.31	23.6	33.3	37.4	12.9				
13.5	25.09	28.9	46.4						
15.1	12.9	20.7	24.7						
		35.9	23.7						
		12.2	22.6						
		12.24	30.81						

Table 2. shows the Distribution of active smokers according to smoking index:

Smoking index = number of cigarettes per day multiply in duration by a year.

Table 2

Smoking index	No.	%
< 200	4	20
200 -600	11	55
> 600	5	25
Total	20	100

CONCLUSIONS

Our cross-sectional study suggested that smoking habits related with increasing Plasma homocysteine level which considered as a risk for cardiovascular system.

Our study concentrated on healthy 100 volunteers of Libyan population which show that plasma homocysteine level will be increased with the smoking index of those active smokers which divided the smokers to mild, moderate and heavy, homocysteine increased as the number of cigarettes per day and duration per year increase in both active and passive smokers. Effect of passive smoking on homocysteine level more in female than in male. may be due to exposure time to smokers more than in males Both sexes of non-smokers have less levels of homocysteine, comparedwithsmokers but in non-smokers females homocysteine level higher than in non- smokers male group and from this observation we can make homocysteine profile (standard reading) for Libyans and to be compared with others. We conclude from

the study the level of homocysteine increased with age and more slightly in males than in females.

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REFERENCES

1. Ridker PM, Rifai N, Clearfield M, Downs JR, Weis SE, Miles JS, Gotto AM Jr; Air Force/Texas Coronary Atherosclerosis Prevention Study Investigators Measurement of C-reactive protein for the targeting of statin therapy in the primary prevention of acute coronary events. N Engl J Med 2001; 344: 1959-1965.
2. Biesenbach G, Janko O and Zazgornik J Similar rate of progression in the predialysis phase in type I and type II diabetes mellitus. Nephrol Dial Transplant 1994; 9: 1097-1102.
3. Orth SR, Stöckmann A, Conradt C, Ritz E in collaboration with Ferro M, Kreusser W, Piccoli G, Rambausek M, Roccatello D, Schaefer K, Sieberth HG, Wanner C, Watschinger B, Zucchelli P Smoking as a risk factor for end-stage renal failure in men with primary renal disease. Kidney Int1998; 54: 926-930.
4. Bleyer AJ, Shemanski LR, Burke GL, Hansen KJ, Appel RG Tobacco, hypertension, and vascular disease: Risk factors for renal functional decline in an older population. Kidney Int 2000; 57: 2072-2079.
5. Kasiske BL, Klinger D Cigarette smoking in renal transplant recipients. J Am Soc Nephrol 2000; 11: 753-759.
6. Singh R, Barden A, Mori T, Beilin L Advanced glycation end products: Areview. Diabetologia 2001; 44: 129-214.
7. Banchaabouchi M, Marescau B, Possemiers I, D'Hooge R, Leviallain O, De Deyn PP NG, NG-dimethylarginine in renal insufficiency. Eur J Physiol 2000; 439: 524-531.
8. Kielstein JT, Böger RH, Bode-Böger SM, Schäfer J, Barbey M, Koch KM, Frölich JC Asymmetric dimethylarginine plasma concentrations differ in patients with end-stage renal disease: Relationship to treatment method and atherosclerotic disease. J Am Soc Nephrol 1999; 10: 594-600.
9. Nygard O, Vollset SE, Refsum H, Stensvold I, Tverdal A, Nordrehaue JE, Ueland PM, Kvale G Total plasma homocysteine and cardiovascular risk profile. The Hordaland homocysteine study JAMA 1995; 274: 1526-1533.
10. Refsum H, Helland S, Ueland PM. Radioenzymic determination of homocysteine in plasma and urine. Clin Chem. 1985; 31: 624-628.
11. Malinow MR, Nieto FJ, Kruger WD, Duell PB, Hess DL, Gluckman RA, Block PC, Holzgang CR, Anderson PH, Seltzer D, Upson B & Lin QRThe effects of folic acid supplementation on plasma total homocysteine are modulated by multivitamin use and methylenetetrahydrofolate reductase genotypes. Arterioscler Thromb Vasc Biol 1997; 17: 1157-1162.
12. Morabia A Markowitz S Extended lung cancer incidence follow-up in the Mayo Lung Projectandoverdiagnosis.J Natl Cancer Inst. 2007 Jun 6; 99(11): 898-9; author reply 899.

Sodium Dichromate Poisoning - A case Report

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ABSTRACT

Acute sodium dichromate poisoning is a rare occurrence. But incidences of accidental or suicidal ingestion of compounds like this may increase due to its wide use and easy accessibility in textile, printing and paper works the demand for which has increased over the last few years. Diagnosis in such cases can only be done based on the history, clinical and postmortem findings. Chemical analysis in such cases may not be helpful.

Key words: *Sodium Dichromate, Acute Rare Poisoning.*

INTRODUCTION

Chromium forms a number of compounds in various oxidation states. Those of II (chromous), III (chromic) and VI (chromate) states are most important. Chromium III compounds (chromium oxide) is used in the tanning of leather and the compounds containing chromium in the Cr^{VI} state are sodium dichromate, potassium dichromate and chromium trioxide are used extensively in textile industries, printing and painting works[1]. These works are becoming more and more popular. Hence the availability of dichromate is likely to become easier, which can increase the incidence of accidental and suicidal exposure[2]. Studies have been conducted on workers chronically exposed for several months or years to chromium (VI) dust or vapour which leads to perforation of the nasal septum, reduced lung function, asthma, dermatitis etc. But the effects of acute poisoning from ingestion of a large amount of sodium dichromate are rarely reported in medical literature. Important features in a case of acute sodium dichromate poisoning are presented in view of these considerations.

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CASE HISTORY

A female aged 23 years was brought to the casualty with the history of ingestion of Sodium bichromate at her residence. The poison was procured from the printing factory where she worked. The empty bottle with the label of the poison was found at the bed side which confirmed the consumption of Sodium dichromate.

She was taken to a local hospital where she developed bleeding manifestations from gums and gastrointestinal bleed because of which she was referred to our Hospital for further management.

On admission to our hospital she was found to be in a semiconscious state with a low volume pulse rate of 30 per min. The BP was not recordable.

On investigation Urea and Creatinine were raised 66 and 3.4 mg/dl respectively, Liver function tests showed - bilirubin total and direct within normal limits, SGOT and SGPT were raised 252 and 148 U/L, Alkaline phosphate was found to be within normal limits. Total protein, S.Albumin, was within normal limits. Haematogram was as follows -Hemoglobin 15 gm%, platelet 84,000/ cubic meter. At the Hospital she was treated with 6 units of frozen plasma, 2 units of platelets and 2 pints of fresh blood. IV drips and antibiotics were continued. Her condition improved briefly but slowly deteriorated and she died on the 3rdday of ingestion of the poison.

AUTOPSY FINDINGS

On postmortem examination, her face was congested and there was bluish discolouration of the finger and toe nails. Blood stains were seen over the angle of the mouth and nostrils. Internal examination showed Tardieu's spots over the lungs, pericardium and under the capsule of the liver. Trachea was filled with blood stained froth. The main site of findings was the GIT. The stomach showed haemorrhagic erosive mucosa which was covered with blood stained tenacious mucous (Fig.1). No normal gastric mucosa was seen. The intestine was filled with reddish fluid and mucosa was intensely congested all along the small intestine with partial congestion of the large intestine and the sigmoid colon. Mesentery showed petichial haemorrhages. All the abdominal organs liver, spleen, kidneys were congested. Brain was oedematous and hyperemic and cut section showed petechial haemorrhages. Peritoneal cavity contained around 500 ml of straw coloured fluid. The uterus was seen with congested endometrium and blood clots.

Fig.1. Haemorrhagic erosion of the gastric mucosa.

Viscera and organs were preserved for chemical analysis and histopathology report.

DISCUSSION

In mammals, chromium is an essential trace element involved in lipid and glucose metabolism[3].Selected chromium compounds, particularly hexavalent (Cr^{VI}) ones, are carcinogens, corrosives, delayed contact sensitizers, and have the kidney as their primary target organ[4]. The hexavalent chromium present in dichromate binds nonspecifically to proteins and nucleoproteins and is specifically taken up by red cells and platelets. It is then reduced to trivalent chromium in the cytoplasm. This conversion causes severe free radical damage to mitochondria, particularly in kidney tubules and hepatocytes[5].

Sodium dichromate appears as reddish to bright orange crystals (Fig.2) and easily dissolves in water to give yellow chromate ions[6]. It has been reported that accidental or intentional ingestion of high doses of chromium (VI) compounds, the exact quantity of which is not usually known, results in acute, potentially fatal, effects in the respiratory, cardiovascular, gastrointestinal, hepatic, renal, and neurological systems.A number of case reports have indicated that the lethal oral dose of dichromates and chromium trioxide is within the range 2.5-195 mg chromium (VI).In one fatal case, a woman who ingested 400 ml of a leather tanning solution containing 48 g basic chromium sulphate died of cardiogenic shock 36 hours after hospital admission despite haemodialysis treatment. Post-mortem revealed haemorrhagic erosive gastroenteritis of the entire gut, severe haemorrhagic pancreatitis, pulmonary congestion and oedema, peritonitis, ascites and widespread petechial haemorrhages[3]. Many of the features in this case correlate with our findings in the case being reported.

Fig.2. Sodium Dichromate crystals.

Histopathology examination of the organs revealed that the kidneys showed acute tubular necrosis, liver showed extensive congestion, the spleen, lungs and brain were congested. Chromium compounds, particularly hexavalent ones, have the kidney as their primary target organ4 was found in this case. The viscera report came negative even though we had mentioned the poison.

CONCLUSION

Though this is a rare case of death due to Sodium dichromate poisoning by ingestion there is a likelihood that such cases of poisoning may increase due increasing demands in printing and painting works in which these chemicals are used extensively. This will

go in the literature as a document of death due to it and the features that it produces which can be seen at post mortem. It is also important to note that in such cases chemical analysis may not be helpful and the diagnoses should be made base on the history, clinical findings and a meticulous postmortem.

REFERENCES

1. Metal: Chemical Properties and Toxicity; chapter 63, part IX, chemicals: Encyclopaedia of Occupational Health and Safety.[internet]. 4th ed. Available from: http://ilocis.org/documents/chpt63e.htm
2. Job C. Ammonium dichromate poisoning: A case report Anil Aggrawal's Internet Journal of Forensic Medicine and Toxicology, 2005; 6, (1).
3. L Assem and H Zhu, Toxicological Overview. Institute of Environment and Health Cranfield University 2007 Version 1: 2-6. HPA Compendium of Chemical Hazards. [internet]. Available from: http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1194947365800
4. Shayne C. Gad. Acute and chronic systemic chromium toxicity. Science of The Total Environment 1989 Oct; 86 (1-2):149-157.
5. Stift A, Friedl J, Längle F et al. Successful treatment of a patient suffering from severe acute potassium dichromate poisoning with liver transplantation. Transplantation 2000 Jun 15; 69(11): 2454-2455.
6. Sodium chromate (VI), anhydrous [Internet] Updated: 2002 Dec Available from: http://chemicals.etacude.com/s/sodium_chromate.php.

Age Estimation by Permanent Teeth Eruption Among School Children in Madurai (South India)

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ABSTRACT

The estimation of age of children and adolescents has medico legal significance particularly in India. Tooth enables accurate estimate of the age in the young as tooth formation has less variability. Since several known varying geographical factors influence development of the tooth, a universally applicable, standard eruption time for the permanent teeth poses difficulty in estimation of age. This resulted in a need for a regional study and this study was undertaken. The subjects of the study were 300 children and adolescents (200 males and 100 females) studying in a school in Madurai (South India) and belonging to middle and lower socio-economic groups. The age of subjects ranged from six to sixteen years.

Sequence of eruption of the maxillary teeth in the males were: first molars, lateral incisors, central incisors, first premolars, second premolars, canines and second molars, while the mandibular teeth slightly differed, first molars, central incisors, lateral incisors, first premolars, second premolars, canines and second molars. Sequence of eruption of the maxillary teeth in the females were, first molars, central incisors, lateral incisors, first premolars, second premolars, canines and second molars, while the mandibular teeth slightly differed, with first molars, central incisors, lateral incisors, second premolars, first premolars, canines and second molars. Sequence of eruption in both the males and females were first molars, central incisors, lateral incisors, first premolars, second premolars, canines and second molars. In conclusion, the mandibular teeth erupted earlier than the maxillary teeth excluding the slightly earlier eruption of the maxillary lateral incisors, premolars and canines in the males and the maxillary lateral incisors, first premolars and canines in the females. When both jaws compared in both the sexes, all the teeth erupted earlier in the females excluding the slightly earlier eruption of the canines in the males.

Key words: *Age, Children, Eruption, Mandibular, Maxillary, Permanent teeth.*

INTRODUCTION

Estimation of the age of children and adolescents from the dental age has medico legal applications. The present study was to determine the median age of eruption of the permanent teeth by oral examination[4] of the erupted permanent teeth out of the gingiva in children and adolescents and to develop a regional standard.

MATERIAL AND METHODS

Subjects selected were 300 children and adolescents (200 males and 100 females) studying in a school in Madurai (South India) and belonging to middle and lower socio-economic groups. The age ranged from six to sixteen years. Children with crowding of teeth, supernumerary teeth, trauma to teeth and caries were not included. The types of permanent teeth on either side designated as follows in the present study. They are central incisor (I_1), lateral incisor (I_2), canine (C), first premolar (PM_1), second premolar (PM_2), first molar (M_1), second molar (M_2), third molar (M_3), the upper teeth as maxillary teeth (Mx), and the lower as mandibular teeth (Mn).

Written consent of parents of the subjects btained. Date of birth from school records and gender of each subject noted. Age of the subject calculated as on the day of dental examination. The subjects were divided uniformly into 20 half-yearly age groups such as 6.0 - 6.5, 6.5 - 7.0 … up to 15.5 - 16. Each group contained ten males and five females.

The erupted permanent teeth observed by oral examination with naked eye in bright day light, which overcame the deficiencies of other anatomical, radiological and microscopic methods. A wooden

spatula used to open the mouth and observe the posterior teeth. A tooth considered to have erupted only when a portion of the crown of that tooth, however small, had penetrated the gingiva and was visible. Between 12 and 20 years of age, development of the root of the third molar tooth was variable and less reliable, hence that tooth excluded from the study. The types of permanent maxillary or mandibular or both maxillary and mandibular teeth that had erupted in each subject in an age group recorded and the same was repeated for an earlier age group and tabulated. From that information, the 50th percentile value was calculated using computerized Probit analysis technique1. The 50th percentile value considered the median age of eruption of a type of tooth. It also gave the median age in which 50% of the population would be expected to have a particular type of tooth erupted.

RESULTS

In the present study, the distribution of age groups for eruption of permanent teeth was the least for M_1 and greatest for I_2 and PM_2 (Table 1).

Table 1. Distribution of type of tooth eruption among schoolchildren according to their age

Type of Tooth	Age group in Males (in yrs)			Age group in Females (in yrs)		
	Maxillary	Mandibular	Both	Maxillary	Mandibular	Both
M_1	6.0-8.0	6.0-8.0	6.0-8.0	6.0-7.5	6.0-7.5	6.0-7.5
I_1	6.0-10.5	6.0-10.5	6.0-10.5	6.0-9.5	6.0-9.5	6.0-9.5
1_2	6.0-12.5	6.0-12.0	6.0-12.5	6.0-10.5	6.0-10.5	6.0-11.0
PM_1	8.0-13.0	8.0-13.0	8.0-13.0	7.0-12.0	7.0-12.0	8.5-12.5
PM_2	7.5-14.0	8.0-14.0	9.0-14.0	7.0-12.0	7.0-12.5	8.5-12.5
C	8.5-15.0	8.5-15.0	9.5-15.0	8.0-13.5	8.0-13.5	9.0-14.0
M_2	9.5-15.5	9.5-15.5	10.5-14.0	9.0-14.5	9.5-14.5	10.0-15.0

The median age (50th percentile value) (in yrs) of eruption of maxillary, mandibular, and both maxillary and mandibular permanent teeth in males and females. The consensus is that the permanent mandibular teeth erupted earlier than the maxillary and the teeth erupted earlier in the females than males (Table 2).

Table 2. Median age (50th percentile) (in yrs) of eruption of maxillary, mandibular, and both maxillary and mandibular teeth of each type in males and females

Tooth	Median Age in Males			Median Age in Females		
	Maxillary	Mandibular	Both	Maxillary	Mandibular	Both
M_1	7.40	7.10	7.45	6.90	6.60	7.20
I_1	8.80	8.40	9.45	7.50	7.30	8.20
I_2	8.60	9.00	9.50	8.50	8.70	9.30
PM_1	10.30	10.70	11.40	9.60	10.40	10.70
PM_2	11.30	11.50	11.80	10.00	9.60	10.75
C	11.60	11.90	12.35	10.60	11.20	12.45
M_2	13.20	12.90	13.40	12.40	12.00	13.00

Maximum difference in the eruption age between the maxillary and mandibular teeth in the males found in I1, PM1, and the minimum difference in PM2; maximum difference in the eruption age between the maxillary and mandibular teeth in the females was in PM1 and the minimum difference in I1 and I2. Maximum difference in the eruption age between the males and females was in PM2 and minimum difference in C. Eruption was earlier in the females except C very slightly later, but completed earlier in females [Table 3].

Table 3. Teeth and duration (yrs) of earlier eruption in males and females.

Tooth Type	Duration of earlier Eruption									
	Males		Females		Maxillary		Mandibular		Both	
	Maxillary	Mandibular	Maxillary	Mandibular	Male	Female	Male	Female	Male	Female
I_1	-	0.4	-	0.2	-	1.3	-	1.1	-	1.25
I_2	0.4	-	0.2	-	-	0.1	-	0.3	-	0.2
C	0.3	-	0.6	-	-	1.0	-	0.7	0.1	-
PM_1	0.4	-	0.8	-	-	0.7	-	0.3	-	0.7
PM_2	0.2	-	-	0.4	-	1.3	-	1.9	-	1.05
M_1	-	0.3	-	0.3	-	0.5	-	0.5	-	0.25
M_2	-	0.3	-	0.4	-	0.8	-	0.9	-	0.4

DISCUSSION

In Northern Ireland, each mandibular tooth erupted before its maxillary counterpart except for the premolars. Females tend to have earlier teeth eruption before males with the exception of maxillary and mandibular second molar[2]. In Niedersachsen, Germany, the eruption times of teeth in females were earlier than in males. Permanent dentition in females completed earlier than in males. The mandibular and maxillary tooth eruption occurred symmetrically, with a slightly advanced eruption of the mandibular teeth for both sexes. The eruption of the canine preceded second premolar[3]. In Athens (Greece), eruption of mandibular and maxillary teeth of males and females was symmetrical. Mandibular teeth had a tendency for earlier eruption in both genders[4]. In Paderborn, Germany, mandibular teeth erupted earlier. Teeth erupted earlier in females[5]. In Tehran (Iran), mandibular teeth erupted earlier excluding premolars[6]. In German state of Saarland, mandibular teeth erupted earlier excluding premolars and first molar in males[7]. In Latakia (Syria), mandibular teeth erupted earlier excluding premolars and first molar[8].

The classic sequence of eruption of the maxillary teeth was, M_1 - I_1- I_2- PM_1-C- PM_2- M_2 and of the mandibular teeth was, I_1- M_1 - I2-C- PM_1- PM_2- M_2 [cited in 2].

In the Present study, the Sequence of Eruption was as follows:

Maxillary teeth-Male	M_1 -I_2- I_1- PM_1- PM_2-C- M_2
Mandibular teeth-Male	M_1 - I_1- I_2- PM_1- PM_2-C- M_2
Maxillary teeth - Female	M_1 - I_1- I_2- PM_1- PM_2-C- M_2
Mandibular teeth - Female	M_1 - I_1- I_2- PM_2- PM_1-C- M_2
Both male and female	M_1- I_1-I_2- PM_1- PM_2-C- M_2

These sequences did not match the classic sequences. The present sequence of eruption for the maxillary teeth in the females and for both the males and females corresponded to[2] Kochher and Richardson (1997). The present sequences for mandibular teeth in males and maxillary teeth in females were identical but the maxillary teeth in males and mandibular teeth in females differed. Eruption of the maxillary I_2 in males preceding I_1 was unique and not reported elsewhere. Though the basic structure of the sequence was similar, the present sequences differed from those of a few other studies in and out of India by a tooth exchanging with another tooth or a tooth preceding another tooth or positioning after another tooth. Exchange of maxillary I_1- I_2 and C- PM_2 in males, exchange of mandibular C- PM_2 in males, exchange of maxillary C- PM_2 in females and exchange of mandibular C- PM_2 in females from Niedersachsen[3]. Exchange of maxillary I_1- I_2 in males and exchange of mandibular PM_1- PM_2 in females from Athens[4]. Exchange of maxillary I_1- I_2 in males, maxillary C positioning after PM_2 in males and exchange of mandibular C- PM_2 in females from Paderborn[5]. Exchange of I_1- I_2 in males and mandibular C positioning after PM_2 in males from Tehran[6]. Exchange of maxillary I_1-I_2 in males, mandibular C positioning after PM_2 in males, exchange of maxillary C- PM_2 in females and exchange of mandibular C- PM_2 in females from Saarland[7]. Exchange of maxillary I_1- I_2 and C- PM_2 in males and mandibular PM2 preceding C in the females from Latakia8. Exchange of maxillary I_1 - I_2 and C - PM_2 and mandibular C positioning after PM_2 in males from Delhi[13].

Indian studies from Madras and Lahore[9], Chandigarh[10], Delhi girls[11], Jats from Haryana[12] and Delhi boys[13] showed similar dentition pattern. Small differences found in eruption pattern of permanent teeth in children from Madras city and Lahore[9]. In well-nourished Delhi boys, median age of eruption of maxillary and mandibular teeth (wheat eating)[13] was much advanced to the present finding in middle and lower socioeconomic group (rice eating). In Delhi boys, maxillary first premolars erupted before canines; mandibular canines erupted before first premolars[13], which was comparable to other Indian studies. This pattern also observed in Kenyan and Asian[14], Chinese[15] and Japanese[16] children. However, was not so in the present study. The median age of eruption was also similar in these studies. In American[17] and north Ireland, children[2] dentition delayed and the pattern varied as the mandibular central incisors erupted before first molars. In Asian children, dentition was advanced by one year and nine months as compared to the American[17] (Eveleth 1966) and Ireland[2] (Kochhar and Richardson 1998) children respectively. Several differences in the dentition pattern such as eruption age, earlier eruption of maxillary or mandibular, or in males or females, maximum and minimum difference in eruption age of counterpart teeth, and sequences of children and adolescents in this study with those in studies in and out of India suggest ethnic variations in dentition and need for regional standards.

The estimated age (in yrs) of a young individual in many combinations as more than the rounded off median age of eruption of the erupted permanent teeth in males and females (Table 1).

Table 1. Estimation of age from rounded off median ages of eruption of erupted permanent

Rounded off ages (in Years)							
	M_1	I_1	I_2	PM_1	PM_2	CM_2	M_2
Maxillary in Males	7	8	9	10	11	11	13
Mandibular in Males	7	8	9	10	11	12	13
Maxillary in Females	7	7	8	9	10	10	12
Mandibular in Females	6	7	8	10	9	11	12
Maxillary & Mandibular in Males	7	9	9	11	11	12	13
Maxillary & Mandibular in Females	7	8	9	10	10	12	13
Maxillary in Males or Females	7	8	8	10	10	11	12
Mandibular in Males or Females	6	7	8	10	10	11	12
Maxillary or mandibular in Males	7	8	8	10	11	11	13
Maxillary or Mandibular in Females	6	7	8	10	9	11	12
Maxillary & Mandibular in Males & Females	7	8	9	11	11	12	13

Age of an individual also estimated from as many types of permanent teeth that had erupted (Table 2).

Table 2. Estimated age indicated by as many types of erupted permanent teeth.

Females Age (in years)	Male Age (in years)							
6	7	M_1						
7	8	M_1	I_1					
8	9	M_1	I_1	I2				
9	10	M_1	I_1	I2	PM_1			
10	11	M_1	I_1	I2	PM_1	PM_2		
11	12	M_1	I_1	I2	PM_1	PM_2	C	
12	13	M_1	I_1	I2	PM_1	PM_2	C	M_2

REFERENCES

1. D.J. Finney, Statistical Method in Biological Assay, third ed.., Charles Griffin and Co., Ltd., London, 1978.
2. R. Kochhar, A. Richardson, The chronology and sequence of human permanent teeth in Northern Ireland, Int. J. Paediatr. Dent. 8 1998 243-152.
3. J.S. Wedl, V. Schoder, R.E. Friedrich, Tooth eruption times of permanent teeth in male and female adolescents in Niedersachsen, Arch. Kriminol. 213 2004 84-91.
4. J. S. Wedl, S. Danias, R. Schmeizle, R.E. Friedrich, Eruption times of permanent teeth in children and young adolescents in Athens (Greece), Oral Investig. 9 2005 131-134.
5. R.E. Friedrich, H. Katerji, J.S. Wedl, H.A. Scheuer, Eruption times of permanent teeth in children and adolescents of Pederborn, Westphalia, Germany, Arch.Kriminol 217 2006 20-35.
6. R.E. Friedrich, N. Kamalwand, S.W. Jurgen, H.A. Scheuer, Eruption times of permanent teeth in male children and adolescents of Tehran (Iran), Arch. Kriminol. 219 2007 145-168.
7. R.E. Friedrich, A. Leist, H.A. Sheuer, Eruption times of permanent teeth in children and adolescents in the German state of Saarland, Arch. Kriminol. 222 2008 73-104.
8. R.E. Friedrich, S. Habib, H.A. Scheuer, Eruption times of permanent teeth in children and adolescents in Latakia (Syria), Arch. Kriminol. 223 2009 84-97.
9. K.L. Shourie, Eruption age of teeth in India, Indian.J. Med. Res. 34 1946 105-118.
10. S. Kaul, S. Saini, B. Saxena, Emergence of permanent teeth in school children in Chandigarh, India. Arch. Oral. Biol. 20 1975 587-593.
11. B. Kour, R. Singh, Physical growth and age at eruption of deciduous and permanent teeth in well nourished Indian girls from birth to twenty years, Am. J. Hum. Biol. 4 1992 757-766.
12. S. Kaul, S. Prakash, Eruption of permanent teeth in Jats in Haryana, in: Human growth & development, J. Borns, R.S. Itauspie, S. Sunanne, Hebbelink, Ney York, Plenum Press, pp. 343-352.
13. K.N. Agarwal, R. Gupta, M.M.A. Farridi, N. Kalra, Permanent dentition in Delhi boys of age 514 years, Indian Paediatr. 41 2004 1031-1035.
14. J. Hassanali, J.W. Odhimbo, Age of eruption of the permanent teeth in Kenyan African and Asian children, Ann. Hum. Biol. 8 1981 425-434.
15. M.M.C. Lee, W.D. Low, K.S.F. Chang, Eruption of the permanent dentition of southern Chinese children in Hongkong, Arch. Oral Biol. 10 1965 849-861.
16. P.B. Eveleth, de Souza, J.A. Freites, Tooth eruption and menarche of Brazilian born of Japanese ancestry, Hum. Biol. 41 1969 176-184.
17. P.B. Eveleth, Eruption of permanent dentition and menarche of American children living in the tropics, Hum. Biol. 38 1966 176-184.

Bilateral Optic Neuritis Following Ressell's Viper's Bite-A Rare Complication

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ABSTRACT

Snake bite is an important health problem in the Indian subcontinent with nearly 60,000 people being bitten every year. Though ocular complications are not uncommon, optic neuritis is very rare and only a few case reports are available in the literature.

We hereby present a case of bilateral optic neuritis following a bite by Russell's Viper which is extremely rare, the treatment of which consists of maximum dose of ASV and early treatment of anticipated ocular complications to prevent permanent loss of vision.

Key words: *Bilateral Optic Neuritis, Snake bite, Russell's Viper*

INTRODUCTION

Snakebites are a common medical emergency in the tropics, particularly in rural and farming areas. Venomous snakebites may result in neurologic or hemostatic dysfunction. Reports of ophthalmic manifestations after viperine snakebites include subconjunctival hemorrhage, hyphema, retinal hemorrhage, vitreous hemorrhage, central retinal artery occlusion, visual loss due to cortical infarction, and macular infarction. Bilateral optic neuritis following snake bite by Russell's viper is very rare and so the treating physician might be in a dilemma for the treatment of the same. Here we will discuss the role of immediate and complete treatment of the same complication which can lead to complete recovery of the patient.

CASE

A 40 year old female got admitted to Krishna hospital with history of a snake bite(Russell's viper-as identified by the patient) on scalp on left side on entering her home 1 hour before admission to the hospital. Following this, she developed complete blindness in both eyes with no perception of light also bilaterally within 30 minutes of the snake-bite. There was no local swelling or tenderness. On examination, her vitals were stable, her haemogram was within normal limits except her 20 minutes whole blood clotting time which was positive(>20 minutes). The neurological examination revealed bilateral ophthalmoplegia with bilateral complete blindness and convergent squint bilaterally. Except for these, there were no other neurological abnormalities. Her other systemic examination was grossly normal. She was immediately treated with three cycles of injectable atropine(0.6mg) with neostigmine(0.5mg) each dose of both given at an interval of 15 minutes and meanwhile, 150 units of lyophilised polyvalent antisnake venom was also given after sensitivity test and injection tetanus toxoid was also given. The vision didn't improve and the repeat whole blood clotting time after 6 hours of first dose of ASV was again positive(>20 minutes) and so 100 units of lyophilised polyvalent antisnake venom(ASV) was repeated and meanwhile, ophthalmologist was called for expert opinion. There was no improvement in vision even after maximum full dose of ASV. Ophthalmoplegia was reversed immediately after first dose of ASV. Ophthalmologist diagnosed it to be having bilateral optic neuritis on fundoscopy. MRI brain, done to rule out other causes of acute onset blindness, was normal. Patient was given pulse doses of steroid [injection methylprednisolone 1 gram diluted in 100 cc of normal saline and transfused over 1 hour] once daily for 5 days. Patient's vision started improving gradually and

it was normal after 5 days of starting methylprednisolone. Patient was discharged with normal vision and haemodynamically stable condition.

Fig. Bilateral Convergent Squint

DISCUSSION

Snake bite is an important health problem in the Indian subcontinent.1 There are very few cases of optic neuritis following a snake bite[2-8]. To our knowledge, there are only 7 case reports of optic neuritis following snake bite. Interestingly, in three of them the fall in vision was reported on the 6th day[3-5]. All these patients had received ASV. Optic atrophy following a bite from a viper is extremely rare. The literature search revealed only one case from Nigeria which was reported in 1956[3]. No case of blindness was seen in a series of 115 cases of carpet viper bites reported by Warrell et al. in Nigeria[10]. In our case, blindness immediately followed the bite by Russell's Viper which is the second only case in the world.

The possible causes of the optic neuritis include the snake venom, allergy to the antivenim serum, and the multiple and extensive haemorrhages. However, there is no evidence that viper venom does have a direct toxic effect on the optic nerve, nor has it been reported as a manifestation of allergy to anti-snake venom. It is possible that, in this case, the optic nerve changes resulted from a severe haemorrhage. It is known that haemorrhage in other parts of the body can result in optic neuritis which generally appears 3-7 days after the haemorrhage.

Mofredj et al. reported a case of loss of vision due to ischaemic anterior optic neuropathy following massive gastrointestinal bleeding from a peptic ulcer.[11]

Terson (1922) gives the following statistics on the time of appearance of blindness after haemorrhage:

During haemorrhage 8.3 Immediately after ... 11.6 Within 2 days ... 19.2 3-16 days ... ... 39.2 Over 16 days ... 7.5	Total 100

Though treatment by ASV still forms the mainstay of treatment of a poisonous snake bite we should be aware of its possible reactions[5]. Some newer, less allergenic substitutes are being developed, namely more purified ASV using polyacryalamide gel affinity chromatography,[12] purified Fab fragments of TgG,[13] monoclonal antibodies (cobra venom[14]), and highly refined purified antivenom from sheep or chicken.

Early treatment with ASV and appropriate and immediate treatment of the ocular complications can prevent further damage of vision and in some cases, can revert back to normal if treated appropriately.

REFERENCES

1. Nigam P, Tandon VK. Snake bite: a clinical study. Indian J Med Sci 1973: 27: 697-704.
2. Chopra PC. Optic neuritis after cobra bite. Indian Med Gaz 1939; 74:420.
3. Davenport RC, Budden FH. Loss of sight following snake bite. Br J Ophthalmol 1953; 37: 119-121.
4. Guttmann-Friedmann A. Blindness after snake bite. Br J Ophthalmol 1956; 40:57-59.
5. Mathur SP. Allergy to antivenine serum. Br J Ophthalmol 1959; 43: 50-51.
6. Sahai AS, Sinha RH. Bilateral blindness following snake bite. Indian J Ophthalmol 1978;26:16.
7. Rao KV. Optic neuritis and ophthalmoplegia caused by snake bite. Indian J Ophthalmol 1981; 29: 243-245.
8. Sanghavi NG, Amin SK, Naik RS. Bilateral optic neuritis following snake bite.JAssoc Physicians India 1982; 30: 117-118.
9. Guttman-Friedman A. Blindness after snake bite. Brit J Ophthal 1956;33: 119-121.
10. Warell DA, Davidson NMcD, Greenwood BM, et al. Poisoning by bites of the saw-scaled or carpet viper in Nigeria. Q J Med 1977; 46: 33-62.
11. Mofredj A, Curan D, d'Arondel C, et al. Blindness following gastrointestinal haemorrhage. Eur J Gastroenterol Hepatol 2000; 12: 1339-1341.

12. Russell FE, Sullivan JB, Egen NB, Jeter WS, Markland FS, Winger WA, et al. Preparation of a new antivenin by affinity chromatography. Am J Trap Med Hyg 1985; 34:141-150.
13. Sullivan Jr JB. Past, present and future immunotherapy of snake venom poisoning. Ann Emerg Med 1987; 16: 938-944.
14. Theakston RD. New techniques in antivenom production and active immunization against snake venom. Trans R Soc Trop Med Hyg 1989; 83: 433-435.

Palm Length - A Predictor of Stature in Indian Population

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ABSTRACT

Stature estimation of an individual is considered as one of the vital parameters in identification. Present study was conducted over 257 South and North Indian subjects in Tumkur, India to establish stature of an individual using palm length. Statistical analysis indicated that bilateral variation was insignificant. Sex differences were found to be highly significant. Linear regression equations for stature estimation were calculated and multiplication factors computed. The correlation coefficients between stature and palm length were found to positive and statistically highly significant.

Key words: *Stature, Palm length, Identification, Indian Population.*

INTRODUCTION

Identification of an individual is the mainstay in forensic investigations. In medico-legal autopsies, establishing personal identity of the victims is often required.[1] When a complete dead body is found, stature determination is rather an easy task, but in cases where only some parts of the body are available, the determination of stature of the individual is difficult. Estimation of stature of an individual from the skeletal material or from the mutilated or from amputated limbs or from parts of limbs has obvious significance in the personal identification in the events of the murders, accidents or natural disasters mainly concerned with the forensic identification analysis.[2]

Many factors like racial, ethnic and nutritional factors play an important role in human development and growth; therefore different nomograms become necessary for different population[3]. The present study is an attempt to understand the relationship between stature and palm length among South Indian and North Indian subjects who belong to different geographical locations & of different food habits.

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MATERIALS AND METHODS

The present study was undertaken in the departments of Anatomy and Forensic Medicine and Toxicology, Sri Siddhartha Medical College, Tumkur, amongst 257 right handed medical students, aged between 20-30 years. The student population of Tumkur comes from all over India. The division of subjects into South and North Indians was based on their region of origin and taking into account other zonal divisions of India (Srinivasan, 2002)[4]. The total sample consisted of 106 South Indian females, 51 South Indian males, 50 North Indian females and 50 North Indian males.

Inclusion Criteria

- All the subjects included in the present study were healthy male and female students of North and South Indian origin.

Exclusion Criteria

- Students with deformities of vertebral column and limbs, students with poorly defined distal flexion crease of forearm & proximal flexion crease of middle finger were excluded.
- Left handed individuals were excluded to maintain uniformity.

In the present study, palm lengths of both hands along with stature of each individual were measured using standard anthropometric instruments (anthropometer and sliding calipers) in centimeters to the nearest millimeter. All the measurements were

taken by one observer in order to avoid inter-observer bias. Diurnal variations have been reported in the stature of an individual; thus all measurements were taken during afternoon hours to avoid diurnal variations[5].

Stature was measured as vertical distance from the vertex to the floor, where vertex is the highest point on the head when the head is held in Frankfurt Horizontal plane. Measurement was taken by making the subject to stand erect on a horizontal resisting plane, bare footed with back of the shoulders and buttocks touching the wall. Palms of hand were turned inwards and fingers horizontally pointing downwards. Anthropometer was placed in straight vertical position in front of the subject with head oriented in eye-ear-eye plane (Frankfurt Plane). The movable rod of the Anthropometer was brought in contact with vertex in the mid sagittal plane. Precautions were taken not to exert pressure as that may affect the contact measurement.

Subjects were asked to place their hands supine with fingers extended and adducted on a flat horizontal surface. Palm length (PL) was measured using sliding calipers (graduated in mm) from mid-point of distal transverse crease of forearm to mid-point of proximal flexion crease of middle finger. Measurements were taken to accuracy of 0.1 cm[6].

Analysis was done using Statistical software namely SPSS 15, Stata 8.0, MedCalc 9.0.! and Systat 11.0 to calculate linear regression equations and compute multiplication factor. Multiplication factor for palm length was calculated by dividing the stature of an individual by palm length, respectively for each subject in males and females and dividing the sum by the sample size. Pearson's correlation coefficient was calculated to establish the correlation between the stature and palm length. Paired sample t-test was performed to find the right and left side differences in palm lengths among males and females. The significance of results was tested using Student's t-test. P-value of less than 0.01 was considered as significant. Predictive value of all equations was compared.

RESULTS AND DISCUSSION

Descriptive statistics for bilateral palm length measurements and stature among South Indian and North Indian males and females are shown in table 1. Stature and palm length on both sides were significantly greater ($p<0.001$) in males when compared with females, which is in concurrence with other studies.[7-11] An association of Y-chromosome with stature has been documented. In addition, age of puberty being two years later in males as compared with females gives them extra time for growth[12].

Statistical analysis indicates that bilateral variation was insignificant for the measurements of palm length in both sexes. No statistically significant differences were found in the mean stature and palm length of South and North Indian population when compared for the sex. These findings are compatible with those arrived at by Rastogi et l, which suggests that in persons of different population groups(belonging to the same race) geographical variations do not have much influence on body proportions. Thus stature can be estimated using any of the mentioned formula irrespective of person being South or North Indian. Correlation coefficient (r) was determined as depicted in Table 2. Correlation coefficient ranged from 0.583 in North Indian males to o.686 in south Indian males which is nearly the same as calculated by Rastogi et al (0.597 to 0.684). Linear Regression equations, multiplication factors and coefficient of determination (R^2) are derived for the estimation of stature for the study groups as shown in Table 3. R^2 value ranged from 33.9% to 47.0%.

Table 1. Comparison of stature estimation of palm length in Indian population

Variables	SIF	SIM	NIM	NIF	Overall sig
Height	156.82±5.17	169.62±6.89	168.86±5.28	156.39±5.18	<0.001**
Rt palm length	9.57±0.52	10.51±0.57	10.5±0.49	9.52±0.51	<0.001**
Lt palm length	9.54±0.52	10.51±0.56	10.53±0.47	9.52±0.48	<0.001**

Table 2. Correlation coefficient of Height and Palm measurements

Variables	r value (SIF)	r value (SIM)	r value (NIM)	r value (NIF)
Height vs Rt palm length	0.672	0.619	0.674	0.640
Height vs Lt palm length	0.672	0.619	0.674	0.640

Table 3. Regression analysis for predicting the stature using the palm length

Variables	Regression equation with R^2value
SIM	Ht=83.82+8.16 RPL; R2=45.1%
	Ht=80.11+8.52 LPL; R2=47.0%
NIM	Ht=98.35+5.72 RPL; R2=38.3%
	Ht=99.25+6.61 LPL; R2=33.9%
SIF	Ht=92.99+5.67 RPL; R2=45.4%
	Ht=93.55+6.62 LPL; R2=45.2%
NIF	Ht=94.02+6.56 RPL; R2=41.0%
	Ht=89.24+7.06 LPL; R2=42.0%

CONCLUSION

Estimation of stature is an important parameter in determining the partial identity of unidentified & dismembered bodied. In the present study reveals that

palm length can be used successfully to predict stature in the Indian populations & it is highly reliable for the estimation of stature in forensic examinations. The results of this study are however applicable only amputated hand is found & brought for examination and other parts of the body are unavailable. Similar studies are proposed on individual's palms that may be helpful, if the mutilated remains of a hand are brought examination.

REFRENCES

1. Momonchand A and Devi Merra Th. Determination of Stature from the Interacromial length. JFMT; 1999: 16(1): 72-73.
2. Tyagi AK, Kohli A, Verma SK and Aggarwal BBL. Correlation between stature and fingers length. International Journal of Medical Toxicology and Legal Medicine;1999: 1(2): 20-22.
3. Qamra R.Suneel, Jit Indar and Deodhar. A Model for reconstruction of height from foot measurements in an adult population of North West India. Indian J Med Res; 1980: 71: 77-83.
4. Srinivasan P. (ed) (2002) The TTK Zonal Road Atlas of India. Chennai, TTK Health care Limited, 7-89.
5. Kanchan T et al. Stature estimation from foot dimensions. Forensic Science International; 2008: 179: 241-245.
6. Singh IP and Bhasin MK. Anthropometry -A Laboratory manual of biological anthropology. Delhi: Kamla Raj Enterprises; 1968: 1-35.
7. Malek AKA, Ahmed AF, Aziz El Sharkawi SAE and Abd EL Hamid NAEM. Prediction of stature from hand measurements. Forensic Science International; 1990: 46:181-187.
8. Habib SR and Kamal NN. Stature estimation from hand and phalanges lengths of Egyptians. J Forensic Legal Med; 2010:1-5.
9. Krishan K and Sharma A. Estimation of stature from dimensions of hands and feet in a North Indian population. J Forensic Legal Med; 2007: 14 (6):327-332.
10. Rastogi P and Yoganarsimha K. Stature estimation using palm length in Indian population. International journal of medical toxicology & legal medicine; 2008:11(2):37-40.
11. Jasuja OP and Singh G. Estimation of stature from hand and phalange length. JIAFM 2004; 26(3): 0971-3.
12. K. Yamada, M. Ohta, K. Yoshimura and H. Hasekura, A possible association of Y chromosome heterochromatin with stature, Hum. Genet; 1981: 58: 268-270.

List of abbreviations used

- SIF - South Indian female - 110 subjects
- SIM - South Indian male - 50 subjects
- NIM - North Indian male - 50 subjects
- NIF - North Indian female - 51 subjects
- HT- Height
- RPL - Right palm length
- LPL - Left palm length

Crime, Society and Role of Forensic Medicine

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ABSTRACT

The bondage of Law and Forensic Medicine has always been a boon to the society, resulting in numerous intriguing cases being correctly investigated, retaining the sanctity of the Judicial system. The role of Forensic Medicine fraternity in assisting the various national and state agencies in maintenance of law and order has been paramount. Forensic medicine focuses on the areas in which medicine and human behavior interface with the law. The basic function of Forensic Medicine is to assist in the administration of justice. From postmortem examination and scene investigations, it is often possible to reconstruct the course of events leading to death. This is useful for police investigations and subsequent pro

secution in the law courts. Due to lack medical and paramedical staff, scientific awareness and knowledge among victims, investigating agencies, the end result is either 'justice hurried is justice buried' or 'justice delayed is justice denied' to the victims.

Key words: *Society, Crime Forensic Medicine, Forensic Pathologist, Criminal Justice.*

INTRODUCTION

As technology continues to progress and provide further degrees of convenience to our lives, the world of criminal justice is also influenced by the by-products of this new and exciting era. The rapid availability of new technology in the world of crime has not only provided law enforcement officials with a greater array of tools and new methods to aid them in the pursuit of criminal justice, but it has also provided them with new challenges, as criminals now have a similar advantage in aiding them in their illegal activities. Since the invention of modern policing and corrections in the 19th century, progress has often been measured in terms of technical innovation. Thus we have seen the advent of Virtopsy[1], DNA Profiling, fingerprinting, wireless communications, the motor car, and other devices which have long since become mundane. The adaptability, and the malign creativity of criminals, however, requires the ongoing development of means to prevent, or at least to minimise, their harmful activity. In this context forensic medicine has found its existence.

The bondage of law and medicine is ancient one, the earliest medico-legalist being the priest who was both law giver and healer. The oldest known law code that of Hammurabi, King of Babylon at around 2200 B.C. contained legislation of medical practice and the rights and duties of medical men. It provided controls on medical and surgical practice, dealt with medical malpractice, and clearly set out the civil and criminal liability of the physician. In Egypt, too, laws were laid down for the practice of medicine and stab wounds, for example, were defined in the seventeenth century B.C. Hippocrates of Greece (460-355 B.C.) discussed the lethality of wounds. Subsequently, the Hippocratic Oath became the foundation of medical ethics. The first medico-legal examination on record was performed on Julius Caesar on March 15, 44 B.C. by Antistius, a Roman physician, who externally examined the great emperor's body and found that only one of his twenty-three wounds was mortal. Over the time the concept of forensic science evolved. The Justinian Code, which made its appearance in Rome around 550 A.C., stated that physicians were witnesses in a dispute before a tribunal or the government. This was the first recognition of the expert witness in history[2].

By the 1600s, legal medicine had advanced beyond the status of just a theoretical pursuit and was introduced into legal texts and courtrooms throughout Europe. In the 17th century medico-legal institutes and departments were established in Universities in Germany, Austria, France, and Italy. This overwhelming reality in the continental Europe was followed by similar changes in the United Kingdom

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where in 1807 the first chair of Legal Medicine in the English-speaking world was established in Edinburgh. In parallel the Scotland Yard in the United Kingdom has been developed over the last century into a modern multidisciplinary investigative police agency[3].

The interdependence and association of Law and Forensic Medicine has always been a boon to the society, resulting in numerous intriguing cases being correctly investigated, retaining the sanctity of the Judicial system. The role of Forensic Medicine fraternity in assisting the various national and state agencies in maintenance of law and order has been paramount. As European nation, states and their judicial systems developed, physicians and surgeons participated more frequently in legal proceedings. Increasingly, high profile criminal and civil legal actions around the world highlight the interactions between medicine and the law. Forensic and legal medicine describes the body of knowledge that encompasses this interaction. However, the nature of forensic and legal medicine is broad and may extend beyond medical and legal issues, into scientific and technical areas, and include specialist roles such as anthropology, toxicology, odontology and psychiatry. Separate from these issues is the much more widely recognized need to apply moral, ethical and human rights principles in the investigation of certain crimes, whether considering victims or perpetrators. Those involved in the practice of forensic and legal medicine and those in legal, judicial, police and other investigative organizations which require knowledge of aspects of forensic and legal medicine often need to identify appropriate and relevant information.

DISCUSSION

Forensic medicine plays a vital role in the Criminal Justice system. The role of forensic medicine has been expressly designed to provide solutions to some of the most urgent concerns in our society. Forensic medicine focuses on the areas in which medicine and human behavior interface with the law. Strictly speaking, the basic function of Forensic Medicine is to assist in the administration of justice. From postmortem examination and scene investigations, it is often possible to reconstruct the course of events leading to death. This is useful for police investigations and subsequent prosecution in the law courts. Murders disguised as suicides or accidents are exposed and the perpetuators brought to book. Lies are Countermanded and the truth upheld. Cause and manner of a death are not always evident even after visual examination and dissection of the body. Over the centuries, forensic medicine has developed technologies of visibility, ways of seeing things that would otherwise be undetectable.

- Post-mortem examination explore into the human body for concealed wounds, foreign objects, and other evidence.
- Chemical analysis explore invisible traces of poison, establishes the identity of body, body parts and fluids, and helps link the victim's body to the perpetrator and the crime scene.
- The microscope helps to see tiny lesions, crystals, microbes, and distinguish hairs and fibers.
- Spectroscopic analysis of blood, tissues, and material found on or near the body, helps distinguish and match trace elements that link the body of the victim, crime scene, and perpetrator.

But no method is infallible. The tests and procedures of forensic medicine have not always stood up to the scrutiny of judges, expert witnesses, lawyers, professional peers, and the public. Over time, the experience of forensic medicine in the courtroom has led to improvements in science, technology, and investigative procedures[4].

Another role for Forensic Medicine is to serve in various governmental committees dealing with medico-legal matters. This includes advising on legislation relating to medicine such as on matters pertaining to autopsy and human organ transplantation, drunken driving, use of seat belts and other like matters. It is also an important component of the Quality Assurance Committee to ensure good medical standards by investigating complaints against doctors and nurses, looking into medical mishaps and, to enquire into possible criminal negligence in the deaths of patients. The clinical part of Forensic Medicine deals with the examination of living patients in cases of assault, poisoning, drunken driving, rape, etc. and the provision of medico-legal advice and opinion on these cases.

Crime is prevalent in every country and society, most of them either goes undetected or unreported. In India, the crimes are increasing day by day. As per National crime records bureau[5], in 2010 a total of 22, 24,831 crimes were reported under Indian Penal Code. Crime in 2010 has increased by 4.9% compared to 2009. At the same time Conviction rate is not up to expectation, Less than ten percent of crimes finally end in conviction and societal demoralization is inevitable. With above crime scenario, forensic medicine plays an important role in helping to prevent, early detection, providing expert medico legal and scientific reports

at earliest, which will result in quick trial and instant administration of justice to victims. If any further evidence for the crucial role of forensic medicine for the society is needed, it is worth pointing out that proper forensic investigation results in a marked increase in conviction rate. Available data shows that the conviction rate in CBI cases is 67.12 compared to merely 6% in those investigations by various state police.[6] The difference is basically because the CBI utilises expert (forensic) scientific evidence which is neglected by the state police.

Due to lack of legal or medico legal, scientific awareness and knowledge among victims, investigating agencies, medical and paramedical staff, the end result is either 'justice hurried is justice buried'or 'justice delayed is justice denied' to the victims. One accused acquitted means breeding of hundred criminals. Prevention and detection is better than conviction. As has been happening all these years, forensic medicine is an ornamental and cosmetic utility of the investigating agencies which completes the formality of legal process and satisfies the lay public. It is showcased and remembered only when major or sensational crimes occur to satisfy the inquisitive and demanding media and citizens. Compared to other disciplines of science and technology, forensic medicine is static and stunted in India. It is not being utilised in its own right with the full thrust to help the investigating law enforcement agencies and the criminal justice system. The benefits of improving, regulating and re-organising forensic medicine vis-à-vis other technologies are obvious as it virtually assists the law enforcement agencies in criminal investigations, provides proactive assistance, enhances internal security, helps criminal justice administration and reduces the risk of wrongful conviction/ exoneration[7].

In India there is huge shortage of forensic doctors and forensic Pathologist. This could be due to the stigma attached to an autopsy surgeon, or it could be due to the poor working conditions, or the pressures of doing work that is disliked by many in society. Except in those hospitals attached to medical colleges, medico-legal autopsies are usually carried out not by forensic medicine doctors but by MBBS doctors who have insufficient experience of such work. As a result, there is a chance that the medical evidence is not properly presented in court. On the other hand, graduates do not consider that a forensic scientist's job is lucrative enough, though it is very challenging. Due to shortages of staff in most central and state-run forensic laboratories, reports are delayed (and justice delayed is justice denied) and there are greater chances of overworked staff making mistakes. By having forensic experts on, both, the prosecution and defence side in a case, we can eliminate bias. By having private forensic science laboratories we can have independent testing of samples by non-government laboratories. But, unfortunately, with a shortage of staff - both forensic doctors and forensic scientists - the idea of a forensic expert for the defence remains a distant dream in India.[8]

Recently, a ray of hope has come from the Ministry of Home Affairs, Govt. of India regarding the Bill on Forensic Science Services[9] which will also include Forensic Medicine to develop a redressal statuary mechanism to provide Forensic Pathology Services as a right of victim and dead persons and state obligation, as well as to ensure quality, on time credible Forensic pathology services commensuration with crime and population statistics. Once the new Forensic Science Service (Regulatory Board) Bill 2011 is passed, the government expects that private forensic laboratory will take the head of over burden national and state forensic facilities, but the government has failed to realise that when the private labs come to exist the Indian criminal justice system their bound to confusion, for instance in Shanti Bhushan case, the Hydrabad lab reported that the CD is genuine while private lab reported them to be doctored. Thus, to privatise the forensic laboratories is perilous and any compromise will jeopardise the whole criminal justice system in the country. Therefore, the government should stop this Bill from being enacted in present shape and increase manpower and infrastructure in government lab to dispose of cases urgently. The developed countries are using the private sector to the minimum extent after their federal and State laboratories are fully developed. Thus, in India the forensic laboratories have to first develop fully and then think about involving the private sector.

Globally the judiciary is depending more & more on scientific evidences in comparison to eye witnesses. The future of judicial system in India, as loaded with galore of cases like Nithari rape and murder cases, Arushi and Hemraj murder case, Jessica Lall case etc. They all have suffered because of the inferior forensic facilities in our country. The case highlights the hazards of a tutor (medical officer without any expertise in the subject) conducting shabby post-mortems. The Supreme Court has taken steps to modernize the judiciary investigating system & gradually consider the scientific evidences when delivering the judgments. In Muniammal vs The Superintendent Of

Police[10] the Honourable Madras High Court has directed the Medical Council of India in following words:

"Medical Evidence is a scientific factor, which plays crucial role for determining many of the crimes perpetrated against the human body. Further, the know-how in Forensic Medicine on the part of Medical Officers is of utmost significance for Justice Delivery System. At this juncture, this Court, after going through the materials available in this case and in the backdrop of the authorities on the subject, deems it appropriate and feels compelled to place some suggestions for consideration and implementation by the authorities concerned, in the following manner : (a) Directorate of Medical Sciences and Directorate of Medical Education with the concurrence of the State Government may contemplate imparting periodical training to the Medical Officers, who are in Government service, on Forensic Medicine, to make their efficiency updated in the field. A standardised format of noting down the injuries and their signs can be evolved so that a uniform procedure for issuing medical certificate be followed state wide. The said authorities may constitute a team of experts to prepare the format, so as to make the job of doctors, who perform medico-legal functions, easier. (b) Every doctor posted in any Government hospital may undergo a week's training in Forensic Science Department in the nearby Government Medical College periodically and the State Government may evolve a scheme in this regard. (c) The Government may provide sufficient infrastructural facilities to the mortuaries and places where autopsies are conducted." Time and again several courts have reprimanded and pulled down the doctors for their callous attitude and also suggested for increase importance to be given in training in forensic medicine. Thus, the legislature and the authorities concerned, should take immediate steps to implement these directions to make forensic medicine more viable, more teeth and trendsetting.

CONCLUSION

Today's is world of science and technology, criminals and terrorists are equipped with the latest technology tools. In such situation, the success of lawsuits and prosecutions is dependent on utilizing the best services available, and knowing when to use them appropriately. Improved methodology of detecting and documenting evidence, ensuring chains of custody and scientifically testing evidence ensures that appropriate judicial outcomes are achieved. Thus, the branch of science of Forensic Medicine is an effective scientific method, which plays a vital role in assisting the Justice Delivery System in traditional and techno-crime to render justice to the society. The service rendered by the Forensic Medicine Experts in this regard is unique and deserves for admiration, but the real state of affairs remain that medico legal cases are handled in this country by the non-forensic experts and none could be blamed in this regard. No one can expect that a Forensic Medicine Expert alone would be deputed for conducting post-mortem examination and to attend the medico legal cases, since the strength of such expert is comparatively minimal, to cater to the needs. Similarly, in most of the cases the court fails to deliver the justice due to deficiency in medico legal reports. The investigation, if carefully and meticulously carried out, will help ascertain the truth. This will not only assist the law courts in the administration of justice but also benefit the profession, community and society. It is a dynamic field, never static, and uses all the advanced technologies to meet the challenges ahead.

REFRENCES

1. Virtopsy, is a new imaging horizon in forensic pathology. The concept of "virtopsy," are promising enough to introduce and evaluate cause of death, relevant traumatological and pathological findings, vital reactions, reconstruction of injuries, visualization, by using postmortem multislice computed tomography (MSCT) and magnetic resonance imaging (MRI).
2. Aurore Schmitt, Forensic Anthropology and Medicine (Human Press, 2006) at p.6.
3. Camps F.E., (ed.) The history of legal medicine. (Bristol: John Wright &Sons, 1976): 1-13 at p.2.
4. T.C. Chao, "Forensic Medicine: The Changing Roles for the Challenges Ahead" , (1991) vol. 13 (1) Malaysian journal of Pathology, 1-4 at p.2.
5. "Crime in India -2010" Press Information Bureau, Government of India Ministry of Home Affairs; 27 October 2011.
6. www.cbi.nic.in/newsarticles/newsreport/poi_reportno24.htm; retrived on 20 March 2012
7. Dr. T. R. Baggi, 'Why is forensic science stunted and static in India?,' The Hindu, (September 11, 2011).
8. Jagdeeh N. Reddy, "The status of forensic medicine in India," Indian Journal of Medical Ethics Vol. V; 4 October-December 2008) at p.154
9. Forensic Science Service(Regulatory Board) Bill 2011.
10. www.indiankanoon.org/ retrieved on 30 march 2012.

Sudden Death Due to Pulmonary Embolism: Two Case Reports

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ABSTRACT

Deaths due to pulmonary thromboembolism are common and the diagnosis is often missed because patients present with nonspecific signs and symptoms. This may lead to allegations of negligence against the operating surgeon or the hospital. Only a meticulous postmortem examination and histopathlogical findings can help in determining the immediate cause of death as pulmonary embolism. In this paper we discuss two rare cases of pulmonary thromboembolism one in which the embolus was found in the right atrium and a case of saddle embolus.

Key words: *Pulmonary Embolism, Medical Negligence, Sudden Death.*

INTRODUCTION

Pulmonary embolism is the most under diagnosed cause of sudden death and less than half of fatal pulmonary embolism[1]. As a cause of sudden death, massive pulmonary embolism is second only to sudden cardiac death. The diagnosis is often missed because patients with pulmonary embolism present with nonspecific signs and symptoms. Despite diagnostic advances, delays in pulmonary embolism diagnosis are common and represent an important issue[2]. If left untreated, approximately one third of patients who survive an initial pulmonary embolism die from a subsequent embolic episode[3].If a patient dies under this situation, there is a strong possibility that the operating surgeon or the anaesthetist, who assisted the surgical procedure, can be charged with medical negligence by the ignorant relatives[4]. The following two cases presented below, one of which is a rare case of saddle embolism.

Case 1

On 25-11-10, a female aged 55 years was hit by a two wheeler while walking along the side of a road and was admitted in a local hospital. As her condition did not improve after being treated for over a month, her relatives decided to shift her back home on 03-01-11. She expired on 08-01-11 at her residence. The police suspected foul play and asked for the viscera to be preserved for chemical analysis. Post-mortem was conducted on 09-01-11 at 02:35pm in our hospital with findings as below:-

External examination:

1. Surgical scar 4cms x 3cms over the left side of the forehead 4cms above the left eyebrow.
2. Surgical scar 6cms x 1cm over the outer aspect of the lower 1/3rd of the right forearm.
3. Surgical scar 20cms x 1cm over the outer aspect of the right thigh (Fig.1).

Fig. 1. Surgical scar over the thigh.

Internal examination

- Heart showed a saddle embolus extending from the right ventricle to the right and left pulmonary arteries (Fig. 2).

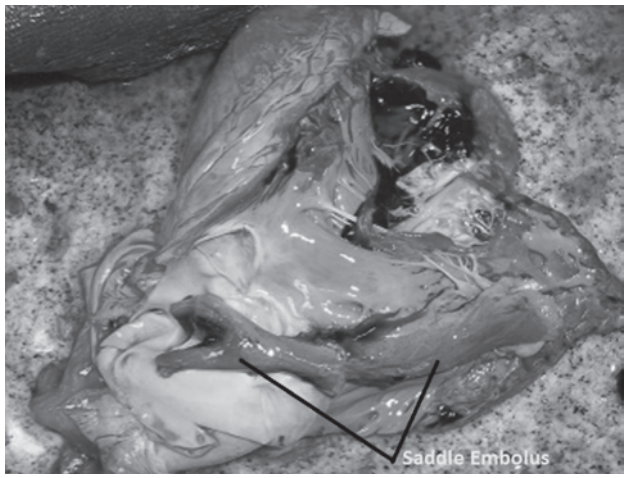

Fig. 2. Saddle embolus in the right ventricle.

- All organs were congested.

Section of the embolus along with other organs were sent for histopathology examination.

Significant findings of the Histopathology Report

Lungs shows patchy areas of mixed inflammatory infiltrate composed of polymorphs and lymphocytes within the alveoli. Areas of necrosis seen.

Section of the material sent as embolus show features compatible with thrombus.

Case2

On 02-04-11, a female aged 50 years, was hit by a two wheeler sustaining injury to her right hand and right leg, she underwent treatment at a hospital and showed signs of recovery but on the night of 20-04-11 she collapsed and died suddenly, the probable cause of death was diagnosed as myocardial infarction. Post-mortem was conducted on 09-01-11 at 02:35 pm in our mortuary with the following findings:

External examination:

1. Surgical sutured wound 7cms in length over the lateral aspect of the upper 1/3rd of the right leg (Fig.3).

Fig. 3. Surgical sutured wound over the right leg.

2. Contusion 8cms × 6cms over the back of the upper 1/3rd of the right leg.
3. Abrasion 1cm × 1cm over the back of the right hand.

Internal examination

Heart showed a grayish white embolus measuring 4cms × 3cms × 2cms in the right atrium and the right ventricle showed number of floating thromboemboli covered with a sheath of post mortem clot (Fig.4) extending up to the pulmonary arteries.

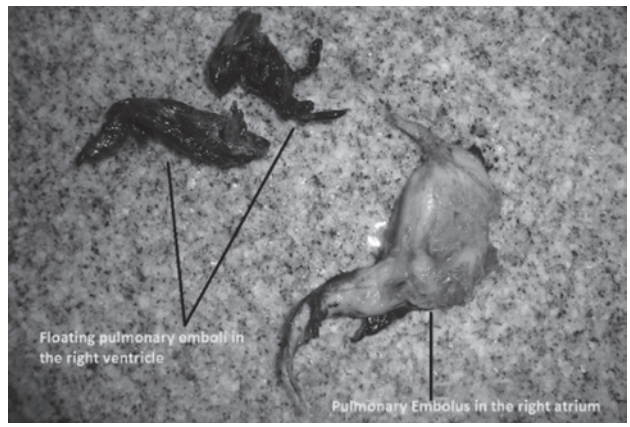

Fig. 4. Pulmonary embolus in the right atrium and floating emboli in the right ventricle.

All organs were congested.

Section of the embolus along with other organs were sent for histopathology examination.

Significant findings of the Histopathology Report

Section from the lung showed large areas of infarction with acute inflammatory cell infiltrate and emphysematous changes.

Specimen sent as thromboembolus show features compatible with thrombus.

Cause of death in both cases was due to

Based on the postmortem and histological findings the Final Opinion was given as the deceased would have appeared to have died of pulmonary embolism as a result of injuries sustained.

DISCUSSION

The incidences of pulmonary embolism are difficult to calculate and are commonly underdiagnosed. Pulmonary emboli have been estimated to occur in more than 600,000 per year in the United States and result in 50,000 to 200,000 deaths annually. Venous thromboembolism is a common immediate cause of death diagnosed by the forensic pathologist because of its sudden and unexpected presentation[5].

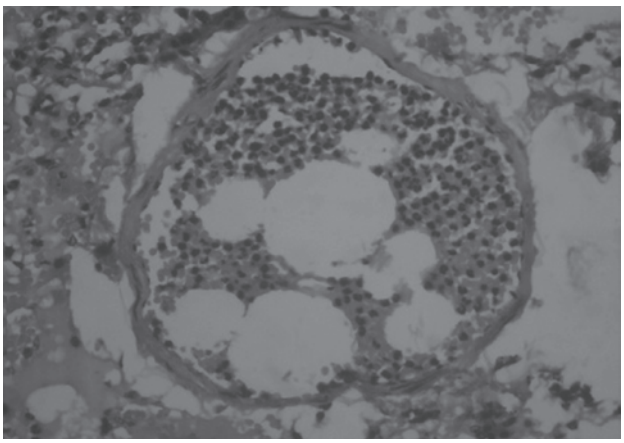

Fig. 5. Slide showing pulmonary thromboembolus.

The victims of many forms of trauma are at risk from pulmonary embolism because:

- Tissue trauma increases the coagubility of blood for several weeks.
- Injury to the tissues, especially the legs or pelvic region, may cause local venous thrombosis in the contused muscles or around fracture bones.
- The injury may confine the victim to bed. Prolonged immobility leads to pressure on the calves and reduced venous return and stasis. The common result is thrombosis of the deep veins of the legs; small emboli may break off and impact in more peripheral branches of the pulmonary arteries that may be precursors of a massive embolus that impacts in the major lung vessels causing rapid death.

At autopsy, large emboli are readily visible and can usually be easily distinguishable from post mortem clot. The latter is dark red, soft and jelly like, with a shiny glistening surface. It is often separated into chicken fat plasma clot and dark red cell clot by sedimentation after death. Post-mortem clot may be adherent to the ante-mortem embolus and sometimes forms a sheath around it, so that the true nature is obscured unless a careful examination is made. Histological confirmation of an antemortem clot must be made if there is any doubt[1].

At autopsy, the pulmonary arteries should be incised during routine removal of the lung and close attention to the cut surfaces of the blood vessels (including the vena cava) should be given. Some emboli may lodge in the right heart and can be missed if they drop out of the vena cava during removal of the heart. Typically, adherent embolism will bulge from the incised pulmonary arteries. Even after removal of the lungs from the thorax, the thromboemboli usually will remain lodged in the hilar pulmonary arteries[5].

As a cautionary approach, in such cases, once the sternum is removed it is always better to dissect the heart first by opening the right ventricle along the border of the interventricular septum up to the pulmonary trunk extending into the right and left pulmonary arteries followed by dissection along the lateral border of the right ventricle up to the right atrium exposing the vena cava in order to rule out any presence of pulmonary emboli.

In the above two cases, there was no direct allegation of medical negligence against the operating surgeons in either of the cases. Though in the second case, doubts were raised by the relatives of the deceased as to why an apparently healthy individual who they assumed had recovered would die all of a sudden. In the latter case the responsibility lies on the Forensic examiner to explain the unexpected consequences in a case of trauma in order to avoid allegations of negligence against the surgeon and vicariously the hospital.

CONCLUSION

Though pulmonary embolism is not new to the world, there still lies a burden on the Forensic examiner to rule out any consequences to avoid allegations of negligence against the hospital or an individual. The purpose of writing this was to highlight the importance of conducting a meticulous autopsy in all cases of sudden deaths not forgetting of pulmonary embolism. This article should go into the literature and add up to the previous work done on the topic. As in the cases mentioned above it is valuable to elucidate a detailed history before conducting an autopsy as a pulmonary emboli can be over looked unless a careful examination is made. A meticulous postmortem examination and histopathlogical findings only can help in determining the immediate cause of death as pulmonary embolism.

REFERENCES

1. Pekka Saukko, Bernard Knight. Knight's Forensic Pathology; 3rd edition; pg 340-341.
2. Ozsu S, Oztuna F, Bulbul Y, et al. The role of risk factors in delayed diagnosis of pulmonary embolism. Am J Emerg Med. Jan 2011; 29(1): 26-32.
3. Daniel R Ouellette, MD, FCCP. Pulmonary Embolism [Internet] Updated: Aug 18, 2011. Available from: http://emedicine.medscape.com/article/300901-overview.
4. Dr DS Bhullar, Dr KK Aggarwal, Dr Ramesh Kundal. Pulmonary embolism & Medical Negligence: Two case reports; J Punjab Acad Forensic Med Toxicol, 2009; 9(1): 35-41.
5. Michael Tsokos. Forensic Pathology Reviews, Volume 3; 2005; pg 286-291.

Use of Carpal Bones as Skeletal Maturity Indicators in Indian Pediatric Age Group

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ABSTRACT

Background: Bone age assessment is frequently performed in children and adolescents for the evaluation of growth, management of limb length discrepancies, scoliosis and the diagnosis and management of a multitude of endocrine disorders and paediatric syndromes. Carpals are the most important bones in determining skeletal age.

Objective: Aim of the study is to find out ossification of carpal bones in Indian children and to find which carpals are more affected in their ossification.

Materials and Methods: The ages of ossification centers of carpals at wrist joint have been studied in pediatric age group in Maharashtra state of India. The study included 306 apparently healthy children belonging to lower economic group of both sexes (161 male and 145 female) up to 14 years of age.

Result: Capitate and hamate were first (during first year) and Pisiform (9-14 years) was the last to ossify. In the Indian children first four ossifying carpals were less affected, while later four carpals ossifying during 4 to 12 years were more affected in their development.

Conclusions: Ossifications of the 8 carpals have a definite relation with age and can be considered as a good indicator for age assessment and skeletal maturation in pediatric age groups.

Key words: *Skeletal Age, Carpals, Ossification.*

INTRODUCTION

In pediatrics, skeletal maturity (bone age), an expression of biological maturity of a child, is an important quantitative measure used for evaluation of growth, management of limb length discrepancies, scoliosis and the diagnosis and management of a multitude of endocrine disorders and paediatric syndromes. Successive skeletal age interpretations indicate the direction of the child's development and show the progress under treatment.

In law, the crime and punishment is entirely based on criminal responsibility and this in turn is dependant on the age of a person[7]. In the modern society the crimes against the children and by the children are increasing. The pediatric age group has got significant medico-legal importance.

There are various methods for age determination of an individual, in which eruption of teeth and ossification activities of bones are important. For the assessment of child skeletal maturity, radiologists use a hand and wrist radiography because of low level of radiation. Carpals are the most important in determining the age of a child as there are eight numbers of carpals, first appears in first year of life and the last at 12th year[9]. Many medical practitioners use number of carpals in determining the age of child. But exact age of appearance of carpal is not fixed, so in this study we are verifying the age of appearance of each carpal in Indian children and finding which carpals are most affected in the process of ossification.

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Table 1. Showing distribution of number of cases according to age groups and sex

Age Group	No of Cases	
	Male	Female
0-1	11	11
1-2	13	10
2-3	12	9
3-4	10	11
4-5	12	10
5-6	10	8
6-7	11	12
7-8	12	10
8-9	11	8
9-10	10	11
10-11	10	9
11-12	12	11
12-13	13	12
13-14	14	13
Total	**161**	**145**
	53%	47%

Table 2. Showing percentage of each carpal and total number of carpals in each age group of males

Male	0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13	13-14
Capitate	66.7	100	100	100	100	100	100	100	100	100	100	100	100	100
Hamate	66.7	100	100	100	100	100	100	100	100	100	100	100	100	100
Triquetral		23	25	60	100	100	100	100	100	100	100	100	100	100
Lunate					33.3	70	81.8	100	100	100	100	100	100	100
Scaphoid						20	63.6	75	63.6	90	100	100	100	100
Trapezium						20	63.6	66.6	81.8	90	100	100	100	100
Trapezoid						20	54.4	83.3	81.8	100	100	100	100	100
Pisiform											20	33.3	69.2	100
No.Carpal	2*	2+1*	2+1*	2+1*	3+1*	3+4*	3+4*	4+3*	4+3*	5+2*	7+1*	7+1*	7+1*	8

Table 3. Showing percentage of each carpal and total number of carpals in each age group of females

Female	0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13	13-14
Capitate	45.5	100	100	100	100	100	100	100	100	100	100	100	100	100
Hamate	45.5	100	100	100	100	100	100	100	100	100	100	100	100	100
Triquetral		20	33.3	81.8	100	100	100	100	100	100	100	100	100	100
Lunate					50	75	100	100	100	100	100	100	100	100
Scaphoid					20	37.5	75	100	100	100	100	100	100	100
Trapezium					20	37.5	66.7	80	100	100	100	100	100	100
Trapezoid					30	50	83.3	100	100	100	100	100	100	100
Pisiform										27.7	44.4	81.8	100	100
No.Carpal	2*	2+1*	2+1*	2+1*	3+4*	3+4*	4+3*	6+1*	7	7+1*	7+1*	7+1*	8	8

** indicate variable number of carpals which may or may not be present in that age group.*

MATERIAL AND METHOD

The present study was carried out in the department of Anatomy, of Government Medical College, Nagpur during the period from November 2007 to November 2009. Study was performed in total 306 children having ages from 1 day to 14 years out of which 161 were males and 145 were females. The children were apparently healthy and from middle socioeconomic status families. The date of birth (chronological age) and consent for radiographic examination was obtained from the parents of each child. Children examined were from the schools near the medical college and the children who came to the radiology department for x-ray for injury. Children with specific bone age studies or with chronic diseases, endochrinopathies, metabolic disorders or any other disease affecting skeletal maturation were excluded from the study. For the study of carpals, wrist and hand radiographs with AP view were taken. The conventional left side radiographs were used whenever possible. When the left side was not available, the right side was used, since there is little,

non-significant difference in skeletal ossification between left and right hand[6].

All the subjects were divided into 14 chronological age groups according to their age and sex as shown in Table 1. Age group 0-1 consisted of children aged 1 day to 0.99 year, age group 1-2 consisted of children aged 1 year to 1.99 year and like this the other groups were formed.

RESULTS

Ossification of all the eight carpal bones studied according to age groups and sex. Presence of each carpal and its percentage in each age group was noted as shown in Table 2 and 3.

Capitate and hamate ossified during the first year of life in 66.7% children of both sexes and found 100% in 1-2 year age group children of both sexes. The triquetral showed its appearance at 1 to 4 years and 100% at 4-5 years in both the sexes. The lunate showed its appearance at 4 to 7 years and 100% at 7-8 years in males while in females it appeared at 4 to 6 years and 100% at 6-7 years. In case of males, trapezoid appeared at 5-9 years while scaphoid and trapezium at 5-10 years. In case of females, scaphoid and trapezoid appeared at 4-7 years while trapezium at 4-8 years. The pisiform showed its appearance at 10 to 13 years and 100% at 13-14 years in males while in females it appeared at 9 to 12 years and 100% at 12-13 years.

In present study, capitate and hamate ossified during the first year of life and found not to be affected in all age groups. As during first year of life child is on breast feeding, there is less chance of getting malnutrition and once ossification center is appeared it develop progressively. Triquetral and lunate appeared on third and fourth numbers are less affected than the other carpal bones except capitate and hamate. Scaphoid, trapezium and trapezoid appeared near about same age range but earlier in female by 1-2 years. Pisiform is the last carpal to be ossified, found earlier in females. These last four carpals were found to be more affected in ossification.

Table 4. Showing the ages of appearance of carpals according to sex.

	Male	Female
Capitate	0-2	0-2
Hamate	0-2	0-2
Triquetral	1-5	1-5
Lunate	4-7	4-7
Scaphoid	5-11	4-8
Trapezium	5-11	4-9
Trapezoid	5-10	5-8
Pisiform	10-14	9-13

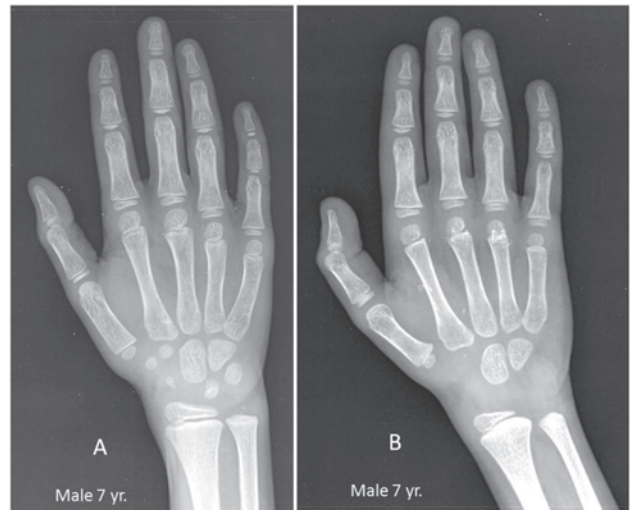

Fig. 1. Showing the radiographs of male children of chronological age 7 years. A- Shows normal appearance of carpals. B- Shows normal development of capitate and hamate bones but no ossification of other carpals. In A and B epiphysis of phalanges and metacarpals show same development.

DISCUSSION

Each carpal bone ossifies from one center, capitate first and pisiform last; the order in other carpal bones varies. The capitates begins to ossify in the second month, the hamate at the end of the third month, the Triquetral in the third year, the lunate, scaphoid, trapezium and trapezoid in the fourth year in females and fifth year in males. The pisiform begins to ossify in ninth or tenth year in females and twelfth in males. The order varies according to sex, nutrition and possibly race[9].

According to Errol Levine[5] carpals are greater source of observer error in skeletal assessments than other hand wrist epiphyses. In addition carpals are more variable in times of onset of ossification than epiphyseal centers, they more frequently show bilateral asymmetry in maturation and appear to be of little predictive value in measurement of ossification status as a whole, but the carpals may provide significant information regarding skeletal development and the health status of a child.

The rate of ossification is primarily affected by the socio-economic development of population concerned8. Malnutrition is one of the most important factors which disrupt postnatal ossification in bones of hand and wrist. This has been demonstrated by many workers[1,4].

Bansari M Maniar et al[2] studied on effect of malnutrition on bones of hand in Indian children. Author stated that between the age of 4 and 8 the upper income group Indian boys are behind the Greulich and

Pyle standards by 2 years. However, upper income group girls closely follow the Greulich and Pyle standard except for a lag between 3 to 5 years. He also wrote that in malnourished every bone of the wrist except for capitate and hamate is delayed in its time of appearance as compared to the upper income group children.

Castelanos JJ et al[3] studied skeletal maturation of wrist and hand ossification centers in normal Spanish children and found that capitate and hamate bones in girls evolve most in accord with chronological age. On the other hand trapezium and trapezoid bones are statistically furthest from chronological age in both sexes.

CONCLUSIONS

In Indian children first four ossifying carpals are less affected, while later four carpals ossifying during 4 to 12 years are more affected in their development. Nutritional need during the age group 4-12 years is not fulfilled, so causing skeletal growth retardation. If once the ossification center for a carpal appears later development is not affected but if there is nutritional deficiency at the time of ossification, delay in the appearance of center is observed (as shown in fig.1). Carpals are more affected by malnutrition than the other epiphysis of the hand. So the size, shape and progress in development of carpals can determine the skeletal maturation.

CONFLICT OF INTEREST

We, the authors declare that we have no financial relationship with any organization for sponsorship of the research. So we have no conflict of interest.

REFERENCES

1. Abbott OD, Towensend RO, et al.(1950) Carpal and epiphysial development; another index of nutritional status of rural school children. Am J Dis Child. Jan;79(1): 69-81.
2. Bansari M.Maniar, Prabha L et al (1973) Effect of malnutrition on bones of hand in children. Indian Paediatrics. vol XI , 3 213-226.
3. Castellanos JJ. Carmona A. et al (1996) Skeletal maturation of wrist and hand ossification centres in normal Spanish boys and girls: A study using the Greulich-Pyle method. Acta Anatomica. 155: 206-211.
4. Dreizen S, Spirakis CN, Stone RE (1964) Chronic under nutrition and postnatal ossification, effect on time and sequence of appearance of the postnatal ossification centers in the human hand skeleton. Am J Dis Child. Jul;108: 44-52.
5. Levine Errol (1972) Contribution of the carpal bones and epiphyseal centres of the hand to the assessment of skeletal maturity. Hum Biol , 44, 3, 317-327.
6. Loder Randall et al (1993) Applicability of Greulich and Pyle skeletal age standards to black and white children of today. Ame J Dis Children. 147:1329-1333.
7. Rao NG (2000) Text book of forensic medicine and toxicology. 1st ed. Jaypee Brothers (P) Ltd. New Delhi, India; 89
8. Schmeling A.et al. (2000) Effect of ethnicity on skeletal maturation: consequences for forensic age estimations. Int J Legal Med. 113: 253-258.
9. Standring S. Gray's anatomy (2008) The anatomical basis of clinical practice. 40th ed. London: Elsevier Churchill Livingstone; 864.

Age Estimation Radiologically from Epiphyseal Union at Wrist Joint Among Subjects in Davanagere District

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ABSTRACT

Determination of age is the common problem that demands forensic expertise. Various studies conducted in India and abroad for estimation of age from long bones revealed that there exists no uniform pattern for epiphyseal union of long bones. The pattern is also different in different regions of the same country. The present study was conducted to estimate age from epiphyseal union at wrist joint.

Radiographs (PA View) of left hand including wrist joint of 120 subjects (60 boys and 60 girls) of age ranging from 15-19 years were studied among subjects in Davangere district of central Karnataka . Stevensons classification of epiphyseal union was used to study the radiographs.

The results of the study showed that the average age of fusion of lower end of ulna is 15-16 years in females and 18-19 years in males and the average age of fusion of lower end of radius is 16-17 years in females and 18-19 years in males. It was concluded from the study that the exact and precise age of an individual cannot be stated, but a range can be given by radiological examination. Females show early union of epiphysis by 1-3 years when compared to males. The results of present study also show appreciable variation in time of union of epiphysis when compared with the observation made by previous workers.

Key words: *Age Estimation, Epiphyseal Union, Radiographs, Wrist Joint, Forensic Radiology.*

INTRODUCTION

Estimation of age of a person is a common problem that demands forensic expertise and is an important criterion in the establishment of identity of an individual. In addition to identification of a person, age is an important feature in day to day legal system, in both civil as well as criminal cases.[1] Age estimation in civil cases is required in situations like making of will, attainment of majority, marriage, voting rights, employment etc. In criminal cases age estimation becomes important in cases of rape, kidnapping, prostitution, criminal abortion and criminal responsibility etc. The gravity of offence depends upon particular age and also certain rights are given to the persons only at certain age. Thus the estimation of age is a task of considerable importance from the view point of administration of justice.[2]

There are various means by which medical personnel can arrive at a fairly accurate opinion about age of a person. Of these, the principal means accepted globally are the eruption of teeth and progress of skeletal ossification. The most common age involved in civil and criminal cases are 16 and 18 yr. Such a study by radiological observation of the period of appearance and fusion of ossification centers of various bones is matter of great medicolegal importance.[3]

Adequate data for each geographical area based on sufficient research is not available at present. Hence the present study was taken up in Davangere district of central Karnataka, to estimate the age of individuals from the epiphyseal union at wrist joint and prepare a data of its population. The data obtained from this study should be of great help to the medical experts, law enforcing authorities and judiciary of this region

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in arriving at a fairly accurate opinion regarding the age of a person concerned with legal matters.

MATERIALS AND METHODOLOGY

The study comprised of 120 subjects from Davangere district of central Karnataka, India. Out of these 60 were boys and 60 were girls. The age group of study ranged from 15-19 years. The subjects were chosen from different schools and colleges of Davangere and also among the subjects visiting the outpatient department of Bapuji and Chigateri General Hospital, Davangere, Karnataka, India. Apparently healthy subjects, having age proof in the form of either birth certificate or school / college admission register and a domicile of Davangere district were selected for the study.

The subjects were categorized into four age groups at an interval of 12 months. Thus among the total of 120 subjects, 30 (25%) of them were selected in each of the four different age groups.

Table 1. Provides the age and sex wise breakdown analysis of the study population

Age in Years	No of Cases		Total (n)
	Male (%)	Female (%)	
15.1 - 16	15	15	30
16.1 - 17	15	15	30
17.1 - 18	15	15	30
18.1 - 19	15	15	30
Total	**60**	**60**	**120**

Informed consent was taken from every individual, prior to examination in the prescribed consent form.

Radiograph of left hand including wrist joint was taken by a single exposure, which included the lower end of radius and ulna, all the carpal bones, metacarpal bones and the phalanges. Stevensons[2,4,5] classification of epiphyseal union was adopted as follows to interpret the radiographs of our study.

Stage I : No union - a clear gap or space is seen between the epiphysis and diaphysis.

Stage II: Begining union - partial closure of gap or space between epiphysis and diaphysis.

Stage III: Recent union - the active process of bony union is over but there is retention of thin line of demarcation at the epiphyseo-diaphyseal junction.

Stage IV: Complete union - completion of process of union and epiphyseal space is bony in architecture.

However for our study purpose, the stage I and II was considered as not fused while stage III and IV as fused.[2,3]

OBSERVATION AND RESULTS

In the present study, a total of 120 the radiographs (PA view) of left hand including wrist joint were studied and following observations were made. The age interval at which, 75% of the cases show fusion was taken as the average age of epiphyseal union for the corresponding centre.

Table 2. Showing fusion of lower end of radius and ulna

Sex	Age in year	Total No.	Lower end of redius fused		Lower end of ulna fused	
			No	%	No	%
Male (n=60)	15-16	15	-	-	4	27
	16-17	15	2	13	8	53
	17-18	15	6	40	11	73
	18-19	15	12	80	14	93
Female(n=60)	15-16	15	7	47	13	87
	16-17	15	12	80	14	93
	17-18	15	14	93	15	100
	18-19	15	15	100	15	100

The results show that, the average age of epiphyseal union of lower end of ulna was 18-19 years as 93% of male subjects showed epiphyseal union in that age group, where as in female subjects, the average age of epiphyseal union of lower end of ulna was 15-16 years where in 87% of subjects showed epiphyseal union. Similarly the average age of epiphyseal union of lower end of radius was 18-19 years as 80% of the subjects showed fusion of lower end of radius in that age group, where as in females the average age was 16-17 years as 80% of females showed fusion of lower end of radius.

It was observed from the study that the average age of fusion of distal end of radius and ulna is much earlier in females than that of males indicating, the epiphyseal union occurs much earlier in females compared to that in males.

Table 3. Showing statistical analysis of mean age of fusion between boys and girls

	Sex	Distal end of ulna	Distal end of radius
Mean Age + SD	M	17.1 ± 1.1 (n = 37)	17.7 ± 0.7 (n = 20)
	F	16.7±1.2 (n = 57)	16.9 ± 1.2 (n = 48)
	M/F	t = 0.12	t = 3.15
		p > 0.05 NS	p < 0.01 S

The mean age at which the females show fusion of distal end of ulna is 16.7 ± 1.2 year and in case of males it is 17.1 ± 1.1. The t value is 0.12 and $p > 0.05$ indicating that there is no significant age difference in male and female with reference to fusion of distal end of ulna.

The mean age at which the females show fusion of distal end of radius is 16.9 ± 1.2 years and in case of males it is 17.7 ± 0.7. The t value is 3.15 and $p < 0.01$ indicating that there is significant age difference in male and female with reference to fusion of distal end of radius and also indicates early fusion in females than the males.

DISCUSSION

A Forensic expert or doctor is often required to give opinion about the age of a person involved in various civil and criminal cases. The important ages with legal implications are 7, 10, 12, 14, 16, 18 and 21 years. In India, 16 years of age is significant for a girl as for as consent for sex is concerned, in alleged cases of rape. Similarly in number of cases false representation of age is made, particularly with respect to the age 18 years. In India 18 years is the age for attainment of majority. At this age the individual enjoys a number of privileges like entry into a government job, making a valid will, voting power and in case of females it is the minimum age for marriage.

Various parameters like height, weight, pubertal changes, eruption and root calcification of teeth etc have been used for estimating age before 25 years. Since all these parameters are variable in both sexes, the study of epiphyseal union of bones is considered universally as a reasonable, scientific and accepted method for estimation of age by the court of law.[6]

Many workers of India and abroad have conducted the studies and recorded appreciable variation in the time of union of epiphysis with their respective diaphysis. It was also observed by them, that the age at which epiphyseal union in bones occurs is influenced by climatic condition, hereditary, nutritional, socioeconomic factors and geographical location.[7] As India is a vast country with diversity of afore mentioned factors, it is not possible to make uniform standards for the entire country.

Keeping in view all the above factors an attempt is being made in the present study to estimate the age among the subjects in Davangere district by the fusion of epiphysis around wrist joint. In the present study, epiphyseal union of distal end of ulna and radius are studied and the results compared with that of the previous workers (Table 4).

Table 4. Comparative study of age of epiphyseal union of lower ends of radius and ulna

Sl No	Research worker	Population studied	Lower end of radius (age in yr)		Lower end of ulna (age in yr)	
			Male	Female	Male	Female
1	Present study	Central Karnataka	18-19	16-17	18-19	15-16
2	Kangne (1999)[2]	Maharashtra	-	17-18	-	16-17
3	Greulich & Pyle(1959)[8]	USA	18	17	18	17
4	Saksena& Vyas (1969)[9]	Madhya Pradesh	18	17	19	19
5	Dasgupta SM (1974)[10]	Uttar Pradesh	20-21	19-20	20-21	20-21
6	Kothari DR (1974)[11]	Rajasthan	18-19	17-18	18-19	17-18
7	Suresh Sankhyan (1991)[12]	Himachal Pradesh	21	20	21	20
8	Sheetal Jain (1999)[13]	Rajasthan	19-20	19-20	19-20	18-19
9	Patil DT (1994)[14]	North Karnataka	17-18	16-17	17-18	16-17
10	Galastun (1937)[15]	West Bengal	18	16-17	18	16-17
11	Basu and basu (1938)[16]	Bengal	-	16-17	-	16-17

The study conducted by Saksena et al[9], Kothari DR[11], Patil DT[14] and Galastun[15] showed that the average age of fusion of lower end of radius and ulna are consistent with the findings of the present study in both the sexes. But the study done by Dasgupta[10], Suresh Sankhyan[12] and Sheetal Jain[13] showed delayed fusion of epiphysis of lower end of radius and ulna when compared with that of the present study in both sexes. However all the previous workers observed that the fusion of epiphysis of lower end of radius and ulna occurs 1-3 yrs earlier in females compared to that in males, which is similar to that observed in present study.

CONCLUSION

Radiological examination is an important and reliable method of visualizing the epiphyseal union of the bones. Even though exact and precise age of the individual cannot be stated, but a reasonable age range can be assessed by the timings of the epiphyseal union.

The fusion of epiphysis is subjected to variabilities as it occurs at different age in different countries and in same country at different regions. Fusion of ossification centres occurs 1-3 years earlier in case of females compared to that of males and is consistent with the observations made universally. It was also

observed that appreciable variation with respect to time of union of epiphysis occurs within the country and also within same state of the country.

REFERENCES

1. Sekhon HS, Piyush K, Mishra VK and Rao CM. Roentgenologic study of epiphyseal union of metatarsals in lower Himachal Pradesh. J Forensic Medicine and Toxicology. 2004 ; 21(2) : 8-18.
2. Kangne RN, Sami SA and Deshpande YL. Age estimation of adolescent girls by radiography. JFMT. 1999 ; 16(1): 20-26.
3. Bhat VJ and Kamath GP. Age estimation from root development of mandibular third molars in comparison with skeletal age of wrist joint. Am J Forensic Med Pathol. 2007; 28: 238-241.
4. Krogman WM and Iscan MY. Skeletal Age : Early years. In: The human skeleton in forensic medicine. 2nd ed. Illinois USA : Charles C Thomas (Publisher) ; 1986. p. 50-102.
5. Evans KT and Knight B. Radiology in Identification. In : Forensic Radiology. London : Blackwell scientific publications ; 1981. p.3-46.
6. Banarjee KK and Agarwal BBL. Estimation of age from epiphyseal union at the wrist and ankle joints in the capital city of India. Forensic Sci Int. 1998 ; 98(1-2) : 31-39.
7. Narayan Reddy KS. Identification. In : The essentials of forensic medicine and toxicology. 25th ed. Hyderabad: Suguna Devi K; 2006. p. 50-86.
8. Greulich WW and Pyle SI. Skeletal age of individual bones and epiphyses. In: Radiographic atlas of skeletal development of the hand and wrist. 2nd ed. California: Stanford University press;1959: 61-183.
9. Saksena JS and Vyas SK. Epiphyseal union at the wrist, knee and iliac crest in residents of Madhya Pradesh. JIMA. 1969 ; 53(2): 67-68.
10. Dasgupta SM, Prasad V and Singh S. A roentgenologic study of epiphyseal union around elbow, wrist and knee joints and the pelvis in boys and girls of Uttar Pradesh. JIMA. 1974 ; 62(1): 10-12.
11. Kothari DR. Age of epiphyseal union at elbow and wrist joints in Marwar region of Rajasthan. JIMA. 1974 ; 63(8) : 252-256.
12. Sankhyan Suresh and Rao CM. A radiological study of epiphyseal union in long bones of upper limb to assess age in Himachal Pradesh. JIAFM.1991; 13(2): 33-36.
13. Sheetal Jain. Estimation of age from 13 to 21 years. JFMT. 1999; 16(1) : 27-30.
14. Patil DT. Estimation of age from epiphyseal union at wrist joint and of hand bones in north Karnataka (A radiological study). A dissertation submitted to Karnataka university, Dharwad 1994.
15. G Galastun. A study of ossification as observed in Indian sublects. Ind J Med. 1937;25:267-324.
16. SK Basu and S Basu. Medicolegal aspects of determination of age of Bengali girls. Ind Med Res.1938;58: 97-100.

A Study of Burn Deaths in North Karnataka

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ABSTRACT

This is an epidemiological profile of the burn fatalities brought to the morgue of Bidar Institute of Medical Sciences & Teaching Hospital Bidar. The study explores the incidence, age-gender distribution, time of occurrence, place of occurrence, socio-economic status, survival period, place of death, cause of death, body surface area involved, manner of death and monthly distribution. From the observations and analysis, certain etiologies are elicited and their preventive measures are suggested.

Key words: *Burn, Death, Cause of Death*

INTRODUCTION

Homicidal burning of married women in India is a major concern for the Government, law-enforcing authorities, the judiciary, the police and medico legal experts all over the country who are associated with dowry disputes. Dowry death, a heinous crime is gradually engulfing and polluting the entire society. To know the trend of the changing profile this study has been taken up.

MATERIAL AND METHODS

A study of all the burn cases that were brought to the morgue of Bidar Institute of Medical Sciences & Teaching Hospital Bidar. During the period January 2008 to December 2008 was done. Altogether, 65 cases were studied. The age and sex of the deceased, the venue and time of sustaining burn injuries, socio-economic status of the victims, body surface area involved, survival period and cause of death, circumstances of burns, etc. were ascertained from the autopsy records. The findings are tabulated in various tables to analyze the whole picture.

OBSERVATIONS

During the period of January 2008 to December 2008 a total of 480 medico legal autopsies were conducted by the Dept. of Forensic Medicine, Bidar Institute of Medical Sciences & Teaching Hospital Bidar, out of which 65 were deaths due to burns. There is no regular pattern in the incidence of burns over the study period. Regarding the gender distribution slight male preponderance was observed, 50.76 and 49.23 in males and females respectively. (Table 1) The age group most involved was 21-30 yrs with an incidence of 38.46%, which was more in females. In the extremes of age i.e., less than 10 yrs, there were 2 cases but there was no case above 60 yrs. (Table 1) On the whole, 53.84% sustained burn injuries during daytime. (Table 2) Taking the place of occurrence into consideration, 24.61% occurred in the husband's house which was the maximum among the studied categories. (Table 3) Most of the victims i.e., 53.84% belonged to lower socio-economic strata. (Table 4) Maximum percentage of victims survived for less than 1 hr and 21.53% for more than one week. (Table 5) 50.76% died in the hospital whereas 49.23% at the site of occurrence. (Table 6) The cause of death was burn shock in 67.69% cases. 49.23% died within an hour of sustaining the burns, 21.53% cases survived for more than 1 week. (Table 7)

Taking the body surface area involved into consideration it was observed that in about 73.84%, >80% body surface area was involved (Table 8). However, in 4 cases there death was due to smoke suffocation.

Most of the cases were accidental, 35.38%, followed by homicidal, 29.23% and suicidal, 24.61%. There was also a case of self immolation as a protest against the Armed Forces Special Powers Act. (Table 9) Highest incidence, 32.30%, was seen in January. (Table 10)

DISCUSSION

Dowry death is not traditionally prevalent in North Karnataka. However, sporadic instances of burn

Table 1. Age-gender distribution

Age in Years	Male (%)	Female (%)	Total (%)
<10	2 (6.06)	3 (9.37)	5 (7.69)
11-20	5 (15.15)	9 (28.12)	14 (21.53)
21-30	9 (27.27)	16 (50)	25 (38.46)
31-40	12 (36.36)	3 (9.37)	15 (23.07)
41-50	2 (6.06)	1 (3.12)	3 (4.61)
51-60	3 (9.09)	0(0)	3 (4.61)
>60	0(0)	0(0)	0(0)
Total	**33 (50.76)**	**32(49.23)**	**65 (100)**

Table 2. Time of occurrence

	Day	%	Night	%	Total
No. of Cases	**35**	**53.84**	**30**	**46.15**	**65**

Table 3. Place of occurrence

Age in Years	Male (%)	Female (%)	Total (%)
Quarter	4(12.12)	4(12.5)	8(12.30)
Own/rented hose	5 (15.15)	8(25)	13(20)
Husband's house	0(0)	17 (53.12)	17(26.15)
Shop	2 (6.06)	1 (3.12)	3(4.61)
Work place(hotel)	2 (6.06)	0(0)	2 (3.07)
Paddy field	1 (3.03)	0(0)	1 (1.53)
Roadside	13 (39.39)	0(0)	13(20)
Master's house	0(0)	1 (3.12)	1 (1.53)
Bazaar	3 (9.09)	0(0)	3(4.61)
Misc.(Riot)	2 (6.06)	2 (6.25)	4(6.15)
Total	**33**	**32**	**65 (100)**

Table 4. Socio-economic status

Socio-economic status	Male (%)	Female (%)	Total (%)
High	1 (3.03)	3 (9.37)	4(6.15)
Middle	19(57.57)	7(21.87)	26 (40)
Low	13 (39.39)	22 (68.75)	35 (53.84)
Total	**33 (50.76)**	**32 (49.23)**	**65(100)**

Table 5. Survival period

Period	No. of cases	%
<1 hr	32	49.23
1-24hrs	11	16.92
24-48 hrs	2	0.30
2-3 days	1	1.53
3-7 days	5	7.69
>l week	14	21.53
Total	**65**	**100**

Table 6. Place of death

Place	No. of cases	%
Hospital	33	50.76
Burn site	32	49.23
Total	65	100

Table 7. Cause of death

Cause of death	No. of cases	%
Burn shock	44	67.69
Toxaemia	9	13.84
Septicaemic shock	4	6.15
Acute tubular necrosis	1	1.53
Complications	2	3.07
Smoke suffocation	4	6.15
Total	**65**	**100**

Table 8. Body surface area involved

Area	No. of cases	%
0%(only smoke suffocation)	4	6.15
<30%	0	0
31-40%	0	0
41-50%	0	0
51-60%	5	7.69
61-70%	2	3.07
71-80%	6	9.23
>80%	48	73.84
Total	**65**	**100**

Table 9. Manner of death

Manner	No. of cases	%
Suicidal	16	24.61
Homicidal	19	29.23
Accidental	23	35.38
Riot	5	7.69
Self-immolation(AFSPA)	1	1.53
Unknown	1	1.53
Total	**65**	**100**

Table 10. Monthly distribution

Month	No. of cases	%
Jan	21	32.30
Feb	3	4.61
Mar	4	6.15
April	1	1.53
May	8	12.30
June	3	4.61
July	6	9.23
Aug	4	6.15
Sept	3	4.61
Oct	4	6.15
Nov	5	7.69
Dec	7	10.76
Total	**65**	**100**

deaths of newlywed women suggest the possibility of a sinister trend slowly creeping into an erstwhile placid society. Slight male preponderance was observed, 50.76 and 49.23 in males and females respectively. This may be because males are generally more active and involved in activities of all kinds. But the difference is not much. Females are not far behind and mainly comprised.

The age group most involved was 21-30 yrs with an incidence of 38.46%, which was more in females. Taking the place of occurrence into consideration 24.61% occurred in the husband's house. These observations are in conformity with other studies from the various regions of India[2-10] and in contrast to the studies from other developing and the developed countries.[11-16]

53.84% sustained burn injuries during daytime. This may be due to the fact that people are usually occupied in their work during daytime and therefore the burns are sustained in the course of their activities.

Maximum percentage of victims survived for less than one hr and 21.53% for more than one week because majority sustained more than 80% burns. Most cases were accidental followed by homicidal and suicidal burns.

Among those who die in suspicious circumstances, family quarrels and marital disharmony are the two important predisposing factors. Illiteracy, arranged marriage, joint family structure, unemployment, economic dependence of the husband on the parents, complete dependence of the women on their husband and in-laws and lack of social security were other contributory factors affecting the incidence in some way.[17-20] This is supported by the observation that 53.84% of the victims in our study belonged to low socio-economic stratum and also that 24.61% occurred in the husband's house which was the maximum among the studied categories. Most of the cases occurred in January. This may be due to use of fire for warming during winter.

CONCLUSION

Burn injuries have been a major cause of concern since pre-historic days to the present era of modern medicine. However, the general belief that burns usually occur at the two extremes of age, indicating the accidental nature of infliction does not hold true in the present Indian setup where the majority of reported cases belongs to second or third decade of life. However, the female preponderance in the ID-20, 21-30 yrs age groups. and should be a strong reason to start thinking of the sneaking intrusion of dowry harassment in a traditionally non-dowry oriented society.

REFERENCES

1. Shaha KK and Mohanty S: Alleged Dowry Death: a study of homicidal burns. Med Sci Law. 2006 Apr; 46(2): 105-110.
2. Dhillon K, Sekhon HS, Mishra VK. Study of burn deaths in and around Shimla district. JPAFMAT 2005; 5: 18-19.
3. Rao NG: Study of fatal female burns in Manipal. JFMT 1997; 14(2): 57-59.
4. Subrahmanyam M: Epidemiology of burns in a district hospital in Western India. Burns 1996; 22: 439-442.
5. Kumar V: Burnt wives-a study of suicide. Burns 2003; 29: 31-35.
6. Murty OP, Paul G: Bride burning and burns-certain differentiating aspects thereof. JFMT 1995; XII (3&4): 13-26.
7. Ghuliani KK. An epidemiological study of burn injuries. Ind Jr. Public Health 1998; 32(1) 34.
8. Naik RS, Anjankar AJ, Sharma BR: Epidemiology of burnt, females. The Antiseptic 1991; 88(11): 570-572.
9. Bangal RS: Thermal Injuries-A study of mortality patterns. JFMT; 1995, XII (1&2), 1-4.
10. Davies JWL: The problem of burns in India. Burns (Supplement 1) 1990.
11. Calder F: Four years of burn injuries in a Red Cross Hospital in Afganistan. Burns 2002; 28: 563-568.
12. Fernandes Marales et al: Epidemiology of burns in Molaga o Spain. Burns 1997; 23: 323-331.
13. Laloe V: Epidemiology and mortality of burns in a general hospital of Eastern Sri Lanka. Burns 2002; 28: 778-781.
14 Lari AR: Epidemiological study of 3341 burn patients during three years in Teheran, Iran. Burns 2000; 26: 49-53.
15. Mahaluxivala A: A retrospective study of etiopathological and preventive factors in a burns unit in Saudi Arabia. Burns 1997; 23: 333-406.
16. Parks JG: Epidemiology of fatal burn injuries. Jr. of Forensic Sciences. 1989; 34(2), 399-406.
17. Sharma BR, Harish D, Sharma A, Sharma S, Singh H: Accidental Fatal burns in Indian Kitchens: Are they really accidental? JIAFM; 2006: 28(1), 14-17.

Variations in ABH Antigenic Stability of Dried Bloodstains from Different Surfaces with Age

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ABSTRACT

During the present study the effect of age on ABH antigenic property were examined in dried blood stains collected from different surfaces. Dried blood stained exhibits of cotton fabric, synthetic fabrics and wooden objects stored at room temperature between the aged groups of three to twelve years were examined for ABH antigens by absorption elusion methods following some modification in absorption time and elusion temperature. We have identified ABH antigenicity in the exhibits of 12 years aged cotton fabric although the ABH antigenicity were found to be negligible in exhibits of synthetic fabric and wooden objects of more than five years aged group.

Key words: *Forensic Serology, ABH Antigens; Absorption-elution, Agglutination, Ulex Europaeus.*

INTRODUCTION

In routine serological examination, the ABH antigen typing play an important role to make a link between criminal with crime or victim, although from the blood stains of aged group it become very difficult to do ABH typing due to the loss of ABH antigenicity of the red blood cells with the storage period. In criminal case work, a serological expert examines various types of bloodstains exhibit depend on case to case. A lot of studies have been made by various scientists on blood grouping, but as per literature we have not found any report on the effect of age on stability of ABH antigenic properties from different types of bloodstains such as synthetic fabrics, cotton fabrics, wooden objects etc[1-7]. Therefore, in present study we have tried to find out a correlation between age and stability of A, B and H (responsible antigen for O blood group) antigens in dried bloodstains on different types exhibits; for this we have selected absorption elution method for identification of ABH antigens due to high sensitivity[8, 9]. This technique is based on detection of antibodies and anti-H lactin that have bounded by A, B and H antigens on immobilized fragments of erythrocyte surface. The bounded antibody is eluted with heating and then eluted antibody is detected with the help of indicator cells[10, 11].

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MATERIAL AND METHODS

2.1 Sample preparation

Dried bloodstained exhibits of cottons fabric (n=52), synthetic fabric (n=32) and wooden objects (n=16) stored at room temperature in dried environments since 3-12 years were used from the different aged groups. Anti-A, anti-B and anti H lactin (extract of Ulex europaeus seeds) were used for the identification of A, B and H antigens. Freshly prepared 0.1% suspensions of A B and O red blood cells in normal saline were used as indicator cells. Known dried bloodstains of A B and O individuals prepared on cotton gauze were used for comparison of results[12,15].

2.2 Absorption elution method

The dried bloodstains exhibits (cotton fabrics and synthetic fabrics) of the size of one centimeter were fixed in areas marked as ABO on cellulose acetate sheets. However, for wooden objects ammonical extract of bloodstains on cotton substrata were used for examination[16,18], then fixed samples were treated with anti-A, anti-B and anti-H lactin and incubated for minimum 16 hours at 4 °C. The unbound antiserum

was washed with chilled normal saline and treated with freshly prepared 0.1% suspension of indicator cells (A B and O) in respective areas, the samples are incubated at 58 to 60°C for 10 minutes for elution of antibodies. Then the samples were shaken at low rpm for 20-30 minutes at room temperature for maximum clumping between eluted antibodies and indicator cells and the results were observed with stereomicroscope (Olympus, Japan). The experiment was repeated for each sample for confirmation of results.

RESULTS AND DISCUSSION

To evaluate the stability of ABH antigens proteins, the results were recorded positive as well as negative for ABH antigens on the basis of presence or absence of clumping in appropriate marked areas for ABO typing of blood stains from different surface.

Table 1. ABH antigenicity of bloodstains from different type surfaces

Age in years	Cotton fabrics			Synthetic fabrics			Wooden objects		
	TN	PN	NN	TN	PN	NN	TN	PN	NN
3-5	24	18	6	10	4	6	6	2	4
5-8	12	6	6	10	0	10	4	1	3
8-10	9	6	3	6	0	6	2	0	2
10-12	7	4	3	6	0	6	4	0	4
Total no	52	34	18	32	4	28	16	3	13
% ABH +ve		65.8			12.5			18.7	

TN = Total number of samples

PN = numbers of samples positive for ABH antigen

NN= Number of samples negative for ABH antigens

As per result table, the number of bloodstains from cotton fabrics were found positive for ABO antigens in the manner; 18 sample out of 24 in aged group of 3 to 5 years, 6 samples out of 12 in aged group of 5-8 years, 6 samples out of 9 in aged groups of 8-10 years and 4 samples out 7 in aged groups of 10-12 years. Therefore the number of samples of blood stains from cotton fabrics are decreasing, and this decrease in number indicate the weakening of ABO antigenicity in bloodstains from cotton fabrics stored more than 5 years but have not lost their properties till 12 years. The positive numbers of samples for ABO antigens of bloodstains from synthetic fabrics are as follows: 4 samples out of 10 in aged group of 3-5 years. However, the ABH antigens could not be detected from the blood stains of synthetic fibers in the age groups of more than five years. In blood stains from wooden objects the ABH antigenic properties detected in age group of 5 years (2 samples out of 6) and ABH antigenicity more than five years blood stains was found to be negligible, so these shows the loss of interaction properties or degradation of ABH antigen with corresponding antibody of more than 5 years aged groups .

The ABO blood group system typing remains one of the basic laboratory tasks in a forensic practice but problem rise when sample is old or degraded. Some serological workers have worked on different type bloodstains. Habashi et al (1991), stored bloodstains from various substrata for six month at different temperature ranges between 29 to 44 °C, and not found any effects on ABH grouping of bloodstains from various substrata with storage period4, but our study reveals the variation in stability of ABH antigenic properties of blood stains of various substrata with the storage of period more than five years. The A, B and H (O) typing was determined by applying PCR-based ABO genotyping in various types of biological materials of forensic interest such as bloodstains, vaginal swabs, cigarette butts, and hair roots, Liechti et al19 (1996), they also extracted DNA from 10 to 12 years-old stamps and correctly typed at the A, B, and O (H) locus but this methods was costly than classical method. Zachová et al4 (2004) stored bloodstains on cotton substrata for different time period (3 months, 6 months, 1year) at various conditions, for the A, B, and H (O) blood group typing used Polymerase Chain Reaction to amplify the glycosyltransferase gene, they also applied some classical methods such as mix agglutination and Therkelsen method, found that by DNA analysis the problem of ABO typing can be solved in degraded samples but classical methods for ABO typing were found to be better in some cases.

CONCLUSION

Thus, the present study reveals the stability of ABH antigens in aged groups of 3-12 years from dried blood stains. The total percentage of bloodstains samples from cotton fabrics, synthetic fabrics and wooden objects detected for the ABH antigen are as 65.8%, 18.7% and 12.5% respectively. Therefore, much variation in ABH antigenic stability was found in blood stains from wooden, synthetic fabric, and cotton fabric exhibits. Most of the bloodstains from wooden and synthetics fabrics were found to be lost their ABH antigenic properties after 5 years, but ABH antigens was detected in age group of 12 years in bloodstains from cotton fabrics. Therefore, the loss in ABH antigens was found in blood stains from wooden and synthetic fabrics but not in cotton fabrics.

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REFERENCES

1. Nishi K, Rand S, Nakagawa T, Yamamoto A, Yamasaki S, Yamamoto Y, Kobayashi A, Kane M, Morimoto A, Spalthoff H , Annuss B. ABO Blood Typing from Forensic Materials - Merits and demerits of detection methods utilized in our laboratories, and biological significance of the antigens. Anil Aggrawal's Internet Journal of Forensic Medicine and Toxicology. 2005; 6(2).
2. Hoste B, Brocteur J, and Andre A. Indefinite storage of dried Gm and Km (Inv) antigens: (examination of bloodstains 33 years old). Forensic Science. 1978; 11(2): 109-113.
3. Nishi K. ABO blood group typing in forensic autopsies. Nihol Hoigaku. Zasshi.2005; 59: 111-117.
4. Habashi A A, Jado A, Farag A M, el-Assam O. Study on the factors affecting ABO grouping of blood stains, Journal of Egypt Soc Parasitol.1991; 21(1) 151-161.
5. Zachova M, Zeleny M, Pexa T, Mazura I, Hirt M. Determination of ABO blood group system from degraded blood stains on serological and molecular genetic level, Soud Lek.2004; 49(3): 43-46.
6. Rao D V, Kashyap V K. A simple Dipstick Immunoassay for detection of A and B antigens. Journal of Immunoassay. 1992; 13: 15-30.
7. Davie M J. ABO, Gm and Km grouping of bloodstains on microtitre plates. Journal of Forensic Science Society. 1979; 19(1): 59-64.
8. Yada S, Ishimoto G, Okane M. Blood grouping an 88-years-old hair rope, Acta Crime Apon. 1968; 34: 90-92.
9. Kaur G, Sharma VK. Comparison of absorption inhibition and absorption -elusion methods in the detection of ABO (H) antigens in the sweats stain. Current Science. 1988; 57: 1221-1222.
10. Richard Li. Forensic Biology, CRC press New York, 2008, 160-163.
11. James S H, Nordby J J. Forensic science: an introduction to scientific and investigative techniques. 2nd edt. CRC press 2005, 250-251.
12. Bashinski J S, Davis J E. A simple procedure for ABO typing of dried blood stains on fibers by the absorption- elution technique. Journal of Forensic Science Soc. 1973; 13(3): 217-222.
13. Bargagna M, Pereira M. A study of absorption elution as a method of Identification of Rhesus antigens in dried blood stains. Journal of Forensic Science Soc.1967; 7(3): 123-130.
14. Kind SS. Absorption-elution of dried blood stains on fabrics. Nature. 1960; 187: 789-790.
15. Kind SS, Cleevely RM. The use of ammonical blood stain extracts in ABO groupings. Journal of Forensic Science Soc. 1969; 9(3): 131-134.
16. Kind SS, Lang B. G. Some observations on the ammoniacal extraction method in Blood Stain Grouping and its Comparison with other Extractive and Non-Extractive Methods. Journal of Forensic Science Soc. 1976; 16: 47-54.
17. Gaensslen RE, Lee HC, Carrol JE. Evolution of monoclonal anti-A and anti-B and affinity purified Ulex europaeus lectin I for forensic blood grouping. International Journal of Legal Medicine. 1984; 93: 259-268.
18. Gaensslen RE, Lee HC, DeGraw SS, Carrol- Reho J. Studies on ABH antigen grouping of ammoniacal extracts of bloodstains by absorption- elution. Forensic Science International. 1986; 14(3): 145-157.
19. Liechti- Gallati S, Neeser D. Efficient and reliable PCR-based detection of the ABO blood group alleles: genotyping on stamps and other biological evidence samples. Journal of Forensic Science. 1996; 41(4): 653-657

Ossification of Bones of the Knee Joint in 17 to 20 Years of Age in South Indian Population

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ABSTRACT

A radiological study was done in south Indian subjects between 17 years to 20 years of age. 32 Males and 18 female's knee joint were studied.

The attempt was made to differentiate the fusion between males and females and it is observed that majority of subjects were having mixed diet, with healthy stature. The fusion in the lower end of femur in males was 17 years of age to 20 years and in females it was before 17 years of age to 19 year. The fusion of upper end of tibia and fibula in males was observed between 17 to 19 years of age while in females before 17 year of age to 18 year. This study shows that there is to one year earlier fusion occurs in female than males. This study will certainly help the Anatomist, Anthropologist Orthopedician and Medico-legal expert.

Key words: *Epiphysis, Fusion, Tibia, Fibula, Femur.*

INTRODUCTION

It is rightly said that, the skeleton of a particular individual is able to adapt to its owners way of life[1]. Hence environmental factors and dietary habits of south Indian population certainly differ from other parts of the country, moreover south Indians are regarded as "Dravidian race"[2]. Hence their genetic makeup, architecture of knee joint has to differ from other parts of the country because many factors known to influence and modify the joints development such as deprivation of raw materials and vitamins, hormonal imbalances and abnormal mechanical situations. Hence attempt is made to study the ossification of knee joint in 17 to 20 years of age through radiological study. Because normal life span of osteoclast is 20 years. Hence complete normal development of knee joint occurs at this age moreover this period of age is also notorious for crimes such as rape, fight, murder etc. Hence it has medico-legal importance apart from anatomical and anthropological study.

MATERIAL AND METHODS

Fifty (50) healthy students (32 Males and 18 females) from south Indian studying in MBBS course at Sri B.M Patil Medical College are selected and their age was 17 to 20 years. There age was confirmed through office documents.

They are made to stand in un-locking position and X-ray was taken AP and lateral view by qualified radiologist. Prior to X-ray it is tested that, no student had any knee joint problem. Study of patella was excluded as it is a seasmoid bone having multiple secondary centres.

RESULTS

Table 1

Out of 50 cases 32 were male and 18 were females. Among 32 males maximum i.e. 15 (46%) were in the age group of 17 years to 18 years and 5 (15.6%) were in the age group of 18 to 19 years and 12 (37.05%) were in the age group of 19 to 20 Years.

Among 18 females maximum i.e. 10 (55.5%) were in the age group of 17 to 18 years and 7 (38.8 %) were in the age group of 18 to 19 years. And 1 (5.5%) were in the age group 19 to 20 Years.

Table 1. Distribution of cases by age & sex

Age Group	Male	Female	Total
17 - 18	15 (46.8%)	10 (55.5%)	25 (50%)
18 - 19	5 (15.6%)	7 (38.8%)	12 (24%)
19 - 20	12 (37.5%)	1 (5.5%)	13 (26%)
Total	32 (100%)	18 (100%)	50 (100%)

Table 2

Out of 50 cases 9 were vegetarian (18%) and 41 (82%) were having mixed diet.

Table 2. Distribution of cases by food habit

Food Habit	Number	Percentage
Vegetarian	9	18%
Mixed	41	82%
Total	**50**	**100%**

Table 3

Out of 32 males 4 (12.5%) were vegetarian and 28 (87.5%) were having mixed diet out of 18 female 5 (27.5%) were vegetarian and 13(72.2%) were having mixed diet.

Table 3. Distribution of cases by sex & food habit

Food Habit	Male	Female	Total
Vegetarian	4 (12.5%)	5 (27.7%)	9 (18%)
Mixed	28 (87.5%)	13 (72.2%)	41 (82%)
Total	**32 (100%)**	**18 (100%)**	**50 (100%)**

Table 4

The average weight of males and females in the study was 60 kgs and 49 kgs respectively.

Table 4. Average weight of cases by sex

Weight	Male	Female
Average Weight	60 Kg	49 Kg

Table 5

Average height of males and females was 166.1 cms and 155.2 cms respectively.

Table 5. Average height of cases by sex

Height	Male	Female
Average Height	166.1 Cm	155.2 Cm

Table 6

It was observed that 10 males out of 32 (31.25%) had the fusion of lower end of femur in the 17 years to 17½ years and in 15 males (46.5 %) had fusion in the age of 19 years and 7 male (21.8%) fusion completed at the age of 20 years.

Table 6. Fusion of lower end of femur in males

Age	17 - 17.5	17.5 - 18.5	18.5 - 20
No of Cases	10 (31.25%)	15 (46.5%)	7 (21.8%)

Table No 7

In females out of 18 females 9 female (50 %) had fusion of lower end of femur at the age of before 17 years only and 6 female (33.00%) had fusion at the age of 18 years and remaining 3 (16.6%) female the fusion occurred in 19 years.

Table 7. Fusion of lower end of femur in females

Age	< 17 Years	18 Years	> 18 Years
No of Cases	9 (50%)	6 (33.3%)	3 (16.66%)

Table No 8

Out of 32 males 6 males (18.75%) had fusion of upper end of tibia and fibula at the age of 17 to 171/2 years of age while and 10 males (31.25 %) had complete fusion occurred of 18 to 181/2 years of age while in 17 males (53.2%) complete fusion occurred in 19 years of age.

Table 8. Fusion of upper ends of tibia & fibula in males

Age	17 - 17 ½	18 - 18 ½	19 & Above	Total
No. of Cases	6 (18.75%)	10 (31.25%)	17 (53.12%)	32 (100%)

Table No 9

Out of 18 females 10 female (55.55%) the upper ends of tibia and fibula had complete fusion at the age before 17th years and in 8 females (44.15%) the fusion completion observed at the age of 171/2 to 18 years.

Table 9. Fusion of upper ends of tibia & fibula in females

Age	Before 17 Years	Between 17 - 18	Total
No. of Cases	10 (55.55%)	8 (44.45%)	18 (100%)

DISCUSSION

In the present study out of 50 subjects 32 were males among them 15 (46%) were in the age group of 17 to 18 years and 5 (15.6%) were in the age group of 18 to 19 years and 12 were in the age group of 19 to 20 years. While in the female out of 18 cases 10 (55.5%) were in the age group of 17 to 18 years and remains 7 cases (38.8%) were 18 to 19 years and 1 (5.5%) was 19 to 20 Years of age (Table 1) Hence our study was an ideal attempt to observe the ossification of bones of knee joint i.e. lower end of the femur and upper ends of tibia and fibula.

It was also observed that the subject studied were 41 were mixed diet while 9 were vegetarians (Table 2) in males 28 (87%) were mixed diet and female 13 (72.2%) were mixed diet (Table 3)because for the proper formation and development of cartilage canals and the secondary centre of ossification in the distal chondro epiphysis of human femur linked with osteogenesis[3]. It occurs only in healthy subjects and present study was carried out in quite healthy subject

whose body weight was more or less 60 kg in males and 49 to 50 kg in females (Table 4) and average height of males and females was 166.1 cms and 155.2 cms respectively (Table 5) because ossification of long bones is influenced by dietic, climatic, hereditary, nutritional, sociological, racial, environmental and geographical factors [4].

In the present study it was observed that, 10(31.25%) of males had complete ossification of lower end of femur was between 17 years to 171/2 of age and complete ossification observed at 15 males (46.5%) at the age of 19 years.

It was also observed that 7 (21.8%) males had complete ossification between 181/2 years to 20 years of age (Table 6).

Among 18 females 9 female (50%) had complete ossification of lower end of femur completed at 17 years only while 6 female (33.3%) it was observed that ossification completed at the age of 18 years and 3 female (16.66%) had complete ossification was observed in 19 years of age (Table 7).

The present study was in agreement with previous workers[5, 7] and earlier ossification in female was also more or less in agreement with other workers[8] as bones of lower end remains distinct until seventieth in females, and 18 to 19th in males when epiphyseal line ossifies, so the lower end is the growing end of the bone [9].

In the present study upper ends of tibia and fibula out of 32 males 6 males (18.75%) had ossification of tibia and fibula completed at the age of 17 to 171/2 years and 10 males (31.25%) had complete ossification in the age of 18 to 181/2 years. While in 17 males (53.00%) complete ossification observed in 19 years of age (Table 8).

In the present study of upper ends of tibia and fibula in females. Out of 18 females 10 females (55.5 %) had complete ossification before 17 years of age (already ossified) while remaining 8 females (44.4%) had shown complete ossification at the age of 17 to 18 years (Table 9) which was earlier than males. The study was in agreement with other workers who observed complete ossification between 16 to 18 years of age[10,12]. Although exact process of ossification is still obscure but skeleton of a particular individual is able to adapt to its owners way of life because bone is a dynamic tissue that responds to the variety of environment and cope with biomechanical stress. Hence variation in the fusion of bones of knee joint could be to cope with environmental stimuli and to overcome biomechanical stress[13] Hence it can be hypothesized that, bones of the female are more adaptive than male bones hence fusion occurs earlier in females.

CONCLUSION

In the present study it was observed that majority of subject were having mixed diet and male's weight was near about 60 kg and females was about 48 to 50 kg.

The secondary fusion in males of lower end of femur was 17 years to 20 years and in females it was between before 17 years of age (probably 16 years) to 19 years.

In the upper end of tibia and fibula in males the secondary fusion was between 17 to 19 years of age while in females it was before 17 years to 18 years.

It was observed that females bones of knee joint fuse six months to one years earlier than males.

This study of south Indians ossification or fusion of bones of knee joint believed as Dravidians race will certainly help the anatomist, anthropologist, orthopedician and medico-legal experts. Because books and literatures always cannot be relied upon.

REFERENCES

1. Pritchard. JJ, Repair of fracture bones in the rat. Journal of Anatomy (1946), 80, 55-60.
2. Bhasin. MK, Genetics of caste and tribes of India, Indian population milieu. International Journal of Human Genetics 2006, 6(3), 233-274.
3. Burkus JK, Ganey TM, Ogden.JA Development of the cartilage canals and the secondary centre of ossification in the distal chondroepiphysis of the Prenatal Human Femur. Yale J. Biol Med. Vol. 66(3), 193-202 (1993).
4. Modi JP, Modis text book of Medical Jurisprudence and Toxicology page no 351-352, 22nd edition (2001).
5. Basmajian. JV, Knee joint, chapter 28, joints of lower limb, grants method of anatomy 8th edition. The Williams and Wilkins Company Publications, 395/402(1972).
6. Pillai. MS, The study of epiphyseal Union for determining the age of south Indians. Journal of Indian Medical Research. Vol, 23, 1015-1017 (1936).
7. Basu SK, Age order of epiphyseal Union in Bengali girls. Journal of Medical Association Vol. 7 (11), 571-578(1938).
8. Sakshena IS and Vyas Sk, Epiphyseal union at the wrist, knee, and iliac crest in resident of

Madhya Pradesh. Journal of Indian Medical Association vol.53, 67-68(1969).

9. Breathnach. AS, Frazers Anatomy pages 152, 170 Vth editions. J and A Churchill London Publication (1965).
10. Galastaun, Study of ossification as observed in Indian subjects. Journal of Indian Medical Research Vol. 25, 267-324 (1937).
11. Das gupta SM, Prasad and Singh, A Roentogenic study of epiphyseal union around elbow, wrist, knee joints and pelvis in Boys and Girls of U.P. Journal of Indian. Medical Association Vol.62, 10-13 (1974).
12. Peepre and Dubey, Radiological examination of epiphyseal union of elbow and knee joint for determination of age. Forecon 96 XVII Annual conference of Indian Association of Forensic Medicine (1996).
13. Roff C.B, Walker A and Trinkaus, Post cranial robust city in Homo III Ontogeny. American Journal of Physical Anthropology (1994) 93, 35-54.

Evaluation of Dowry Related Crimes in Bijapur City

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ABSTRACT

The present modern day culture still has the ancient evils of civilization. In spite of the increase in educational status of most parts of the country, dowry related harassments and deaths are on the rise with every coming year. Marriage is considered more of a business alliance rather than a religious rite and bride as a source of money and materials.

The objectives of our study is to find out the cause and mode of death in the victims, to study age of victims, married life, educational, occupational and socioeconomic status of the victims, to study the type of family, type of marriage and number and gender of the children born to the victims and to suggest concrete steps to Government and NGO's, especial women organization to reduce incidence of dowry harassment and deaths. The present study was conducted at Department of Forensic medicine, Shri B.M. Patil Medical College, Bijapur, on all married women dying within 7 years of married life, booked under section 304 (B), 306 and 498 (A), IPC and 176 Cr PC, whose inquest was conducted by executive magistrate.

Key words: *Dowry deaths, Bride-burning, Harassment, Dowry, Homicide, Suicide.*

INTRODUCTION

Dowry related violence occurs among all subgroups of Indian population, but the rates are higher among the poor &lower castes, In many cases, the husband &his family continues for years to demand additional dowry by physically &verbally abusing the bride.

In majority of cases, young wives,are unable to fulfill their husband's demand for dowries continue to be victimized until they eventually commit suicide by fire .The husband's response to the tragedy is often, It was her time, in other words her predetermined fate, The news papers report on back paper simply that so &so died of burns, without giving background or details is a routine.

Indeed in India today,torture of the young house wives by their husbands and in-law's for failure to bring sufficient dowry has become the order of the day all over the country. On an average, one indian women commits suicide every four hours over a dowry dispute, as for official data, despite a series of laws to empower them.

To know the various incidences of dowry related crimes in our region, This project has been undertaken.

OBJECTIVES

1. To know the incidence of dowry deaths.
2. To identify all the factors that lead to the young women take the extreme step of ending her own life.
3. To assess the physical and mental trauma that coexists in the victim

MATERIAL AND METHODS

Source of data and materials:

a) Source: Method of collection of data:

The Data will be collected from the Following Sources:

- In patient case sheets in case of hospital admission.
- Postmortem examination reports of the cases.

Inquest reports

Period of Study

1st July 2009 to 1st June 2010.

c) Sample size

35 cases of dowry related crimes in Bijapur City

Inclusion criteria:

1. Autopsy conducted on all the dead bodies of married women brought under the umbrella of dowry death by law, at BLDE University's Shri B.M Patil Medical College, Bijapur.
2. Interrogation of the victim while in the hospital

Exclusion criteria:

- The incidents not related to 304(B)I.P.C, 498(A) I.P.C., 306 I.P.C. 176 CrPC

Table 1. Dowry death cases with reference to age of the victims and duration of married life

Sl No	Age of Victims	Total		Duration Married life in year						
0		No	%	O to <1	>1 to <2	>2to <3	>3to <4	>4 to <5	>5 to <6	>6 to <7
1	18-21	15	42.85	02	03	03	02	02	01	02
2	22-25	09	25.71	01	02	02	01	01	00	00
3	26-29	05	14.29	02	02	01	00	00	01	01
4	30-33	01	8.5	01	01	00	01	00	00	00
5	34-37	00	5.8	00	01	00	00	00	00	00
6	>38	00	2.85	00	00	00	01	00	00	01
	Total	**35**	**100**	**06**	**09**	**06**	**05**	**03**	**02**	**04**
	Percentage			**17.1**	**25.8**	**17.23**	**14.35**	**8.65**	**5.7**	**10.8**

Table 2. Mode and manner of dowry deaths observed during Study period

SI No.	Date of Death	Manner of Death				Total	
		Homicide		Suicide			
		No	%	No	%	No	%
1	Burning	04	11.4	12	34.28	16	45.71
2	Hanging	03	8.58	06	17.15	09	25.71
3	Poisoning	04	11.4	03	8.58	07	20
4	Drowning	00	00	02	5.8	02	5.8
5	Strangulation	01	2.85	00	00	01	2.85
	Total	**12**	**34.2**	**23**	**65.81**	**35**	**100**

Table 3. Religion wise distribution of dowry deaths

Sl No	Religion	No	%
1	Hindu	18	51.43
2	Muslim	10	28.58
3	Christian	07	20.00
	Total	**35**	**100**

Table 4. Time of occurrance of incidences leading to dowry deaths

Sl No	Time of Incidence	No of Death	%
1	Morning (6A M to 10AM.)	07	20.0
2	Working Hours (10 AM to 4PM.)	03	8.58
3	Evening Hours 4 PM to 9PM.)	11	31.5
4	Night (9PM to 6 AM.)	14	40.00
	Total	**35**	**100.0**

Table 5. Location of incidence leading to dowry deaths

Sl No	Location of incidence	Total		Homicidal		Suicidal	
		No	%	No	%	No	%
1	In-law's House	16	45.11	04	11.42	12	34.28
2	Husband's House	09	25.71	03	8.58	06	17.15
3	Parent's House	05	14.29	02	5.8	03	8.58
4	Road side /Field	05	14.29	02	5.8	03	8.58
		35		11		24	
		100		**31.6**		**68.59**	

Table 6. Offenders involved in causation of dowry deaths

Sl. No.	Offenders Involved	Dowry Deaths	
		No	%
1	Husband	17	48.5
2	Relative Husband	03	8.58
3	Both Husband / Relation	13	37.14
4	No offenders	12	5.8
	Total	**35**	**100**

Table 7. Offenders involved in causation of dowry deaths

Sl. No.	Type of Family	Dowry Deaths	
		No	%
1	Joint Family	22	62.85
2	Nuclear family	13	37.15
	Total	**35**	**100.0**

Table 8. Occurrence of dowry death's in relation to type of marriage

Sl. No.	Type of Marriage	Dowry Deaths	
		No	%
1	Arranged Marriage	26	74.28
2.	Love Marriage	9	25.72
	Total	**35**	**100.00**

Table 9. Occurrence of dowry death's in relation with socio-economic status of the family

Sl. No.	Socio-Economic status	Dowry Deaths	
		No	%
1	Very Lower Class	08	22.85
2	Lower Class	10	28.58
3	Lower Middle Class	08	22.85
4	Higher Middle Class	06	17.15
5	Higher Class	03	8.58
6	Very Higher Class	00	00
	Total	**35**	**100**

Table 10. Occurrence of dowry death's in relation with educational status of victim

Sl. No.	Educational Status	Dowry Deaths	
		No	%
1	Illiterate	09	25.72
2	Non Metric	12	34.28
3	Matriculation	08	22.85
4	Graduate	05	14.29
5	Postgraduate	01	2.85
	Total	**35**	**100**

Table 11. Occurrence of dowry death's in relation with occupational status of the victim

Sl. No.	Occupational Status	Dowry Deaths	
		No	%
1	House Wife	18	51.43
2	Private Sector Employee	09	25.71
3	Government Employee	05	14.29
4	Student	03	8.58
	Total	**35**	**100**

Table 12. Occurrence of dowry death's in relation with number and gender of the children born to victim

Sl. No.	Children	Dowry Deaths	
		No	%
1	No Child	08	22.85
2	Only Female Child	13	37.14
3	Only Male Child	05	14.29
4	Male &Female	02	5.8
5	Below Child Bearing Duration	07	20
	Total	**35**	**100.0**

DISCUSSION

In dowry a deep routed cause of voilence against women in India, has been reported. Sharma B R, Harish D, Manish Gupta and Veerendra pal singh[1] 2005, write as even the world has entered new millenium, the harsh reality is that the women in India have long been illtreated in our male dominant society.

PradeepKumar M V and F S Kulagoud[2] 2004 says as the dowry is a continuing demand it is not an one time payment initially at the time of marriage alone, but the series of gifts before and after marriage till the in-laws and husbands are satisfied.

The age of victims of the dowry death's studied in our study are mostly between 18-21 yrs, accounting for i.e.,15 cases(42.85%), out of 35 cases in one year. Maximum incidence of dowry death's observed during first 2-3 years of married life.These findings are in accordance with Sharma B R[1].

Regarding the causative factors modes of dowry deaths such as burns, hanging, poisoning, drowning etc.,majority of dowry deaths are due to burns i.e.,16(45.71%) followed by hanging 09(25.71%), poisoning 07(20%) and drowning 02(5.8%). These findings are comparable to other previous studies conducted by Nagesh Kumar 4, where 60% of dowry deaths were due to burns Bhullar D.S[3].

When dowry deaths are analyzed according to religion, Hindu females comprised of an alarming large group i.e.,18(51.43%) followed by other communities like Muslims 10(28.58%), Christian's 07(20%). These findings are comparable with findings of Anil Agnihotri[5],where 90% were Hindus other communities like Muslims, Christians and Sikhs, the number of dowry deaths were negligible.

The analysis of the cases with reference to time of occurrence of vital events leading to death's in our study showed that most of the cases have occurred at night hours, between i.e., 14(40%) between 9pm to 6am followed by i.e., 11(31.5%)between 4pm to 9pm and i.e.,7(20%)between 6am to 10am. It is in comparison with Anil Agnihotri[5] study where 35.76% cases during night and 32.45% cases in the evening hours and Bhullar D.S[3] reported (68.75%) cases between 4pm to 6pm.

These findings suggest most of the victims are killed by suicide i.e., 24(68.59%) or homicide i.e., 11(31.6%), when the other members of the family members were at home. Most of the incidence have occurred i.e., in-laws house 16(45.11%) followed by i.e., 09(25.71%) in husband's house.

The study clearly suggests that young house wives from joint families are major targets i.e., 22(62.85%) cases. These findings are in collaboration with Anil Agnihotri[5] and Manjunath K. H study[6], who observed more cases in joint families. In our study i.e., 17(48.5%) cases we found, husbands are the offenders.

With regard to type of marriage, most of the victims were got married by arranged marriage i.e., 26(74.28%) cases where as only i.e., 09(25.72%) cases are love marriage. This is because low understanding between husband and wife in arranged marriage and most of the brides going to stay in joint family after marriage. These findings are in corroboration with Anil Agnihotri[5] and Manjunath K H studies[6].

The socio-economic status of the family has a relevance in occurrence of dowry death cases, In the present study, most of the cases have been reported from lower class and lower middle class i.e., 08(22.85%) cases from lower class and i.e., 10(28.58%) cases from lower middle socio-economic class families and only 03(8.58%) cases in higher class. These findings are in agreement with the study of Sharma B.R, Harish D1 who observed 85% of victims belong to the low socio-economic group while only 0.8% belongs to high socio-economic group and in Anil Agnihotri5 study, most of the cases (54.97%) were found in low socio-economic group. These findings correspond to observation made by viz Gupta R.K and Srivastava A.K[7].

In relevance to educational status of the victims,most of the cases were below matriculation

12(34.28%) followed by illiterate in 09(25.72%) cases,Among matriculated, 08(22.85%) victims were graduate. These findings correspond to other studies in this field by viz Gupta.R.K and Bhullar D S[3].

When dowry related death cases are studied in relation to occupational status of the victims,the house wives constitute alarmingly a large group i.e., 18(51.43%) cases followed by private sector employees 09(25.71%) and only 05(14.29%) cases of government employees and students 03(8.58%) . These findings are more or less similar to Anil Agnihotri[5] study in which 95% of cases were house wives. This is because of their economic and social dependence on husband and in-laws for one or other means.

Most of the occurrence of dowry deaths in relation with number and Gender of the children Born to victim. i.e., 13(37.14%) in only female child and no child i.e., 8(28.85%). These findings are similar to the observations made by Sharma B R and Harish D[1].

CONCLUSION

1 . Most of the dowry deaths occurred during evening and night hours between 4pm to 6am (61.5%).
2. Most of the dowry deaths occured in Hindu religion (51.43%). And spreading to other religions too, Muslims (28.58%), Christian (14.28%).
3. Most of the victims are in the age between 18 to 21 years (42.85%).Maximum incidence of dowry deaths observed during first 2-3years of married life.
4. Commonest location of incidence leading to dowry deaths. being the in laws house (45.72%) followed by husband's house (25.71%).
5. Commonest offenders being husband, in laws and both husband's relatives (82.85%).
6. Most of the victims belong to joint families(62.85%). In nuclear family, husband being alcoholic (37.15%).
7. Most of the victims were of arranged marriage(74.28%).
8. Most of the victims belong to lower and lower middle socio economic families(28.58%).
9. Most of the victims were educated below matriculation or just matriculate(57.13%).
10. Burning is the most common mode of dowry deaths(34.28%), followed by hanging(17.15%) and poisoning (8.58%).
11. Most of the victims were house wives (51.43%).
12. Most of the victims gave birth to only female child(37.14%) or no child at all(22.85%).

SUMMARY

1. Burning and hanging were the commonest methods employed by the victims in case of suicide but strangely enough the same methods were employed in homicides also
2. Between the ages of 18 to 21 years and married life of 2 to 3 years maximum incidence were observed
3 . Hindu victims were more in number probably due to larger population
4. 4pm to 6am appears to be very sensitive period of the day as can be seen here with maximum number of deaths
5. Maximum number of deaths have taken place in in laws house.
6. Husband appears to be the main perpetrator of the crime.
7. Strangely maximum number of deaths have taken place in joint families.
8. Maximum incidence is in the arranged marriage.
9. Incidence is more in very low, low and lower middle class families.
10. Even the low educational level has gobbled up maximum number of victims.
11. Similarly, the incidence is maximum in housewives.
12. Unfortunately, the incidence was seen to be more in mothers of female children.

"It is very important to know the demographic pattern of Dowry Deaths in order to curb this menace and save the lives of women from their helplessness. Retributory measures alone have not significantly reduced any crime so far education is the key."

RECOMMENDATIONS

In the view of the worrying results of present study following measures are suggested to reduce the number of dowry related deaths and harassments.

A. Social and Economical measures
   1. Public opinion against the magnitude and gravity of dowry deaths should be mobilized through various agencies.
   2. Costly and ostentaneous marriage rituals should be discouraged by society and preferably a ban on such marriages be imposed by government.
   3. Newly wedded couples should be allowed to live separately from their in laws during first few formative years of their marriage as couples develop good understanding between each other.

4. Social boycott should be done to those tainted boys and their families in future marriage negotiation by the society.
5. Effective measures to be taken to promote education among the women to increase their educational status.
6. Women to be educated regarding their legal rights and special provisions under which they can fight the crimes against them.
7. Inter caste and love marriages to be encouraged.
8. Crimes against women cells should be opened in large number to tackle the immediate provocation.

B. Measures by NGO's and government

The government in consultation with women NGO's working on gender violence issues should develop a national initiative against crimes against women.

C. Legal and Administrative measures

1. More strict laws should be framed for offenders of dowry deaths and use of various explosive and inflammable materials and poisons to present unnatural female deaths.
2. A well equipped task force with modern techniques for proper and prompt investigation should be sanctioned as dowry death cases are relatively high.

REFERENCES

1. Sharma. B.R., Harish .D. ,Manish Gupta and Virender Pal Singh: Dowry - A deep rooted cause of violence against women in India: Medical Science & Law: 2005:45(2).pg.161-168.
2. Pradeep Kumar. M .V. and F S Kulogod: Dowry Death A- Social Menance: JKAMILS: 2004: 13(2) pg.41-44.
3. Bhullar. D .S., Gorea. R.K., Aggarwal. A.D.: Study of Profile of un natural female death between 18-30 years of age in Government Medical College, Rajindra Hospital, Patiala (India): JFMT: 1996: Vol.13 (3 & 4),pg.5-8.
4. Nagesh Kumar .G. Rao : Study of fatal female burns in Manipur (India): JFMT: 1997: 14 (2): pg 57-59.
5. Anil agnihotri: The edidemiological study of dowry death cases with special references to burial cases in Allahabad zone : Anil Aggrawal's Internet Journal of F.M & T : Vol.2(1)
6. Manjunath.K.H, Crimes Against women with special reference to Dowry harassment and Death. Thesis submitted to the National law School of India University Bangalore.
7. Gupta R.K and Srivastav A.K.: Study of burns cases in Kanpur: Forensic science International: Kanpur, India : 1998:Vol.237:pg 81-89.
8. Narayana Reddy.K.S : Essential of Forensic Medicine and Toxicology: 29th edition, Medical Book Company hyderabad-2010: pg.263-264.
9. Krishan Vij: Text Book of Forensic Medicine and Toxicology 11th edition. ELSEVIER 2005:pg.242-266.
10. Parikh.C. K : Text Book of Medical Jurisprudence and Toxicology, 5th edition: 1995 page 384 -389.
11. Pillay, V.V: Text book of Forensic Medicine and Toxicology: 14th edition Paras Publishing: Hyderabad: 2004: pg.478-497.
12. Shashidhar.C. Mestri: Role of doctor in Dowry deaths: JFMT: 1993: Vol.10.(3 & 4) , pg: 36-37.
13. Pekka Saukko and Bernard Knight: Knight's Forensic Pathology: 3rd edition Arnold Publishers: London: 2004: pg 312-324
14. Rajesh Bardale : principles of Forensic medicine and Toxicology : first edition 2011 Page 271-273.

An Autopsy Study of Sudden Natural Deaths in Victoria Hospital, Bowring and Lady Curzon Hospitals, Bangalore

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ABSTRACT

Sudden deaths are mostly natural deaths which occur immediately or within 24 hours of the onset of terminal symptoms, which may be totally different from the symptoms which the patient was having so long.

Such cases pose medico legal problems to relatives/friends/police officers/judiciary/doctors and other law enforcing agencies.

Key words: - *Sudden Natural Death, Autopsy, Medico Legal Problems, Coronary Vessels.*

INTRODUCTION

Sudden deaths are mostly natural deaths which occur immediately or within 24hours of the onset of the terminal symptoms, which may be totally different from the symptoms which the patient was having so long. The explanation does not essentially exclude or rule out deaths due to means other than natural diseases, but no unnatural factor/cause need be apparent.

Not uncommonly, medicolegal autopsies are conducted in cases of sudden and unexpected deaths primarily to establish the cause of death in cases where such deaths have occurred in apparently healthy individuals under suspicious circumstances. The outcome may reveal some natural disease, the presence which may trigger issues like association of the disease with trauma, work, crime etc and its relative contribution towards death.

REVIEW OF LITERATURE

Definition and terminology of sudden natural death: Recent concepts Sudden natural death is a phrase very commonly used. The unexpected nature is more important than its suddenness. Although many unexpected deaths will be sudden, that is occurring immediately upon collapse or within minutes, there may be delay of hours or even with a diagnosis being clinically evident.

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Dr. K.S. Narayana Reddy defines death is said to be sudden or unexpected when a person not known to have been suffering from any dangerous disease, injury or poisoning is found dead or dies within 24 hours after the inset of terminal illness. Natural death means that the death was caused entirely by disease and trauma or poison did not play any role in bringing it about. The incidence is 10% of all deaths. No period in life is exempt.

The causes are
1. Diseases of the cardiovascular system. 45 to 50%
2. Respiratory System. 25-40%
3. Central nervous system. 10 to 18%
4. Alimentary system. 6 to 8%
5. Genito urinary system. 3 to 5%
6. Miscellaneous. 5 to 10%

Sydney Smith and Fiddes have expresses their opinion as regarding "sudden death". They have opined that in some cases the most minute post mortem examination fails to show any cause of death. But if the examination can exclude death from violence, poisoning etc., of justice are sufficiently served.

Keith Simpson observes, though SND quite free suspicious and presumed natural are subject of inquiry in most of the civilized communities.

Dr. M.K.R. Krishnan observes "sudden deaths" are those which are not preceded or preceded only for a short time by signs and symptoms of the disease. There is always a suspicious of foul play and so they warrant a thorough investigation.

Dr. P.V. Guharaj had defined "sudden death" as the sudden or unexpected termination of life of apparently healthy people from natural diseases.

John Hunter's prophecy: "My life is at the mercy of any scoundrel who chooses to put me in a passion". Defines: "sudden natural"- deaths within 2hours, between emotional stress event & death, which is witnessed.

In various published analysis, the range for frequency of the sites of occlusion in coronary arteries, with or without thrombosis are [1].

- LAD- Left Anterior Descending (45-64%)
- Right main coronary (24-46%)
- Left circumflex coronary (3-10%)
- Left main coronary (0-10%)

If a person dies within 24hours without suffering from a recognizable cause, the likely reason for death, his death would be called a 'Sudden death'. Most of these deaths occur by natural causes but obvious etiology is not known at that time. All such cases which are brought before a doctor should not be certified as a natural one, till all other causes like trauma, poisoning or assault have been ruled out[2].

The medicolegal autopsy is conducted in cases of sudden and unexpected deaths, including apparently accidental death, primarily to establish the cause of death. The definition of sudden death varies according to authority and convention. WHO defines sudden death as 'death that is unknown or sudden and occurring within 24hours from onset of symptoms'. However, some pathologists and clinical will only accept sudden death as the one occurring within one hour from the onset the symptoms.

In sudden deaths, the immediate cause is usually in the cardiovascular system.

Causes of sudden death

- Cardiovascular system 45-50%
- Respiratory system 10-15%
- Central nervous System 10-18%
- Alimentary system 6-8%
- Genitourinary system 3-5%
- Miscellaneous 5-10%[3].

Hypertrophic cardiomyopathy is a genetic disease with an autosomal dominant inheritance. The exact cause is not yet known. Majority of patients with hypertrophic cardiomyopathy have atleast one other affected first degree relative. It affects males. Nine genes have been implicated; other genes may be uncovered with continued research. In approximately 50-60% of families, affected individuals are found to have a mutation in the gene for myosin, Troponin T, alpha tropomyosin, cardiac myosin binding protan-C, or essential and regulatory ligut chains. These are important proteins for the contraction of the heart.

The first clinical presentation of the disease may be unfortunately sudden death or sudden death may the only indication of the disease occurring frequently in children and young adults often occurring during or after exertion[4].

Need for the PM examination

Circumstances under which natural death occur:

I. Deaths in the presence of witnesses and under circumstances in which factors of physical and emotional strain may have played a role: Thus it may have occurred during physical exertion, during altercation, during sexual excitement or without any obvious precipitating factor.

II. Deceased is found dead: Suspicious death

Sudden natural death is less frequent in younger persons; a suspicion of violent death is more likely to arise. In older individuals it is more frequent.

Insurance claim: finding the correct cause of death may discourage unnecessary and unwarranted litigation. Fraudulent claimants will be settled by autopsy.

Workmen's compensation: lesions found at the autopsy can be evaluated in their relationship to alleged occupational accident or injury.

He/She also functions as Public Health Officer by first recognizing & first calling attention to early fatalities in epidemic disease.

MATERIALS AND METHODS

The materials used in this study were obtained from the postmortem examinations conducted in the department of Forensic medicine. Bowring & Lady Curzon Hospital and Victoria Hospital attached to Bangalore medical college, Bangalore.

The study period was from January 2001 to November 2001 during which 1590 postmortem examinations were conducted. Of these 100 cases accounted for sudden natural deaths as shown in Table 1.

Information was collected from the relatives and friends of the deceased who accompanied the body, the investigating officer of the concerned case and his requisition form.

Each case was studies giving particular importance to the time of incident, circumstances and participating causes, physical strain preceding death, premonitory symptoms, history of previous illness, and personal habits of the deceased, and a thorough postmortem was conducted in each case to establish the cause of death and autopsy findings were recorded in detail and investigations such as histopathological examination of the diseases organs and chemical analysis of the viscera and blood were undertaken in cases wherever it was found necessary.

EXAMINATION

External

a. Length
b. Physical State: Moderate/asthenic/obese/ muscular
c. Postmortem staining: Appreciable/ not appreciable
d. Rigormortis: present/absent, if present details
e. Cyanosis: present/absent
f. Associated injuries

Internal

Cranium

1. Skull
2. Vertebra and spinal cord
3. Membrane
4. Brain

Thorax

1. Walls: Ribs:
2. Pleura -
3. Larynx & trachea
4. Lungs: a. Right b. Left:
5. Pericardium
6. Heart:
   a. Size
   b. Weight
   c. Walls & Chambers
   d. Valves
   e. Coronaries

Abdominal Organs

a. Peritoneum
b. Liver
c. Spleen
d. Stomach and intestine
e. Kidneys
f. Genital organs

STATISCAL DATA

During the study period from January 2001 to November 2001 1590 autopsies were conducted in the department of Forensic medicine, Victoria Hospital & Bowring/Lady Curzon Hospital, Bangalore. Of these 100 cases accounted for sudden natural deaths. i.e.6.36% of the total cases.

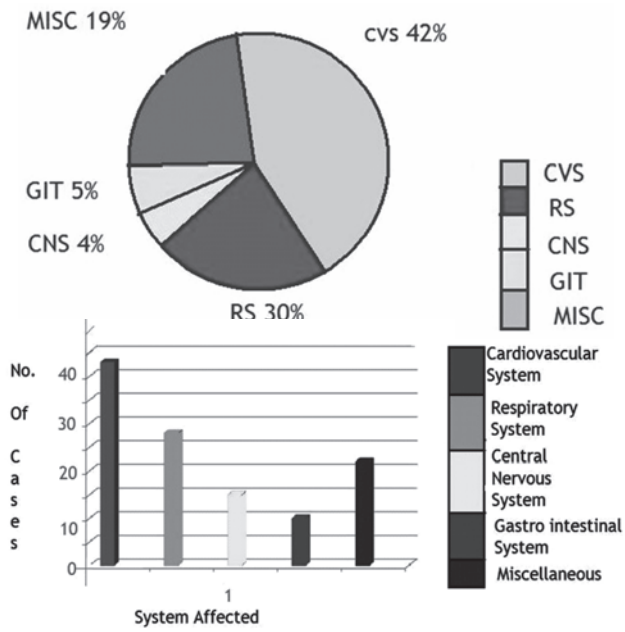
Statistics of the different cases were taken in the lines of etiological classification, age incidence, sex incidence, religion distribution and the period of survival and the results were noted in the tabular columns.

Table 1. Table showing incidence of sudden natural death

Sl No.	Description	Jan' 01 to June 30 at Bowring & Lady Curzon Hospital	June 01 to Dec 31 at Victoria Hospital	Total
1.	Post mortem conducted	540	1030	1590
2.	Sudden natural deaths	37	63	100
	Percentage	6.85%	6.11%	6.36%

Table 2. Aetiological Classification

I.	**Diseases of the Cardiovascular System**	
	1. Occlusive Coronary Artery disease:	
	a. Descending branch of the left coronary artery	27
	b. Common trunk of left coronary artery	2
	c. Circumflex of the left coronary artery	1
	2. Coronary artery insufficiency	6
	3. Cardiac tamponade	1
	4. Rupture of aortic aneurysms	0
	5. Valvular diseases of the heart	3
	6. Congenital Heart diseases	2
	Total	**42 cases 42%**
II.	**Diseases of the Respiratory System**	
	1. Pulmonary tuberculosis	19
	2. Lobar Pneumonia	03
	3. Bronchopneumia	5
	4. Emphysema Thoracis	2
	5. Lung Abscess	1
		30%
III.	**Diseases of the Respiratory System**	
	1. Intracerebral haemorrage	4
	2. Meningitis	1
		5%
IV.	**Diseases of the Respiratory System**	
	1. Rupture of Oesophageal Varices	1
	2. Hepatic Failure	3
		4%
V.	**Miscellaneous**	
	1. Exhaution secondary to infection and malnutrition	**19%**



Aetiological Classification Of Sudden Natural Death

Out of 100 cases of Sudden Natural Deaths studied, diseases of the cardio vascular system constituted 42% group. Diseases of the respiratory system constituted the next major group i.e., 30%. Central Nervous system accounted for 5%. Gastrointestinal system accounted for 4% other cases like malnutrition added with infection accounted 19 cases.

Of 42 cases of Sudden Natural Deaths due to cardio origin, coronary occlusion accounted for 30 cases. Coronary artery sufficiency was the next major cause accounting for 6 cases. Cardiac tamponade accounted for 1 case of which was due to rupture of myocardial infarction. There were 3 cases of valvular diseases the heart. Congenital heart disease -2 cases.

In the respiratory system out of 30 cases, pulmonary tuberculosis accounted for 19 cases. Consolidation of lungs accounted for 3 cases. Emphema thoracis, lung abscess and bronchopneumonia accounted for 8 cases.

Among 5 cases of central nervous system, intra cerebral haemorrage accounted for 4 cases and meningitis accounted for 1 case.

Of 4 cases of sudden natural deaths due to gastrointestinal system, one case was due to rupture of oesophageal varices due to cirrhosis of liver. Hepatic failure accounted for 3 cases.

The other 19 cases were due to exhaustion as a result of infection and malnutrition especially among the destitute that were found dead.

AGE INCIDENCE IN SUDDEN NATURAL DEATHS

The age distribution of 100 cases of sudden natural deaths studies in this series is shown in Table 3.

The maximum number of cases i.e. 22 (22%) were seen in the age group of 46 to 50years. The next common age group was between 36 to 40years (16%).

Table 3

Age Group in years	No. of cases	Percentage
0 to 5	-	-
6 to 10	-	-
11 to 15	-	-
16 to 20	2	2
21 to 25	1	1
26 to 30	5	5
31 to 35	6	6
36 to 40	16	16
41 to 45	10	10
46 to 50	22	22
51 to 55	14	14
56 to 60	13	13
61 to 65	2	2
66 to 70	6	6
71 to 75	1	1
76 to 80	1	1
81 to 85	1	1
86 to 90	-	-

The maximum number of cases i.e. 22 (22%) were seen in the age group of 46 to 50years. The next common age group was between 36 to 40years (16%).

The maximum number of cases decade wise was seen in 5th decade i.e. 41 to 50years-32 cases (32%)

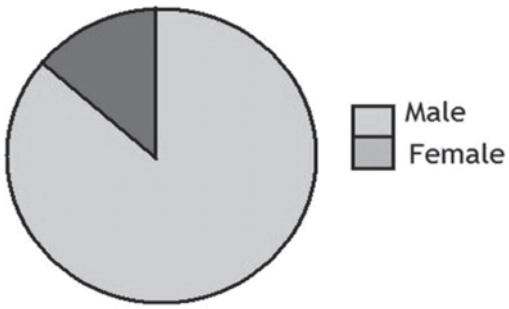
Only two cases were there above the age of 70 years and there were no cases below the age of 15 years.

SEX INCIDENCE IN SUDEN NATURAL DEATHS

The sex incidence if the 100 cases of sudden natural deaths studies are shown in table 4.

Table 4

Sex	No. of cases
Male	91%
Female	9%

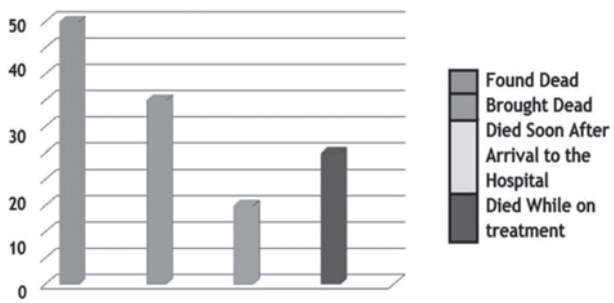


In this series, males constituted 91% and females constituted 9%.

PERIOD OF SURIVAL IN SUDDEN NATURAL DEATHS

Table 5

Period of Survival	No. of cases	Percentage
Found dead	51	51
Brought dead	30	30
Died soon after arrival to Hospital	5	5
Died while on treatment	14	14



In this series, nearly one-third of the cases i.e.30 (30%) presented with history of brought dead to the hospital, i.e. died on the way to Hospital, while 51 were found dead elsewhere. Only 5 cases died soon after arrival to the hospital, and 14 cases during the treatment.

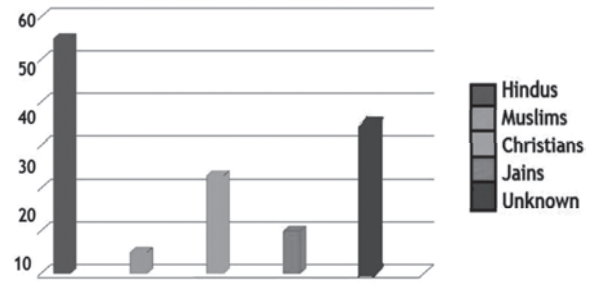
RELIGION DISTRIBUTION IN SUDDEN NATURAL DEATHS

The religion distribution of 100 cases of sudden natural deaths is shown in Table 6

Table 6

Religion	No. of cases	Percentage
Hindus	54	54
Muslims	1	1
Christians	10	10
Jains	2	2
Not known	33	33

Majority of cases of sudden natural deaths is seen in Hindus, followed by Christians, then Jains and then by Muslims.



DISCUSSION

100 cases of sudden natural deaths were studies in this series and accounted for 6.36% of the total autopsies (1590 cases).

Stress and strain of modern life, [security guards, police constables, drivers, changing dietetic pattern-alcoholism, concentration of fatty foods, ice cream, fast foods, bakery items, high salt intake, tobacco, lack of physical activity, use of two wheelers or four wheelers, increased population, increased competitive life, industrialization are the contributory factors.

In the cardiovascular system diseases, occlusion of the descending branch of left coronary artery accounted for 90% of total coronary artery occlusion. Comparison of this date with other series clearly confirms the pre-ponderance of occlusion of descending branch of left coronary artery.

On the other hand atherosclerosis of the right coronary artery does contribute for the coronary artery insufficiency because combined luminal narrowing was noted in majority of the cases.

Three cases of valvular diseases of hear was noted, one having mitral valve and others having both mitral and tricuspid valves. In all three cases aetiology was rheumatic disease and shows that this disease is quite common in our country compared to western countries.

Two congenital heart diseases contributed to sudden death. One had died during his 'honey-moon'. Other had earlier gone cardiac surgery 7years earlier.

In the respiratory system diseases, the main contributor is pulmonary tuberculosis 19 out of 30 cases. (57%). Majority of them were unidentified destitutes who were found dead. This is one of the commonest of disease in developing countries which is responsible for higher incidence of sudden natural deaths due to this disease.

In the central nervous system diseases major contributor is intra-cerebral hemorrhage coincides with other series.

Diseases of the gastro intestinal system accounted for only 4 cases. This decreased incidence could be due to revolution in the treatment of ulcers and other gastro-intestinal diseases.

In this study no case of disease of genitor-urinary system resulted in sudden natural death. Probable reason could be many of the diseases of the urinary system are gradual giving rise to symptoms like swelling of the face & body, haematuria, etc., whereby there is sufficient time for the person to have medical attention and hence chances of giving rise to suspicion are negligible.

It has been shown in this study that the maximum number of cases of sudden natural deaths is in the age group of 41 to 50 years. In Helpern and Rabson series the greatest incidence was in the age group of 45 to 54 years which was 10 to 15 years later than the percentage peak of population, i.e., 35 to 39 years.

In Lauren's series the maximum cases were between 40 to 45 years and shows that the age distribution of this study coincides with other series.

Male to female ratio of sudden natural deaths in all was 91:9(10:1). The result when compared with Nagpur series (5:3:1), Helpern and Rabson series (4:2:1), Lauren's series (3:1) reveal that male affliction in sudden natural deaths is certainly higher as reported in this study. Valid reason for this cannot be accounted scientifically. In deaths due to cardiac diseases, male preponderance is most striking between the ages of 33 and 55 years, with a male to female risk in these years of about 6:1. During reproductive life, women for reasons still unknown remarkably spared unless they have an underlying predisposition to atherosclerosis such as diabetes mellitus. In the later decades the male's preponderance steadily diminishes and approaches 1:1 in extreme old age. Also the habits of smoking and alcoholism common in males may contribute for the high incidence in them, and females predominantly being housewives rarely fall under circumstances that would necessitate an autopsy.

Religion distribution of sudden natural deaths shows that the maximum number of cases i.e., 54 cases is in Hindus followed by Christians & Jains. This is not only seen in sudden natural death but it is the same case in any disease or disorders on clinical side and is a reflection of the religion distribution of the general population. Hence, religion has no bearing on sudden natural death.

CONCLUSION

From this study we can definitely conclude that diseases of the cardiovascular system are the major contributing factor to sudden natural deaths. Respiratory system diseases contributed the next major share and diseases compared with Western countries. This can be attributed to the poor hygiene, malnutrition, socio-economic status, and tuberculosis in particular.

In diseases of the cardiovascular system, coronary artery disease is the major contributing factor to which valvular diseases of heart especially of rheumatic aetiology and congenital heart diseases also contribute.

The age distribution curve points out the fact that younger age group involvement appears to be slightly higher. This could be explained by bearing in mind the changing social concepts, ways of living, progressive excessive indulgence of younger age groups in predisposing factors like smoking, alcoholism, stress & strain of modern life and so on.

Attempts at determining the relationship between physical constitution, occupation and sudden natural deaths revealed that contrary to the popular belief, moderately built and muscular individuals and those engaged in strenuous activity also had a considerable risk of sudden natural deaths of cardiac origin.

Personal habits when compared indicated that in cases of adequate history, smoking with alcohol an especially smoking are definitely high risk factors in causing sudden natural deaths.

This drives home the conclusion that even under circumstances suggestive of foul play one need to bear in mind the possibility of sudden natural death and there by emphasize the medico legal importance of sudden natural death. This unbiased open mind when investigating medico legal cases helps to serve the ends of justice to the best.

This study of sudden natural deaths has clearly demonstrated the vast, interesting and intriguing to the profession. We can only conclude by saying that we have focused the importance of entity by this study leaving the portal of research in this specially open and alive for posterity to prove its best.

REFERENCES

1. Krishan Vij Textbook of Forensic Medicine and Toxicology: Principles and practice.4th ed Newdelhi: Elsevier, a division of Reed Elsevier India Private Limited; 2008. P134-135.
2 Sharma R K. Concise Textbook of Forensic medicine and Toxicology.2nd ed. Newdelhi: Elsevier, a division of Reed Elsevier India Private Limited; 2008.P40.
3 Dikshit PC Textbook of Forensic Medicine and Toxicology. Icd. Newdelhi.Peepee publishers and distributors (P) Ltd: 2007. P.147
4. Sudden death - A case of hypertrophic cardiomyopathy JIAFM, 2004; 26(4). ISSN 0971-0973

Role of Nature of Anions in the Toxicities of Various Lead Salts Lead Nitrate, Lead Sulphate & Lead Acetate on the Different Organs of Female Rats

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ABSTRACT

Heavy metal toxicity is a serious worldwide problem which adversely affects the growth, health, reproductive performance and life span of all living organisms. In my previous work1-4 I (Dhir) have worked on the toxicological aspects on the reproductive functions in female rats, physicochemical interactions in between the biomolecules with series of cations and also studied the importance of hydrophobic character of big biomolecules. Therefore; in this project I studied the importance of nature of anion in the salt of lead. Lead being a toxic cumulative poison and an environmental pollutant, experiments were conducted on an oral chronic dose (@35 mg/kg/day) for 90 days on adult female rats (Rattus Norvegicus) and its effect on the reproductive functions in relation to the biochemical effects were studied. It was observed that the chronic dose of various salts of lead caused an elevation in the level of proteins, acid phosphatase, alkaline phosphatase, alanine aminotransferase and aspartate aminotransferase in all the soft tissues which indicated tissue damage and the effects are received in the following order (as in case of anions):

$$SO_4^{2-} > NO_3^- > CH_3COO^- \text{ (in terms of toxicity)}$$

However no literature was available so far as to compare the effect of anion on the toxicity level of lead. The doubly charged sulphate anion (SO_4^{2-}) interacts physicochemically with the various biological biomolecules (imidazole, cysteine sulfhydryls & amino group of lysine) results in tissue damage as in comparison with nitrate and acetate which are singly charged. Histological studies of ovary showed atresia (Figures 1-4) in all the stages of folliculogenesis sustaining the poor fertility observations.

Key words: - *Proteins, Acid Phosphatase, Alkaline Phosphatase, Alanine Aminotransferase, Aspartate Aminotransferase.*

INTRODUCTION

Lead have no known biological function and any lead (lead nitrate, lead sulphate & lead acetate) absorbed by man or animals may be potentially toxic. All spheres which are affected by lead can causes 33% increased absorption of lead which interferes with blood forming processes, vitamin D metabolism and other kidney and neurological processes[5]. The toxic effects are many, ranging from morphological tissues damage at higher concentration to lesser biochemical effects at lower concentrations[6]. Lead is known to be toxic when present in traces and enters human body as a result of environmental pollution[5]. Occupational hazards due to lead exposure produce reversible changes in mood and personality as fatigue, irritability, depression, deficits in vascular motor functioning, memory and verbal ability[1,4,5]. Lead has high affinity for various complexing groups such as imidazole, cysteine sulfhydryls and amino group of lysine. By complexing with these moieties lead may interfere with biochemical processors through alterations of structural integrity of enzyme or by disruption of substrate binding. Children exposed to lead are reported to have adverse effects on central nervous system and kidneys[6]. Maternal blood lead level as an environmental factor is an apparent predictor of low birth weight and child body mass ratio[6] and low to moderate environmental exposure increases the risk for spontaneous abortion[5] Anaemia which is frequently observed in lead poisoning was a result of decrease lifetime of erythrocytes and synthesis of heme[3,7]. Mating involving one lead toxic parent have recorded significant decrease in litter size, birth weight and survival rate[5,7]. A variation in the time of vaginal opening and a significant disturbed oestrous cycle was also observed in lead toxicity[8].

MATERIALS AND METHODS

Disease free albino rats 2-3 months were maintained on rat feed (Ashirwad Industries, Chandigarh-India) and black gram. Water was provided ad *libitum*. Blood samples were drawn into heparinised tubes and plasma was separated after centrifugation at 3000 rpm for 5 minutes at room temperature. The plasma was diluted in the ratio of 1:10. The tissue samples were homogenized in the homogenizer in potassium phosphate buffer in the ratio of 1:10. The effect of various salts of lead on aspartate aminotransferase, alanine aminotransferase, acid phosphate and alkaline phosphatise was estimated by the method of Wootton (1964). The cholinesterase activity was determined according to the method of Voss and Sachsse (1970) and total proteins were determined by Lowry et al. (1951). Statistical significance of biochemical parameters was obtained by students t- tests at 1% level (P 0.01) and at the 5% level (P 0.05). State of the estrous cycle of each animal was determined by taking vaginal smears[7] daily between 10:30 a.m. to 12:30 a.m. In order to take vaginal smears, the vaginal was washed with physiological saline (0.9 per cent) by injecting a drop of solution with a dropper.

RESULTS AND DISCUSSIONS; BIOCHEMICAL PARAMETERS:

Daily oral administration of lead nitrate, lead sulphate & lead acetate (@35 mg/kg/day) for 90 days produced a significant rise in the levels of acid phophatase in lever, kidney and ovary and a non-significant increase of enzyme in plasma following daily exposure of lead. Acid phosphatase is a lysosomal enzyme and is stimulated in cases of tissue damage[4]. Increase in level of acid phosphatase in liver and kidney might be suggestive of increase physiological phagocytosis[3] and the moderate amount of acid phosphatase activity in regressing luteal cells of the ovary indicated lysosomal activity in luteolysis[4]. The increase in acid phosphatase activity estimated biochemically would therefore mean a destruction of the luteal cells which is in support of the fact that absence of acetylecholinesterase activity in ovary also causes lack of steroidogenesis. It has been further suggested that in follicle cells, lysosomal enzymes affects estrogen receptor by dephosphorylation which led to atresia and also the enzyme acid phosphatase is an excellent indicator of atrophy[4]. lead nitrate, lead sulphate & lead acetate caused a significant increase in alkaline phosphatase level (Table 1) in plasma, liver, kidney and ovary. It has been suggested that an increase in alkaline phosphatase level occur due to the

Table 1. Effect of Lead Acetate, Lead Nitrate and Lead Sulphate on Tissue Phosphatases

Organ	Control	Treatment					
		15 days	30 days	45 days	60 days	75 days	90 days
Acid phosphatase (n mol phenol liberated / min/ml) (Mean±S.D.)							
Plasma (Lead Acetate)	118.93±2.95	0.699±1.43	0.701±3.32	0.711±1.95	0.723±2.95	0.715±1.22	0.750±2.22
Plasma (Lead Nitrate)	118.93±2.95	0.622±0.092	0.656±0.061	0.670±0.273	0.699±0.158	0.715±0.099	0.729±0.043
Plasma (Lead Sulphate)	118.93±2.95	0.7789±0.052	0.795±0.045	0.8214±0.353	0.8344±0.556	0.856±0.194	0.877±0.267
Liver (Lead Acetate)	118.93±2.95	120.22±2.22	121.22±1.23	134.33±1.55[ab]	136.20±1.43[a]	143.22±1.20[ab]	198.23±1.24[ab]
Liver (Lead Nitrate)	118.93±2.95	119.41±1.92	116.43±2.48	130.72±0.97[ab]	125.30±2.69[a]	147.92±2.40[ab]	196.52±3.69[ab]
Liver (Lead Sulphate)	118.93±2.95	118.34±3.88	119.56±3.45	132.56±0.88[ab]	134.55±4.88[a]	138.99±4.40[ab]	178.58±4.45[ab]
Kidney (Lead Acetate)	9.315±0.258	9.234±1.30	16.23±1.23[ab]	27.36±2.34[ab]	28.021±1.78[ab]	29.350±0.928[ab]	30.023±2.102[ab]
Kidney (Lead Nitrate)	9.315±0.258	9.000±2.240	15.590±3.120[ab]	26.326±1.77[ab]	26.058±2.880[ab]	25.550±0.938[ab]	29.055±1.301[ab]
Kidney (Lead Sulphate)	9.315±0.258	11.245±1.35	11.345±4.24[ab]	28.453±4.66[ab]	28.994±3.670[ab]	30.657±0.787[ab]	31.567±2.454[ab]
Ovary (Lead Acetate)	4.069±0.65	4.644±0.089	4.342±0.123	4.55±0.234	5.88±0.234	9.99±0.234[ab]	23.124±0.539[ab]
Ovary (Lead Nitrate)	4.069±0.65	4.527±0.078	4.222±0.056	4.54±0.403	5.73±0.698	9.71±0.146[ab]	21.934±0.639[ab]
Ovary (Sulphate)	4.069±0.65	4.543±0.178	4.768±0.248	6.765±0.243	7.789±0.897	18.675±0.344[ab]	29.657±0.874[ab]
Alkaline Phosphatase (n mol phenol liberated / min/ml) (Mean S.D.)							
Plasma(Lead Acetate)	13.81±0.215	13.621±0.845[a]	18.999±0.234[ab]	23.183±1.230[ab]	25.121±1.232[ab]	30.245±0.234[ab]	43.234±0.234
Plasma (Lead Nitrate)	13.81±0.215	12.609±0.880[a]	18.487±0.955[ab]	22.214±1.090[ab]	24.535±1.190[ab]	29.54±0.455[ab]	40.912±0.346
Plasma (Lead Sulphate)	13.81±0.215	15.775±0.678[a]	17.298±0.788[ab]	22.564±2.676[ab]	26.534±1.34[ab]	37.57±0.679[ab]	47.881±0.789
Liver (Lead Acetate)	27.15±0.786	28.230±0.563	29.332±0.332[ab]	36.231±2.22[ab]	32.223±0.787[ab]	36.734±2.123[ab]	43.234±1.235[ab]
Liver (Lead Nitrate)	27.15±0.786	27.950±0.673	29.530±0.600[ab]	35.091±1.630[ab]	30.630±0.304[ab]	35.841±1.013[ab]	42.349±1.960[ab]
Liver (Lead Sulphate)	27.15±0.786	30.567±0.883	28.490±0.597[ab]	39.247±3.645[ab]	39.989±0.456[ab]	42.689±2.345[ab]	44.897±2.978[ab]
Kidney (Lead Acetate)	1630.03±12.930	1855.345±26.234[ab]	1859.897±21.232[ab]	1811.521±18.234[ab]	1875.457±22.320[ab]	1966.122±22.320[ab]	28422.50±19.330[ab]
Kidney (Lead Nitrate)	1630.03±12.930	1846.310±24.140[ab]	1857.760±20.980[ab]	1801.551±18.490[ab]	1874.277±39.950[ab]	1964.194±21.380[ab]	2846.2510±12.330[ab]
Kidney (Lead Sulphate)	1630.03±12.930	1956.7±34.367[ab]	2089.9±18.967[ab]	1999.01±34.678[ab]	2078.9±43.123[ab]	1999.78±22.675[ab]	2789.7±2.378[ab]
Ovary (Lead Acetate)	12.193±3.050	15.223±0.243	22.124±3.430	24.123±1.234[ab]	27.323± 4.321[ab]	35.343±0.234[ab]	47.342±4.501[ab]
Ovary (Lead Nitrate)	12.193±3.050	14.280±0.495	21.550±7.690	22.261±2.480[ab]	26.998±2.970[ab]	31.460±0.500[ab]	45.260±9.900[ab]
Ovary (Lead Sulphate)	12.193±3.050	15.338±0.345	22.586±8.560	23.1246±3.568[ab]	27.998±3.560[ab]	33.680±0.2430[ab]	47.356±9.879[ab]

damage of the cells of liver, kidney, small intestine and bone resulting in liberation of this enzymes in the blood systems (Zimmerman 1969). Alkaline phosphatase helps in ionic movement across the cell membrane and in also associated with secretory and absorption processes of the cell[5]. Wise (1987) in bovine follicles also postulated AKP as an excellent indicator of atresia since AKP activity was greater in ovary. The changes in enzymes system had been correlated with the steroid biosynthesis in the granulose cells of maturing follicles of mammalian ovary[8]. Lead nitrate, lead sulphate & lead acetate also produced significant increase in aspartate aminotransferase in liver, plasma and ovary while the effect of SO_4^{2-} anion (sulphate) in lead on aspartate aminotransferase in liver, plasma and ovary is more as compared to NO_3- & CH_3COO^- salts of lead (Table 2). This is a very important observation. Aspartate aminotransferase SGOT occur mainly in muscles (Doxey 1971) and increase in its activity related to the leakage of enzyme from muscles because of muscular activity induced by intoxication. Direct effect of lead on muscles increasing the permeability of cell membrane cannot be excluded (Thomson 1971). Thus decrease in AChE activity in the rat ovary might be an indicator of the lack of steroidogenesis resulting in poor fertility. Elevation of proteins might also be due to destruction of tissues, which cause release of proteins.

Fertility test

Five sets of experiments which were set up for the testing effect of various salts of lead on the fertility of rats indicated that lead sulphate at a dose of 35 mg/kg caused 50% reduction in the fertility rate while lead nitrate & acetate at a dose of 35 mg/kg caused 40% & 35 % reduction in the fertility rate, respectively (Table 2) as compared to control group of rats which showed 100% results. The decrease in fertility has been related to the decrease in AChE concentration which is considered important in the process of steroidogenesis and increase in level of other enzymes which might be damaging to the tissue leading to atresia (Figures 1-4). Chronic dosage of various salts of lead probably imbalances this delicate interplay of hormones and disallows implantation in rat6. In addition to the observations made above, the treated females showed irregularity in estrous cycle. Female pups of treated

Table 2. Effect of Lead Acetate, Lead Nitrate and Lead Sulphate on the Body Weights of Pups of Treated Mothers and Dose after Lactation.

Body weight at birth (mean±S.D.)		15 days	30 days	45 days	60 days
Control	7.06±0.24	18.40±1.94	40.86±3.42	59.86±2.43	81.92±4.61
A	5.250±0.900[ab]	11.290±1.25[ab] (nitrate), 10.388±2.45[ab] (acetate), 11.399±3.23[ab] (sulphate).	21.290±2.46 (nitrate), 19.38±3.58 (sulphate), 20.11±2.79 (sulphate).	35.21±0.21[ab](sulphate), 30.99±0.234[ab] (acetate), 31.29±0.258 (nitrate).	-
B	5.306±0.370[ab]	9.8260±2.27[ab] (sulphate), 9.768±3.14[ab] (acetate), 9.892±2.34[ab] (nitrate).	16.440±4.29(sulphate), 15.230±3.03(acetate), 16.089±2.15(nitrate).	33.80±0.39[ab] (sulphate), 31.67±0.58[ab] (acetate), 32.34±0.24[ab] (nitrate).	-
C	5.570±0.233[ab]	10.912±1.03[ab] (sulphate), 09.876±1.05[ab] (nitrate), 08.896±1.11[ab] (acetate).	20.990±1.56(sulphate), 18.678±1.89(nitrate), 19.453±1.99(acetate).	31.82±0.00[ab] (sulphate), 30.65±0.03[ab] (nitrate), 28.232±0.42(acetate).	30.21± 0.00[ab] (nitrate, died on day 63), 26.61±0.05[ab] (acetate, died on day 55), 32.12±0.25[ab] (sulphate, died on day 52).
D	5.490±0.150[ab]	10.560±1.97[ab] (sulphate), 09.384±1.82[ab] (nitrate), 05.642±0.78[ab] (acetate).	20.765±1.14(nitrate), 19.325±2.28(acetate), 21.221±1.02(sulphate).	31.48±0.00[ab] (sulphate), 18.98±0.04[ab] (acetate), 23.122±2.12 (nitrate).	

Table 3. Survival Rate of Pups

Days of treatment (female)	Number of pups	Survival at birth time	Survival after 15 days	Survival after 30 days	Survival after 30 days	Survival after 45 days	Survival after 60 days
Control (no treatment)	6-10	6-10	6-10 / (nitrate/ sulphate acetate)	6-10	6-10	6-10	6-10
60	11	9	8(nitrate), 6(acetate), 9(sulphate).	6	6	3	Died
60	Nil	Nil	Nil	Nil	Nil	Nil	Nil
60	8	5	5 (acetate), 7(nitrate), 8(sulphate).	4 (nitrate), 3(acetate), 5(sulphate).	4(acetate), 6(nitrate), 7(sulphate).	2(acetate), 4(nitrate), 5(sulphate).	Died (nitrate / /sulphate / acetate).
60	9	5	5(nitrate), 7(acetate), 8(sulphate).	3(nitrate), 5(acetate), 6(sulphate).	3 (acetate), 6(sulphate), 5(nitrate).	1(acetate), 2(nitrate), 7(sulphate).	1(acetate), 3(sulphate), 2(nitrate).
60	7	5	3(nitrate), 2(acetate), 4(sulphate).	2(nitrate), 1(acetate), 3(sulphate).	2(sulphate/nitrate), 1(acetate).	1(sulphate), Nil-(acetate), 1(nitrate).	Died (nitrate / sulphate / acetate).

Table 4. Effect of Lead Acetate, Lead Nitrate and Lead Sulphate on Body Weight of Treated Female

Treatment dose (mg/kg)	0 day	15 days	30 days	45 days	60 days
0	122±4.52	132±3.33 (nitrate), 120±4.24 (sulphate), 135±2.99(acetate)	145±2.26(sulphate), 135±2.04(nitrate), 123±1.03(acetate).	151±2.61(nitrate), 149±2.20(acetate), 155±3.23(sulphate).	165±4.21(sulphate), 155±2.93(acetate), 160±2.03(nitrate).
60	120±2.95	122±4.32ab (all dead, sulphate), 119±2.12ab(acetate), 125±2.12(nitrate).	125±2.26ab (nitrate), 118±1.86ab(acetate), 128±2.51(sulphate).	130±1.56ab(sulphate), 120±1.33ab(nitrate), 128±2.35(acetate).	127±4.56ab(nitrate), 120±3.44ab(acetate), 130±2.14(sulphate).

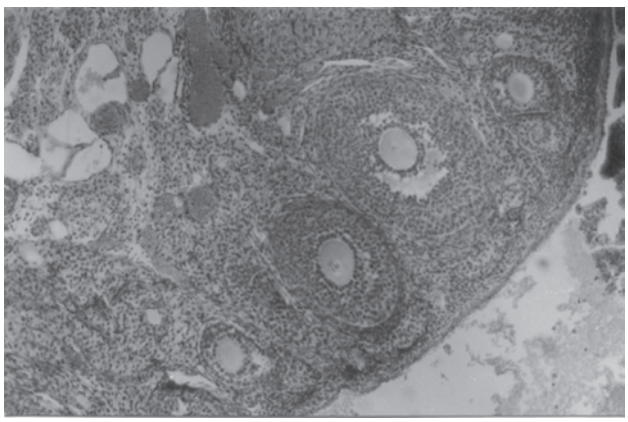

Fig. 1. Various Stages of Follicles undergoing atrsia (HE stain) 100X(Lead Nitrate).

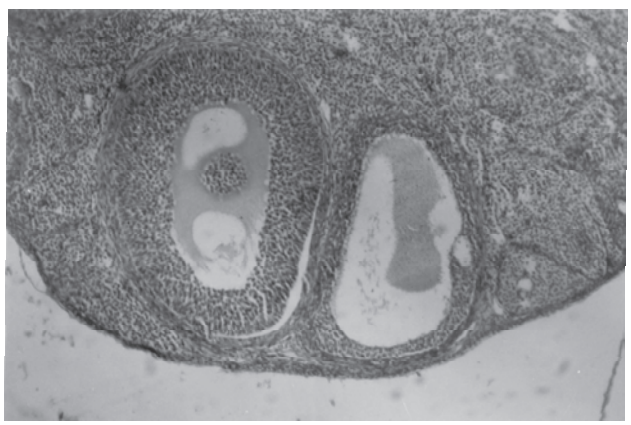

Fig. 2. Incipient Antral Stage Follicle undergoing atresia (HE stain) 100X(Lead Sulphate).

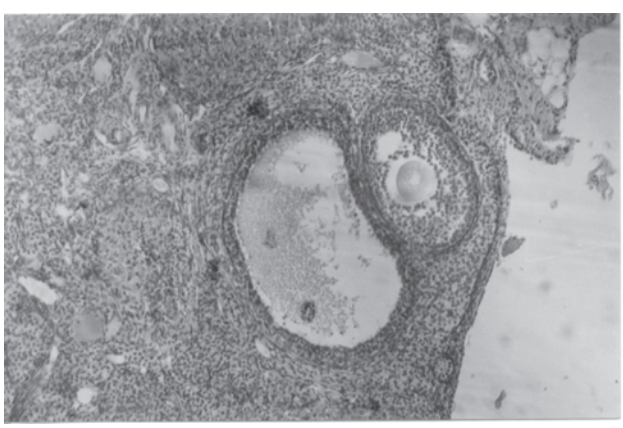

Fig. 3. Antrum formed Graafian follicle undergoing atresia with complete detachment of Granulose from theca shows advanced stage in atresia (HE stain) 100X (Lead Acetate).

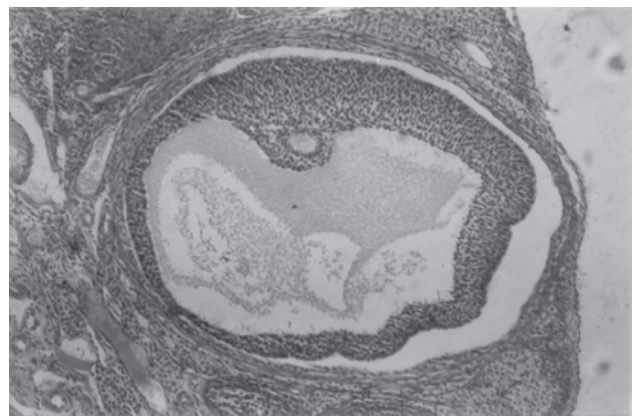

Fig. 4. Antrum formed Graafian follicle undergoing atresia with complete detachment (arrowheads) of Granulose from theca shows advanced stage in atresia (HE stain) 100X (Lead Nitrate).

mother also showed late vaginal opening, poor fur growth, significantly lower body weight (Table 3) and decrease foetal survival ratio. Rat fed on various salts of lead showed significant decrease in body weight (Table 4) while the decrease in weight is more in case of :

$SO_4^{2-} > NO_3^- > CH_3COO^-$ (Various Salts of Lead)

Parshant et al.[9] (2009) in medico-legal update (journal) also mentioned the detailed method of analysis of Pb in blood samples but with the help of Flame Atomic Absorption Spectrophotometer which also shows good observations.

The above study concluded that lead has interaction with the vital body functions and reproductive parameters in rats. The dosage administered caused significant biochemical alterations and reduction in the weight of pups as well as the treated mothers.

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REFERENCES

1. Dhir, Vaneet, Dhand P (2010) Toxicological Approach in Chronic exposure of lead on reproductive functions in female rats (Rattus Norvegicus) Toxicology International., 17:01-07.

2 Dhir, Vaneet (2009) Physicochemical interactions in between eccrine and series of cations (according to hofmeister series). Indian Journal of Forensic Medicine & Toxicology., 9(2): 46-48.

3 Dhir, Vaneet (2009) Comparative study of latent fingerprint impression over different materials like plastic sheets, mica, aluminium, copper and their interpretation in terms of potential surge as compared to old classical theory. Medico-Legal Update., 9(2): 48-52.

4 Dhir, Vaneet (2011) Comparitive studies of some toxic ions like Pb2+ & Cd2+on the reproductive finctions in female rats: A case study. Medico-Legal Update., 11(1): 75-80.

5 Aurrichio F, Migliacci A and Castoria Y (1981) Dephosphorylation of oestradiol receptor in vivo. Biochem. J., 198: 699.

6 Borja-Arburito, Victor H, Irva HP, Magdelena, RL, Paurline F, Camilo R and Julia Blenco (1999) Blood lead levels measured prospectively and risk of spontaneous abortion. Ame.r J. Epidemiol., 150 (6): 590-597.

7 Zimmerman HJ (1969) Serum enzymes determination as an aid to diagonosis In : Clinical diagnosis by Laboratory methods Dawidson I and Henry J B (eds.) pp 719, Saunders W B Co., Philadelphia.

8 Goody WW, Schrader WT and Malley BWO (1982) Activation, transformation and subunit structure of steroid hormone receptor. Endocr. Rev., 3: 141.

9 Mittal Anugya, Agrawal Prashant, Jain Madhu, Basu Sriparna, Tripathi S.K.(2009) Detailed method of analysis of Pb in blood samples with the help of Flame Atomic Absorption Spectrophotometer Medico-Legal Update., 9(2): 24-25.

Dental Profiling- A Boon for the Forensic World

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ABSTRACT

A dental profile will typically provide information on the deceased's age, ancestry background, sex and socio-economic status. In some instances it is possible to provide additional information regarding occupation, dietary habits, habitual behaviours and occasionally on dental or systemic diseases. In many cases the tentative identification of the individual is unknown and therefore antemortem records cannot be located. In such a situation a dental profile of the individual is developed to aid the search for the individual's identity. With such a profile a forensic odontologist can identify and report indicators for age at time of death, race, and sex. In addition to these parameters the forensic dentist may be able to give more insight into the individual. This paper reviews for the non-expert, some of the additional personal information that can be derived from the teeth of the deceased, and which may assist in their ultimate identification.

Key words : *DNA Profiling, Dental Profiling, Forensic Dentistry.*

INTRODUCTION

The use of unique features of the human dentition to aid in personal identification is well accepted within the forensic field[1]. Despite leaps in modern technology, medical breakthroughs and the geographical changes that the last century has brought, crime still persists in all aspects of our lives. Through the specialty of forensic odontology, dentistry plays a small but significant role in this process. By identifying the victims of crime and disaster through dental records, dentists assist those involved in crime investigation[2]. A dental profile will typically provide information on the deceased's age, ancestry background, sex and socio-economic status. In some instances it is possible to provide additional information regarding occupation, dietary habits, habitual behaviours and occasionally on dental or systemic diseases[3].

The most common role of the forensic dentist is the identification of deceased individuals[4].Dental identification takes two main forms. Firstly, the most frequently performed examination is a comparative identification that is used to establish (to a high degree of certainty) that the remains of a decedent and a person represented by antemortem (before death) dental records are the same individual. Information from the body or circumstances usually contains clues as to who has died. Secondly, in those cases where antemortem records are not available, and no clues to the possible identity exist, a postmortem (after death) dental profile is completed by the forensic dentist suggesting characteristics of the individual likely to narrow the search for the antemortem materials[5].

The forensic dentist will often work with a forensic anthropologist to help in identification of an individual or the development of a profile from remains. The determination of sex and ancestry can be assessed from skull shape and form. Generally, from skull appearance, forensic dentists can determine race within the three major groups: Caucasoid, Mongoloid and Negroid. Additional characteristics, such as cusps of Carabelli, shovel-shaped incisors and multi-cusped premolars, can also assist in determination of ancestry[6]. Sex determination is usually based on cranial appearance, as no sex differences are apparent in the morphology of teeth. Microscopic examination of teeth can confirm sex by the presence or absence of Y-chromatin and DNA analysis can also reveal sex[7-9].

Because of the resistant nature of dental tissues to environmental assaults, such as incineration, immersion, trauma, mutilation and decomposition, teeth represent an excellent source of DNA material. When conventional dental identification methods fail, this biological material can provide the necessary link to prove identity[7].

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Indeed, despite advances in DNA and other identification methodologies, comparative dental identifications still play a major role in identifying the victims of violence, disaster or other misfortune. The classic comparative dental identification employs the use of post-mortem and antemortem dental records to determine similarities and exclude discrepancies. In many cases the tentative identification of the individual is unknown and therefore antemortem records cannot be located. In such a situation a dental profile of the individual is developed to aid the search for the individual's identity. With such a profile a forensic odontologist can identify and report indicators for age at time of death, race, and sex. In addition to these parameters the forensic dentist may be able to give more insight into the individual1. This paper reviews for the non-expert, some of the additional personal information that can be derived from the teeth of the deceased, and which may assist in their ultimate identification.

POSTMORTEM IDENTIFICATION

When antemortem dental records are unavailable and other methods of identification are not possible, the forensic dentist can assist in limiting the population pool to which the deceased is likely to belong and thus increase the likelihood of locating antemortem dental records. This process is known as postmortem dental profiling. The information from this process will enable a more focused search for antemortem records. A postmortem dental profile will typically provide information on the deceased's age, ancestry background, sex and socio-economic status[5].

The most common role of the forensic dentist is the identification of deceased individuals. Dental identification of humans occurs for a number of different reasons and in a number of different situations[10]. The bodies of victims of violent crimes, fires, motor vehicle accidents and work place accidents, can be disfigured to such an extent that identification by a family member is neither reliable nor desirable[11-13]. Dental identifications have always played a key role in natural and manmade disaster situations and in particular the mass casualties normally associated with aviation disasters[14,15].

Many people are familiar with the concept of dental identification; it is frequently mentioned on television news. But the nuances and complexities of the process are rarely understood. The central dogma of dental identification is that post-mortem dental remains can be compared with dental records, including written notes, study casts, radiographs, etc, to confirm identity.

The forensic dentist produces the post-mortem record by careful charting and written descriptions of the dental structures and x-rays. Once the post-mortem record is complete, a comparison between these and dental records can be carried out. A range of conclusions can be reached when reporting a dental identification. The American Board of Forensic Odontology recommends that these be limited to the following four conclusions:

- **Positive identification:** The antemortem and post-mortem data match in sufficient detail, with no unexplainable discrepancies, to establish that they are from the same individual.
- **Possible identification:** the antemortem and post-mortem data have consistent features but, because of the quality of either the post-mortem remains or the antemortem evidence, it is not possible to establish identity positively.
- **Insufficient evidence:** The available information is insufficient to form the basis for a conclusion.
- **Exclusion:** the antemortem and post-mortem data are clearly inconsistent.

Unlike fingerprints there is no minimum number of concordant features that are required for a positive identification. A single tooth can be used for identification if it contains sufficient unique features. Equally, a full-mouth series of radiographs may not reveal sufficient detail to render a positive conclusion. The discretion of identification lies with the odontologist who must be prepared to justify the conclusions in court, surely the ultimate in peer-review[16].

RECONSTRUCTIVE POSTMORTEM DENTAL PROFILING

When forensic odontologists can't readily identify the remains of a deceased individual through dental record cross-referencing, the use of reconstructive postmortem dental profiling may be helpful in giving the odontologists clues to the profile of the deceased, and can narrow the search parameters for investigators. By studying characteristics like tooth development, corrosion and stains present on tooth artifacts, factors like alcohol use, substance abuse, smoking habits and age can be determined or approximated[5].

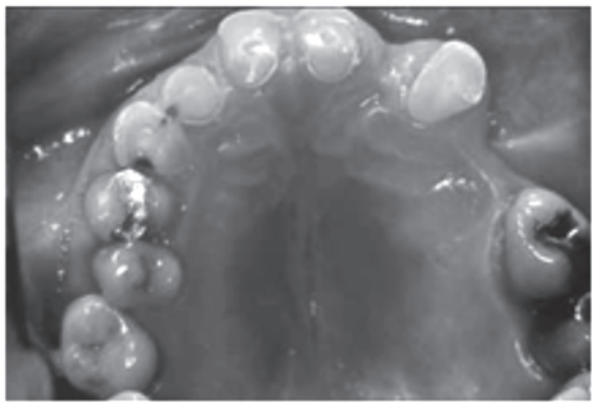
DENTAL POSTMORTEM PROFILING GUIDE

The dental postmortem profiling guide highlights a range of profiling factors. Here are some examples of some of the features which can help narrow the search for antemortem records when no putative identification exists. While each, on their own, cannot

identify an individual, they can all help to produce a more accurate profile of the deceased.

Case 1

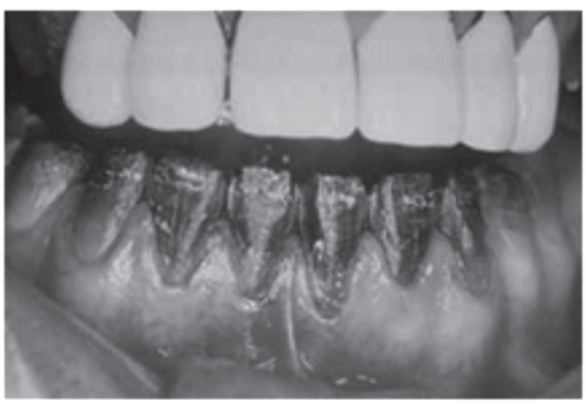
This is an example of dental erosion. Notice on the palatal surfaces of the teeth (i.e. facing inside the mouth) that enamel has been lost. This can be caused by a number of different conditions, such as anorexia, chronic alcoholism and gastric problems. Each causes repeated vomiting, which causes acid erosion of the teeth[17].



Case 1

Case 2

This individual has very pronounced staining visible on their lower teeth. It was also present on their upper teeth, but this has been covered by some (very poor) porcelain crowns or caps. This staining has been caused by the anti-biotic tetracycline which was taken when the teeth were developing[18].



Case 2

Case 3

This slide shows the tooth wear associated with pipe-smoking. Individuals found with wear of this sort are likely to have smoked a pipe and this information could be useful to the police in narrowing the search for antemortem records[1].

Case 3

Case 4

In this image we can see a wear facet on the upper and lower teeth. This individual is an electrician who wore his teeth by using them to strip electrical wire. This is a common finding in such professions, and a similar wear pattern can often by seen in seamstresses who hold pins between their teeth[19].

Case 4

Case 5

The quality and/or type of dentistry can often tell us where an individual received that treatment. This can prove valuable information in the search for antemortem records. This work was performed in Russia and is a classic example of the type of dentistry performed in the former Soviet Union. Note that the porcelain has been lost from 3 units on this individual[1].

Case 5

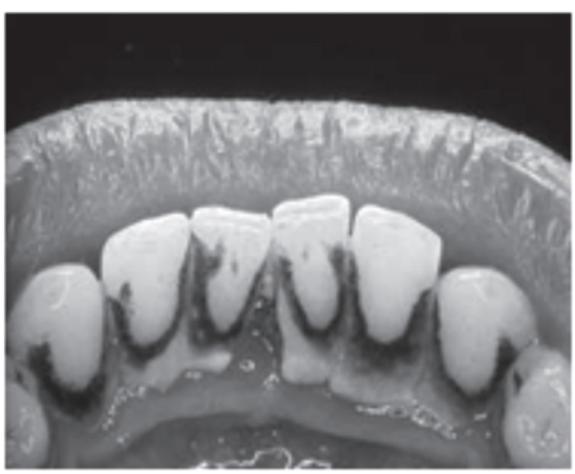
Case 6

Pink teeth can occur during life mainly due to traumatic or iatrogenic injuries to the pulp in dental procedures such as pulpal hemorrhage following restorative and periodontal treatment. In forensic pathology context, pink teeth is often observed in victims of drowning[20].

Case 6

Case 7

The staining on this individual's lower teeth have been caused by cigarette smoking. This finding can be useful to help include or exclude individual when determining an identity. The yellow material adhered to the teeth is dental calculus - this shows that this individual does not practice good oral hygiene or visit their dentist often[1].



Case 7

ROLE OF DNA IN DENTAL IDENTIFICATIONS

Because of the resistant nature of dental tissues to environmental assaults, such as incineration, immersion, trauma, mutilation and decomposition, teeth represent an excellent source of DNA material[21]. When conventional dental identification methods fail, this biological material can provide the necessary link to prove identity[22]. With the advent of the polymerase chain reaction (PCR), a technique that allows amplification of DNA at pre-selected, specific sites, this source of evidence is becoming increasingly popular with investigators. Comparison of DNA preserved in and extracted from the teeth of an unidentified individual can be made to a known antemortem sample (stored blood, hairbrush, clothing, biopsy, etc) or to a parent or sibling[5].

DENTAL IDENTIFICATION IN MASS DISASTERS

The identification of large numbers of casualties in mass disasters are complex and fraught with hazards, both physically and emotionally. The identification process is fundamentally the same as that in a routine comparative dental identification, but the inherent problems are magnified. Problems of body fragmentation, mutilation, commingling and incineration, idiosyncratic dental records from numerous regions, poor working conditions and psychological stresses all confound the identification process. The key to successful mass disaster identification is preparedness. Many jurisdictions have dental identification teams and disaster plans in place. Mock disaster scenarios that help dentists prepare for the disaster situation have proven to be successful[23,24].

SCREENING TOOL FOR DENTAL PROFILING

Besides DNA, dental radiographs play a major role in the identification of victims in mass casualties or in corpses with major postmortem alterations. Computed tomography (CT) is increasingly applied in forensic investigations and is used to scan the dentition of deceased persons within minutes. The use of dental processing software for computed tomography (CT) data (Dentascan) is described on postmortem CT data for the purpose of pm identification. The software allows reconstructing reformatted images comparable to conventional panoramic dental radiographs by defining a curved reconstruction line along the teeth on oblique images[25].

The teeth are by far the most durable part of the human body. In cases of fires and explosions, the teeth are often the only remains with which officials can work to identify the victim. In order to identify a person based on his or her teeth, it is essential to have pre and post mortem dental records for the person. The art of forensic odontology is so precise that it is possible to positively identify a person from one tooth. There are approximately 200 forensic odontology methods that exist internationally[25].

American/Universal method

Each of the 32 adult teeth is assigned a specific number. The upper right third molar is number one and the teeth are numbered until the lower right third molar is reached. The odontologist then records information about each tooth, which may be helpful to the identification process. The information is used to create an odontogram for the individual. An interesting development began in 1986 when the American Dental Association started a new program. Here coded microdisk is adhered to the tooth of an individual. The microdisk contains the details of the person's identity and its code can be translated by a computer. The teeth are also very useful in age determination. The markings on the teeth can be used to determine age within twenty days if the person is a child/teenager, and within 4-5 years if the person is an adult[25].

CONCLUSION

Forensic dentistry plays a major role in the identification of those individuals who cannot be identified visually or by other means. The unique nature of our dental anatomy and the placement of custom restorations ensure accuracy when the techniques are correctly employed. Unfortunately in developing countries like India forensic dentistry is not developed up to the mark, and services of forensic dentist are not being utilised. Government should instruct the Indian Dental association and other responsible agencies to direct the dental surgeons of the country to maintain the dental records casts, x-rays etc. and sample of tissue (tooth in case of extraction and debris of tooth material in case of restorations) of the patients treated by dental surgeons. So the preserved materials (Antemortem specimen) may be used in identification of deceased individuals.

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REFERENCES

1. A. Pretty, L.D.Addy. Associated post-mortem dental findings as an aid to personal identification. Science and Justice, 2002 ; 22 (2) : 0-10.
2. I.A. Pretty, D.Sweet. A look at forensic dentistry-Part 1: The role of teeth in the determination of human identity. British Dental Journal, 2001; 190 (7) : 359-366.
3. Malkowski F S. Forensic dentistry, a study of personal identification. Dent Stud 1972; 51:42-44.
4. Jones D G. Odontology often is final piece to grim puzzle. J Calif Dent Assoc 1998; 26: 650-651.
5. Sweet D, DiZinno J A. Personal identification through dental evidence-tooth fragments to DNA. J Calif Dent Assoc 1996; 24: 35-42.
6. Whittaker D K, Rawle L W. The effect of conditions of putrefaction on species determination in human and animal teeth. Forensic Sci Int 1987; 35: 209-212.
7. Sweet D, Hildebrand D, Phillips D. Identification of a skeleton using DNA from teeth and a PAP smear. J Forensic Sci 1999; 44: 630-633.
8. Murray M O, Wilson N H. Ecstasy related tooth wear. Br Dent J 1998; 185: 264.
9. Gupta B N. Occupational diseases of teeth. J Soc Occup Med 1990; 40: 149-152.
10. Weedn V W. Postmortem identifications of remains. Clin Lab Med 1998; 18: 115-137.
11. Whittaker D K, Richards B H, Jones M L. Orthodontic reconstruction in a victim of murder. Br J Orthod 1998; 25: 11-14.
12. Rothwell B R, Haglund W, Morton T H. Dental identification in serial homicides: the Green River Murders. J Am Dent Assoc 1989; 119: 373-379.
13. Andersen L, Juhl M, Solheim T, Borrman H. Odontological identification of fire victimspotentialities and limitations. Int J Legal Med 1995; 107: 229-234.
14. Tuominen M.L, Tuominen R.J, Fubusa F, Mgalula N. Tooth surface loss and exposure to organic and inorganic acid fumes in workplace air. Community Dental and Oral Epidemology 1991; 19(4): 217-220.
15. Florescu M. Dental lesions in workers from the sulphuric acid industry. Stomatologia 1972; 19 (5): 395-403.
16. American Board of Forensic Odontology. Body identification guidelines. J Am Dent Assoc 1994; 125: 1244-1254.
17. Bartellet D.W, Smith B.G. The dental impact of eating disorders. Dental Update 1994; 21 (10) : 404-407.
18. Kashyap A.S, Sharma H.S. Discolouration of permanent teeth and enamel hypoplasia due to tetracycline. Postgraduate Medical Journal 1999; 75 (890) :772.
19. Gupta B.N. Occupational diseases of teeth. Journal of the Society of Occupational Medicine 1990 ; 40 (4) : 149-152.
20. Carlo.P et al. Pink teeth in a series of bodies recovered from a single shipwreck. The American Journal of Forensic Medicine and Pathology 2006; 27 (4): 313-316.
21. Schwartz T R et al. Characterization of deoxyribonucleic acid (DNA) obtained from teeth subjected to various environmental conditions. J Forensic Sci 1991; 36: 979-990.

22. Sweet D, Hildebrand D. Recovery of DNA from human teeth by cryogenic grinding. J Forensic Sci 1998; 43: 1199-1202.
23. Pretty I A, Webb D A, Sweet D. The design and assessment of mock mass disasters for dental personnel. J Forensic Sci 2001; 46: 74-79.
24. Woodward J D. Identification of victims following a mass disaster. Ky Dent J 1982; 34: 37-41.
25. Thali M.J et al.Dental CT imaging as a screening tool for dental profiling: advantages and limitations. J Forensic Sci. 2006 Jan;51(1): 113-139.

Aluminum Phosphide Poisoning: An Agent of Sure Death

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ABSTRACT

Aluminum phosphide as a poison is used for deliberate self-poisoning by inhalation/ingestion route especially in North India .The poison effects all systems of the body with predominantly CVS and GIT. Till Date no Antidote is available although man is claiming his conquer over the life and death. Medical critics still say if a patient with suspected aluminum phosphide(ALP) poisoning is saved in a hospital then either that was not ALP or the poison was exposed one .This poison which is reputed in medical field as "An Agent of Sure Death" has been forgotten by the medical world and not much of treatment is available for ALP poisoning though Magnesium sulfate is well documented in reducing the cardiac arrhythmias and the mortality, but the problem needs multifaceted approach in the form of preventive measures, restrictions on supply of ALP in market, updating the management techniques and overall a search for an effective antidote.

INTRODUCTION

Aluminum phosphide(ALP) is a solid fumigant poison. The first case of poisoning was reported in India in 1981 and thereafter the number of cases is increasing throughout the North India.[1-3]

Aluminum phosphide is marketed as Alphos/celphos, Commonly known as "The Wheat Pill" and labeled as "An Agent of Sure Death" is available as dirty white/gray colored 3g tablet/10g powder pack.

Chemical composition

It contains Aluminum phosphide(56%), ammonium carbonate (44%) which is inert, & paraffin, and has a capacity to liberate 1g phosphine(PH_3) gas on exposure[4,5] i.e.

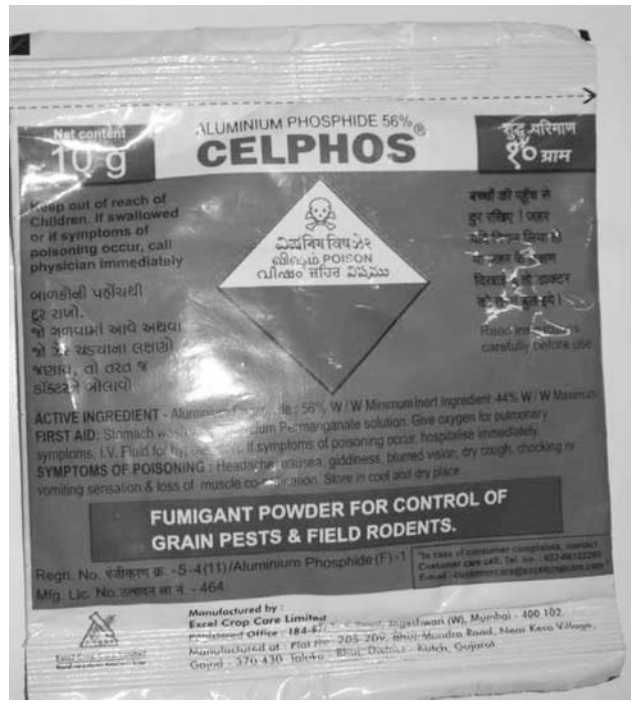
$ALP + H_2O \rightarrow Al(OH)_3 + PH_3 \uparrow$(with Moisture).

$ALP + HCl \rightarrow AlCl_3 + PH_3 \uparrow$(with HCl in Stomach).

Phosphine is a colorless gas with fishy/garlic odor with vapor density of 1.17[6,7]. The non-toxic residues, phosphates & hypophosphates of aluminum, left in grains are less than 0.1mg/kg of phosphine, the WHO/FAO recommended permissible levels for human consumption[4,5].

Pathophysiology

Irrespective of the routes of exposure, the toxic effects of PH_3 are same and dose dependent. Some of the aluminum phosphide is directly absorbed from liver, liberates PH_3 slowly prolonging the toxic effects of the poisoning[4].



Aluminium phosphide 56% powder available in the market

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Phosphine leads to the non-competitive inhibition of cytochrome oxidase causing cellular hypoxia and small vessel injury, further potentiated by cardiovascular damage due to anoxic myocardial damage and shock[8].

It damages the cell membrane and enzymes important for respiratory metabolism e.g. decreases the catalase and increases superoxide dismutase(SOD) and Malanoyl dealdehydase(MDA)[9].

Hypomagnesaemia brought on by focal myocardial damage leads to arrhythmias.

Hypotension and shock ensue within 3-6hours of ingestion of ALP.

PH_3 inhaled reacts with moisture in the lungs forming phosphoric acid causing blistering and edema leading to ARDS[10].

Denaturing of oxyhemoglobin decreases oxygen delivery to the tissues.

Local trauma to the gastric tissues can cause gastritis. Toxic chemical myocarditis leads to varied fatal ECG changes 6-24hours after ingestion of ALP in non-survivors in the form of VPC, conduction disturbances, LBBB/RBBB, Ventricular fibrillation, idio-ventricular rhythm leading to asystole .Death in first 24 hours appears to be cardiogenic as enhanced by shock and ECG abnormalities[11-14].

Widespread capillary damage leads to bleeding diathesis, DIC, ATN. Shock and DIC lead to terminal liver failure[15].

Direct toxic effects of ALP and its absorbed products or metabolic acidosis on muscles cause muscular ischemia and degeneration of non-specific nature leading to myopathy[16].

The occurrence of intravascular hemolysis with ALP in patients with normal

A G6PD level is of significance as jaundice in patients with poisoning is often attributed to the hepatic damage alone[17].

Direct toxic effects on adrenal cortex accompanied by decreased cortisol levels, leads to shock, high mortality and hypo/hyperglycemia[18-19].

In addition to effects on adrenals, ALP also effects glucose metabolism leading to hypo/hyperglycemia or no change effects which can be attributed to the wide variety of changes in Mg2+, Ca2+, phosphate, citrate and cortisol levels which acts as active stimulatory/ inhibitory modulators to enzymes/hormones that catalyze and regulate glucose metabolism[20].

Epidemiology

Incidence of ALP poisoning was 0.06/1000 hospital admissions which progressively increased to 10/1000 in 1989-90 with male: female ratio of 2:1 and now is continuously increasing so as to surpass any other poisoning in North India and particularly in Haryana, Punjab and J&K. ALP poisoning is more common in rural areas of North India where agricultural community irrespective of sex is more at risk which correlates with the easy availability of ALP in household [1,2,3,21].

Fatal dose is 150mg for a 70 Kg man and fatal period is 1 to 96 hours average being 24hours[1,2,3].

Clinical features

Depend on the dose and severity of the poisoning.

1. **Non-specific features:** nausea, vomiting, headache, abdominal pain, etc.

2. **Specific features:** involves the following systems.

   - **GIT:** In 100% patients. Manifests as nausea, vomiting, diarrhea, in 20% patients. Pain epigastrium, epigastric burning sensation in 60%patients.
   - **Hepatobiliary system:** In 20-30% patients. Jaundice, tender hepatomegaly, raised trans-aminases(SGOT/SGPT). Acute hepatic failure is seen in 6% patients[22].
   - **CVS:** In 60-100% cases. Hypotension, bradycardia, arrhythmias, myocarditis, pericarditis, raised JVP, CCF, Cardiac arrest[23,24] are the main manifestations.
   - Respiratory system: manifestations seen within first 2-3hours in 50-60% cases in the form of cough, dyspnea, crackles, ronchi, chest tightness, ARDS, and respiratory failure[10].
   - **Renal:** In 5-10% cases in the form of proteinuria, hematuria, acute renal failure.
   - **CNS:** 50% patients have CNS involvement presenting as headache, dizziness, diplopia, paraesthesias, ataxia, altered sensorium and hypoxic encephalopathy, delayed hemorrhagic stroke[11,25].
   - **Locomotor system:** myopathies, proximal muscle weakness are the main manifestations[11,25].
   - Haematopoietic system: bleeding diathesis, DIC and shock[15,17].
   - **Endocrine system:** hyperglycemia, hypoglycemia.

- **Electrolytes:** Hypomagnesaemia, hypocalcaemia and metabolic acidosis.
- **Bad prognostic signs in ALP poisoning:** Include intractable shock, anemia, hemolysis, DIC, electrolyte disturbances, metabolic acidosis, severe hypoxia, coma, severe chest infections, aspiration pneumonia, oliguria, arrhythmias[3,26,27].

Lab diagnosis

1. **Routine investigations:**

   Hemogram: leucopoenia

   Routine examination urine: hematuria and proteinuria

   ECG changes: present in about 80% cases. ST-T changes seen in 40% cases as ST depression, ST elevation/coving, hyper-acute T-waves, multifocal resistant ectopic beats, tachycardia, bradycardia, atrial fibrillation(AF), aberrant conduction, LBBB/ RBBB, asystole[13].

   ABG: Hypoxia, hypercapnoea/eucapnoea, decreased bicarbonate levels, metabolic acidosis.

   X-ray chest: normal except in ARDS patients.

   Blood biochemistry: Increased transaminases (10-12 times), decreased serum choline esterase activity, increased plasma rennin activity, deranged renal function tests(RFT) in cases of severe renal failure. Increased serum amylases and CPK(MM) is seen in myopathy[24,29].

   Echocardiography: marked LV systolic dysfunction with mean EF= 43.52+4.97% on day 1 of poisoning[24,29].

2. **Specific investigations:**

   Silver nitrate impregnated filter paper test: Based on the property of phosphine to reduce AgNO3 to silver giving black color on filter paper. It has a high sensitivity and is positive even at low concentrations and even on day 2 or 3of poisoning in survivors. It can be done with gastric contents/ breath.

   Gastric contents: take 5ml of gastric contents with 15ml of water in flask, cover the mouth of the flask with filter paper impregnated with 0.1N $AgNO_3$, heat for 15-20 minutes, blackening of the filter paper indicates the presence of PH_3. The test is 100% positive with gastric juice/ lavage fluid.

   Breath test: filter paper impregnated with 0.1N $AgNO_3$ is used in the form of face mask, and the patient is asked to breathe in and out for 15-20 minutes, blackening of the filter paper indicates the presence of PH_3. The test is positive in 50% patients.

   Management of Aluminum phosphide poisoning:

   No specific antidote is available till date[32,33].

General measures

- Maintain ABC i.e. Airway, Breathing and Circulation. Secure two i.v lines with wide bore cannulae.
- Measures to decrease the absorption of Phosphine gas: Gastric lavage with $KMNO_4$(1:10,000) repeated 3-4 times or till the $AgNO_3$ filter paper test is negative. It removes unabsorbed poison as well as oxidizes it.

  Slurry of Activated Charcoal 1mg/Kg weight (60-90 mg) through the nasogastric tube is given which absorbs PH_3 from GIT.

  Liquid paraffin, vegetable oil/ mineral oil accelerate excretion of ALP and PH_3 from GIT and inhibit the release of PH_3 from ALP[31].

  Reduction of the organ toxicity can be achieved by membrane stabilizing agents as $MgSO_4$[32].
- Measures to increase the excretion of PH3:

  Fats/mineral oil.

  Adequate hydration with 2-3L of intravenous fluids, at least 50% should be Normal Saline.

  When systolic blood pressure(SBP) is more than 90 mmHg, 40-60 mg of furosemide can be tried.

  For a hemodynamically stable patient, consider hemodialysis although not much data is available on this.

  Dopamine @ 4-6mcg/kg can be tried to increase the renal perfusion.
- Treatment of hypoxia:

  Keep airways patent.

  Oxygen inhalation through facemask @5-10 l/min 100% oxygen to achieve a PaO2 of 60-70% with low FiO2.

  Endotracheal tube should be considered for the unconscious patients.
- Treatment of shock:

  IV fluids with 3-4 l of fluids, at least 50% should be normal saline.

  Dopamine @6mcg/kg with dobutamine to keep SBP>100 mmHg.

  IV Hydrocortisone 200-400mg every 4-6hours decreases the dose of dopamine, checks the capillary leakage in lungs, potentiates the response of shock to endogenous catecholamines, and compensates for the low levels of the cortisone found in cases with severe poisoning.
- Treatment of arrhythmias:

  $MgSO_4$ due to its membrane stabilizing action is effective against the arrhythmias in first 24 hours in a dose of 1g i.v. stat followed by 1-1.5g every 6 hours for 5-7days as IV infusion in D5% to maintain Mg2+ levels between 3.4-4.6mEq/l.

Other regimen given is 3g $MgSO_4$ in 500ml of D5% in first 3hours followed by 6g in 500ml D5% over 24hours starting the next day and continuing for 3-5 days.

$MgSO_4$ modulates the sympathetic, parasympathetic, and slow channel kinetics[32-38], thus prevents the arrhythmias. Goal is to keep the levels below 10mEq/l (the toxic levels).

Some workers advocate the role of rapid digitalization with IV digoxin in failing heart.

Conventional anti-arrhythmics have no role.

- Treatment of metabolic acidosis:

  IV sodium bicarbonate 100mEq in 1L Normal saline every 8 hours to keep the bicarbonate levels between 18-20 mEq/l and pH>7.1.

  If acidosis persists in a hemodynamically stable patient, consider haemodialysis.

- Treatment of ARDS:
- 100% oxygen through the facemask@ 5-10 l/min to achieve a PaO2 of 60-70% with low FiO2.

  Complications viz. pericarditis/myocarditis, acute massive UGI bleed require appropriate treatment[39].

  Mortality ranges from 50-90% depending on factors as dose consumed, freshness of the compound, arrival at the hospital, duration and severity of the shock, presence/absence of the complications, delay in therapy.

  Mortality with supportive measures alone is 70-100% while with supportive care and $MgSO_4$ is 25-45%[33,40,41].

Recent advances in the treatment of ALP poisoning:

A trial with N-acetyl-cysteine(NAC) at a dose of 6.25mg/kg/min for 30minutes had shown a significant hemodynamic, biochemical changes. Pre and post treatment of NAC with ALP significantly improved the survival time, stabilizes the heart rate, blood pressure, and ECG; decreased the MDA and increased Glutathione reductase levels which was due to reduction in the myocardial oxidative injury[42,43].

The L-nitroarginine-methyl-ester(L-NAME) at 1mg/kg/min showed significant improvement in BP but precipated the ECG abnormalities and was thus less effective than NAC[42,43].

REFERENCES

1. Bajaj R,et al. Epidemiology of alphos poisoning. Need for a survey. JAPI 1990, 30:3: 197-198.
2. Chugh SN, et al. Incidence and out come of ALP poisoning in a hospital study. Ind J Med Res 1992; 94: 232-235.
3. Siwach S.B, et al. Acute aluminum phosphide poisoning. An epidemiological, clinical and histopathological study. JAPI 1988; 36: 594-596.
4. Chugh SN et al. Aluminum phosphide poisoning present status and management. An update. JAPI 1992; 40: 401-405.
5. Singh S,et al. aluminum phosphide ingestion. Br Med J 1985; 290: 1110-1111.
6. National Occupational Hazard Survey Publication(NIOCH). US Department of Health Education and Welfare 1977; 3: 78-114.
7. Jefferey L.,et al. "Fumigant related illness, Washington State's five years experience". Clin toxicol 2000; 38 (1): 7-14.
8. Chelurka W, et al. The effect of phosphine on electron transport in mitochondria. Pestic Biochem Physiol 1976; 6: 65-81.
9. Chugh SN,et al. Free radical scavengers and Lipid peroxidation in acute aluminum phosphide poisoning. Ind J Med Res 1996; 104: 190-193.
10. Sant Ram et al. Adult respiratory distress syndrome following aluminum phosphide poisoning. JAPI 1989; 37: 271.
11. Chugh SN, et al. significance of heart rate response in shock due to aluminum phosphide poisoning. JAPI 1989; 37: 708-709.
12. Jain SM,et al. electrocardiographic changes in aluminum phosphide poisoning. JAPI 1985; 33: 406-409.
13. Katira R, et al. A study of aluminum phosphide poisoning with special reference to electro-cardiographic changes. JAPI 1990: 38: 471-474.
14. Juggal K.L, et al. Hypomagnesaemia and Atrial fibrillation in a case of aluminum phosphide poisoning. JAPI 1990; 38: 376-77.
15. Mehta LK,et al. Aluminum phosphide poisoning: two cases with rare presentation. JAPI 1990: 38: 509-510.
16. Nityanand,et al. Muscle involvement in aluminum phosphide poisoning. JAPI 1988; 36: 289-290.
17. Aggarwal P, et al. intravascular hemolysis in aluminum phosphide poisoning. American Journal of Emergency Medicine 1999; 17(5): 488-489.
18. Khosla SN,et al. Systemic involvement in aluminum phosphide poisoning. JAPI 1986; 34: 227-230.
19. Sant Ram et al. Adreno-cortical involvement in aluminum phosphide poisoning. Ind J Med Res 1989; 90: 289-294.
20. Abder Rahman H. Effect of aluminum phosphide on blood glucose levels. Vet Hum Toxicol 1999; 41(1): 31-32.
21. S.B. Siwach, et al. The profile of acute poisoning in Haryana-Rohtak Study. JAPI 1995; 11: 756-779.
22. Chittra MD, et al. Acute Hepatic Failure in

aluminum phosphide poisoning(letter). JAPI 1994; 42(11): 924.
23. Ictali B, et al. Acute phosphotoxin poisoning. Press Med 1995; 124(13): 611-612.
24. Gupta MS,et al. Cardiovascular manifestations in aluminum phosphide poisoning with special reference to Echocardiographic changes. JAPI 1995; 43(11): 779-780.
25. Dave HH, et al. Delayed hemorrhagic Stroke following accidental aluminum phosphide ingestion. JAPI 1994; 42(1): 78-79.
26. Chugh SN, et al. aluminum phosphide poisoning: Analysis of 226 cases (abstract). JAPI 1989; 37: 28.
27. Chopra JS,et al. aluminum phosphide poisoning a prospective of 16 cases in one year. Post-grad Med J 1986; 62: 1113-1115.
28. Singhal HR, et al. Plasma Rennin Activity in shock due to aluminum phosphide poisoning. JAPI 1990; 38(6): 398-400.
29. Mittal HS et al. Echocardiographic study in aluminum phosphide poisoning (abstract) JAPI 1991; 39: 851.
30. Chugh K, et. al. Spot Diagnosis of aluminum phosphide ingestion .An application of simple test. JAPI 1989; 37: 219-220.
31. Goswami M, et al. Fat and Oil inhibit phosphine release from aluminum phosphide its clinical implications. Ind Exp Biol 1994; 32(a): 467-469.
32. Prem Kumar, et al. Magnesium sulfate therapy in acute aluminum phosphide poisoning JAPI 1994; 42: 373.
33. Chugh SN, et al. Magnesium status and parenteral Magnesium Sulfate therapy in acute aluminum phosphide poisoning. Magnesium Research 1994; 7: 289-294.
34. Chugh SN, et al. Is there any residual effect or sequel of phosphine (abstract). JAPI 1991; 39: 87.
35. Mahajan SK, et al. Efficacy of magnesium sulfate in aluminum phosphide poisoning- comparison of two different dose schedules. JAPI 1994; 42(5): 373-375.
36. Siwach SB, et al. Serum tissue magnesium content in patients of aluminum phosphide poisoning and critical evaluation of high dose magnesium sulfate therapy in reducing mortality (see comments). JAPI 1994; 42(2): 373-375.
37. Ram A,et al. a study of aluminum phosphide poisoning with special reference to therapeutic efficacy of magnesium sulfate. JAPI 1988; 36: 23.
38. Chugh SN, et al. efficacy of magnesium sulfate therapy in acute aluminum phosphide poisoning. JAPI 1991; 39(11): 850.
39. Chugh SN, et al. Acute pericarditis in aluminum phosphide poisoning(Abstract). JAPI 1991; 11: 851.
40. Kolley T et al.A critical evaluation of anti-peroxidant effect of intravenous magnesium in acute aluminum phosphide poisoning. Magnesium Research 1997; 10(3): 225-230.
41. Chugh SN, et al. Successful reversion of supra-ventricular and ventricular tachycardia with magnesium sulfate in aluminum phosphide poisoning. JAPI 1991; 39: 642.
42. Zhang H,et al. Protective effect of NAC in Endotoxemia. Am J Physiol 1994; 266: H1746-H1754.
43 Archana Azad, et al. Effect of N-Acetyl Cysteine (NAC) and L-Nitro Arginine Methyl Ester (L-NAME) on ALP induced cardiovascular toxicity in rats. Acta Pharmacol Sin 2001; 22(4): 298-304.

Meckel Gruber Syndrome- A Case Report

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ABSTRACT

Meckel-Gruber syndrome (MKS) is a rare congenital polymalformative syndrome with an autosomal recessive mode of inheritance. It is characterized by a classic triad of renal cystic disease, CNS malformation and polydactyly. The outcome of the syndrome is usually lethal. Prenatal diagnosis of the syndrome can be made by ultrasound examination between 11-14 weeks of gestation. Termination of the pregnancy can be offered if the diagnosis is made before viability. The parents have to be counseled regarding the chances of recurrence in the subsequent pregnancy. We report a case of a stillborn male fetus with features compatible with Meckel-Gruber syndrome.

Key words : *Meckel-Gruber Syndrome, Encephalocele, Cystic Renal Dysplasia.*

INTRODUCTION

Meckel-Gruber syndrome (MKS) was first described in 1822 by Meckel[1] and later in 1934 by Gruber[2]. It is a rare congenital polymalformative syndrome with an autosomal recessive mode of inheritance and the postnatal outcome of affected fetuses is invariably lethal.[3]

It is characterized by anomalies of the central nervous system resulting in mental retardation, cystic dysplasia of the kidneys, and malformations of the hands and feet. Children with MKS may also demonstrate cleft palate, ductal plate malformation of the liver, cardiac abnormalities and incomplete development of external or internal genitalia. The phenotypic manifestations vary among cases; however, patients with MKS invariably die from it, primarily of lung and renal failure.[4]

CASE REPORT

We report a case of a 21 year old lady with G3P2L0, with 34 weeks of gestation presenting with inability to appreciate fetal movements. Both the previous pregnancies had resulted in stillbirths. Ultrasonographic examination showed a single intrauterine dead fetus with multiple anomalies showing encephalocele and cystically dilated kidneys with oligohydramnios. Labour was induced and the fetus was sent for autopsy. On external examination an encephalocele (figure 1) in the occipital region and cleft palate were noted. No anomalies of digits were made out. Internal examination showed bilateral enlarged kidneys with multiple cysts in cortex and medulla (figure 2). The liver was enlarged with homogenous appearance on cut section. Histopathological examination showed bilateral cystic renal dysplasia (figure 3) and ductal plate malformations in liver (figure 4). The constellation of anomalies was compatible with the diagnosis of Meckel-Gruber syndrome. The parents were counseled about the risk of recurrence in the future pregnancies.

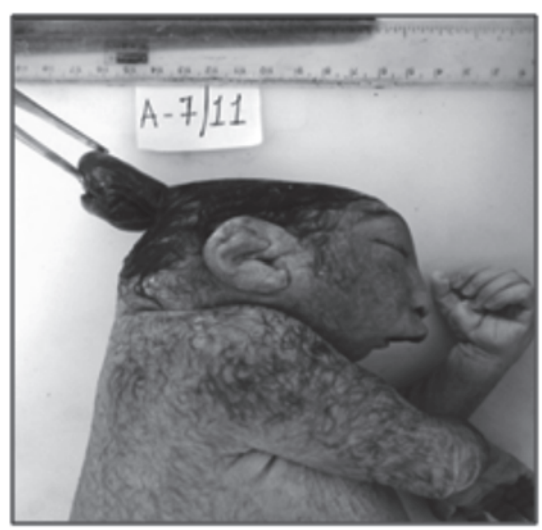

Fig. 1. Encephalocele

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Fig. 2. Bilateral polycystic kidneys.

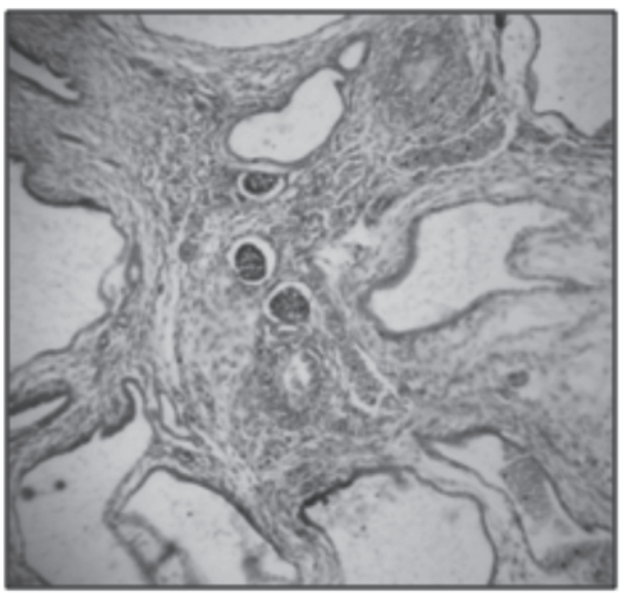

Fig. 3. Cystic renal dysplasia, (H&E, x100).

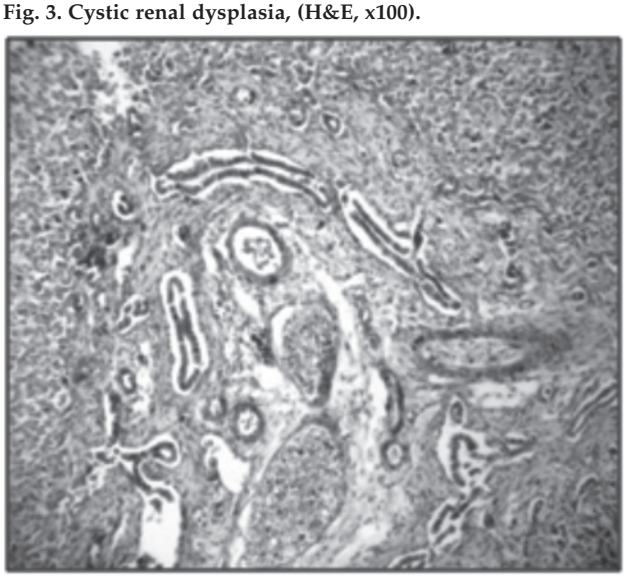

Fig. 4. Ductal plate malformations of Liver. (H&E, x100).

DISCUSSION

The worldwide incidence of MKS varies from 1 in 13,250 to 1 in 140,000 live births. There is a predilection for Belgian (1/3000) and Finish (1/9000) populations.[5] The disease affects males and females in equal numbers. Once diagnosed in reproductive couples, the chances of recurrence in subsequent pregnancies is 1 in 4 (25% chance) for each pregnancy.[4]

The minimum diagnostic criteria for the diagnosis of Meckel-Gruber syndrome have been variously defined. Hsia et al[6] and Mecke et al[7] suggested that at least two of the three criteria constituting the classic triad of occipital encephalocele, polydactyly, and cystic kidneys should be present. Sergi et al[8] reported cystic renal dysplasia, occipital encephalocele, and postaxial polydactyly in 100%, 90%, and 83.3% of the fetuses, respectively. However, Fitch and Pinsky[9] opined such criteria to be too restrictive. Salonen R[10] reviewed the clinico-pathologic ?ndings in 67 cases in Finland, where the disorder is unusually frequent. She proposed that bilateral multicystic kidneys, ?brotic changes (ductal plate malformation) in the liver, and occipital encephalocele or some other central nervous system malformation are minimal diagnostic criteria.

Table.1. Manifestations of Meckel- Gruber syndrome[4]

System	Malformations
Central nervous system	Occipital encephalocele, Dandy-Walker malformation Others: microcephaly, holoprosencephaly, cerebral and/or cerebellar hypoplasia, anencephaly
Genitourinary system	Cystic renal dysplasia, External genital ambiguity in males Others: renal hypoplasia/ aplasia, horse- shoe kidneys, double ureter
Hepatobiliary system	Ductal plate malformation of the liver
Skeletal system	Postaxial polydactyly Others: club feet, short limbs
Others	Cleft lip/palate, micrognathia, microphthalmia, nasal anomalies, sloping forehead, heart malformations

The identification of the disease causing genes has enabled the DNA based diagnostics for MKS. Five genes and six loci have been identified in MKS at the time of writing this review and the number will evidently increase since these genes explain only a fraction of known MKS cases. Analysis of polymorphic DNA markers from Finish families revealed the MKS locus to be on chromosome bands 17q21-q24, telomeric to the homeobox B region.[11] A second locus (MKS2) has been mapped to band 11q13 in a subset of Middle

Eastern and Northern African families, demonstrating the clinical and genetic heterogeneity of MKS.[12] A third locus (MKS3) has been localized to chromosome 8q24 by a genome-wide linkage search using autozygosity mapping.[13] Comparison of the clinical features of MKS3 linked cases with those of MKS1 and MKS2 linked kindred suggests that polydactyly appears less commonly in MKS3 linked families.

The diagnosis of MKS can be made prenatally during routine ultrasonographic screening for fetal chromosomal abnormalities at 11 to 14 weeks of gestation.[14] Ickowicz et al[3] reported CNS anomalies in all their cases and found encephalocele as the most common anomaly seen in 88% of cases. On ultrasound examination, a liver lesion could be visualized in only one of their patients, conforming that hepatic anomalies can only be ascertained on pathology.

The differential diagnosis should include trisomy 13, Zellweger syndrome, Agostino syndrome and Jeune syndrome. In each of these syndromes, complementary investigations, in particular morphological, karyotypic and genetic analyses are necessary to help differentiate them from MKS. In all these conditions, amniotic fluid volume generally remains normal. This is in contrast to MKS, in which oligohydramnios occurs earlier in the pregnancy. Autosomal recessive polycystic disease is another differential diagnosis to be discussed, but medullary lesions are very unusual, especially during the ?rst trimester.[3] Meckel-Gruber syndrome is a lethal disorder. The mortality is 100%. Most infants are stillborn or die hours or days after birth. When MKS is suspected, a karyotype study should be obtained to exclude chromosomal disorder (trisomy 13). If the diagnosis is made before viability, termination can be offered.[4]

REFERENCES

1. Meckel JF. Beschreibung zweier, durch sehr aehnliche. Bildungsabweichungen entsellter Geschwister Dutsch. Arch Physiol 1822; 7: 99-172.
2. Gruber GB. Beitraege zur frage "gekoppelter" missbildungen (akrocephalo-syndactylieund dysencephalia splanchnocystica).Beitr Path Anat 1934; 93: 459-476.
3. Icowicz V, Eurin D, Laulom BM, Didier F, Garel C, Gubler MC et al.Meckel-Gruber syndrome : sonography and pathology.Ultrasound Obstet Gynecol 2006; 27 : 296-300.
4. Alexiev BA, Lin X, Sun CC, Brenner DS. Meckel-Gruber Syndrome.Pathologic Manifestations, Minimal Diagnostic Criteria, and Differential Diagnosis Arch Pathol Lab Med. 2006; 130: 1236-1238.
5. Salonen R, Norio R. The Meckel syndrome in Finland: epidemiologic and genetic aspects. Am J Med Genet. 1984; 18:691-698.
6. Hsia YE, Bratu M, Herbordt A. Genetics of the Meckel syndrome (dysencephalia splanchnocystica). Pediatrics 1971; 48: 237-247.
7. Mecke S, Passarge E. Encephalocele, polycystic kidneysand polydactyly as an autosomal recessive trait simulating certain other disorders: the Meckel syndrome. Ann Genet (ParisJ1971; 14: 97-103.
8. Sergi C, Adam S, Kahl P, Otto HF. Study of the malformation of the ductal plate of the liver in Meckel syndrome and review of other syndromes presenting with this anomaly. Pediatr Dev Pathol. 2000; 3: 568-583.
9. Fitch N,Pinsky L.The Meckel syndrome with limited Expression in relatives.Clin Genet 1973; 4: 33-47.
10. Salonen R. The Meckel syndrome: clinico-pathological findings in 67 patients. Am J Med Genet. 1984; 18: 671-689.
11. Paavola P, Salonen R, Weissenbach J, Peltonen L. The locus for Meckel syndrome with multiple congenital anomalies maps to chromosome 17q21-24 [letter]. Nature Genet. 1995; 11: 213-215.
12. Paavola P, Salonen R, Baumer A et al. Clinical and genetic heterogeneity in Meckel syndrome. Hum Genet. 1997; 101: 88-92.
13. Morgan NV, Gissen P, Sharif SM, et al. A novel locus for Meckel-Gruber syndrome, MKS3, maps to chromosome 8q24. Hum Genet. 2002; 111: 456-461.
14. Sepulveda W, Sebire NJ, Souka A, Snijders RJ, Nicolaides KH. Diagnosis of the Meckel-Gruber syndrome at eleven to fourteen weeks' gestation. Am J Obstet Gynecol. 1997; 176: 316-319.

Clinical Profile and Outcome of Organophosphorus Poisoning at Tertiary Care Centre in Western Maharashtra

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ABSTRACT

Background: Organophosphorus (OP) compounds are the most common suicidal poison in developing countries and mortality is high. In Maharashtra state, India the widespread use of organophosphates (OPs) in agricultural and household environments results in numerous OP poisoning.

Material & Methods: Total 97 patients admitted in medical intensive care unit between January 2010 to December 2010 at Krishna Institute of medical sciences Karad, were included in this retrospective study.

Results: Out of total 97 patients with OP poisoning 57 (58.76%) were males and 40 (41.23%) were females. Patients those who succumbed to death had mean time of consumption of OP and institution of treatment was >6 hours which was high compared to those who survived ('p'<0.01). Total 56 (57.73%) patients were required ventilatory support of which 36 (63.15%) were for >7 days and 20 (36.85%) for <7 days. Acute cholinergic crisis was present in 41 (42.26%) patients and intermediate syndrome was present in 34 (35.05%) patients. Dimethoate (12/9) had highest mortality compared to other OPs with 'p' <0.001. Total 12(21.05%) male and 5 (12.5%) female patients developed ventilator associated pneumonia with ARDS. Total 10 (16. 94%) patients have developed central venous catheter related infection. Delayed peripheral predominant motar neuropathy was seen in 2 (2.06%) patients.

Conclusions: There was significantly high mortality in moderate and severe type of OP poisoning (15.46%). The low mean serum cholinesterase level was significantly correlated with delayed admission and institution of treatment, fasciculation's, acute cholinergic crisis, intermediate syndrome and nosocomial infections. Dimethoate poisoning associated with highest mortality.

Key words : *Organophosphorus (OP) Compounds, Intermediate Syndrome, Acute Cholinergic Crisis, Dichlorovas, Dimethoate.*

INTRODUCTION

Acute organophosphorus pesticide poisoning is widespread in the developing world and its frequency has increased over the years. It is the most common poisoning in many developing countries including India. Organophosphate compounds are a diverse group of chemicals used in both domestic and industrial settings.[1] Organophosphates can be absorbed cutaneously, ingested, inhaled, or injected. Organophosphates may cause serious life-threatening conditions, such as an initial acute cholinergic crisis (ACC) and *intermediate syndrome* (IMs) which potential for respiratory failure requiring ventilatory support. Worldwide mortality studies report mortality rates from 3-25%.[2] In Maharashtra, India the widespread use of organophosphates in agricultural and household environments results in numerous OP poisoning. This study was undertaken to better understand the clinical significance of associated parameters on respiratory failure and patient outcome. So far there is scanty literature and studies about the Organophosphorus poisoning in western Maharashtra.

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MATERIAL & METHODS

This retrospective and observational study was conducted at tertiary care, referral centre, Karad to study various factors affecting outcome, complications and mortality associated with organophosphorus poisoning and to find out relation of the serum cholinesterase levels to the severity of OP poisoning. All patients with alleged history of organophosporous compound consumption with signs and symptoms of OP poisoning of both genders of age more than 18 years were included in this study. This study was approved by ethical committee Krishna Institute of Medical Sciences University karad. The bio-data clinical examination findings and biochemical profile were recorded on standard sheet and analysed by *statistical software SPSS -10 package*.

Patients presenting with organophosphorus poisoning and admitted to the Medical intensive care unit were included in this study. Total 97 patients were included in this study. Out of 97 patients 57 (58.76%) were male and 40 (41.23%) were females. Patients admitted with OP poisoning were classified as mild, moderate and severe poisoning based on the Namba criteria. *Mild* if plasma cholinesterase 20 - 50%, *moderate* if cholinesterase 10 - 20% and *Severe* if cholinesterase <10%.[3] Serum cholinesterase activity was measured by kinetic/ DGKC calorimetric method.[4] Respiratory failure was labeled if SpO_2 < 90% with oxygen supplementation by non invasive means.[3] Diagnosis of VAP was based on clinical and microbiological criteria.

INVESTIGATIONS

Immediately after clinical assessment, blood samples were sent for investigations including haemoglobin level, total count, differential count, blood sugar, blood urea, serum creatinine, serum electrolytes and serum cholinesterase level, arterial blood gas analysis (ABGA), ECG, chest radiograph, blood/CVC catheter tip/ urine/ sputum culture & sensitivity.

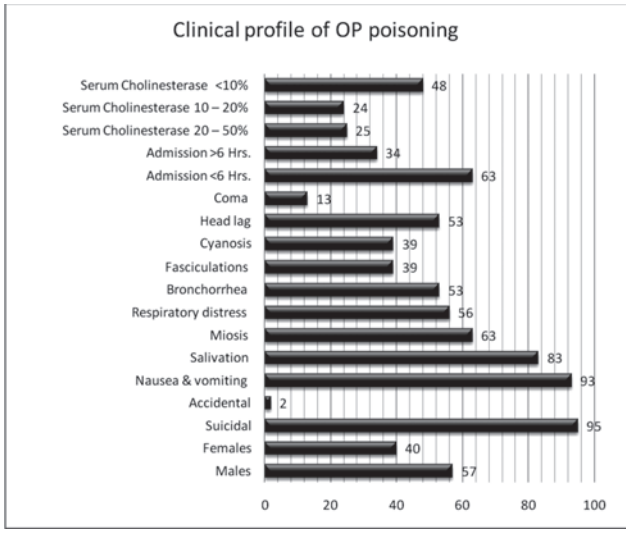
TREATMENT

Routine protocol regarding informing police and medico-legal aspects were followed. Ryle's tube was inserted and first aspirate was preserved for chemical analysis. All patients were given stomach wash. Activated charcoal was given and cathartics were administered as early as possible. Injection PAM, bolus dose intravenous (IV) and Inj. Atropine bolus 5-10 mg IV were given according to standard protocol and requirement. P2AM 12 grams infusion per day for 3-5 days was used for severe cases. Four grams/ day only for 3-5 days was used for moderate cases. Patients were reassessed for respiratory failure, and if so, intubated kept on artificial ventilator. Adult respiratory distress syndrome (ARDS), VAP, hypotension and arrhythmias were all managed as per standard protocol. Patients were assessed every day for cholinergic signs, muscle power (head lag), atropine requirement and level of consciousness.

RESULTS

A total 97 patients were included in this retrospective observational, study with history and findings suggestive of OP poisoning. Out of total 97 patients with OP poisoning 57 (58.76%) were males and 40 (41.23%) were females. The mean and standard deviation for age in male was 27/±9.7 yrs and female was 25/±6.8 yrs. Total 56 patients were consumed OP as a suicide and one were accidental consumption in male. Total 39 female patients consumed OP as suicidal and one was accidental consumption. Mean time of consumption of OP and institution of treatment was 4.5 ± 3 hours. Nausea and vomiting was presenting complaint in 93 (95.87%) patients. Excessive salivation was present in 83 (85.56%) patients. Miosis was present in 63 (64.94%) patients. Respiratory distress was present in 56 (57.73%) patients. Bronchorrhea was present in 53 (54.63%) and fasciculation's were present in 39 (40.20%) patients. Cyanosis was present in 39 (40.20%) and head lag was present in 53 (54.63%) patients. Total 13 (13.40%) were in coma at the time of admission to intensive care unit. Total 63 (64.94%) patients had duration of admission after consumption of OP compound was <6 hours. Total 34 (35.05%) patients had duration of admission after consumption of OP compound was >6 hours. Total 25 (25.77%) patients had serum cholinesterase 20 - 50% (>2000U/L). Total 24 (24.74%) patients had serum cholinesterase 10 - 20% (600 - 1246 units/L). Total 48 (49.48%) patients had serum cholinesterase <10% (60 - 450 units/L). Patients those who succumbed to death had mean time of consumption of OP and institution of treatment was >6 hours which was statistically significantly high compared to those who survived ('p'<0.01). (*Graph 1*).

Out of total 97 patients 48 (49.48%) patients had serum cholinesterase level <1000, 24 (24.74%) had 1000-2000 and 25 (25.77%) had > 2001. Acute cholinergic crisis was present in 41(42.26%) patients and intermediate syndrome was present in 34 (35.05%) patients. Acute cholinergic crisis with intermediate syndrome present in 8 (8.24%) patients. Delayed peripheral neuropathy was present in 2 (2.06%) patients. Delayed peripheral predominant motor neuropathy was recovered over 6-8 weeks. One patient has right sided ulnar nerve palsy and one had medial nerve palsy probably pressure palsy which was recovered over 10-12 days.



Graph 1: Clinical profile of Organophosphorus poisoning.

Total 56 (57.73%) patients were required ventilatory support of which 36 (37.11%) were for >7 days and 20 (20.61%) for <7 days. Mean serum cholinesterase level for those without ventilatory support was 1231±478 U/l which was significantly high compared to those who required ('p'<0.001). Mean serum cholinesterase level for those with ventilatory support for >7 was 375±178 U/l. Mean serum cholinesterase level for those with ventilatory support for <7 was 785±138 U/l. The mean serum cholinesterase level for those with ventilatory support for >7 was significantly low compared to those with <7 days ('p' = 0.02). Ventilator associated pneumonia (VAP) was present in 17 (30.35%) patients (12: male and 5: female). Total 12 male patients developed VAP with ARDS (21.05%). Total 5 (12.5%) female patients developed VAP with ARDS. Total 6 (10.52%) male patients and 1 (2.5%) female patient had UTI. Total 7 (12.28%) male and 3 (7.5%) female patients had developed central venous catheter (CVC) related infection. The overall incidence of CVC infection was 10 (16.94%). Mean serum cholinesterase level was negatively correlated with VAP, UTI and CVC related infection.

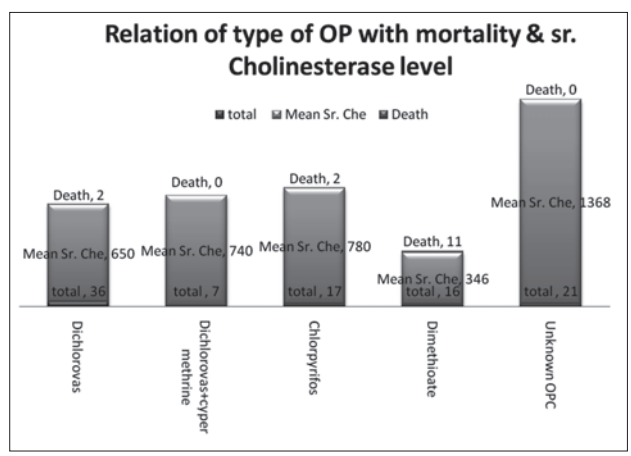
Total 36 (37.11%) patients have consumed dichlorovas with mean serum cholinesterase level 650±270. Total 7 (7.21%) patients have consumed cypermethrine with dichlorovas combination with mean serum cholinesterase level 740±180. Total 17 (17.52%) patients have consumed chorpyrifos with mean serum cholinesterase level 780±130. Total 16 (16.49%) patients have consumed dimethoate with mean serum cholinesterase level 346±198. Total 21 (21.64%) patients have consumed unknown OP compound with mean serum cholinesterase level 1368±345. The mean serum cholinesterase level was lowest in Dimethoate poisoning with mortality of 68.75% (11/16). The mean serum cholinesterase level was significantly low those who succumbed compared to those who survived ('p' =0.02). (Table.1)

Table 1. Type of OPCs and relation to serum cholinesterase level & mortality

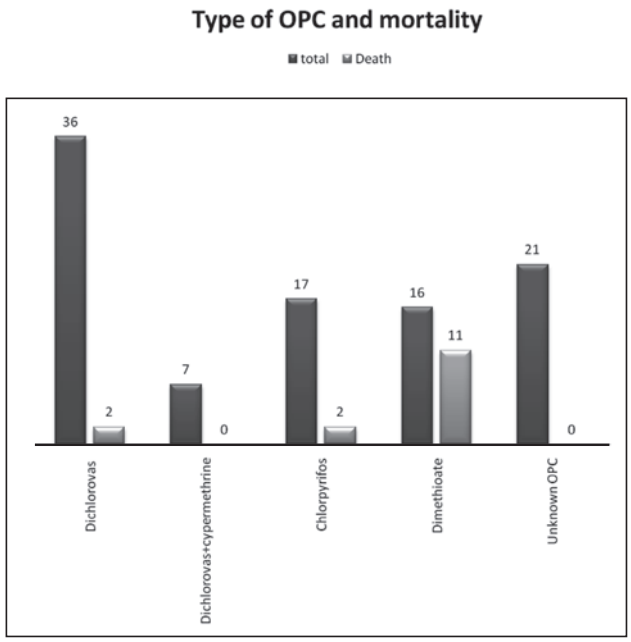
Type of OPC	n=97	Mean Serum cholinesterase (U/L)	Mean atropine (mg.)	Death
Dichlorovas	36 (M:1, F:1)	650±270	123±27	2(8.33%)
Dichlorovas & Cypermethrine	7	740±180	117±19	Nil
Chlorpyrifos	17 (M:2)	780±130	127±19	2(11.76%)
Dimethoate	16 (M:9, F:2)	346±198	321±56	11(68.75%)
Unknown OPC	21	1368±345	97±17	Nil

Dimethoate had highest mortality compared to other OPs. Out of 12 death 9 (75%) were because of dimethoate in male patients. Out of three death two death in female was because of Dimethoate poisoning with 'p' <0.001. Total there were 12 (21.05%) death in male patients and 3 were in female patients (7.5%). Case fatality rate was significantly high in male patients (21.05%) compared to female (7.5%) patients with 'p' <0.001. The mean serum cholinesterase level was lowest with dimethoate (346±198). (Table 1, Graph 2 & 3).

Serum cholinesterase level at the time of admission was negatively correlated with duration of hospital stay, duration of ventilatory support and overall morbidity and mortality with 'p' <0.05. Lower the serum cholinesterase level higher the mortality and morbidity. Time interval between consumption of OP and institution of treatment was significantly more in patients those who succumbed to death with 'p' <0.01. In *multivariate analysis* the low mean serum cholinesterase level was significantly correlated with delayed admission and institution of treatment, fasciculation, acute cholinergic crisis, intermediate syndrome, nosocomial infections.



Graph 2: Relation of type of OPC and mortality



Graph 3: Relation of mortality to serum cholinesterase level

DISCUSSION

Ingestion of OP compounds for suicidal purposes is a major problem, especially in developing countries including India. Organophosphate (OP) insecticides inhibit both cholinesterase and pseudo-cholinesterase activities. The diagnosis of organophosphate poisoning is based essentially on a clinical assessment, followed by laboratory examinations. The primary mechanism of action of organophosphate pesticides is inhibition of carboxyl ester hydrolases, particularly acetylcholinesterase (AChE). In present study patients those who succumbed to death had mean time of consumption of OP and institution of treatment was >6 hours ('p'<0.01). Acute cholinergic crisis (ACC) was present in 41(42.26%) patients and intermediate syndrome (IMs) was present in 34 (35.05%) patients. Acute cholinergic crisis with intermediate syndrome was present in 8 (8.24%) patients. Delayed peripheral neuropathy was present in 2 patients. Total 56 (57.73%) patients were required ventilatory support of which 36 (37.11%) were for >7 days and 20 (20.61%) for <7 days. The mean serum cholinesterase level for those with ventilatory support for >7 was significantly low compared to those with <7 days ('p' = 0.02). Ventilator associated pneumonia (VAP) was present in 17 (30.35%) patients. The mean serum cholinesterase level was lowest in Dimethoate poisoning with mortality of 68.75% (11/16). Overall mortality with OPC was 15.46% (15/97). We have compared our results with various studies.

Paudyal et al[5] in their study found that, the total 75% were with intentional consumption. Intensive care unit was required in 17% of patients and 25% developed complications. Aspiration pneumonia and respiratory failure were the most frequently observed complications with mortality rate of 5%. These findings are comparable with our study. *Noshad et al*[6] in his study stated that majority OP was consumed as a suicide. Ten patients were mechanically ventilated and the mortality rate was 50%. Similarly in our study mortality was highest amongst patients on mechanical ventilator.

Goswamy et. al.[7] in their study found that dyspnea and vomiting were the most common symptom and miosis and cyanosis were the most frequently observed signs. Twenty-seven subjects developed type I respiratory failure and 7 had type II respiratory failure. A miosis, unconsciousness, fasciculation's and plasma cholinesterase levels to predict ventilator requirement and mortality. *Lin CL et al*[8] in their study of 42 patients with OP poisoning show, nausea, vomiting and salivation were the leading manifestations. The dosage of atropine administered for treatment was significantly higher in the patient group with respiratory failure compared to those without respiratory failure ($p<0.05$). These findings are comparable with our results.

Sungur M et. al.[9] in their study of 25 female and 22 male patients found that (68%) were suicide attempts and 15 (32%) were accidental exposure. The mortality rates for the patients who did and did not receive pralidoxime were 32 and 18.7%, respectively. Ten patients (21.2%) required mechanical ventilation. Intermediate syndrome was observed in 9 (19.1%) patients. Complications were observed in 35 (74.4%) patients. These findings are comparable with present study. Similar to our results *Aygun D et al*[10] stated that the serum acetylcholinesterase activity may be a useful parameter in following the acute prognosis of organophosphate poisoning.

Moretto A et. al.[11] in their study of 11 patients with OPP 3 patients developed OP induced delayed neuropathy. In present study we documented two (2.061%) patients with delayed peripheral neuropathy. Similarly *N Nand et. al.*[12] reported cases of organophosphate induced delayed neuropathy. *Cherian et. al.*[13] in their study found that BuChE levels did not correlate with severity of poisoning nor did it correlate with type I or II paralysis, but in present study we found that serum cholinesterase level was well correlated with mortality, outcome, duration of ventilator and nosocomial infection.

Michael et. al.[14] in their study with OPP found that death were significantly higher with dimethoate 23·1% than chlorpyrifos. These findings are similar to our study were highest mortality was with dimethoate (68.75%) compared to dichlorovas and chlorpyrofos.

Güven et. al.[15] stated that fresh frozen plasma therapy increases BuChE levels in patients with organophosphate poisonings. High dose of PAM, $MgSo_4$, obidoxime, plasma transfusion and hemofiltration are used with variable results in recent studies and still many studies are required to prove their benefit in management of organophosphorus compound poisoning.[16, 17]

CONCLUSIONS

Organophosphate compounds poisoning is a lethal condition and needs early diagnosis and appropriate treatment. In patients with respiratory failure the mortality is very high. Total 56 patients required ventilator immediately after admission. The case fatality rate was significantly high with dimethoate and they died sooner than other OPs. Overall mortality in moderate and severe type of OP poisoning was 15.46%. Respiratory paralysis and ARDS requiring ventilatory support was the commonest complication in OP poisoning. Uncommon complication like delayed peripheral neuropathy was also seen in present study. Fasiculation's, headlag, low serum cholinesterase activity at the time of admission was associated with more complications mortality. Early diagnosis, immediate management, early prediction of complications and timely ventilatory support will definitely reduce the morbidity and mortality associated with OPC poisoning. We also recommend to use less toxic insecticides and to keep control over availability of more toxic OP compounds.

REFERENCES

1. Bawaskar HS, Joshi SR. Organophosphorus poisoning in agricultural India: status in 2005. J Assoc Physician India 2005; 53: 422-424.
2. Karalliedde L, Baker D, Marrs TC. Organophosphate-induced intermediate syndrome: aetiology and relationships with myopathy. Toxicol Rev. 2006; 25(1): 1-14.
3. Namba T, Nolte C T, Jackrel J, et al. Poisoning due to organophosphate insecticides. Am J Med 1971; 50: 475-92.
4. Worek F, Mast U, Kiderlen D, et al. Improved determination of acetylcholinesterase activity in human whole blood. Clin Chim Acta 1999; 288: 73-90.
5. Paudyal BP. Poisoning: pattern and profile of admitted cases in a hospital in central Nepal. JNMA J Nepal Med Assoc. 2005; 44(159): 92-96.
6. Noshad H, Ansarin K, Ardalan MR, Ghaffari AR, Safa J, Nezami N. Respiratory failure in organophosphate insecticide poisoning. Saudi Med J. 2007; 28(3): 405-407.
7. Goswamy R, Chaudhuri A, Mahashur AA. Study of respiratory failure in organophosphate and carbamate poisoning. Heart Lung.1994; 23(6): 466-472.
8. Lin CL, Yang CT, Pan KY, Huang CC. Most common intoxication in nephrology ward organophosphate poisoning. Ren Fail. 2004; 26(4): 349-354.
9. Sungur M, Güven M. Intensive care management of organophosphate insecticide poisoning. Crit Care. 2001; 5(4):211-215.
10. Aygun D, Doganay Z, Altintop L, Guven H, Onar M, Deniz T, Sunter T. Serum acetylcholinesterase and prognosis of acute organophosphate poisoning. J Toxicol Clin Toxicol. 2002; 40(7): 903-910.
11. Moretto A, Lotti M. Poisoning by organophosphorus insecticides and sensory neuropathy. J Neurol Neurosurg Psychiatry. 1998; 64(4): 463-468.
12. N Nand, HK Aggarwal, Komal Bharti, D Chakrabarti. Organophosphate Induced Delayed Neuropathy. JAPI. 2007; 55.
13. Cherian MA, Roshini C, Visalakshi J, Jeyaseelan L, Cherian AM. Biochemical and clinical profile after organophosphorus poisoning-a placebo-controlled trial using pralidoxime. J Assoc Physicians India. 2005;53:427-431.
14. Michael Eddleston, Peter Eyer, Franz Worek, Fahim Mohamed, Lalith Senarathna, Ludwig von Meyer. Differences between organophosphorus insecticides in human self-poisoning: a prospective cohort study. The Lancet. 2005; 366: 1452-1459.
15. Güven M, Sungur M, Eser B, Sari I, Altunta? F. The effects of fresh frozen plasma on cholinesterase levels and outcomes in patients with organophosphate poisoning. J Toxicol Clin Toxicol. 2004; 42(5): 617-623.
16. Peter JV, Moran JL, Graham P. Oxime therapy and outcomes in human organophosphate poisoning: an evaluation using meta-analytic techniques. Crit Care Med. 2006; 34(2): 502-510.
17. Yaguang Zhou, Chengye Zhan, Yongsheng Li, Qiang Zhong, Hao Pan, Guangtian Yang. Intravenous lipid emulsions combine extracorporeal blood purification: A novel therapeutic strategy for severe organophosphate poisoning. 2010; 74(2): 309-311.

Current Status of Hair Analysis in Drug Detection

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ABSTRACT

Hair sample, like blood, sputum or urine can act as trustworthy biological specimen for tracing drugs and chemicals. The drugs can be detected regardless of the pharmaceutical dosage form of the drug or it's mode of administration. Hair analysis may be regarded as a convenient technique for tracing the presence of drugs in particular drugs of abuse in a very low nanogram levels in blood and various body parts, even after gap of several months.The hair sample can be collected easily without causing perplexment to the patient. Furthermore, they can be stored and transported easily without refrigeration or use of preservatives. Recent reports from forensic and research laboratories argue the utility of hair analysis in tracing different xenobiotics, opiates, benzodiazepines, doping agents (steroids, diuretics etc) and environmental toxicants. Main factor affecting drug incorporation into hair are-melanin affinity, liphophilicity and alkalinity. Hair analysis can be applied in work laboratory. Furthermore it can be used not only for therapeutic drug monitoring but also for confirming pre-natal drug exposure and drug abuse. In coming future, hair analysis might depict to be promising tool in criminal investigations.

Key words : Hair microtoming, Drug detection, Atomic absorption spectroscopy, Forensic research, Gas / Liquid chromatography.

INTRODUCTION

Hair analysis is fast gaining acceptance as a powerful tool for the detection of drugs in the fields of forensic sciences and clinical toxicology. Hair matrix, like blood, sputum or urine can act as a reliable biological specimen. Therefore hair analysis may be regarded as convenient technique for tracing the presence of drugs(particularly abused drugs)in minute amounts even after a gap of several months. The drugs can be detected regardless of pharmaceutical dosage form of the drug or it's mode of consumption. Hair matrix can provide preliminary clues about the previous exposure of drugs, thus complimenting the information obtained from other biological fluids. Sample[hair] collection is not only a simple process, but also a economical one. The samples can be stored without refrigeration or use of preservatives. The transportation is also not a cumbersome process. Recent reports[1,2] from forensic and research laboratories advocate the utility of hair analysis in tracing different xenobiotics, opiates, benzodiazepines, doping agents, (steroids, diuretics), agricultural toxicants.

Mechanism of Drug incorporation into hair:

Hair consists of mostly proteins, melanin pigments, and lipids. In an fully keratinized hair, all cells are surrounded by a modified cell membrane derived from the original cell membrane, but fused together by intercellular binding material.[3] The scalp hair grows at the rate between 0.3mm to 0.5mm/day. Since, hair is a tissue that is living and growing by supply of nutrients from the blood, it is not surprising that the drugs can be incorporated into growing hairs along with nutrients. The most widely accepted theory for the incorporation of drugs into hair is that the drugs passively diffuses into the hair from the blood supply surrounding the follicle into the dividing matrix cells of the dermal papilla. The drug diffuses passively the systemic circulation into the dividing cells for about 4-5 days, before these cell are keratinized. This process helps in incorporating the drug within internal core of hair fiber. Also, the drug within the systemic circulation may bind to intracellular components like melanin, but this binding contributes very little to the overall concenteration of the drug found in hair.[4,5] The greater challenge to this concept of passive diffusion mechanism is the consistent observation of high drug metabolite ratio found in hair eg high heroin to 6 monoacetyl morphine, high cocaine to benzoylecgonine ratio respectively.[6] This shows that there must be a rapid and selective incorporation mechanism for parent drug, which occurs before it is subjected to its conventional metabolism. It may be that, the parent drug is incorporated and the

production of the metabolite may occur intracellularly with in hair rather than by the incorporation of metabolite from systemic circulation. From this observation, Henderson suggested a multicompartmental model for the entry of drugs into hair.[7] He suggested that drugs could be incorporated into hair from multiple pools at various times, during the life cycle of hair. Finally, the drugs can be incorporated from systemic circulation,sebum, sweat, post-keratinization, but pror to its emergence from scalp and skin. The drug can be accumulated in hair by i) incorporation into hair at follicular level, from vascularisation including both capillary and circulatory contact with hair papilla.ii) entry into hair from contact with sweat or sebum and iii) contact with hair shaft from environmental contaminants such as smoke.

Factors affecting Drug incorporation into hair:

The three key factors affecting drug incorporation into hair are-hair melanin content, liphophilicity and alkalinity of a drug. These factors are crucial for incorporation and retention of drugs in hair.

Hair melanin content:

The melanin, especially eumelanin, plays an important role as a host compound for drug incorporation in hair. It is observed that the pigmented hair possessed a greater capacity to bind and incorporate drugs than non-pigmented hair.thus, the drug concenteration in hair was affected by hair color ie black,brown, blonde, white etc. The drugs like cocaine, LSD and nicotine have been tested for their melanin activity.[8,9] Joseph et al suggested that drugs were incorporated into the hair through ligand binding to specific sites with in protein matrix of the hair , eg cocaine binding in hair was a reversible stereo selective and the binding site for cocaine was melanin.[10] Nakakara et al[11] showed that cocaine had a highest incorporation rate and that their was a huge difference between the incorporation rate of cocaine and that of LSD.They studied incorporation rate into hair of various drugs using gas-chromatography[GC]/mass spectrometry [MS] / liquid chromatography[LC] and found that the drug more affinitive to melanin was cocaine followed by benzphetamine, phencyclidine, methamphetamine and LSD.[11] Zahlsen and Nilsen[12] demonstrated that there was a clear statistically significant difference in the concenteration of nicotine deposited in hair of smokers as compared to non-smokers.[12] Ishyama et al[13] found that methamphetamine bound to same degree to both light and dark hair suggesting that drug binding might be drug specific and for some drugs involved protein binding sites.[13]

Liphophilicity:

Normally the lipid content of hair is high. It was observed that there was a positive correlation between lipophilicity and the drug incorporation into hair. It has been documented that more polar substances like benzoylecgonine/amphetamine entered the hair to a lesser extent than their more liphophilic precursors ,cocaine or methamphetamine.[5,9]

Alkalinity:

The isoelectric point/pH of hair is 3.7 due to acidic nature of melanin. Therefore, the basic drugs were incorporated into hair much faster and in higher concenterations than acidic or neutral drugs.[13] So ,we can safely generalize that non-pigmented hair would be less acidic than black hair due to low melanin content. There are some chemical structural factors[14,15], which affect drug incorporation. Molecular structure of a drug determines its physicochemical properties,[11] which in turn clearly influence the degree of drug incorporation into hair. Chemical mechanisms of the drug transport into hair matrix and the dynamics of drug partitioning with in hair matrix is important for rate of drug incorporation into the hair.[14] For example, accumulation of fentanyl in hair after in vivo administration differed in the nature of storage site from fentanyl accumulation from external solutions.[15] Furthermore, the color and structure of hair also played an important role, e.g. pigmented hair showed higher drug content than blonde/gray/white hair after the same dosage.[16,17] Esterification of hair resulted in significantly less uptake of non ionized fentanyl and significantly lower recovery of drug from hair relative to untreated hair suggesting that carboxylic acid functionalities were necessary for the incorporation of drugs into hair. On the other hand, acetylation of hair resulted in increased removal of fentanyl from methylene chloride solutions and increased recovery of fentanyl from hair samples.[15] Other factors that modified the structure of the hair includes diet, genetic variation and cosmetic treatment of the hair, affecting the degree of drug incorporation into the hair.[17,18]

The basic steps contributing to hair drug analysis in Forensic lab are:

i. collection of hair samples,

ii hair decontamination,

iii sample preparation and derivatization,

iv analysis employing sophisticated instrumentation and

v interpretation and conclusion.

Specimens of hair up to 50 strands (10gms) are cut with surgical scissors at scalp level from each subject usually from the crown of the head. These hair samples

are then placed in small plastic bags, labeled and stored at ambient temperature(15-30°C) till analysis. These hair samples are washed at 37°C for 15 minutes with dry isopropranol to remove greasy contaminants and loosely adhering substances from hair surface. This is followed by three washings (each of 30 minutes duration) with phosphate buffer at 37°C, with shaking at 100rpm. If these washings fail to remove the adherent material, additional washing by phosphate buffer for 1 hr is performed. This is followed by two additional 60 minutes phosphate buffer washes. For washing, various extracting mediums with different incubation media can be used. The inaccessible domain of the hair is extracted by enzymatic dissolution of hair specimens prior to analysis. The melanin bound drug is removed by digestion of hair using centrifuge technique. It is observed that the drug recovery from hair powder obtained with ball mill was higher than obtained from hair snippets due to higher surface in contact with extraction media. Heavy metals were among the first toxic substances, which could be traced in hair matrix by means of atomic absorption spectroscopy[AAS].[19] Hair analysis has its own unique problem in that passive contamination of the exterior surface of the hair can taint the analysis with false positive. Infrared microscopy examined the interior of hair and differentiated passive contamination from drugs absorbed into hair from ingestion. By microtoming the hair either cross-sectionally or laterally, infrared spectra could be obtained of the cortex and medulla of a single hair with a nominal spatial resolution of 10microns.[3] In addition, three dimensional infrared imaging was possible with fourier transform infrared [FT-IR] microscopy, which yielded a plot that could be understood by the layman.[3] GC-MS was a method of choice in general practice and various tandem mass spectrometry methods (GC-MS-MS or LC-MS-MS) were used for target analysis of low dose compounds or for detection of some important metabolites present in trace concenterations in hair.[1] Baumgartener et al 20 extracted opiates from hair of heroin users and analysed the final buffer extract employing radio-immunoassay[RIA]. The concenteration of the drug along the hair shaft corresponded to the time of drug intake.[20] A GC/MS method for analysis of nicotine in scalp hair was developed by Zahlsen and Nilsen[12] was found to be precise, highly sensitive and yielded reproducible results.[12] Goulle and Kintz[21] reported that GC / MS / Liquid chromatography coupled to a diode array detector could be taken as a powerful tool for detecting xenobiotics[21] in human hair. This method could also detect criminal drug exposure, even when blood sample was missing.[21] Ninety doping agents /abused drugs were detectable in human hair using highly sophisticated techniques.[22] Even the minute concenterations of fentanyl /flunitrazepam/ buprenorphine[22] could be detected employing these sophisticated techniques. Hair samples from suspected drug abusers were analysed by radioimmunoassay [RIA] for the cocaine metabolite benzoylecgonine.[23]

Various drugs which can be detected by Hair-analysis includes:

1. Hallucinogens,sedatives and tranquilizers.[2] steroids, minerals and heavy metals.
2. drugs of abuse like alcohol, LSD, opiates, cocaine, amphetamine and benzodiazepines.
3. central analgesics like tramadol.[5] Catha edulisis an herbal euphoric drug chewed by African people.

Advantages of Hair-Drug analysis:

1. It can reveal retrospective information about drug use and the drug addict.
2. It can give clues about quantity and frequency of drug used.
3. It can also detect the drug after long time of exposure(weeks,months)
4. It is an easy technique for analysis.
5. No special storage criteria required.
6. Hair samples are easy to transport.
7. As hair is washed several times prior to analysis, tampering is not possible.
8. Sample collection is easy without any embarrasement to the patient.

Application of Hair-drug-analysis:

1. It can be used in work place.[24]
2. It is effective in identifying prenatal drug exposure.[2,25]
3. its main application is in forensic sciences for investigating criminal cases.[1,26]
4. Hair analysis can serve as a useful aid in clinical diagnosis and toxicology.[21,22]
5. Hair analysis is a powerful tool for confirming drug abuse particularly morphine ,LSD and cocaine abuse.[9,11,23]
6. It can easily detect chronic excessive alcohol consumption.[27]
7. It is useful for therapeutic monitoring of plasma levels in patients undergoing chronic treatment.[28-30]

CONCLUSION

In foreseeable future, this novel technique of tracing drugs through hair analysis holds promise and might turn to be a powerful tool in the hands of a competent analyst finding wide applications in clinical diagnosis ,therapeutic drug monitoring, toxicology and criminal investigations.

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Conflict of interest: Nil

REFERENCES

1. Kintz P.Value of hair analysis in postmortem toxicology.Forensic Sci.Int 2004; 142: 127-134.
2. Vinner E,Vignau j, Thibault D, Codaccioni X, Brassart C, Humbert L,et al,.Hair analysisnof opiates in mothers and new borns for evaluating opiate exposure during pregnancy. Forensic Sci Int 2003; 133: 57-62.
3. Kalasinsky KS, Magluilo j Jr, Schaefer T.Hair analysis by infrared microscopy for drugs of abuse.Forensic Sci Int 1993; 62: 253-260.
4. Potsch l, Skopp g, Moeller MR,. Influence of pigmentation on the codeine content of hair fibres in guinea pigs.j Forensic Sci 1997; 42: 1095-1098.
5. Harrison WH, Gary RM, Solomon LM. Incorporation of D-amphetamine into pigmented guinea pig hair.Br J Dermatol 1994; 91: 415-418.
6. Cone EJ, Yousefnejad d,Darwin WD, Maguire T. Testing human hair for drugs of abuse. II. Identification of unique cocaine metabolites in hair of drug abusers and evaluation of decontamination procedures. J Anal Toxicol 1991; 15: 250-255.
7. Henderson GL. Mechanisms of drug incorporation into hair. Forensic Sci Int 1993; 63: 19-29.
8. Joseph Jr RE, Tsung-Ping Su, Cone EJ. In vitro binding studies of drugs to hair: influence of melanin and lipids on cocaine binding to Caucasoid and africoid hair. J Anal Toxicol 1996; 20: 338-344.
9. Nakhara Y, Ochiai T, Kikura R. Hair analysis for drugs of abuse .V.The facility in incorporation of cocaine into hair over its major metabolites, benzoylecogonine and ecogonine methyl ester. Arch toxicol 1992; 66: 446-449.
10. Joseph RE, Tsai W, Tsao l, Su T, Cone EJ. In vitro characteristion of cocaine binding sites in human hair. J Pharmacol Exp Ther 1997; 282: 1228-1241.
11. Nakahara Y, Takahashi K, kikura R. Hair analysis for drugs of abuse X. Effect of physicochemical properties of drugs on incorporation rates into hair. Biol Pharm Bull 1995; 18:1 223-227.
12. Zahlsen K, Nilsen OG. Nicotine in hair of smokers and non-smokers : sampling procedure and gas chromatographic / mass spectrometric analysis. Pharmacol Toxicol 1994; 75(3-4): 143-149.
13. Ishyana I, Nagai T, Tosshida S. Detection of basic drugs(methamphetamine, anti-depressants, and nicotine) from human hair . J Forensic Sci 1983; 28: 380-385.
14. Nakhara Y, Kikura R. Hair analysis for drug of abuse III. Effect of structural factors on incorporation of drugs into hair: the incorporation rates of amphetamine analogs. Arch Toxicol 1996; 70: 841-849.
15. Stout PR, Claffey DJ, Ruth JA. Fentanyl in hair: chemical factors involved in accumulation and retention of fentanyl in hair after external exposure or in-vivo deposition. Drug metabolism and disposition 1998;26(7): 689-700.
16. Rothe M, Pragst F, Thor S, Hungen j. effect of pigmentation on the drug deposition in hair of grey haired subjects. Forensic Sci Int 1997; 84: 53-60.
17. Henderson GL, Harkey MR, Zhou C. Incorporation of isotopically labeled cocaine into human hair: Race as a factor. J Anal Toxicol 1998; 22: 156-165.
18. Hamilton E, Howard A, Potten CS, Cell kinetics in the sebaceous glands of the mouse. Cell tissue Kinet 1974;7: 399-405.
19. Sachs H. History of hair-analysis. Forensic Sci Int 1997; 84: 7-16.
20. Baumgartner AM,Jones PF, Baumgartner WA, Black CT. Radioimmunoassay of hair for determining opiate abuse histories.J Nucl Med 1979; 20: 748-752.
21. Goulle JP, Kintz P. Hair: a powerful biological marker for exposure to Xenobiotics. Ann Biol Clin(Paris) 1997; 55(5): 435-442.
22. Gaillard Y, Pepsin G. Testing hair for pharmaceuticals . J Chromatogr B Biomed Sci Appl 1999; 733(1-2) 231-246.
23. Valente d, Cassini M, Pigliapochi M, Vansetti g. Hair as the sample in assessing morphine and cocaine addiction. Clin Chern 1981; 27: 1952-1953.
24. Cone EJ, Legal workplace and treatment drug testing with alternate biological matrices on a global scale. Forensic Sci int 2001; 21: 7-15.
25. Kintz P, Kieffer I.,Mangin P. Nicotine analysis in neonates hair for measuring gestational exposure to tobacco.J Forensic Sci 1993; 38: 119-123.
26. Villain M, Cheze M, Tracqu A, Ludes B, Kintz P. Testing for zopiclone in hair application to drug-facilitated crimes.Forensic Sci Int 2004; 145(2-3): 117-121.
27. Yegles M, Labarthe A, Auwarther V, Hartwig S, Vater H, Wening R, Pragst F. Comparison of ethylglucuronide and fatty acid ethyl ester concenterations in hair of alcoholics , social drinkers and teetotalers. Forensic Sci Int 2004; 145: 167-173.
28. Pragst f, Roth M, Spiegel K, Sporkert F. Illegal and therapeutic drug concenteration in hair analysis in hair segment- A time table of drug exposure?. Forensic Sci Rev 1998; 10/2:1-111.
29. Gygi SP, Colon F, Raftogianis RB, Galinsky RE, Wilkins DG, Rollins DE. Dose related distribution of codeine and its metabolites into rat hair. Drug metabolism and Disposition 1995 1995; 24: 282-287.
30. Goulle JP, Noyon J, Layet A, Rapoport NF, Vaschalde Y, Pignier Y, Bouige d, Jouen F. Phenobarbital in hair and drug monitoring. Forensic Sci Int 1995; 70: 191-202.

Study of Pattern of Head Injury in Drivers of Two Wheeler Auto Vehicle Accidents

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ABSTRACT

Every life has an end on one day by natural or unnatural means. Death due to an accident either Road traffic or Air crash or Train leaves a bad taste in society. WHO defines accident as unwanted, unplanned occurrence that may involve injury. During 1990 road traffic accidents was the 9th leading cause of death in the world. If the same trends continue, it will be 2nd leading cause by 2020. Head injury is most important cause of mortality worldwide. In India , with increasing mechanization and greater use of high speed two wheeler vehicles without safety measures , number of the head injury in such driver of are increasing day by day.

Key words : *Auto Wheeler, Bleeding, Contusion, Ecchymosis, Fracture, Haemorrahge, Laceration.*

INTRODUCTION

Two wheeler auto vehicles like motorbike, scooter bike are too unsafe for the reasons mentioned below:

1. Less stable as compared to four wheelers.
2. Relatively less weighed for balance.
3. Mechanized to attain high speed.
4. Cannot remain upright after collision
5. Small size may be the reason for being easily overlooked by larger vehicles.
6. Driver is thrown off and therefore subject to very severe impact force.

Injury to head may result when moving head strikes an object or when a moving object impact on head. Severity of head injury depends on: [1]

1. Speed and mass of moving object.
2. Velocity and direction of the force.
3. Structural properties of the skull and intracranial contents.

A driver of two wheeler is subjected to sudden deceleration, could be thrown off his vehicle and with head first could be flung to the ground[3].

REVIEW OF LITERATURE

J K Mason and B N Purdue, studied a rise in the number of motorcycle casualties from 1994 to 1997: with two wheeled motor vehicles account for 14.1 % of all road fatalities in Great Britain. The 20-39 years age band accounted for 70% of all fatalities in 1995. In our study most common age group was 21 to 30 years and motorcyclist were 69.31% of cases.

Dings SL et al[5] (1993) in their study of 4329 cases of Road Traffic Accidents in China in 1990, showed that there were more men than women in all age groups. The maximum frequency of traffic injuries was noted in patients between 20-39 years.

Javanovi OJ and Javanovi CM[7] (1993) in their study (1985-1992) of Road Traffic Accidents at Zavod Za Zdvastvenu Za Stityrandnika Nis, Hivuv Ska Khiluika Nis showed that traffic accidents most commonly occurred during weekends between 16-20 hrs.

A study of 100 cases of Road Traffic Accidents by Anand Menon and Nagesh KR[2] from 1995-1997 at Manipal shows that most common vehicle involved was two wheelers, most common time was afternoon (12.01-18.00 hrs), male (89%) most commonly involved, predominant age group (21-30 years). Skull fracture was present in total 62% cases (vault fracture in 38 % and base fracture in 34%), fissured fracture was common (57%) with subdural haemorrhage in 77%, subarachnoid haemorrhage in 55% and contusion and laceration was present in 35% of cases almost similar to our study.

A study of Road Traffic Accidents at Jaipur from 2003-2004 by Akhilesh Pathak, N.L. Desania, Rajesh Verma shows that male of middle age most commonly

involved compare to female. Mostly accidents occurred between 6-9 PM. Linear fracture present in 43% cases followed by basilar fracture in 17.73%, comminuted fracture in 7.61 % and subdural haemorrhage in 94% of cases followed by subarachnoid haemorrhage in 83.54% cases, intracerebral haemorrhage in 20.25% cases and extradural haemorrhage in 10.13% cases almost similar to our study.

A study of 249 cases of Road Traffic Accidents at Jammu by Khajuria B, Sharma R, Verma A[6] from 2000-2005 shows that most common age group 20-40 yrs (53%), male (85%) most commonly involved. Accidents mostly occurred in day time 9AM to 7 PM of 54%, Skull fracture present in 68%, subdural haemorrhage in 80%, subarachnoid haemorrhage in 63%, Extradural haemorrhage in 48% and contusion was present in 36% of cases almost similar to our study.

A study of 7008 autopsies from 2001-2005 at AIIMS, Delhi by Arvindkumar, Sanjeev, Deepak, Ravi and TD Dogra[4] shows that total 35 % cases were of Road Traffic Accidents. In Accidents cases, male: female ratio was 7.5: 1. Most common age group 21-40 years (54%). Skull fracture was present in 69%, Temporal bone fracture in 47%, subdural haemorrhage in 89% and Craniotomy was present in 17% of cases.

If the victim's vehicle is high speed , than face to face collision will cause infliction of three types of injuries. If the two wheeler is moving in the direction of offending vehicle, the force of impact will be less which will depend on the speeds of both the vehicles and maximum danger lies with the injuries sustained on head which may be directly hit by the vehicle or may itself strike the ground after being knocked down. The head or for that purpose any part of the body may be crushed subsequently by being run over by the wheels of the offending vehicle and such type of injuries can be reduced by the use of helmet.

AIMS AND OBJECTIVES

1. To find out the pattern of injuries in two wheeler accidents in Ahmedabad city.
2. To find out the changing pattern of two wheeler accidents in Ahmedabad city in last decade.
3. To find out the causes involved in mortality from two wheeler vehicle accidents.
4. To determine pattern of head injury in driver of such vehicle during autopsy at VS General Hospital, Ahmedabad.
5. To define and to compare present study with other available studies.

MATERIAL AND METHODS

The present study consisted of 189 consecutive deaths of driver of two wheeler auto vehicle accidents to determine pattern of head injury whose autopsies were conducted at mortuary of V.S.General Hospital, Ahmedabad, from May 2007 to April 2009.

A total autopsy conducted during this tenure was 2361. In the study, an attempt was made to record all the findings in specially designed Performa for study. These data was reduced to tables, subsequently subjected to computer assisted statistically analysis.

OBSERVATIONS

Table.1. Incidence of cases of head injury in driver of two wheeler auto vehicle accidents at autopsy

Year	Total Autopsies	No of cases Under Study	Percentage
2007-2008	1194	101	8.46
2008-2009	1167	88	7.54
TOTAL	2361	189	8.00

Table shows that total number of autopsies conducted were 2361 during two consecutive years. The number of cases of head injury in driver of two wheeler auto vehicle accidents was 189 [8.00 %].

Table.2. Shows the circumstances of accidents of two wheeler [to which vehicle/object they hit/struck]

Opposite Vehicle	2007-2008	%	2008-2009	%	Total	%
Bus	11	10.89	1	1.14	12	6.35
Car	21	20.79	11	12.50	32	16.93
Truck	19	18.81	24	27.27	43	22.75
Motorcycle	5	4.95	6	6.82	11	5.82
Rickshaw	4	3.96	5	5.68	9	4.76
Scooter	2	1.98	1	1.14	3	1.59
Tractor	2	1.98	0	0	2	1.06
Pedal rickshaw	1	0.99	0	0	1	0.53
Animal	3	2.97	5	5.68	8	4.23
Road divider	0	0	3	3.41	3	1.59
Traffic cabin	1	0.99	0	0	1	0.53
Tree	2	1.98	1	1.14	3	1.59
Slip	30	29.70	31	35.23	61	32.28
Total	101	-	88	-	189	-

Table shows that most common circumstances of accidents were just two wheeler slip [32.28%]. Next was hit by truck [22.75%].

Table. 3. Sexwise distribution of cases

Year	Total	Male	%	Female	%
2007-2008	101	98	97.03	3	2.97
2008-2009	88	84	95.45	4	4.55
TOTAL	189	182	96.30	7	3.70

Total 96.30% of cases were males, & 3.70% of cases were females.

Table 4. Nature of vehicle used

Year	Total Class	Moter Cycle	%	Scooter	%	Moped	%
2007-08	101	64	63.37	19	18.81	18	17.82
2008-09	88	67	76.14	11	12.50	10	11.36
TOTAL	189	131	69.31	30	15.87	28	14.82

Motorcycle is the most common two wheeler [69.31 %], may be due to high peak up, speed and average.

Table 5. Number of cases according to Age distribution

Age (Years)	2007-2008	%	2008-2009	%	Total	%
0-10	-	-	-	-	-	-
11-20	18	17.82	12	13.64	30	15.87
21-30	35	34.65	26	29.55	61	32.28
31-40	16	15.84	21	23.86	37	19.58
41-50	22	21.78	12	13.64	34	17.99
51-60	7	6.93	11	12.50	18	9.52
ABOVE 60	3	2.97	6	6.82	9	4.76
TOTAL	101	-	88	-	189	-

The highest number of cases is seen in the age group 21-30 years. (32.28%) as this is the age group that uses two wheeler maximum and with high speed too. Followed by 31-40 years age group (19.58%).

Table 6. Distribution of cases in a day as per time:

Time of Accident	05-08 AM	08-11 AM	11AM 02PM	02-05 PM	05-08 PM	08-11 PM	11PM 02AM	02-05 AM
TOTAL	6	33	29	30	33	41	15	2
%	3.17	17.46	15.34	15.87	17.46	21.69	7.94	1.06

Most common time of accident was in between 8 PM to 11 PM in 21.69% of cases. This is the time in which most people return from workplace to reach home in hurry with stress of over day work [2].

Table 7. Distribution of external injury on head

Ext. injury	Right				Left			
	F	P	T	O	F	P	T	O
Abrasion	28	3	4	-	23	3	6	1
%	14.81	1.59	2.12	-	12.17	1.59	3.17	0.53
Contusion	3	1	-	-	3	-	2	-
%	1.59	0.53	-	-	1.59	-	1.06	-
CLW	11	7	6	6	2	6	2	5
%	5.82	3.70	3.17	3.17	1.06	3.17	1.06	2.65
SSW	33	31	26	3	16	18	18	2
%	17.46	16.40	13.76	1.59	8.47	9.52	9.52	1.06

[F-Frontal, P-Parietal, T-Temporal, O-Occipital, CLW-contused lacerated wound, SSW-surgically stitched wound]

In study as a result of hospital treatment major part shows surgically stitched wound, followed by abrasion. Third one was contused lacerated wound. Amongst SSW most common part injured was right side on frontal and parietal regions. Abrasion was seen highest on frontal region. CLW were common on right frontal region.

External injury was most common on frontal region. Right side was more prone to injury than left side.

Table 8. Regional distribution of scalp ecchymosis with ecchymosis of temporalis muscle

Scalp Ecchymosis		Total	%
Right	Frontal	80	42.33
	Parietal	98	51.85
	Temporal	81	42.86
	Occipital	57	30.16
	Temporalis muscle	20	10.58
Left	frontal	74	39.15
	Parietal	80	42.33
	Temporal	71	37.57
	Occipital	51	26.98
	Temporalis muscle	17	8.99

Most common site for scalp ecchymosis was right parietal region followed by right temporal, right frontal, left parietal. Injury was more on right side than on left side.

Table 9. Regional distribution of skull vault fracture.

Skull Vault Fracture			Total	%
Right	Frontal	Linear	9	4.76
		Depressed	21	11.11
	Parietal	Linear	18	9.52
		Depressed	17	8.99
	Temporal	Linear	19	10.05
		Depressed	16	8.47
	Occipital	Linear	12	6.35
		Depressed	9	4.76
Left	Frontal	Linear	3	1.59
		Depressed	15	7.94
	Parietal	Linear	13	6.88
		Depressed	12	6.35
	Temporal	Linear	15	7.94
		Depressed	12	6.35
	Occipital	Linear	11	5.82
		Depressed	7	3.70

Linear fracture was more common than depressed fracture. Linear fracture was mostly seen on right temporal bone followed by right parietal bone. Depressed fracture was mostly found involving right frontal bone followed by right parietal bone. Skull fracture was seen more on right side than on left side.

Table.10. Distribution of fracture of cranial fossa

Intra Cranial Fossa		Total	%
Right	Anterior	15	7.94
	Middle	23	12.17
	Posterior	13	6.88
Left	Anterior	16	8.47
	Middle	25	13.23
	Posterior	13	6.88

Middle cranial fossa shows fracture in 17.46 % of cases out of which 13.23 % were on left and 12.17 % were on right side. The middle cranial fossa was more prone to fracture.

Table 11. Distribution of intracranial haemorrhage.

Site of Haemorrhage		Extradural Haemorrhage	%	Subdural Haemorrhage	%	Subarachanoid Haemorrhage	%
Right	Frontal	5	2.65	105	55.56	142	75.13
	Parietal	8	4.23	128	67.72	136	71.96
	Temporal	11	5.82	104	55.03	115	60.85
	Occipital	1	0.53	102	53.97	111	58.73
Left	Frontal	2	1.06	98	51.85	113	59.79
	Parietal	3	1.59	122	64.55	131	69.31
	Temporal	1	0.53	103	54.50	115	60.85
	Occipital	4	2.12	96	50.79	106	56.08

Most common type of haemorrhage was subarachnoid haemorrhage that was most frequent on right frontal brain followed by right parietal region.Second common was subdural haemorrhage that was seen on right parietal, followed by left parietal region. Most common haemorrahge was found on right side of brain.

Table 12. Pattern of intra ventricular haemorrhage.

Intra ventricular haemorrhage	Total	%
Right Lateral	13	6.88
Left Lateral	14	7.41
Third	6	3.17
Fourth	6	3.17

Most common site for intra ventricular haemorrhage was lateral ventricles.

Table 13. Distribution of contusion and laceration of brain

Site of Brain Injury		Contusion	%	Laceration	%
Right	Frontal	20	10.58	8	4.23
	Parietal	8	4.23	7	3.70
	Temporal	18	9.52	7	3.70
	Occipital	7	3.70	5	2.65
Left	Frontal	24	12.70	9	4.76
	Parietal	11	5.82	6	3.17
	Temporal	21	11.11	8	4.23
	Occipital	9	4.76	4	2.12

Contusion was more common than laceration on brain. Contusion was mostly present on left frontal lobe followed by left temporal lobe, right frontal lobe. Laceration was seen mostly on left frontal lobe followed by right frontal and left temporal lobes. Contusion was seen common on left side in comparison to skull fracture which was common on right side due to counter-coup brain injury. Lacerations found on brain were corresponding to skull fractures.

CONCLUSION

The present study consisted of 189 consecutive autopsies of driver of two wheeler auto vehicle accidents conducted during the period from May 2007 to April 2009 at Department of Forensic medicine, Sheth V.S. General Hospital, Ahmedabad.

Autopsy studies in head injury can be of immense value of not only in ascertaining the cause of death but also in planning a better approach for the prevention of head injury in driver of two wheeler auto vehicle accidents, which is burning problem. Present study has contributed in some way towards the better understanding of this problem.

The result is summarized as follow:

1. Total 2361 autopsies were done during the year May 2007 to April 2009. Among them 189 cases were of "driver of two wheeler auto vehicle accident with head injury" total 8% of total autopsies. This number is definitely higher as vehicle accidents are increasing day by day.
2. Motorcycle is the most common two wheeler with 69.31 % of total cases, may be due to high peak up, speed and average.
3. Incidence of mortality was higher in males i.e. 96.30% than in females because males are more involved in outdoor activities.
4. Most common time of accident was in between 8 PM to 11 PM in 21.69% of cases. This is the time in which most people return from workplace to reach home in hurry with stress of over day work.
5. Total 32.28% of cases occurred in 21-30 years of age group as in this age group there is obsession for speed.
6. External injuries on head was more common on right side which suggest that such type of injuries occur by striking the victim to ground as victims lost his balance after an accident. Skull fractures were also more common on right side and most common fracture found was linear fracture. Mid cranial fossa show high rate of fracture suggestive of coup type of injury.
7. Subarachnoid haemorrhage was most common type of haemorrhage and was most common on right side. Subdural haemorrhage was also found common on right side corresponding to fracture and external injury on right side which suggest coup type of injury.
8. However contusion and laceration were more common on left side of brain which suggests counter-coup type injury.
9. The relationship between speed, accidents and the severity of crashes is well known as a result of promotion of faster and faster vehicles. Head injury can be prevented by controlled speed, proper engineered road conditions, well controlled traffic

conditions including lights and safety measures like suitable helmet.

REFERENCES

1. A.K.Banerji : post mortem examination of nervous system, Neurology India vol.45, page 120-125, June 1997.
2. Anand Menon, Nagesh KR: Pattern of fatal head injuries due to vehicular accidents in Manipal, JIAFM, 2005: 27(1).
3. Apurba Nandy:Principles of forensic medicine, 2001.
4. Arvind, Sanjeev, Deepak & et al : Fatal Road Traffic Accidents and their relationship with head injuries - Epidemiological survey of 5 years. Ind Jour Neurotrauma, 2008, vol-5, no 2, pg 63-67.
5. Ding SL et al (1994) : Head injuries in traffic accidents with emphasis on the comparisons between motor cycle - helmet users and non users: Department of Medical Research. Tri-Service General Hospital : National Defense Medical Center, R.O.C. J Forms Med. Assoc. 1994 March, 93 supp. 1: 542-548 (B-17).
6. Khajuria B, Sharma R, Verma A : Profile of autopsies of Road Traffic Accidents victims in Jammu. Jour Clin & Diag Resea 2008, February, 2: 639- 642.
7. Jovanovi CJ : Jovanovi CM (1993) : Epideurilogic characteristics of collisions and injuries in traffic accident in the area of Ni S: Zavod Za Zdravstvenu Za Stitu Randika Ni S, Hirur Ska Khinika Ni S, Vajnosanit pest 1993 Nov-Dec, 50(6): 584-588 (E-20).

A Study of Post-Mortem Histopathological Changes in Septaecemia Due to Burns

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ABSTRACT

The study was carried out over a period of 1-year (January 2010-December 2010), where eight hundred and twenty five autopsies were performed. Out of these One hundred and sixty two autopsies performed were deaths due to burn injuries. The male to female ratio was 0.35:1. Females succumbed to burn injuries twice frequently (65%) when compared to males (35%). The age ranged between 01-80 years(76.54% of them belonged to the 12-35years of age-group, 16% belonged to >35 years of age group and 7.4% belonged to 9 months to 11 years of age group). The total burn surface area ranged between 20% to 95% and 117 patients (72.2%) had major burns (50-95% total burn surface area). More than half of the deaths (58%, 94 cases) from burns were within 48 hrs of the incident, 25 cases (15.43%) died between 3-4 post burn days, 34 cases survived for 5 days(20.98%) and 7 cases(4.32%) survived for 10-11 days of burn injuries.

Almost 74.07% (120 cases) presented with oedema of the glottis and pulmonary oedema due to inhalation of carbon monoxide and carbon dioxide. More than 50 cases had multiple organ failure. Hence the most affected organ was lung (120 cases), followed by kidney (93 cases), heart (64 cases), nervous system (58 cases), digestive system (46 cases) and liver (41 cases).

Key words: *Burns, Postmortem Histopathological Changes, Pneumonia.*

INTRODUCTION

Burn profile closely follows the socioeconomic flux of a country[1]. Economically developed nations with sound prevention policy, organized dwelling and safe kitchen technology and fuel have brought down burn incidence drastically. However, in developing nations, burn continues to be endemic because of massive slum dwelling and large scale use of unsafe stoves and fuel[2].

A recent study by Ahuja et al[3], documents that economic uplift and shift from kerosene to safer LPG stoves has brought down annual burn admission by 43% in a major burn unit of Delhi.

The major difference in the pediatric burn profile of developed and developing nations is that pediatric burn in the former is mainly scalds, whereas, in the latter a large number of children sustain flame burn. This can be attributed to congested living and floor level cooking. This is evident in many reports from India and other developing countries[4-7].

Although the survival from burn injury has increased in recent years with the development of effective fluid resuscitation management and early surgical excision of burned tissue, the mortality of burn injury is still high. In these fire victims, progressive pulmonary failure, cardiovascular dysfunction and nephrosis are important determinants of morbidity and mortality. The morbidity and mortality increases when burn injury is associated with smoke inhalation[8,9]. In this study we attempted to study deaths due to severe burns in order to outline the wide range of visceral alterations caused by thermal traumatism.

MATERIAL AND METHODS

We considered 162 reports specifying the findings of autopsies of victims with complex burns performed at the Vijayanagar Institute of Medical Sciences, Bellary, Karnataka, during the period January 2010-December 2010. Autopsy was performed within 24 hours of death in each patient.

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After death, bodies were transferred to a cooling chamber (temperature 3.8-4°C) which is close to the department. On the morning of the next working day, the corpses were transported to the Department of Forensic Medicine, where the autopsy was performed.

The following information was extracted from each patient's record: burn size percentage, age, sex, address, cause of burn injuries, autopsy findings and histopathological findings. The percentage of body surface involved in burns was determined by "Rule of Nines".

The organs were removed in four blocks: 1) heart and lungs, 2) liver and gastrointestinal tract, 3) urogenital system, 4) brain. Afterwards, all organs were systematically examined for macroscopic pathologies. Whenever macroscopically unclear lesions were detected, tissue samples were taken for histological analyses to either confirm or refute the macroscopic diagnosis.

RESULTS AND DISCUSSION

Of the 162 deaths due to burns, the male to female ratio was 0.35:1. Females succumbed to burn injuries twice frequently (65%) when compared to males (35%). In contrast Haik J et al[10], in their study of burns in Israel reported that male gender was twice as frequent as female gender in burns patients (68.0% vs. 31.9%). The age ranged between 01-80 years(7.4% belonged to 9 months to 11 years of age group, 76.54% of them belonged to the 12-35 years of age-group and 16% belonged to >35 years of age group). The total burn surface area ranged between 20% to 95% and 117 patients (72.2%) had major burns (50-95% total burn surface area). Seventy two cases (44.4%) were from rural areas and 90 cases (55.5%) were from urban area. Forty two cases were homicidal burns, 13 were suicidal and 107 were accidental.

Age of Burn Injury

We tried to correlate the age of burn injury depending on the different stages of the repairative process of burn injuries. Vesication usually develops within about 2-3 hours. The exudates begins to dry in 12-24 hours and within 48-72 hours, it forms a dry, brown crust. The red inflammatory zone disappears in 36-72 hours. If infection happens to occur, pus is formed in 2-3 days but not before 36 hours. Superficial sloughs usually separate out from the 4th to 6th day and deeper sloughs are formed within about 2 weeks[11]. More than half of the deaths (58%, 94 cases) from burns were within 48 hrs of the incident, 25 cases(15.43%) died between 3-4 post burn days, 34 cases survived for 5 days(20.98%) and 7 cases(4.32%) survived for 10-14 days of burn injuries.

On the basis of our observations, we noted a variable incidence of destructive processes in the visceral organs: 35.8% (58 cases) in the neurological system, 57.4.0% (93 cases) in the renal system, 28.3% (46 cases) in the gastrointestinal system, 25.3% (41 cases) in the hepatobiliary system, and 39.5% (64 cases) in the cardiovascular system, with prevalence in the pulmonary system of 74.07%(120 cases). Iliopoulou E et al[12] reported that 70% (28 cases) of their patients presented inhalation injury, and half (20 cases) presented multiple organ failure.

Autopsy and Histopathological changes in the lungs

One hundred and twenty cases (74.07%) showed pulmonary changes like Pulmonary oedema, pulmonary artery embolism and Pneumonias. The tongue, fauces, larynx, trachea and bronchi were inflamed and contained soot mixed with mucus. The mucosa over the tongue and larynx were oedematous and exhibited blistering and shredding. In some cases vomitus was present in the respiratory passages, probably due to bouts of coughing.

Thirty six cases had pulmonary edema where Lungs were 2-3 times their normal weight (800 g) and sectioning revealed frothy, blood tinged fluid. One hundred and two cases had

Pneumonia where there were consolidated lung areas with patchy infiltrations and there was pus on sectioning of the lungs. Thus the leading cause of death in patients hospitalized after burn injury is sepsis, followed by pneumonia.

Six cases had Pleural effusions where there was fluid collection (blood/pus) in one or both pleural cavities. Seven cases had Tracheobronchitis where substantial parts of the tracheobronchial tree had reddened with edematous mucosa. Eleven cases had Pulmonary embolism where there was partial or total occlusion of a pulmonary artery by a venous thrombus. Two to three cases had Pulmonary hemorrhage where there were consolidated lung areas with hemorrhagic infiltrations; pleuritis with reddened, edematous pleura and Pulmonary infarction with raised, red-blue wedge-shaped areas extending to the lung periphery.

Autopsy and Histopathological changes in the cardiac tissue

In this study 64 cases (39.5%) had changes in the cardiac tissue. Four cases had myocardial ischemia with diffuse red-blue lesions of the myocardium rimmed by a hyperemic zone with one case showing occlusion of the supplying coronary artery. Sixteen cases had Pericarditis accompanied by pericardial effusion with reddened and granular pericardium and

fluid collection in the pericardial cavity. Thirty four deceased patients had Myocarditis with diffuse or patchy myocardial lesions of flabby consistence with either pale or hemorrhagic foci. Ten cases had Endocarditis: Friable, bulky, destructive vegetations on cardiac valves containing fibrin, inflammatory cells, and pathogens on histology. Taran et al[9] reported Cardiovascular affections in 74 patients (39.7%) who died at different stages of burn disease development and that their observations reconfirmed the data from other field-related research, showing that diffuse myocardial deteriorations occur during shock, which remain conspicuous during the entire post-burn development interval. Regules JA et al[13] observed that endocarditis occurred in 0.4% of burn unit admissions and in 8.9% of these patients with persistent bacteremia.

Autopsy and Histopathological changes of the digestive tract of deceased burn patients

The ulcerous and erosive affections of the digestive tract were diagnosed in 46 deceased patients (28.3%).The most common findings were gastritis (37 cases) with edematous gastric mucosa with vascular congestion but maintained mucosal barrier; gastrointestinal hemorrhage in 6 cases with intraluminal blood originating from lesions of the gastrointestinal tract; gastroduodenal ulcer in 3 cases with erosions of the gastric mucosa greater than 0.5 cm in diameter.

Taran et al[9] reported nine cases in which acute gastric ulcers were found and seven with intestinal ulcers (five duodenal ulcers and two ulcers in the caecum and colon) and concluded that ulcerous and erosive affections of the digestive tract constituted phenomena that frequently complicated the development of severe burns, often becoming the cause of lethal developments.

Autopsy and Histopathological changes in kidneys in persons with thermal burns

Autopsy and histopathological modifications of the kidneys in persons with thermal burns were identified in 93 cases (57.4%). The different findings were Cystitis with reddened, edematous mucosa of the urinary bladder, diffuse enlargement of the kidney; Kidney ischemia with sharply demarcated, pale areas containing hemorrhagic foci; Pyelonephritis with grayish-white discoloration of the pyelum and ureter. Taran et al[9] reported irreversible renal dysfunctions in 11 (5.9%) of the 186 patients examined by necropsy.

Autopsy and Histopathological changes in liver in persons with thermal burns

Hepatic changes were found in 41 patients (25.3%). There was considerable repletion of the liver, firm when palpated, red-brown in section. Similarly Taran et al[9] are also of the opinion that the most conspicuous modifications at the hepatic parenchyma level were atrophy and fat necrosis of the liver.

Autopsy and Histopathological changes in nervous system in persons with thermal burns

In our study 58 (35.8%) had changes in the nervous tissue. There was edema of the brain with flattened gyri, narrowed sulci and compressed ventricular cavities, localized, pale and swollen areas of the brain; Hemorrhage within the cerebral parenchyma and Encephalitis with Diffuse brain swelling with inflammatory alterations of temporal cerebral areas.

A burn is not only one of the most severe skin injuries but also a very complicated disease with a large variety of morphological and functional multi-system lesions. The main factors determining the deleterious effects of heat are the temperature, i.e. the intensity of heat applied and the duration for which it is applied. This is amply clear from the observations of Moritz and Henriques, who found that the lowest temperature that would produce damage was 44°C, though the time required to produce burn was of the order of about five hours, whereas if the object was at 60°C, it required only three seconds to cause burn.

ARDS (acute respiratory distress syndrome) is one of the major complications of thermal injury. In patients with extensive cutaneous burns in which the burned area exceeds 30% of the TBSA (total body surface area), capillary hyperpermeability occurs not only at the injured site, but also in regions distant from the injury [14, 15]. Vascular hyperpermeability leads to a large amount of fluid flux from the circulating plasma to the interstitial spaces. This lung oedema formation is even more severe when the thermal injury is associated with smoke inhalation[16]. Combined burn and smoke inhalation injury causes pulmonary microvascular hyperpermeability to both fluid and protein[8].

Of the 120 cases, we could detect carbon monoxide poisoining in only 13 cases. There was cherry pink coloration of unburnt skin in protected areas, blood and tissues. The saturation of haemoglobin with carbon monoxide varies greatly from one fatal case to another. Many variable factors exist such as the concentration in the atmosphere, the time of exposure and local changes in oxygen content. When carbon monoxide is the sole cause of death, a blood saturation of atleast 40 percent is required, except in old and debilitated persons where deaths have been reported at 25 percent[17].

Systemic inflammatory response syndrome (SIRS) leading to Multiple Organ Failure (MOF) continues to be a major problem after surgery and trauma. MOF is a critical condition developing in patients with overwhelming bodily injury resulting from severe traumas, extensive burns, and sepsis. MOF accounts for up to 80% of all intensive care unit (ICU) deaths and costs millions of dollars due to prolonged ICU-bed occupancy[18,19]. MOF is one of the major causes of death in patients with severe burns. The pathogenetic mechanism that links these conditions is SIRS. SIRS is defined as a failure of local control of the inflammatory response to injury or infection, allowing the inflammatory process to spill over and damage organs remote from the site of injury[20].

Zhang[21] presents an analysis of the relationship between invasive infection and the Multiple Organ Dysfunction Syndrome (MODS) in 158 burn cases. Morbidity in MODS is high, reaching 81.6%, and there is a 50% possibility that it will become progressive MOF. Death is associated with MOF rather than with MODS. Mortality in MOF is very high - over 90%. Among various microbes, Pseudomonas pyocyanea causes the highest morbidity in MODS. The diagnosis and treatment of MODS are difficult, and it is therefore essential to control invasive infections.

REFERENCES

1. Forjuoh SN. Burns in low and middle income countries: A review of available literature on descriptive epidemiology, risk factors, treatment and prevention. Burns 2006; 32: 529-537.
2. Bhattacharya S. Burn epidemiology - An Indian perspective. Indian J Plast Surg 2009; 42:193-194.
3. Ahuja RB, Bhattacharya S, Rai A. Changing trends of an endemic trauma. Burns 2009; 35: 650-656.
4. Ahuja RB, Bhattacharya S. An analysis of 11,196 burn admissions and evaluationof conservative management techniques. Burns 2002; 28: 555-661.
5. Jayaraman V, Ramakrishnan KM, Devies MR. Burns in Madras: An analysis of1368 patients in one year. Burns 1993; 19: 339-444.
6. Subrahmanyam M. Epidemiology of burns in a district hospital in western India. Burns 1996; 22: 339-444.
7. Laloe V. Epidemiology and mortality of burns in a general hospital of Eastern Sri Lanka. Burns 2002; 28: 778-781.
8. Perenlei Enkhbaatar and Daniel L. Traber. Pathophysiology of acute lung injury in Combined burn and smoke inhalation injury - Review; Clinical Science 2004; 107, 137-143.
9. Taran A., Baciu N., Rafulea V., German A. Clinical and autopsy diagnoses of visceral affections of patients who died because of complicated burns with multi-organ failure. Annals of Burns and Fire Disasters - vol. XVIII - n. 4 - December 2005; 177-184 pp (ISSN 1592-9566).
10. Haik J, Liran A, Tessone A, Givon A, Orenstein A, Peleg K; Israeli Trauma Group. Burns in Israel: demographic, etiologic and clinical trends, 1997-2003. Isr Med Assoc J. 2007 Sep; 9(9): 659-662.
11. Krishnan Vij. Textbook of Forensic medicine and Toxicology. 4th edition; chapter 11, Thermal Deaths, Elsvier Publications., 2008, 209-228pp.
12. Iliopoulou E, Markaki S, Poulikakos L. Autopsy findings in burn injuries. Arch Anat Cytol Pathol. 1993; 41(1):5-8. PMID: 8517764 [PubMed - indexed for MEDLINE].
13. Regules JA, Glasser JS, Wolf SE, Hospenthal DR, Murray CK. Endocarditis in burn patients: clinical and diagnostic considerations. Burns. 2008 Aug;34(5):610-6. Epub 2007 Oct 29. PMID: 18029099 [PubMed - indexed for MEDLINE].
14. Bowen, B. D., Bert, J. L., Gu, X., Lund, T. and Reed, R. K. Microvascular exchange during burn injury. III. Implications of the model. Circ. Shock 1989; 28, 221-233.
15. Lund, T., Bert, J. L., Onarheim, H., Bowen, B. D. and Reed, R. K. Microvascular exchange during burn injury. I: A review. Circ. Shock 1989; 28, 179-197
16. Soejima, K., Schmalstieg, F. C., Sakurai, H., Traber, L. D. and Traber, D. L. (2001) Pathophysiological analysis of combined burn and smoke inhalation injuries in sheep. Am. J. Physiol. Lung. Cell Mol. Physiol. 280, L1233-L1241.
17. Bernard Knight, Forensic Pathology, 2nd edition, Chapter Burns and Scalds, Oxford University Press, Inc., New York.1996, 314-315pp.
18. Baue A.E., Durham R., Faist E.: Systemic in?ammatory responsesyndrome (SIRS), multiple organ dysfunction syndrome (MODS), multiple organ failure (MOF): Are we winning the battle? Shock,1998; 10: 79-89.
19. Davies M.G., Hagen P.O.: Systemic in?ammatory response syndrome.Br. J. Surg., 1997; 84: 920-935.
20. Jansen M.J., Hendriks M.J., Hermsen R. et al.: A monoclonal antibody against tumour necrosis factor-alpha improves survival in experimental multiple organ dysfunction syndrome. Cytokine 1998; 11: 904-910.
21. Zhang M., Zhang P., Wang Y.: Invasive infection and multiple organ dysfunction syndrome (MODS) in burns. Zhonghua Zheng Xing Shao Shang Wai Ke Za Zhi, 1997; 13: 191-194.

Study of Pattern of Injuries in Fatal Railway Accidents

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ABSTRACT

India has the largest network of railway providing the basic mode of transportation for passengers and freight. It carries about 10-12 million passengers everyday, travelling the length and breath of our country covering about 10,000 sq kilometers. Railway related deaths are either accidental or suicidal and use of railway tracks to mask homicides is also encountered often. The railway related deaths are on the rise due to various factors viz, unmanned level crossings, lack of safety precautions during entraining or detraining, poor infrastructure, poor safety measures. The present study is attempted to determine, the various pattern of injuries and correlate them with the available information and to provide solutions to common practical problems, encountered in railway related deaths.

INTRODUCTION

Historically railways dates back to Babylonian times about 2245 BC. The first train in washington was run between Baltimore and Ohio on 24th August 1835. First passenger train accident in US occurred on 9th November 1831 on Camden and Amboy railroad between Spotswood and High town, New Jersey.

Indian railway passes through cities, town and villages. Many dwellings /settlements are located close to the tracks, and are usually unmanned without any barricades/signals. Further trains of our country are usually over crowded with people who are commuting for work, business etc., and hence are prone to meet with fatalities.

All modes of transportation lead to fatalities/death, of which railways are major contributors. The fatalities due to railway injuries are of different types. Deaths due to railway injuries occur during detraining, entraining, collisions, derailments, sabotage etc. Railway tracks are commonly chosen method for committing suicide. It is not uncommon in India using railway tracks for masking homicides. At times cases are brought to morgue as railway deaths, which later prove to be otherwise and hence a proper history from police/relatives, visit to scene of occurrence and meticulous postmortem examination is necessary for identification, to ascertain whether the injuries are ante mortem or post mortem, consistent with railway injuries and to opine regarding the manner of injury.

"India cannot survive without monsoon rains. India cannot survive without its Trains".

AIMS AND OBJECTIVES OF STUDY

1. To study the pattern of injuries in railway deaths.
2. To determine the Age and sex incidence.
3. To differentiate the ante mortem injuries from postmortem.
4. To identify the common medico legal problems associated with railway deaths viz, identity, manner of deaths etc.

MATERIAL AND METHODS

The present study "Pattern of injuries in fatal railway accidents" was carried out in department of forensic medicine, M. S. Ramaiah Medical College and Hospital, Bangalore during the period 2004 and 2005, of all the cases of railway related deaths subjected for medico legal autopsy In some of the instances information was supplemented by either visit to scene of occurrence or from photographs of scene of occurrence. Natural deaths occurring in trains and platforms and highly decomposed bodies were excluded.

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RESULTS

Out of total 763 cases of postmortem, railway related deaths constituted 75 cases. (9.82%)

Table 1. Distribution of study population according to the Age

S. No.	Age Group in years	Males	Females	No. of Cases	Percentage
1	0 to 10	0	0	0	0.00
2	11 to 20	9	4	13	17.33
3	21 to 30	20	4	24	32.00
4	31 to 40	12	3	15	20.00
5	41 to 50	10	4	14	18.66
6	51 to 60	8	1	9	12.66
	TOTAL	59	16	75	100%

In present study it is observed that maximum incidence of fatalities occurred in the age groupm of 21-30 years, may be attributed to unemployment, unsuccessful romantic deeds, academic failure etc.

Table 2. Distribution of study population according to history furnised by police/relatives

Sl. No.	Type	No. of Cases	Percentage
1.	Accident	33	44
2.	Suicide	32	42.6
3.	Allegation	2	2.6
4.	Others	8	10.6
	TOTAL	75	100%

History furnished by railway police, eyewitness, driver of engines and presence of suicide notes in victims pocket, consumption of poison in some cases.

Table 3. Distribution of study population according to level of transection

Sl. No.	Level	No. of Cases	Percentage
1.	Thoracic	11	14.6
2.	Thoraco lumbar	5	6.6
3.	No Transection	59	78.6
	TOTAL	75	100%

In the present study it was observed that transection of bodies at Thoracic or Thoraco abdominal levels was common in suicide cases, the victim were found near to the rail tracks with division of the body into two halves and with the margins contused and stained with oil and grease stains of.

In the present it was observed that head was, common body region involved. Extensive injuries to head (skull and brain) was common in accidents. Single Decapitation was commonly in suicidal cases. In accidental cases it is observed that wide range of injuries were observed.

Table- 4: According to head injuries sustained

Sl. No.	Type	No. of Cases	Percentage
1.	External Injuries	69	92
2.	Fracture of skull	25	33.3
3.	Intra cranial hemorrhage	23	30
4	Brain missing	18	24
5	Head not brought /un traceable	2	2.6

Table 5. Distribution of study population according to injuries to the thorax

Sl. No.	Injuries	No. of Cases	Percentage
1.	External injuries	7	9.3
2.	Fracture of sternum ribs	20	26
3.	Lungs	28	37
4.	Heart	27	36
5.	Diaphragm	5	6.6
6.	Thoracic Vertebrae	11	14.6

Injuries to thorax were observed in accidental, suicidal and fall from train cases. In accidents, injuries to thorax varied from multiple soft tissue injuries to extensive injuries to the ribcage and viscera organs. In suicidal cases, transection at the level of thoracic vertebrae was observed. In case of fall from train, injuries to thorax were present along with injuries to limbs.

Table 6. Distribution of study population according to injuries to the abdomen

Sl. No.	Injuries	No. of Cases	Percentage
1.	External Injuries	9	12
2.	Liver	27	36
3.	Kidney	26	34
4.	Spleen	20	26
5.	Intestines	10	13.3
6.	Thoraco - Lumbar Vertebrae	5	6.6

Injuries to abdomen were observed in accidents, suicides and fall from the train cases, in accidental cases, injuries were due to blunt and sharp forces, like evisceration, laceration of internal organs like liver, spleen and kidney etc.. Liver was lacerated and commonest visceral organ to be injured followed by kidneys.

Table 7. Distribution of study population according to the manner of death

Sl.No.	Marks	No. of Cases	Percentage
1.	Accident	26	34.6
2.	Suicide	35	46.6
3.	Fall	2	2.6
4.	Allegation	2	2.6
	TOTAL	75	100%

Based on the history revealed by police, relatives, eye witness of by stander / passengers, engine driver of train, presence of the suicidal notes with victims,

Table 8. Distribution of study population according to the margins of injuries

Sl.No.	Margins of injuries	No. of Cases	Percentage
1.	Irregularly abraded	71	94.6%
2.	Not consistent with railway injuries	4	5.4%
	TOTAL	75	100%

In present study 71 cases (94.6%) died due railway related injuries and injuries sustained by them were consistent with railway injuries, the margins of injuries were irregularly abraded and contused which were stained with oil and grease of railway engine and tracks.

Table 9. Distribution of study population according to the body region involved

Sl.No.	Body Regions	No. of Cases	Percentage
1.	Head	69	92%
2.	Neck	35	46%
3	Face	32	42%
4.	Thorax	28	37.3%
5.	Abdomen	27	36%
6.	Upper limb	44	58%
7.	Lower Limb	34	45%

Railway fatalities causes extensive damage to the body regions, involving more than one region. In accidental cases polytrauma was common feature probably because of primary injuries and secondary impact injuries sustained. In suicide cases except for few cases, injuries were present in more than one body region. In case of fall from a moving train, limbs and head were involved commonly.

DISCUSSION

Railway injuries are due to kinetic energy imparted by a moving train to a person and are dependent up on the mass (m) and the velocity (v) of the moving train (kinetic energy = 1/2mv2). The mass of a train combined with the velocity results an enormous amount of kinetic energy, and it is transferred on to the body of a person when struck, resulting in massive blunt force injuries.

Rail accidents are classified into three main categories.

1. Train accidents - these are due to trains and rolling stock on or affecting passengers lines, and failure of rolling stock, track and structures.
2. Movement accidents - Accident to people caused by movement of vehicles.
3. Non-movement accidents - accident to people on railway premises but not connected with movement of vehicles.

Accidents among people

Accident to passenger

Accidents to tress passers

Accidents to staff.

Non-train accidents - Injuries to railway staff occur when track workers are struck by moving train or rolling stocks due to inadequate protection or due to human error.[1]

A retrospective study of 127 cases of railway traffic accidents was conducted at south Delhi during 1996-2002. They found majority of the victims where males 112 cases (88.19%) Commonest age group was 21-30 years - 43 victims (33.86%), Head and neck was the most common region in 99 in 46 cases (36.22%). Subdural hemorrhage was found in 78 cases (61.42%).. Lungs were involved in 25 cases (19.69%), the bones of upper limbs & lower limbs damaged in 33 cases (25.98%) & 29 cases (57.48%) respectively.[2]

Corpses found on train tracks demand special attention, so that no homicide goes undetected. Radiographs are helpful in this matter.[3] Traumatic amputation of limbs or fractures are common in accidents or rarely when a person throwing himself in front of moving train. Accidental injuries occurs while trying to board a moving train or falling from it and while walking on rails etc.[4]

Facial and scalp lesions are common in rail accidents involving automobile occupants. Characteristic pattern of injuries in pedestrian - train collision are traumatic amputation of limbs. In train collisions or derailment the passenger may present with blunt and sharp injuries, extensive burns, signs of traumatic asphyxia and sometimes carbon monoxide and hydro cyanide in toxication.[5]

Railway injuries were divided into 5 categories train fatalities, line fatalities, suicide, criminal violence and others. Train fatalities were present in 38 cases (10%), 227 cases (60%) suffered line fatalities, criminal violence was found in 43 cases (11%), 32 cases (8%) were of suicidal nature, 39 cases came under others category.[6]

In case of suicide, traumatic separation of head was commonest, head being most common soft tissue injuries. Trunk injuries resulted either from separation of body parts in thoracic / abdominal level.[7]

In some other cases such passengers may sustain only fatal mechanical injuries particularly over the head, when the train, say for example, passes through

a tunnel or the head strikes any overhead structure like an over bridge.[8]

Collision Lying Between Rails: With less then 80km/hour, no loss of body parts or organs. Single ruptures of liver and spleen were present with closed anatomical cavities. At and with more than 80 km / hour, brain exenteration, smashing of liver, spleen and opening of one or two anatomical cavities were noticed. At and more than the 100 km / hr speed collision, they noticed dismembering of body, opening of 2 or 3 anatomical cavities. At 160 km / per hour speed loss of single part of limbs, abdominal exenteration and loss of parts of truck were noticed.[9]

When the person is crossing the line, primary injuries are seen on side of head and shoulders, if the person is bending down the primary injuries are seen on back and buttocks and secondary injuries on the face and front of the body.[10]

Hooliganism and vandalism affecting rail track or moving trains has also been observed in recent years, deliberate laying of objects on rail track or throwing object at passing train may form a part of malicious strategy.[11]

A clean decapitation is most common injury in suicide cases. A careful autopsy has to be conducted on a body recovered from railway back to determine to injuries sustained are ante mortem or not. In some cases the dead body of homicide may also be placed on rails to simulate suicide. As death is instantaneous in run over, vital reaction in injuries will be minimum, profuse bleeding and absence of staining will differentiate postmortem injuries.[12]

Study conducted at Southern California during 1989 - 1993, with regard to train verses pedestrian injuries. majority of injuries sustained were musculoskeletal. 23 cases (77%) had severe orthopedic injuries with an average of 1.7 extremities involved.[13]

Postmortem examination on dead bodies in India is carried out by medical officer at peripheral level and forensic experts at district hospitals.. Due to lack of forensic knowledge many a times, the medical officer fails to see wounds or injuries, sometimes deliberately or un intentionally overlooking the clearly visible injuries, leading to travesty of justice to the dead and the relatives of deceased may request for a re-postmortem, the results of the re-postmortem of the body usually has a totally different outcome than the initial postmortem examination.[14]

CONCLUSIONS

Railway related deaths subjected for medico legal autopsy contributed for (9.82%) of total autopsies conducted. Railway related deaths included accidents, suicides and fall from train Two cases of homicide masquerading as deaths due to Railway Injuries. Males (86%) out numbered Females (13.3%). Decapitation (26.66%), Transection at thoracic level (14.6%), at thoraco lumbar vertebrae level (6.6%), was common in suicidal cases. Traumatic amputation of upper limbs (10.6%) and lower limbs (10%) with multiple fractures were commonly noticed in cases of fall from moving train. In 2.6% cases injuries noticed were non specific of railway injuries.

Poly trauma was noticed commonly in accidents Railway injuries seen were abrasions, contused abrasions, lacerations, margins of which, were abraded and contused irregularly with stains of oil and grease. Study revealed that in (90%) of cases, more than one body region was injured. 97.33% cases had ante mortem injuries and (2.6%) cases injuries were postmortem in nature. Study of injury pattern revealed the manner of death eg: Decapitation, transaction of body was commonly encountered in suicidal cases, polytrauma was a common finding in accidents and traumatic amputations of limbs were seen in cases of fall from a moving train.

REFERENCES

1. Tedeschi Eckert Tedeschi forensic medicine Vol III Environmental hazards. Edition W.B. Saunder's Company Phildelphia London, Toranto; 1977: 1186-1191.
2. R. Rautji, T.D Dogra, 'Rail traffic - accidents; A retrospective study, Journal Medicine Science and Law; 2004: 44 (1): 67-70.
3. Gregory G. Davis, Bruce Alexander And Robert. M Brissie, 'A 15 year review of railway related deaths in Jefferson country, Albaha' American Journal of Forensic Medicine and Pathology; 1997: 18(4): 363-368.
4. Parikh C.K 'Parik's textbook of medical Jurispendence, Forensic Medicine and toxicology for classrooms and courtrooms. 6th edition CBS Publishers and distributors, New Delhi; 1990: 369-371.
5. Jason Payne James Anthony Busuttil, Williams Mock Forensic Medicine; Clinical Pathological aspects, distributed world wide by plymbridge distributors Ltd; 2003: 538-539.
6. Leonard B. Lerer And Richard G. Matzapoulos 'Fatal railway injuries in Cape Town, South Africa' The American Journal of Forensic Medicine and Pathology; 1997: 18 (2): 144-147.
7. Paran Baran Paul, Suddhodhan Batabyal Tapas Kumar Bose, Rabin Basu 'Railway fatalities -A retrospective study'. Journal of Indian Academy of Forensic Medicine; 24(3): 89-90.

8. Apurba Nandy 'Principles of Forensic Medicine' II nd Edition, Published by New central book agency (P) Ltd. Calcutta - 700 009; 2000: 291-293.
9. Frank Driever, Peter Schmidt Bukhard Madea "About morphological findings in fatal railway collisions" Forensic science International; 2002; (126): 123-128
10. K.S. Narayan Reddy. The essentials for forensic medicine and toxicology 21st edition medical book company, Hyderabad; 2003: 241.
11. Krishnan Vij Textbook of forensic medicine and toxicology, 3rd edition published by Elsevier, Lajpatnagar New Delhi - 110024; 2002: 455-456.
12. Darvesh Tandon, Ajay Aggarwal, K. L. Sharma, P.S. Das Gupta. 'Suicide simulating homicide - A railway track mystery - A case report Journal of Karnataka Medico Legal Society; 2001: 10(2): 60-62.
13. Singer G et. al. Orthopedic Rev Feb 1994: 30(4)
14. Sharma L. Khanagwal V P, Sirohiwal B L. Paliwal And Yadav. D R. "Second autopsy a bane or boon!" Anil Agarwal's internet journal of forensic medicine and toxicology; 2003: 4(2)

Determination of Sex from Clavicle in Vidarbha Region of Maharashtra

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ABSTRACT

Objectives: It has become apparent that anthropological study of clavicle has been somewhat neglected. The present study was undertaken on the samples of Vidarbha region of Maharashtra to determine the sex from clavicle which will be helpful medico-legally in the region.

Materials and Methods: Sixty fully ossified clavicle belonging to 48 males and 12 females were studied. Each bone was measured between its anatomical ends in millimeters with the articular cartilage intact. Parameters used were length, weight, mid-clavicular circumference, mid-clavicular diameter, anterior curved length, and posterior curved length and robustness Index. Demarking Points (DP) were obtained for each parameter.

Results: Mean DP in male for mid-clavicular circumference was 39.6% for right and 50.0% for left clavicle while it was 8.3% and none in right and left clavicle respectively in female. Mean DP in female for length was 8.3% for right and 8.3% for left clavicle while it was 8.7% and 0.67% in right and left clavicle respectively in male.

Conclusion: We conclude that Mid-clavicular circumference of the clavicle is the best parameter for determination of sex for clavicle in males while length is best in female. Length, weight, mid-clavicular diameter and robostness index are also useful parameters.

Key words: *Anthropometry, Clavicle, Sex determination.*

INTRODUCTION

Examinations of the skeletal samples of the burials are often fragmentary and they are found in mixed lots. Determination of sex of the skeletal remains of an individual from an examination of a single bone except the hip bone is considered to be almost an impossible task. Even when entire human body, pelvis and skull are available not more than 90% accuracy can be achieved[1]. For this reason there is a need for developing a technique for sex determination from skeletal parts which are durable. Attempt has been made to study the characters of other bones, which could be helpful in identification of the sex. But it has become apparent that anthropological study of clavicle has been somewhat neglected, as this bone has not appealed to anthropologist to any such degree as it has to comparative anatomist.

Maharashtra is a big state, having various regions like Vidarbha, Marathwada and Western Maharashtra where environmental conditions and nutritional status vary region wise. It necessitates different formula for different region of the state for determination of individual sex. The present study is undertaken on the samples of Vidarbha region of Maharashtra (Eastern Maharashtra) India to determine the sex from clavicle which will be helpful medico legally.

MATERIAL AND METHODS

The present study was carried out in the department of anatomy, Government Medical College, Nagpur, Vidarbha Maharashtra, India. 60 fully ossified clavicle belonging to 48 males and 12 females were

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collected from different medical colleges of the region. The bones of each side of both sexes were numbered and kept separately. Bones showing pathological deformity or fractures were excluded from the study.

Each bone was measured between its anatomical ends in millimeters with the articular cartilage intact[2]. Measurements were taken with the help of osteometric board[2], vernier caliper and spreading caliper[3]. The bony points were first localized and the measurements of both the sides were taken simultaneously. Different parameters used were length, weight, mid-clavicular circumference, mid-clavicular diameter, anterior curved length, and posterior curved length and robustness Index.

Demarking Points (DP)[4] were derived from the calculated range of various parameters and on the basis of demarking point, the percentage of bones in male and females were calculated. Bones were also sexed by using the Identification Points (IP)[4,5] which is the limiting points of the range for males and females. Data was analyzed using Microsoft excel software.

RESULTS

Length

Maximum length of clavicle on right side in females was 136 mm but 70.8 % of male right clavicle had their length exceeding 136 mm. Similarly the smallest male right clavicle was 123 mm and 33% of the female right clavicles were less than this value. Thus by these IP 70.8% male and 33% female right clavicle can be identified. But on the basis on DP (DP = Mean + 3 SD), 8.7% male & 8.3& female bone could be sexed correctly. In case of bone falling in the range between the DP for females and males (116.65 to 148.99) sex could not be identified by this method. Table 1

In left side 64.5% male clavicles were larger than 138 mm whereas 255 of left female clavicles were having values smaller than 124 mm. However by DP only 17.0% male and 8.3% female left clavicles could be sexed accurately. In case of bone falling between the DP for females and males (117.0 mm to 152.5 mm) the sex could not be identified by this method. Table 1

Table 1. Measurements of length of clavicle

Measurements	Right		Left	
	Male	Female	Male	Female
Bones	48	12	48	12
Range in mm	123-156	108-136	124-54	108-138
Mean + SD	140.77+8.04	125.83+7.72	142.00+8.54	128.00+8.32
IP	> 136	< 123	>138	< 124
Identified bones	70.8%	33%	64.5%	25%
Calculated range	116.65-164.89	102.67-148.99	116.62-168.00	102.49-152.35
DP	> 148.99	<116.65	>152.5	<117.0
Beyond DP	8.7%	8.3%	16.67%	8.3%

Weight

We observed right and left male clavicle more in weight than right and left female clavicles. However by DP only 25% of male and none of female right clavicle could be identified while 29.1% of male and none of female left clavicle could be identified by this method. Table 2

Table 2. Measurements of weight of clavicle

Measurements	Right		Left	
	Male	Female	Male	Female
Range in mm	11.0-25.7	10.2-17.3	12.2-25.9	9.0-16.3
Mean + SD	18.08+3.47	12.93+2.51	18.28+3.55	12.3+2.49
IP	> 17.3	< 11.0	>16.3	< 12.2
Identified bones	58.3%	41.67%	31.25%	50.0%
Calculated range	7.63 -28.45	5.4- 20.46	7.63-28.93	4.83-19.77
DP	> 20.46	<7.63	>19.7	<7.63
Beyond DP	25.0%	Nil	29.1%	Nil

Mid-clavicular Circumference

In the study 50% of right male clavicle circumference was more than 34 mm. 58.33% of right female clavicle circumference was less than 28 mm. By DP 39.6% male and 8.3% of the female can be identified. But in left side 50.0% male and none of the female left clavicles can be identified. Table 3

Table 3. Measurements of mid-clavicular circumference

Measurements	Right		Left	
	Male	Female	Male	Female
Range in mm	28-42	24-34	29-49	25-33
Mean + SD	34.52+3.42	28.25+2.83	34.60+3.95	27.79+2.62
IP	> 34	< 28	>33	< 29
Identified bones	50.0%	58.33%	60.4%	75.0%
Calculated range	24.20-44.91	19.75-36.74	22.7-46.4	19.9-35.6
DP	> 36.74	<24.21	>35.6	<22.7
Beyond DP	39.6%	8.3%	50.00%	Nil

Mid-clavicular diameter

By DP only 4.16% male and none of the female right clavicles could be identified. While 2.08% male and none of the female right clavicles could be identified by DP. Table 4

Table 4: Measurements of Mid-Clavicular diameter

Measurements	Right		Left	
	Male	Female	Male	Female
Range in mm	9.66-14.36	8.7-12.2	9.78-14.11	8.9-11.38
Mean + SD	12.23+1.23	10.23+1.19	11.91+0.98	10.16+0.96
IP	> 12.2	< 9.66	>11.38	< 9.78
Identified bones	56.2%	41.67%	72.9%	41.67%
Calculated range	8.53-15.93	6.7-13.8	8.96-14.86	7.3-13.04
DP	> 13.8	<8.4	>13.04	<8.5
Beyond DP	4.16%	Nil	2.08%	Nil

Anterior Curved Length

We observed that by DP none of male and only 8.3% of the female right clavicles could be identified while none of the male and female left clavicle could be identified. Table 5

Table 5. Measurements of anterior curved length

Measurements	Right		Left	
	Male	Female	Male	Female
Range in mm	120-156	104-150	120-157	120-152
Mean + SD	139.81+8.79	125.83+11.09	140.06+9.41	130.67+9.60
IP	> 150	< 120	>152	< 120
Identified bones	10.4%	8.33%	14.6%	0.0%
Calculated range	113.4-166.2	92.5-159.1	111.8-168.3	101.9-159.5
DP	> 159.18	<113.4	>159.5	<111.8
Beyond DP	Nil	8.3%	Nil	Nil

Posterior Curved Length

Present study indicates that by DP 4.16% of male and 8.3% of the female right clavicles and 2.1% of male and 8.3% of the female left clavicles could be identified. Table- 6

Table 6. Measurements of posterior curved length

Measurements	Right		Left	
	Male	Female	Male	Female
Range in mm	134-168	122-161	137-172	131-154
Mean + SD	154.17+8.13	136.16+9.61	156.31+7.0	141.4+7.98
IP	> 161	< 134	>154	< 137
Identified bones	14.6%	41.67%	60.4%	50.0%
Calculated range	129.8-178.6	107.3-164.99	135.3-177.3	117.5-165.4
DP	> 164.99	<129.8	>165.4	<135.3
Beyond DP	4.16%	8.3%	2.1%	8.3%

Robustness Index

On the basis of DP, 6.97% of male right clavicle and none of the female bone while 4.16% of male left clavicle and none of female left clavicle could be identified. Table- 7

Table 7. Measurements of robustness index

Measurements	Right		Left	
	Male	Female	Male	Female
Range in mm	19.33-31.81	19.2-28.33	19.48-36.34	18.52-27.73
Mean + SD	24.82+2.82	22.49+2.35	24.41+2.97	21.89+2.48
IP	> 28.33	< 19.33	>27.73	< 19.48
Identified bones	12.5%	8.3%	8.33%	16.67%
Calculated range	16.34-33.31	15.42-29.57	15.58-33.32	14.45-29.34
DP	> 29.57	<16.12	>29.34	<15.58
Beyond DP	6.97%	Nil	4.16%	Nil

DISCUSSION

Present study reveals that the best metrical character for the determination of sex from the clavicle is the mid-clavicular circumference of clavicle for males. Our findings are in accordance with the study of Jit and Singh[4] who found that DP for mid-clavicular circumference was 71.8 and 48.2 for right and left clavicle respectively. Similar findings were also reported by Shamer Singh and K.C. Gangrade[6,7].

The weight of clavicle was also found to be the next useful parameter while determining the sex in males, also showed by Jit & Singh[4] as they found DP for weight as 23.97 for right clavicle and 35.1 for left clavicle. Study of Shamer Singh & K.C.Gangrade[6,7] also has the similar findings.

In our study the length of clavicle was found to be the best parameter for determining the sex in females. Similar findings were reported by Indar Jit and Daisy sahni[8] as they found 11.25 and 8.75 DP for right and left clavicle in females. Jit and Singh[4] findings are also in accordance of our study as their findings were 13.7 and 11.5 DP for right and left clavicle in female respectively. The mid-clavicular circumference of clavicle was found to be the next useful parameter while determining the sex in females, as shown by Indar Jit and Daisy sahni[8] which is in accordance with the study.

Terry[9] found that the robustness index of the clavicles in Negro males was found to be 26.32 (right) 25.40 (left) while Olivier[10] found that French clavicles have index of 25.0 in males, 22.9 in females. In the present study it is 24.82 (right) and 24.41 (left) in males as compared to22.49 in right female clavicle and 21.89 in left female clavicles. Thus the present study is in accordance with the study of other workers that the robustness index is greater in males than in females.

But DP is different in all the studies conducted in various populations and even in different zones of India as we found 34.65 DP for mid-clavicular circumference in male while Robert J Terry[8] found 39.3 in white Americans and L.J Ray[11] found it 37.8 in Australian aboriginal while Singh[12] found it 39.52 in north Indians.

Thus the data shows that there is a significant difference in some of the mean values of different zones suggesting the need of separate demarking points for different zones if 100% accuracy is required.

CONCLUSION

We conclude that demarking points (DP) vary for each zone and also differ according to sex and side of the bone and thus DP give high degree of accuracy for sexual dimorphism. Mid-clavicular circumference of the clavicle is the best parameter for determination of sex for clavicle in males while length is best in case of female. Also the length, weight, mid-clavicular diameter and robostness index are also useful parameter in sex determination.

REFERENCES

1. Krogman W.M. Skeleton in forensic medicine. Proc. Inst. Med., Chicago. 1946; 16: 154-167.
2. Singh B. and Sohal H.S. Estimation of stature from clavicles of Punjabis. A preliminary report. Indian journal. Med. Research 1952 Jan; 46:(1).
3. Kaur H. Harjeet, Sahani D. and Jit I. Lengths and curves of clavicles in Northwest Indians. J. of Anat Society of India 2002; 51(2): 199-209.
4. Indar Jit and Shamer Singh. Sexing of adult clavicle. IJMR 1966; 54: 551-557.
5. Washburn S.L. Sex differeces in pubic bone of Bantu and Bushman. Am. J. Phys. Anthropology 1948; 7: 425- 432.
6. Singh Shamer & Gangrade K. C. The sexing of adult clavicles verification and applicability of demarking points. J. of Indian Academy of forensic sciences 1968a; 7(1): 20-30.
7. Singh Shamer & Gangrade K. C. Sexing of adult clavicles – demarking points for Varanasi zone. J. of Anat Society of India 1968b; 17(2): 89-100.
8. Indar Jit and Daisy Sahni. Sexing of North Indian clavicles. J. of Anat Society of India 1983; 32(2): 61-72.
9. Robert J. Terry. The clavicle of American Negro. American Journal of Physical Anthropology 1932 Jan- March; 44(3).
10. Olivier G. Anthropologic de, la clavicule III La clavicule due francois Bull,soc Anthrop 1951; 2: 121-157.
11. Ray L. J. Metrical and non-metrical features of clavicles of the Australian Aboriginal. American Journal of Physical Anthropology 1959; 17.
12. Singh S. Jit I., Singh S.P. and Gangrade K.C. Identification of sex from the skeletal remain. Bulletin Institute of Medical Science (BHU) 1972; 3: 65-75.

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