ISSN-0971-720X (Print) • ISSN-0974-1283 (Electronic)

Volume 11 / Number 1 / January - June 2011

Medico-Legal Update

An International Journal

www.medicolegalupdate.org

MEDICO-LEGAL UPDATE

Medico legal update is a scientific journal which brings latest knowledge regarding changing medico legal scenario to its readers. The journal caters to specialties of Forensic Medicine, Forensic Science, D. N. A. fingerprinting, Toxicology, Environmental hazards, Sexual Medicine, Forensic Odontology & Law. The journal has been assigned international standard serial number (ISSN) 0971-720X. The journal is registered with Registrar of Newspaper for India vide registration numbers 63757/96 under Press and Registration of Books act, 1867. The journal is covered by EBSCO database (USA) and by INDEX COPERNICUS, POLAND. The journal is also covered by EMBASE (SCOPUS).

Medico legal update is a quarterly peer reviewed journal. The journal has been assigned E-ISSN 0974-1283 (Electronic version). The first issue of the journal was published in 1996.

Editor

Dr. R. K. Sharma
Aster-06/603, Supertech Emerald Court
Sector – 93 A, Expressway, NOIDA 201 304, UTTAR PRADESH

Published, Printed and Onwed by

Dr. R. K. Sharma
Aster-06/603, Supertech Emerald Court
Sector – 93 A, Expressway, NOIDA 201 304
UTTAR PRADESH

Printed at

Process and Spot
C-112/3, Naraina Industrial Area, Ph-I
New Delhi- 110 028

Published at

Aster-06/603, Supertech Emerald Court
Sector – 93 A, Expressway, NOIDA 201 304
UTTAR PRADESH

MEDICO-LEGAL UPDATE

www.medicolegalupdate.org

Contents

Volume 11, Number 1 Jan. - June 2011

A pilot study of dilated cardiomyopathy (DCM) in western Uttar Pradesh, India: A four year review

Ajoy Deshmukh*, Avnish Deshmukh, Geeta Deshmukh***, Prem K. Garg******

*Assistant Professor, Medicine, Saraswathi Institute of Medical Sciences, Hapur, U.P., **Ex-junior Resident, Muzaffarnagar Medical College, U.P., ***Professor & HOD, Pathology, Saraswathi Institute of Medical Sciences, Hapur, U.P., ****Associate Professor, Pathology, Saraswathi Institute of Medical Sciences, Hapur, U.P.

Abstract

This study was conducted in the Department of Medicine, M.M.C. Muzaffarnagar, U.P, & SIMS, Hapur, UP. India. between April 2006 to June 2010. One hundred cases of dilated cardiomyopathy (DCM) diagnosed echocardiographically were studied. Male to female ratio was observed to be 1.5:1. 48 % patients were above 60 yrs. DCM below 40 years was 09% and was found mainly in females in peripartum period.

Presenting feature were congestive heart failure (90%), acute pulmonary edema (42 %), and thromboembolism (14%). More than half of the patients were smoker (65%) and one third (3o %) were alcoholic.

Echocardiographic findings were, low ejection fraction less than 50 % in all the

Patients and 45 %patients have ejection fraction less than 20 %.

Other associated echo findings were MR (30%), TR (26%), pericardial effusion (09%) and LV thrombus (8%).ECG changes observed were LVH (35%) ST-T changes (90%), LBBB (30%) PVC (50%), VT (10%), AF (5%). Overall mortality observed among D.C.M was 14 %

Betablockers were used both in asymptomatic and symptomatic D.C.M patients whenever there was no definite contraindications.

Cardiac resynchronization therapy (CRT) was not advised in our patients due to their poor socioeconomic status.

Keywords

Dilated cardiomyopathy (DCM), Congestive heart failure (CHF), Arrhythmias, Echocardiography, Cardiac resynchronization therapy (CRT)

Interest of conflict- none

Introduction

Cardiomyopathies are heterogenous group of diseases and are defined as a Myocardial disorders in which the heart muscle is structurally and functionally abnormal, in the absence of coronary artery disease, hypertension, valvular disease and congenital heart disease, sufficient to cause observed myocardial abnormality.

The European society of cardiology working group on myocardial and pericardial diseases has grouped cardiomyopathies into five specific morphological and functional phenotypes – dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM), restrictive cardiomyopathy (RCM), arrhythmogenic Rt.ventricular cardiomyopathy (ARVC) and unclassified cardiomyopathies.

DCM is the commonest type of cardiomyopathy and is characterized by the gradual development of heart failure associated with four chamber dilatation of the heart of unknown cause.[21] Ventricles are more dilated than atrias [2].It affects one or both ventricles [3] and Left ventricle is affected more than rt.ventricle.[4]

Alcohol toxicity, pregnancy associated with nutritional deficiency, genetic defect and post viral myocarditis are considered to be the basis for DCM [5]. DCM was considered to be relatively rare disease but now with advent of echocardiography and its wide availability increasing number of DCM cases are being diagnosed day by day.

Cardiac resynchronisation therapy (CRT) has recently become an additional established treatment for highly selected population of patients of DCM. with NYHA Class III /IV chronic systolic heart failure and L.V. dysynchrony as evidenced by a prolonged Q R S duration beyond 130 ms .The purpose of CRT in patients with Heart failure and L .V. dysynchrony is to optimize arteioventricular conduction and L.V. filling, coordinate right and left ventricular contraction by minimizing interventricular and intraventricular mechanical delay and facilitate interventricular dependence.

Corresponding Address:
Dr. Ajoy Deshmukh MD, DM
E-3, 16B- Shatabdi Vihar, Sector-52, NOIDA.
U.P. India. Drdeshmukh_n17@yahoo.co.in

Materials and methods

Echocardiographically diagnosed cases of dilated cardiomyopathy (DCM) during April 2006 to June 2010 were included in this study.

Diagnostic criteria [21, 3, 4] used in 2D echocardiography study were
1-Global hypokinesia of walls mainly, of Left Ventricle.
2-Dilatation of all chambers of the heart mainly Left ventricle Laboratory clinical, Hematological and Biochemical tests were done to exclude other diseases.

Serological study for viral antibody titre, myocardial biopsy, cardiac catheterization, coronary angiography was not used in the study due to non availability of these facilities.

Results

Total number of 100 patients were studied .Males were 60 % and 40 % females.

Females were mainly in peripartum period.

Results are shown in the following tables and charts.

Majority of DCM cases were complicated by arrhythmias.Laboratory tests did not show any significant finding.Drugs used in DCM were diuretics (95%) Digoxin (75 %) ACE inhibitors (75 %) anticoagulants (3%) and betablockers (75 %)

Discussion

DCM was considered to be rare in the past but with advent of Echocardiographic study and its wide availability, it is increasingly detected nowadays.

One hundred cases of DCM were diagnosed in four years with male predominance (M: F = 1.5:1) .It is mainly disease of middle and

Table 1: Age group distribution & habits in DCM

Age group	Percentage	Habits	Percentage
>60 Yrs	48	Smoking	65
40-60	43	Alcoholics	30
<40	09	Non Smoker& Non Alcoholic	05

Maximum number of patients was elderly persons and smokers.

Table 2 : Presenting features in DCM

Presenting features	Percentage
Congestive Heart Failure	90
Acute Pulmonary Edema	42
Thromboembolism	14

Main presentation of DCM was congestive heart failure.

Table 3: ECHO Findings IN DCM

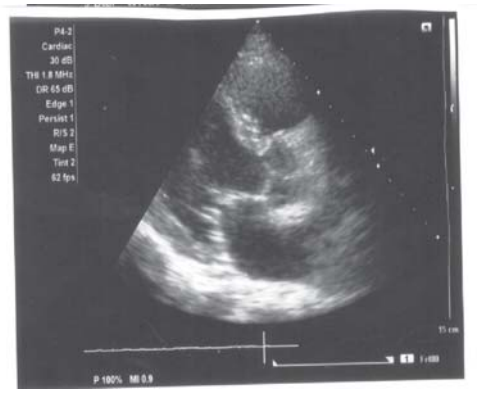
Ejection Fraction (%)	Percentage Findins	Other Echo	Percentage
<20	45	Mr	30
21-30	30	Tr	26
31-40	13	Pericardial Effusion	09
41-50	12	Lv Thrombus	08
>50	None	Lv Systolic Dysfunction	All

Maximum number of patients had mitral regurgitation & all had LV systolic dysfunction.

Table 5: Complications in DCM

Complications	Percentage
Acute Pulm.oedema	45
Arrhythmias	60
Thromboembolism	15
Chest Infection	75

Photograph 1: Parasternal long axis view showing dilated Left atrium, Right atrium and Left ventricle



elderly males .We found 48 % cases of DCM were above 60 years and 09% cases were below 40 years and were mainly young females in peripartum period .However the age range of DCM patients was 8 yrs to 70 yrs.

Dilated cardiomyopathy is characterized by gradual development of cardiac failure associated with four chamber dilatation of heart of unknown cause. Ventricles were dilated more than Atria [2.] DCM can affect one or both ventricles [3]and Lt Ventricle is affected more than right ventricle.

In Western Europe (Denmark 1980-81) overall incidence of DCM was calculated as 7.3 cases per 100,000 populations per year with family history of DCM in 28 %.

Incidence of DCM may be higher in third world countries comprising one third of all cases of CHF [6.]The exact cause of DCM is not known but it is likely that DCM represents a common expression of myocardial damage that has been produced by a variety of un established myocardial insult [3]

It is speculated that an episode of subclinical viral myocarditis initiates an autoimmune reaction that culminates in the development of full blown DCM [7, 8, 9.]

Evidence favouring the concept of post viral disorder includes presence of high titers of viral antibody 10, viral specific RNA sequence and viral particle in patients with idiopathic DCM.

It has been estimated that only 15% patients with myocarditis progress to DCM [10.]and only 5-10 % of patients with DCM have biopsy evidence of myocarditis [8,12,13,14]

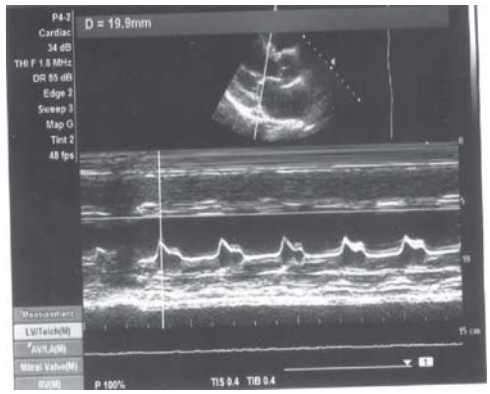
Cardiac biopsy specimens obtained by transvenous or transthoracic

Table 4: ECG Findings in DCM

ECG Findings in DCM	Percentage
ST-T CHANGE	90
LVH	35
LBBB	30
VPC	50
VT	10
AF	05

ST-T changes were found in maximum number of cases followed by VPC.

Photograph 2: 2D and M mode Echocardiography of a Patient showing dilated Lt .ventricle (increased EPSS) with With flat interventricular septum and post .wall in M mode Tracing.



approach demonstrate interstitial fibrosis, cellular infiltrate, cellular hypertrophy and myocardial cell degeneration [15, 16, 17]No virus or other etiological agent has been identified.

Particularly disappointing has been the failure to identify any immunological, histochemical, morphological, Ultrastructural or microbiological markers that could be used for establishing the diagnosis of idiopathic DCM and its cause. Reversible dilated cardiomyopathy may be found with alcohol, pregnancy, selenium deficiency, hypophosphotaemia, hypocalcaemia and chronic uncontrolled tachycardia.[18]

Independent of its important role as risk factor in the development of coronary artery disease, cigarette smoking has been found to be associated with DCM [19.]

In this study 62 % patients were smoker and 30 % patients were alcoholic, 15 % of patients were hypertensive and 09 % of the patients were in peripartum period.

Association of hypertension with DCM could not be explained well, possibility may be failing dilated heart in long standing hypertension may look like DCM in echocardiography and also Hypertension is usually associated with coronary artery disease and ischemic cardiomyopathy may be almost impossible to differentiate in some occasion. Coronary angiography was not done in our study so some ischemic cardiomyopathy may be wrongly diagnosed as cases of DCM.

We detected 09 % of DCM during pregnancy or postpartum period. The exact etiology is not clear but nutritional deficiency (e.g. hypocalcemia, hypophosphataemia, selenium deficiency) associated with multiple pregnancies may be the culprit.

Surprisingly many female patients from well to do families were seen to suffer from CHF during their first pregnancy and found to have DCM. Dilatation usually reverts within few months following delivery but in some cases cardiomegaly fails to recover and they are especially at risk of death during subsequent pregnancies.

Ninety percent patients with DCM had features of CHF and 10% complained of palpitation. Acute pulmonary edema was in 45 %, thromboembolism in 15 % and chest infections in 75 % patients.

High incidence of chest infections in DCM is probably due to real association of DCM with smoking and smoking with chest infection .Other likely reasoning could be susceptibility of infection in CHF and pulmonary edema.

E.K.G .findings observed were ST-T changes in 90 % LVH 35 % LBBB in 30 %. Sixty five percent of Patients had some form of arrhythmias e.g. VPC (50 %) VT (10 %) and AF (5%)

In echocardiography global hypokinesia, dilatation of LV with or without four chamber dilatation was observed most often. 85 % of the patients had Lt. ventricular end diastolic diameter more than 55 mm. EF was less than 20 % in 45 % of cases and none had EF more than 50 % .The E point septal separation is one of the means to quantify the degree of ventricular septal dysfunction [4.] .

The E point septal separation was more than 20 mm in majority of the cases.

Systolic murmurs in DCM are common and are usually due to mitral or less commonly Tricuspid regurgitation. In our study 30% had MR and 26 % had TR 09% pericardial effusion and LV thrombus in 8 % of the patients.

Drugs used in DCM were diuretics (95 %) digoxin (75%) ACE inhibitors (75%) Beta blockers (carvedilol) in 75 % and anticoagulants (3 %)

Whenever there was no definite contraindication, Beta blockers were used both in symptomatic and asymptomatic DCM patients as neurohormoral blockade with Betablockers may reverse ventricular remodeling, prevent progression to severe symptomatic HF and improve clinical out come.

Conclusion

All patients of breathlessness must be subjected to echocardiographic study. Many patients

Clinically diagnosed as cor pulmonale, Rh. heart disease and ischemic heart disease came out to be cases of DCM after echocardiography. As the management of DCM and its prognosis is different from other cardiac ailments, the exact diagnosis is essential before starting the treatment. However echocardiography may wrongly diagnose some cases of ischemic cardiomyopathy as DCM .Cardiac catheterization , coronary angiography, myocardial biopsy are better for firmer diagnosis .Betablockers have been underused because of the perceptions of adverse events, clinical worsening in patients with decompensate heart failure .Blockade of neurohormonal activation with betablockers is important in retarding heart failure progression and death .

Though cardiac resynchronization therapy has emerged as an effective treatment that improves symptoms, exercise capacity and quality of life in most patients with advanced CHF who are refractory to optimal medical treatment.[1], this therapy was not advised to our patients due to their poor socioeconomic status.

As intractable ventricular arrhythmias and sudden death is one of the presentations of DCM. It may be of medico legal significance and autopsy should be done to rule out DCM in cases of sudden death.

References

1. Stellbrink c., Breithardt O, Franke A, et al, Impact of cardiac resynchronization therapy using thermodynamically optimized pacing on left ventricular remodeling in patients with congestive heart failure and ventricular conduction disturbances. J. American Cardiolo. 2001;
2. RobertsW.C, Siegel R.J., and Mc Manus B.M: idiopathic dilated cardiomyopathies Analysis of 152 necropsy patients. American J.Cardiol.60:1340, 1987.
3. WynneJ., Braunwald E, The cardiomyopathies and Myocarditis: Toxic Chemical and physical damage to the heart. In Braunwald E(Ed) Heart disease, W.B.Saunders Company Philadelphia, 43:1398-1402, 1992
4. Fiegenbaun H: Diseases of Myocardium In, Echocardiography, Lea & Febiger Philadelphia l9: 527-531, 1986
5. Cotran R.S.Kumar V., Robbins S.L the Heart in, Robbin's Pathological basis of Diseases, W.B Saunders Company, Philadelphia 642-644, 1989
6. Littler W.A; Dilated Cardiomyopathy. In Julian D.G ., Camma A.J.Fox K.M, Hall R.J.C., Poole –Wilson P.A.(ed.): Diseases of the Heart ,ELBS bailliere Tinadall ,Great Britain,37 ; 916—1921 ,1991
7. Muir P., Nicholson F., Tilsey A J et al: Chronic relapsing pericarditis and dilated cardiomyopathy; Serological evidence of persistent enterovirus infection .Lancet 1: 804, 1989
8. O' Connel J.B. and Mason J.W.: Immunosuppressive therapy in experimental and clinical myocarditis .Pathol.Immunopathology .Res.7:292, 1988.
9. Shabatdi R.: Myocarditis and dilated cardiomyopathy; Twins or Distant relative? Cardiology, 76: 332, 1989.
10. O' Connel J.B: Immunosuppression for dilated cardiomyopathy (editorial) N.Engl. j.Med, 3421: 1119, 1989.
11. Bowles N.E., Rose M.C.Taylor P., et al: End stage dilated Cardiomyopathy: Persistence of Enterovirus RNA in myocardium at cardiac transplantation and lack of immune response.Circulation 80: 1128, 1989
12. Fallon J.T: Myocarditis and dilated cardiomyopathy: Different stages of same disease? Cardiovasc.clinics 18:115, 1988.
13. Maisch N., Bauer E., Jufnagel G .et al: The use of endomyocardial biopsy in heart Failure .Eur.Heart .J.9:59 1988.
14. Popma J .J. Cigaroa R.G., Duja L.M and Hillis L.D.: Diagnostic and prognostic utility of rt .sided catheterization and endomyocardial biopsy in idiopathic dilated cardiomyopathy Am.J.Cardiol.63:955, 1989.
15. Tazelaar H.D and Billingham M.E; Leukocytic infiltrate in idiopathic dilated cardiomyopathy. A source of confusion with active myocarditis .Am.J.Surg.Pathol.10: 10: 405 1986.
16 Edwards W.D, Caiomyopathies .Hum.Pathol.18: 625, 1987
17. Schopper J., Froede R., hein S., et al Impairment of the myocardial ultra structure and Changes of cytoskeleton in dilated cardiomyopathy,Circulation 83,504,1991
18. Wynne J., Braunwald E.: The cardiomyopathies and myocarditis In Isselbacher K.J.,Braunwald E., Wilson J.P.,.Martin J.B.Fauci A.S. (ed) Harrison 'Principles of Internal Medicine .McGraw Hill ,Inc,USA 205:1088-1090,1994.
19. Hartz.A.J. Anderson A.J., Brooks H.L., et al: The association of smoking with cardiomyopathy N.Engl. J.Med.311:1201, 1984.
20. Rabko P.S. and orie J.E.: The clinical presentation and laboratory evaluation of congestive and ischemic cardiomyopathies Cardiovasc.Clin.19:75, 1988.
21. Editorial: Natural history of DCM Lancet, 1:248, 1986

Epidemiological study of non fatal road traffic accidents in Rohilkhand Region

Ajit Singh[1], Anchit Goel[2], Shekhar[3]

[1]Department of Orthopaedics, Rohilkhand Medical College, Bareilly, [2]7th Semester MBBS Student, Rohilkhand Medical College, Bareilly, [3]Department of Physiotherapy, Jaipur College of Physiotherapy, Jaipur

Abstract

The present cross-sectional study evaluates the prevalence and associated epidemiological factors in non fatal Road Traffic Accident (RTA) cases in Rohilkhand region of India. A total of 195 non-fatal RTA cases reporting to orthopedic, surgery or casualty departments of Rohilkhand Medical College and Hospital during 1st March, 2010 to 1stJune, 2010 were included for the purpose of present study. All victims were thoroughly interviewed and followed up-to 30 days where necessary.

In non-fatal RTAs', there were a total of 205 cases of non-fatal road accident victims out of which 195 cases agreed to participate in this study, thus giving a response rate of 95%. Male outnumbered females and male/female ratio was approximately 2.5:1. The maximum number of RTA were recorded in the age group of 25- 45 years accounting for maximum 76(38.97%) of total RTA. Majority of victims were motorcyclist 66 (33.84%). A maximum number of cases were reported on ordinary roads 115 cases (58.97%). There were maximum cases reported of lower extremities injuries 89(45.64%) with fractures of long bones the commonest type of injury. Majority of RTA occurred during daytime. Regarding mode of accident, the sideways accidents were found to be the commonest. Majority of victims showed low level of education, 71% victims were educated up-to 10th standard with 28% being illiterate. More accidents were seen among middle and low socio-economic group of people. 31.67% drivers had no valid license and all of them were two-wheeler motorcycle drivers. None of the drivers or occupants of any motorized two wheeler vehicle was using helmet at the time of the accident.

Non-fatal RTAs' are associated with multiple preventable risk factors. Road traffic injuries affect mainly young males in active and productive period of life causing enormous loss to nation.

Keywords

Epidemiological study; Non-fatal RTA; Road Traffic Accidents.

Introduction

Road Traffic injuries have emerged as a major cause of public health problem in the last decade[1]. According to World Health Organization report, 2004, if the current trend continues Road Traffic Injuries will be the third leading contributor to the global burden of disease by the year 2020[2]. Road traffic accidents are the leading cause of death for young people worldwide between ages 15-45, and kill 1.2 million people each year – the majority in Asia and Africa[1]. There are about 20 million and 50 million non-fatal injuries every year and is important cause of disability. In addition to death and injury, traffic accidents in these countries cost $65 billion to $100 billion annually[1]. These costs include loss of income and the burden placed on families to care for their injured relatives. South East Asia has one-third of Road Traffic fatalities occurring in the world. Southeast Asia is at particular risk because its rapid economic development resulted in a steep increase of motorized vehicles. The situation in India is also alarming, needing the attention of the decision makers. In India 1 .06 lakhs deaths and 4.5 lakh injuries occur due to road traffic accidents annually which are nearly 10% of the total road traffic deaths in the world[3].

Accurate statistics are crucial for understanding the state of road safety and measuring the impact of efforts to improve it. The paucity of surveillance data leads to under-estimates of the size of the actual problem. Data on fatal injuries are easier to ascertain and analyze, but deaths are a small fraction of the overall burden of injury, i.e. the tip of the injury pyramid. Despite considerable human and economic consequences, non-fatal RTA injuries in India have received scant attention. Research in this field is challenged as it depends on to which health care unit the victim reports for treatment and the lack of a single agency that has the responsibility of collecting information on all non fatal RTA[4]. The aim of this study was to describe the characteristics of patients who had non-fatal traffic injuries and to consider the relationship between the injured patients' characteristics and the severity of their injuries and to identify risk factors associated with nonfatal traffic accidents in Rohilkhand region of northern India.

Corresponding Author:
Dr. Ajit Singh
*Department of Orthopaedics , Rohilkhand Medical College , Pilibhit by pass road, Bareilly, U.P. 243006, Phone Numbers: 09319930079 , 05812525018, Fax numbers : 0581-2526152, E-mail : ajitsingh2409@ gmail.com

Materials and methods

This cross sectional study was carried out in Rohilkhand medical college, Bareilly - a rural based tertiary care teaching hospital in Uttar Pradesh, northern India. It was conducted from a time period of 1, March 2010 to 1, June 2010 on convenient based sampling method. Institutional ethical committee reviewed and approved the present study. All non fatal and non comatose cases of road traffic accidents victims reporting to casualty , orthopedics and surgery out patient department were included in this study. In India deaths occurring within 30 days of RTA are considered as being due to road traffic injury and all such cases were excluded from the study.

For the purpose of study, a road traffic accident was defined as any vehicle accident occurring on a public highway or road and which takes place between two or more objects one of which has to be any kind of moving vehicle. A vehicle is defined as any mechanically or electrically powered devices not operated on rails and include cars, buses, trucks vans, motorcycles and bicycles.

The victims/relatives of RTA cases who attained either out patient clinic or admitted in the wards were interviewed to obtain the information about the circumstances leading to the accident. The victims and relatives (in case of unconscious patients) who did not consent to be a part of the study were excluded. The information collected consisted of nature of injuries, category of road users day and time of accidents, treatment outcome and other demographical variables. The collected data was analyzed using SPSS® 17 version.

Results

There were a total of 205 cases of non-fatal road accident victims, out of which 195 cases agreed to participate in this study, thus giving a response rate of 95%. Male outnumbered females and male/female ratio was approximately 2.5:1. The gender distribution in the different categories is as shown in Figure 1. There were 139 (70.91%) males and 56 (29.01%) females. The ages of the victims ranged from 1 year to 81 years and the ages were divided into six groups starting from 0-10 years up to 65+ age groups. The maximum number of RTA were recorded in the age group of 25- 45 years accounting for maximum

Figure 1: Distribution of Non-fatal RTA cases on basis of gender

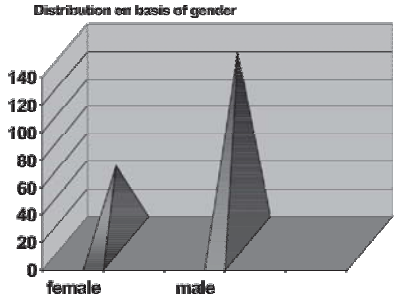


Figure 2:

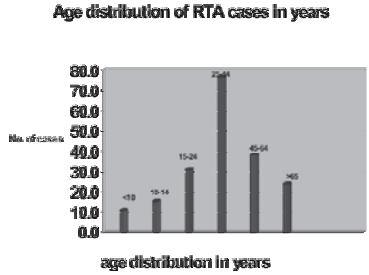


Table 1: Showing different types of Road users involved in RTA

TYPE OF ROAD USERS	N(NO. OF CASES)	%
PEDESTRIANS	23	11.70
BICYCLIST	49	25.12
MOTOR- CYCLIST	66	33.84
VEHICLE OCCUPANTS	35	17.94
OTHERS	14	7.00
UNKNOWN	8	4.10

Table 2: Showing number of accidents in relation to types of Roads

Types of roads	No.	%
Highway	44	22.5
Ordinary Roads	115	58.97
Lane	16	8.20
Other places	20	10.20

Table 3: Site of injury of RTA victims.

S.NO.	PART RECEIVING INJURY	No.	%
1.	HEAD AND NECK	26	13.33
2.	UPPER LIMB	46	23.58
3.	CHEST	6	3.07
4.	ABDOMEN	8	4.10
5.	BACK AND SPINE	6	3.07
6.	PELVIS	14	7.10
7.	LOWER EXTRIMITY	89	45.64

Table 4: E ducational status of the victims.

Education level	No.	%
Illiterate	55	28.2
Primary(5th class)	24	12.30
Junior high school(8th class)	34	17.43
High school(10th class)	26	13.33
Intermediate(10+2)	25	12.82
Graduate and above	31	15.89

Table 5 : Socioeconomic status of RTA victims.

Socioeconomic class	No.	%
Upper	11	5.64
Middle	105	53.84
Lower	79	40.52

Table 6 : Time of injury of RTA victims.

Time of injury	No.	%
Day(6:00am -6:00pm)	109	55.897
Night(6:00pm-6:00am)	76	38.97
Not known	10	5.12

Table 7 : Mode of accident of non-fatal RTA victims.

Mode of accident	No.	%
Sideways accident	90	46.15
Head on collision	51	26.15
Others	54	27.69

76(38.97%) of total RTA whereas 62(31.79%) were recorded for above 45 years. Only 24(12.30%) cases were reported in the age group above 65 and there were 26(13.33%) children's below the age of 15 years [Figure 2]. Majority of victims were motorcyclist 66 (33.84%) followed by bicyclists 49 (25.12%) followed by pedestrians 23(11.79%)[Table 1]. Table-2 shows distribution of accidents in relation to types of roads. A maximum number of cases were reported on ordinary roads 115 cases (58.97%) followed by highway roads 44(22.5%), only 16 (8.20%) occurred in lanes [Table 2]. There were maximum cases reported of lower extremities injuries 89(45.64%) followed by upper limb injuries 46(23.58%) then by head and neck injuries 26(13.33%) then back and spine injuries 14 (7.10%). The least common were of abdomen and chest [Table 3]. There were 75(38.40%) cases reported from urban population and about 120(61.53%) cases reported from rural population. Among 195 victims who were interviewed, 12.3% had education up to 5th class (Table 4), 17.4% were educated up to 8th class while 28.2% were illiterates. Victims with a higher education (matriculation and above) were fewer in proportion. More accidents were seen among middle and low socio-economic group of people (Table 5). Majority of RTA occurred during daytime (Table 6). Regarding mode of accident, the sideways accidents were found to be the commonest (Table 7). Of the 109 drivers involved in RTAs, 49 were bicyclists and others who do not require a license. Of the remaining 60 drivers, 19 (31.67%) drivers had no valid license and all of them were two-wheeler motorcycle drivers. None of the drivers or occupants of any motorized two wheeler vehicle was using helmet at the time of the accident.

Discussion

There is a hidden epidemic on the world's roadways[4]. As developing country vehicle use rises, road traffic injuries are also growing. With exploding population, increasing registration of automobiles every month, rampant encroachment of roads, nasty tendency of violating traffic rules and chaotic traffic systems have greatly contributed rapid strides in road traffic accidents[5].

To best of our knowledge this is first study on non-fatal RTA in Rohilkhand region. The findings of this study revealed that the highest percentage (38.97%) victims were in the age group of 25-44 years. Earlier studies have also reported high incidence of road traffic injuries in similar age groups[4, 5]. This cause's double loss to the nation as this is the young productive age group leading to loss of man-productive days and financial expenditure is incurred in their treatment. It was noticed that age below 10 years and above the age of 50 there is a decreased tendency in accident cases. The reason for this is that the children are taken care by elders and elders are less mobile.

Males are the predominant victims because males being the bread earners for the family besides they are more involved in activities such as driving and traveling are therefore, involved usually in outdoor

activities exposing themselves to accidents. In our society, females are less active and mostly remain indoors mostly due to cultural background and low potential for employment rate owing to poor literacy, along with tendency that some male members mostly accompany females and extra precautions are taken on roads. These are reasons for their less morbidity in RTA. Our findings are in general agreement with those of others[4, 5, 6]. We found a male: female ratio of 2.5:1 which is lower than high ratio of 6:1 and 9:1 reported in previous studies[4, 5]. This is probably due to the fact that many of our victims were from hilly areas where larger percentage of working females are engaged in outdoor activities. Also with increasing modernization more number of females are taking up outdoor employment and this may be one of the factors responsible for increased female morbidity.

Most common mode of transportation involved in road traffic accidents in this study was motorcycles 66(33.84%). It is popular and considered as cost effective vehicle in middle and low income community population and this may also explain the fact that most of the RTA victims were of middle and low socioeconomic status. Similar findings were also observed in studies carried out in other parts of the country[4]. It has been reported that more accidents were seen among low socio-economic group of people[7], but in this study maximum number of cases were from middle class. Thus majority of patients who sustain traffic injuries are young male motorcyclists. The exposure of motorcyclists, their speed, their limited driving experience and their risk-taking behavior are some of the factors related to preventable injury.

People from rural areas suffered more 120(61.53%) as compared to urban people 75(38.40%).This is because rural population are more illiterate , unawareness of traffic laws and having poorly built roads in rural community. This is partly also because of the fact that most of the cases reporting in our hospital are from rural area. Probably because of these factors it was observed that more people with lower levels of education were RTA victims. Similar result were also observed by others[5, 7]. However, this relationship between education and RTA may not be causal. When temporal distribution of the accidents was studied it was observed that most occurred in the day time. This reveals the common out-door working time of the region with increased activities on road during daytime such as commercial activities and attending schools, colleges and offices. This is in agreement with previous studies[4, 5, 7].

Present study depicts that largest number (58.97%) accidents took place on ordinary roads and least number in lanes. This can be explained on the basis that these are the busiest roads with heavy traffic loads, lack proper footpath facility, are crowded and congested with lot of encroachment, and drivers, especially motorcyclists are tempted to drive at high speed. On the other hand, lanes allow fewer numbers of vehicle hence reduced incidence of RTA. Studies on fatal RTA have found that maximum number of fatalities occur on highway roads[8].

The commonest injury was fracture of bones .This was probably due to interplay of gravitational force and velocity of vehicles at the time of accidents. The commonest injury was that of the lower limb 89(45.64%) followed by the upper limb 46(23.58%).the reason was that the mobile activities are performed by the lower limb. It is the most common part which is used in driving and most common part which comes in contact with other vehicles during injury. Motorcyclists are more exposed to injury than other vehicle users. This is due to the higher speed, which can be achieved over short distances and less stability of the vehicle. These findings are in conformity with previous research[4, 7]. The high percentage of patients with face, head, neck and spine injuries (about 17%) is concerning because of the possibility of neurological insults, which may lead to death or the need for long-term care. However, as the number of patients who required admission was small and the details of their injuries were vague, the degree of neurological impairment or the severity of head injury could only be surmised.

This study found 31.7% drivers of different vehicles were without driving license, which is higher compared to 7-15% found in previous studies[7, 8]. The reason may be the easy accessibility of the vehicles and the casual attitude of drivers towards obtaining licenses because of lesser checking of vehicles in rural areas by traffic police. When they were interviewed they mentioned that they would be applying for obtaining the license in future. No protective gear was used by any of the victims in this

Study, because laws regarding helmet use are not strictly enforced in this pat of country.

The commonest mode of sustaining injury was sideway accident accounting for as many as 46% of victims. Similar results were also observed in previous studies from central India[4]. Falling from a moving vehicle, knocked down by a moving vehicle and collision between two vehicles were other modes of injury.

The generalizability of the study results is limited by the characteristic of the sample, which was recruited from a single medical college. Another limitation of this cross sectional study is cause-effect association between demographic variables and RTA cannot be determined from the study. Studies that rely on routine hospital records have limited validity and reliability[9]. Information on some aspects of non-fatal injury was not gathered because data were either scanty or not uniformly recorded: these aspects included the number of pillion passengers, the road conditions, drivers experience, weather conditions, psychosocial conditions, physical fitness of drivers, the number and type of parties involved in the injury, and the use of drugs and alcohol.

Recommendations

On the basis of study findings it is recommended that:

1. Strict licensure procedure should be followed. Licensing authorities should adopt stricter, more comprehensive and scientifically based test laying a stress on road rules, and regulations.
2. There should be deployment of more number of traffic police and regular checking of license and strict enforcement of traffic rules should be done.
3. Road safety education is an area of high priority and it should be directed towards road users, who are frequently involved and injured in RTAs (e.g. young students). Minimum level of education regarding road safety should be imparted especially to the young age group during giving license.
4. An integrated programme of road safety education must be taken up by government and non governmental organizations. School children may be introduced to the concepts of road safety.
5. Efforts should be made to minimize congestion on road particularly during daytime with the help of traffic police.
6. Preventing measures like avoiding high speeding and driving under the influence of alcohol; promoting the use of helmets, seat belts and other restraints must be ensured.
7. Measures should be taken to improving the design of roads and vehicles for better road safety. Setting safety standards for vehicles, especial ly motorcycles are required.

There is no single method that will prevent road traffic accidents, what is required is an organized team work by people in many disciplines like education, engineering, medical, law enforcement agencies for effective prevention of road accidents.

References

1. M Peden, R Scurfield, D Sleet, D Mohan, A A Hyder, E Jarawan, C Mathers. World Report on Road Traffic Injury Prevention Geneva: World Health Organization, 2004.
2. Goff Jacobs and Amy Aeron-Thomas. Estimating global road fatalities, Accessed http://www.grsproadsafety.org/activities/reports/5/50.pdf on June 22, 2010.
3. World Health Organization, Regional Office for South-East Asia. Regional report on status of road safety: the South-East Asia Region. New Delhi, 2009.

4. Ganveer GB, Twiari RR. Injury pattern among non-fatal road traffic accident cases: A cross-sectional study in Central India. Indian J Med Sci 2006 Oct 6;59:9-12.
5. Sharma S, Singh S, Sinha US, Kapoor AK. An epidemiological study of non-fatal road traffic accidents cases in Allahabad region, India. Indian Internet Journal of Forensic Medicine & Toxicology 2006; 4(4). Accessed: http://www.indianjournals.com/ijor.aspx?target=ijor:iijfmt&type=archive.
6. Jha N, Agrawal CS. Epidemiological study of Road Traffic Accident Cases: A Study from Eastern Nepal. Regional Health Forum WHO South-East Asia Region 2004; 8 (1).
7. Jha N, Srinivasa DK, Roy G, Jagdish S. Epidemiological study of Road Traffic accident cases: A study from South India. Ind J Commun Med 2004; 29: 1-8.
8. Singh H, Dhattarwal SK. Pattern and distribution of injuries in fatal road traffic accidents in Rohtak (Haryana). J Ind Aca Forensic Med 2004; 26: 20-23.
9. Beaglehole R, Bonita R, Kjellström T. Basic epidemiology. Geneva: World Health Organization,1993.

Arcus senilis – An indicator of age

S.S.Oberoi*, R.K.Gorea**, Hardev Singh***, Parminder Singh****, A.D. Aggarwal*****

*Associate Professor, Forensic Medicine, Government Medical College, Patiala, **Professor & Head, Forensic Medicine, Gian Sagar Medical College, Banur, Patiala, ***Associate Professor, Ophthalmology, Government Medical College, Patiala, ****Medical Officer, Punjab Civil Medical Services, Jalandhar, *****Assistant Professor, Forensic Medicine, Post Graduate Institute of Medical Sciences, Rohtak

Abstract

The findings suggestive of advancing age in adults include fusion of skull sutures, sternum, pubic symphysis, arcus senilis, elasticity of skin in sun protected areas, evidence of osteoarthritis, gingival shrinkage, greying and thinning of hair, cataracts, kyphosis, etc. In this study, the frequency and size of arcus senilis were found to be positively associated with age, but provided no useful guide to the determination of age. Age should be estimated through physical examination; life history, matching local or national events with personal milestones; and existing nonformal documents. Individual ageing features have a poor positive predictive value for age, but in combination, are suggestive of advancing age.

Keywords

Arcus senilis, incidence, grading, density, age estimation.

Introduction

Many adults do not have documents of birth, either because they have been lost or because there has been no routine recording of birth. In Western countries and now-a-days in India, date of birth is used as a basic identifier, and access to services and support tends to be age regulated. Doctors are not infrequently asked to write formal reports estimating the true age of adults; however, there are no existing guidelines to assist in this task. Determination of age in the living person is a baffling problem and is very important in medicolegal cases especially in old age. As the person grows older, study of sternum, public symphsis and the closure of sutures of skull helps us in estimating the age.

Like other body parts, age related changes also occur in cornea inlcuding change of curvature, diameter, aspherity, thickness, etc. Arcus senilis is a deposition of lipids at the corneal periphery and is dependent on vascularity for formation and is not a degenerative change. Arcus senilis needs accurate measurement of its width with digitizier and biomicroscopic examination of cornea.[1,2] The decrease in clarity seen in all corneal layers with increasing age corresponds with both the decrease in epithelial luster and the increased stromal relucency that is known to occur in the aging eye. It has been demonstrated that a linear decrease in both anterior and posterior keratocyte density is a function of age.[2]

The prevalence of arcus increases with age, and it appears more in blacks and males.[3] The frequency and size of arcus senilis are positively associated with age; there is a positive corelation between the size of corneal arcus and the level of cholesterol and low density lipoporotein in males; and that there is negative corelation between corneal arcus and diastolic blood pressure in both sexes.[4] Arcus senilis appears to be related to cardiovascular diseases but is no reliable predictor of the disease. It has been found that there is no definite corelation between incidence of arcus senilis width and age.[3]

Arcus senilis appears to be related with alcoholism and also with the levels of potassium, sodium, calcium, glycosated heamoglobulin, vascular disease, myocardial infarction, weight of the body, smoking, education, geographic areas, family history, contraceptive drug usage, sex hormone levels, menopausal history, body mass index, race and strain on the eyes.[5]

The presence and pattern of arcus senilis are visible indicators of age. Greying of hair and arcus senilis occur with advancing age. This study was done to find out whether there is any definite correlation between arcus senilis with age or not; focussing on finding a useful relationship between changes of arcus senilis with age in the age group of 40-70 years. This can be of great value for poor patients in remote areas and rural dispensaries where no facility of investigation is present.

Aims and objectives

1. To determine the incidence of arcus senilis in the age group of 40-70 years
2. To generate data for age estimation using the age related changes of arcus senilis

Material and methods

The present study comprised of 500 cases in the age group of 40-70 years of either sex taken randomly. The complete data of the persons was collected including proof of age and filled on a proforma. The persons having diabetes melitus, history of trauma to eye and history of ocular surgery; and with no documentary proof of age were excluded from the study. Each subject was examined for the presence of arcus senilis using the unaided human eye in adequate light. An arcus senilis was said to be present if the cornea showed an opaque arc (which is greater than a quarter of the circumference of the cornea) or ring in either or both eyes. Arcus senilis was graded according to the position it is present in clockwise pattern.

- Grade 0 No evidence of arcus senilis
- Grade 1 Arcus senilis 11'o clock to 1'o clock
- Garde 2 Arcus senilis 10'o clock to 2'o clock
- Grade 3 Arcus senilis 9'o clock to 3'o clock
- Grade 4 Arcus senilis 8'o clock to 4'o clock
- Grade 5 Arcus senilis 7'o clock to 5'o clock
- Grade 6 Arcus senilis complete

Observations

Discussion

The results of our study are also similar with study of Pe'er et al[4] which found that the size and frequency of arcus senilis is positively correlated with age. The present study shows that arcus senilis is absent in 33.33% cases in the age group of 40-45 years and 3.23% in the age group of 56-60 years. Arcus senilis is present in 100% cases in age group of 61-70 years. Arcus senilis is complete in 3 out of 144 cases in the age group 40-45 years and in 22 out of 30 cases in the age group of 66-70 years. Present study shows that the development of arcus senilis starts at 12 o'clock sector of the peripheral cornea. It expands sideways nasally and temporally symmetrically. These results are similar to the study of Phillips et al[6].

A high density of the same was observed in the age group of 66-70 years (24 out of 30 cases). These results show that the frequency and density of arcus senilis is positively corelated with age and is similar with the study of Phillips et al[6]. Further, age is highly significant for frequency and density of arcus senilis. But arcus senilis is of little value in estimation of age; however, it may be used in conjunction with other factors. A recent study has found that greying of the hair, skin inelasticity, arcus senilis, and baldness were not predictors of mortality as a measure of biological age.[7] A study amongst London Civil Servants aged from 37 to 58 years of age found that arcus senilis may be a

Table 1: Age distribution of grades of Arcus senilis

Age	Total cases	Grade of arcus senilis													
		0		1		2		3		4		5		6	
40-45	144	48	33.33%	29	20.14%	42	29.17%	18	12.50%	2	1.39%	2	1.39%	3	2.08%
46-50	144	12	8.33%	38	26.39%	26	18.06%	40	27.78%	10	6.94%	2	1.39%	16	11.11%
51-55	88	4	4.55%	16	18.18%	22	25.00%	32	36.36%	2	2.27%	1	1.14%	11	12.50%
56-60	62	2	3.23%	4	6.45%	4	6.45%	10	16.13%	8	12.90%	6	9.68%	28	45.16%
61-65	32	0	0.00%	0	0.00%	8	25.00%	10	31.25%	2	6.25%	2	6.25%	10	31.25%
66-70	30	0	0.00%	0	0.00%	0	0.00%	2	6.67%	2	6.67%	4	13.33%	22	73.33%
Total	500	66	13.20%	87	17.40%	102	20.40%	112	22.40%	26	5.20%	17	3.40%	90	18.00%

P <0.001 highly significant

Table 2: Arcus senilis according to built

Built	Total cases	Grade of arcus senilis													
		0		1		2		3		4		5		6	
Thin	48	8	16.67%	8	16.67%	8	16.67%	8	16.67%	2	4.17%	2	4.17%	12	25.00%
Average	350	48	13.71%	66	18.86%	66	18.86%	84	24.00%	24	6.86%	11	3.14%	51	14.57%
Obese	102	10	9.80%	13	12.75%	28	27.45%	20	19.61%	0	0.00%	4	3.92%	27	26.47%

P >0.05 not significant

Table 3: Arcus senilis according to nourishment

Nourish ment	Total cases	Grade of arcus senilis													
		0		1		2		3		4		5		6	
Poor	2	0	0.00%	0	0.00%	2	100.00%	0	0.00%	0	0.00%	0	0.00%	0	0.00%
Moderate	276	26	9.42%	56	20.29%	60	21.74%	57	20.65%	14	5.07%	13	4.71%	51	18.48%
Well	222	40	18.02%	31	13.96%	40	18.02%	55	24.77%	12	5.41%	4	1.80%	39	17.57%

P >0.05 not significant

Table 4: Arcus senilis according to sex

Sex	Total cases	Grade of arcus senilis													
		0		1		2		3		4		5		6	
Male	330	40	12.12%	55	16.67%	64	19.39%	84	25.45%	20	6.06%	8	2.42%	59	17.88%
Female	170	26	15.29%	32	18.82%	38	22.35%	28	16.47%	6	3.53%	9	5.29%	31	18.24%

P >0.05 not significant

Table 5: Arcus senilis according to area

Area	Total cases	Grade of arcus senilis													
		0		1		2		3		4		5		6	
Urban	328	42	12.80%	59	17.99%	66	20.12%	68	20.73%	20	6.10%	9	2.74%	62	18.90%
Rural	172	24	13.95%	28	16.28%	36	20.93%	44	25.58%	6	3.49%	8	4.65%	28	16.28%

P >0.05 not significant

Table 6: Arcus senilis in hypertensive persons

Disease	Total cases	Grade of arcus senilis													
		0		1		2		3		4		5		6	
Hypertension	21	1	4.76%	2	9.52%	3	14.29%	6	28.57%	1	4.76%	1	4.76%	7	33.33% P

<0.05 significant

Table 7: Density of arcus senilis

Age/Sex	Total cases	Density of arcus senilis					
		Absent		Low		High	
40-45	144	48	33.33%	81	56.25%	15	10.42%
46-50	144	12	8.33%	100	69.44%	32	22.22%
51-55	88	4	4.55%	53	60.23%	31	35.23%
56-60	62	2	3.23%	20	32.26%	40	64.52%
61-65	32	0	0.00%	10	31.25%	22	68.75%
66-70	30	0	0.00%	6	20.00%	24	80.00%
Total	500	66	13.20%	270	54.00%	164	32.80%
Male	330	40	12.12%	175	53.03%	115	34.85%
Female	170	26	15.29%	95	55.88%	49	28.82%

marker of biological rather than chronological age.[8]

In the age of 40-70 years, 87.88% males and 84.71% females show arcus senilis. The high density of arcus senilis is present in 34.85% males and 28.82% females in the age group of 40-70 years. Thus the frequency and density of arcus senilis is not significant for sex. This is in agreement with the study by Pe'er et al[4] who found that corneal arcus is more frequent in males and with study of Patterson[3] who found that the arcus senilis appears more in males. Our study also agrees with study of Karoly and Balazsy[9], who found that occurrence of arcus senilis, is significantly higher in males, but it increases in strong correlation with age in both sexes.

A general absence of corelative significance of cornea with built, nourishment, sex and area of living has been observed. The built is not significant for presence of arcus senilis. Present study shows that the percentage of high density arcus senilis is higher in obese than that of average built persons and thin built persons, but it is not significant statistically.

Arcus senilis has been found to have an association with hypertension ($p < 0.05$). Similar findings have been reported by larger studies including hypercholesterolemia, xanthelasmas, alcohol, cigarette smoking, diabetes, age, and coronary heart disease. Nevertheless, it is not clear whether or not corneal arcus is an independent risk factor for coronary heart disease.[10]

Conclusions

A comparison of the incidence of arcus senilis with age was made in middle-aged persons and no statistically significant difference in incidence was found. In agreement with previously published observations there was a progressive rise and strong association of corneal arcus with increasing age.[11] The present study shows that age is highly significant for arcus senilis however we cannot estimate age of a person from arcus senilis alone; and the arcus senilis may complete at the age of 40 years and may not even start at the age of 60 years. Further nourishment is also found to be a significant factor for the presence and density of arcus senilis; being inversely corelated. The sex and built are not significant determinants for presence of arcus senilis and density of arcus senilis. The main conclusion from this study must be that the presence or absence of an arcus senilis in middle-aged persons gives no useful guide to the determination of age.

Morphological markers of advancing chronological age tend to be variable and nonspecific, at best providing support for minimum age estimations. The phenomena we typically associate with aging (greying of hair, arcus senilis and loss of skin elasticity) are complex phenomena that also reflect genetic and environmental patterns and occurrence of illness. Although there is little data on cross cultural differences, some of these phenomena may not occur at the same ages across different settings.[12] Arcus senilis, appears to occur at an earlier age in African Americans, than in Caucasian Americans, but this may be confounded by smoking or hypercholesterolaemia. There is no correlation between arcus width and increasing age.[3]

Accurate estimation of age for living adults is of central importance in ensuring access to correct services and medical care. Adults from countries with fragile civil infrastructure often have incorrect dates of birth accorded by clerical error, which they may need to change. Doctors should not rely on physical examination alone to estimate age, as there is significant interpersonal and intercultural variability in the relationship between morphological characteristics of aging and age. A combination of physical examination, careful life history, and corroborating documents is likely to produce a more accurate estimation of age. Accuracy of age estimation should be subject to three tests: biological plausibility, historical plausibility, and corroboration from reputable sources.[12] Estimates of age made by forensic scientists tend to underestimate the ages of older people, and overestimate the ages of younger people.[13]

References

1. Friedlander MH, Smolin G. Corneal Degenerations. Ann Ophthalmol 1979;21:1485-95.
2. Kotulak JC, Brungardt T. Age-related changes in the cornea. J Am Optom Assoc 1980;51:761–5.
3. Patterson L. Arcus senilis: an important forensic physical finding. Am J Forensic Med Pathol 1982;3(2):115-8.
4. Pe'er J, Vidaurri J, Halfon ST, Eisenberg S, Zauberman H. Association between corneal arcus and some of the risk factors for coronary artery disease. Br J Ophthalmol 1983;67:795-798
5. Ewing JA, Rouse BA. Corneal arcus as a sign of possible alcoholism. Alcohol Clin Exp Res 1980;4(1):104-6.
6. Phillips CI, Tsukahara S, Gore SM. Corneal arcus: some morphology and applied pathophysiology. Jpn J Ophthalmol 1990;34(4):442-9.
7. Bulpitt CJ, Antikainen RL, Markowe HL, Shipley MJ. Mortality according to a prior assessment of biological age. Curr Aging Sci 2009;2(3):193-9.
8. Bulpitt CJ, Shipley MJ, Broughton PM, Fletcher AE, Markowe HL, Marmot MG, Semmence A, Rose G. The assessment of biological age: a report from the Department of Environment Study. Aging (Milano) 1994;6(3):181-91.
9. Károlyi G, Balázsy K. Corneal arcus and life expectancy. Orv Hetil 1999;140(49):2755-61.
10. Fernández A, Sorokin A, Thompson PD. Corneal arcus as coronary artery disease risk factor. Atherosclerosis. 2007;193(2):235-40.
11. Fernandez A, Keyes MJ, Pencina M, D'Agostino R, O'Donnell CJ, Thompson PD. Relation of Corneal Arcus to Cardiovascular Disease (From the Framingham Heart Study Data Set). Am J Cardiol 2009;103(1):64–66.
12. Proof of age required – estimating age in adults without birth records. Aust Fam Physician 2010:39(7):518-521.
13. Aykroyd RG, Lucy D, Pollard AM. Regression analysis in adult age estimation. Am J Phys Anthropol 1997;104;259–65.

Implants - defining absolute anchorage

Namrata Rastogi*, Dheeraj Kumar**, Praveen Mehrotra***, Amol Bansal

*M.D.S. (K.G.M.C) Associate Professor Department of Orthodontics and Dentofacial Orthopaedics, Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow, India, **M.D.S. (K.G.M.C.), (Implantologist) Associate Professor Department of Prosthodontics, Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow, India, ***M.D.S. Professor & Head Department of Orthodontics and Dentofacial Orthopaedics, Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow, India, ****B.D.S. (M.D.S. Student) Department of Orthodontics and Dentofacial Orthopedics, Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow, India

Introduction

Anchorage control is one of the most important aspects of orthodontic treatment. There are times when absolute or maximum anchorage, i.e., high resistance to displacement, is needed. However, Newton's Third Law states that an applied force can be divided into an active component and an equal and opposite reactive component. Louis Ottofy[1] defined it as 'the base against which orthodontic force or reaction of orthodontic force is applied." In orthodontic mechanotherapy, even a small reactive force can cause undesirable tooth movement. Any uncontrolled reactive forces can have a negative effect on the outcome of the orthodontic treatment of a malocclusion. Therefore, it is virtually impossible to achieve absolute anchorage in which the reaction force producing no movement, especially with intraoral anchorage.

Anchorage preparation plays a key role in orthodontic treatment. Ottofy[1] also summarized the anchorage categories previously outlined by E.H. Angle and others as Intraoral (simple, stationary, reciprocal, intermaxillary) or Extraoral. The success of orthodontic treatment relies on the anchorage protocol planned for that particular case. Gianelly and Goldman[2] suggested the terms maximum, moderate and minimum to indicate the extent $_{to}$ which the teeth of the active and reactive units should move when a force is applied. Marcotte[3] and Burstone[4] classified anchorage into three categories - A, B, and C - depending on how much of the anchorage unit contributes to space closure. When preparing anchorage, the clinician must be realistic enough to foresee the possibility of losing some anchorage. The type of anchorage is based on the desired type of tooth movement.

Traditionally, extraoral anchorage has been used to reinforce intraoral anchorage. The use of extraoral anchorage ideally demands full cooperation of the patient as well as 24 hours per day of continuous appliance wear, objectives that are difficult to achieve and so they have unpredictable success rate. Adults and teenagers are prone to reject extraoral appliances because of esthetic problems and the discomfort they cause. Ravindra Nanda et a1[5] (2004) conducted a study to assess the effectiveness of skeletal anchorage for intrusion of maxillary posterior teeth, to correct open bite malocclusion, and to evaluate the usage of titanium mini plates for orthodontic anchorage. Therefore, it is extremely difficult to undertake orthodontic treatment without compromising anchorage in some way. Clinicians and researchers have tried to use implants as orthodontic anchorage units for over a half century. Branemark and co-workers[6] (1970) reported the successful osseointegration of implants in bone; many orthodontists began taking an interest in using implants for using orthodontic anchorage. Costa and colleagues[7] (1998) used 2mm titanium miniscrews for orthodontic anchorage. The screws were inserted manually with a screw driver directly through the mucosa without making a flap and were loaded immediately. Of the 16 miniscrews used during the clinical trial, two became loose and subsequently were lost before treatment was finished.

CORRESPONDING AUTHOR:
Dr. Namrataa Rastogi
102-a, Vijay Nagar, Krishna Nagar, Kanpur Road, India
Email id: drdheerajkumarb@gmail.com
Mobile No. 09415303807

Use of orthodontic implants as a source of anchorage has number of advantages as compared to traditional anchorage such as no patient cooperation, easy to use, shortening of treatment time, good control on tooth movements. The present paper is an attempt to analyze all the aspects of mini-implant and its various clinical implications and procedures.

Classification of implants for orthodontic anchorage

1. According to the shape and size

1) Conical (Cylindrical)
   a) Miniscrew Implants
   b) Palatal Implants
   c) Prosthodontic Implants
2) Mini plate Implants
3) Disc Implants (Onplants)

2. According to Implant bone contact

a) Osteointegrated
b) Non-osteointegrated

3. According to the application

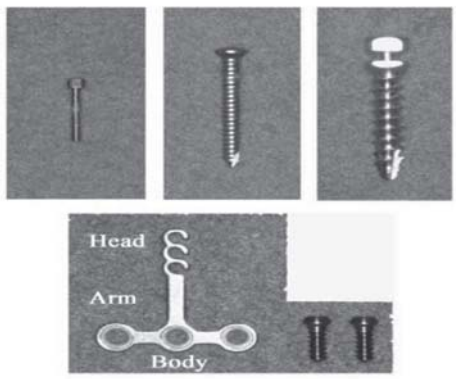
a) Used only for orthodontic purposes. (Orthodontic Implants)
b) Used for prosthodontic and orthodontic purposes. (Prosthodontic Implants)

Miniplates

The Miniplate Implants are comprised of bone plates and fixation screws. The plates and screws are made of commercially pure titanium that is biocompatible and suitable for osseointegration.

The miniplate consists of the three components—the head, the arm, and the body as shown in Fig.1.

Figure 1: Mini-plates



The head component is exposed intraorally and positioned outside of the dentition so that it does not interfere with tooth movement. The head component has three continuous hooks for attachment of orthodontic forces. There are two different types of head components based on the direction of the hooks.

- The arm component is transmucosal and is available in three different lengths—short (10.5 mm), medium (13.5 mm), and

long (16.5 mm) to accommodate individual morphological differences.

- ❖ The body component is positioned subperiosteally and is available in three different configurations—the T -plate, the Y-plate, and the I-plate.

The T-plates can be modified and used as L-plates by cutting off one of the screw holes. The surgical site requires at least 2 mm of cortical bone thickness to fix the anchor plate using monocortical screws, which are 2.0 mm in diameter and 5.0 mm in length. Each screw has an internal tapered square head with a self-tapping threaded body. Buttress is almost always thick enough. The I-plate is most often placed at the anterior ridge of the piriform opening for intrusion of upper anterior teeth or protraction of upper molars.

Mandible

In the mandible, screw fixation is possible on the lateral cortex in most locations except adjacent to the mental foramen. The T-plate and/or the L-plate is usually placed in the mandibular body to intrude, protract, or distalize lower molars, or at the anterior border of the ascending ramus to extrude impacted molars.

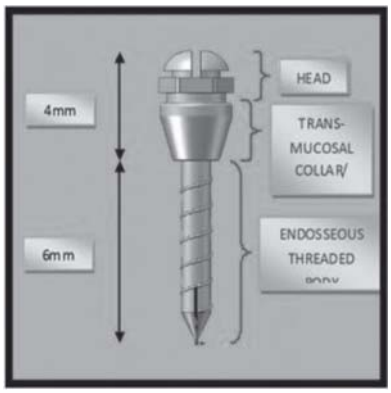
Orthodontic force is usually applied about 3 weeks after surgical placement of the miniplate, waiting only for soft tissue healing, not for osseointegration. Immediately after orthodontic treatment, all of the anchor plates are removed.

Of all orthodontic implants, miniscrews have gained considerable importance due to less surgical procedure and easy installation.

Miniscrews

Titanium miniscrews (Fig.2) may be an ideal anchorage system that fulfills the clinical needs of the orthodontist. This anchorage system is usually made of titanium and can be used to support a variety of orthodontic tooth movements in clinical situations involving mutilated dentitions, poor cooperation, or extraction cases requiring maximum anchorage. This system is available in either 1.5- or 2.0-mm diameters. The 1.5-mm diameter screw comes in 6.0-. 8.0-. or 10.0-mm lengths, while the 2.0-mm diameter screw comes in 7.0-, 9.0-, or 11.0-mm lengths. Both diameters are available in three different transmucosal designs to accommodate the soft tissues— low profile, low profile flat, and regular.

Figure 2: Parts of mini implant



The low profile screw has a longer transmucosal collar combined with a flat head and is utilized in the thick soft tissues of posterior segments, the low profile flat screw has the same head combined with a short collar and is indicated in the thin tissue of the patient's anterior segments, and the regular design has an intermediate length with a raised head, and when combined with a resin core can be used as a temporary prosthetic abutment.

Advantages of mini screws over mini plates-

Some of their benefits include-

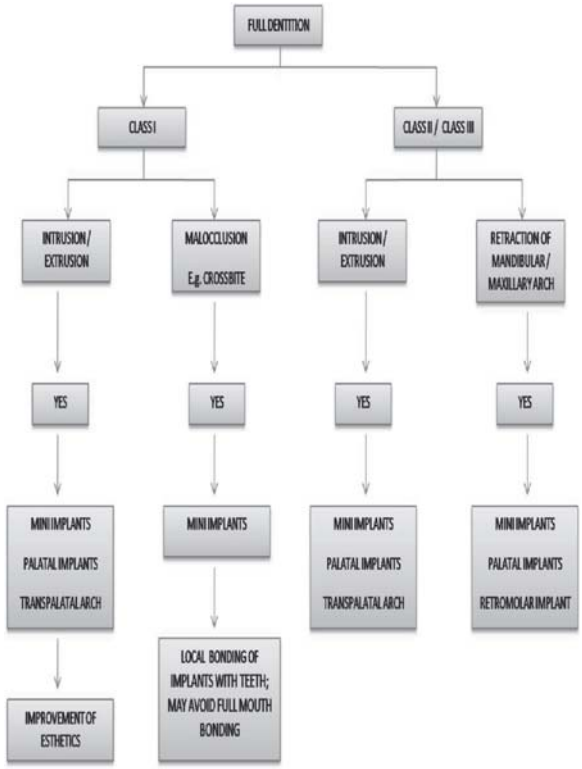
- Less patient compliance
- well acceptance by the patient
- immediate loading,
- simple to insert and remove,
- conform to the anchorage needs of the orthodontist.

Complete osseointegration is neither expected nor desired with this anchorage system.

Diagnosis and treatment planning

Treatment planning must include a careful choice of miniscrew location as well as the criteria as shown in Table 1. The placement location will enable the clinician to control or effect extrusive and intrusive movements of teeth. The placement of the screw requires a location that has sufficient bone depth to accommodate the miniscrew and at least 2.5 mm of bone width to protect the anatomic structures[8].

Table 1: Criteria for diagnosis and treatment planning for implant placement



Sites of placement

Maxilla

- Infrazygomatic crest area.
- Tuberosity area.
- Between 1ST and 2nd molars buccally.
- Between 1St molar and 2nd premolar buccally.
- Between canine and premolar buccally.
- Between incisors facially.
- Midpalatal Area.

Mandible

- Retromolar Area.
- Between 1st and 2nd molars buccally
- Between 1st molar and 2nd premolar buccally.
- Between canine and premolar buccally.
- Symphysis facially.

- Edentulous Area.
- Mandibular Tori.

Implant driving method

There are two methods of placement of minimplants.

(1) Self-tapping method: In this method the miniscrews is driven into the tunnel of bone formed by drilling, making it tap during implant driving. This method is used when we use small diameter miniscrews.

(2) Self-Drilling method: Here the miniscrews is driven directly into bone without drilling. This method can be used when we want to use larger diameter (more than 1.5mm) miniscrews.

Presurgical orthodontics

The surgical armamentarium for miniscrew insertion includes a low-speed contra angle hand piece, a bur with a depth stop, and a hand screwdriver. During surgical planning, the surgical site and screw length are determined. Every effort must be made to avoid contact with local anatomical structures.

Long cone radiographs (Fig.3) are taken to visualize the site locator relative to the delicate anatomical structures. Each screw length corresponds to a bur with a depth stop of equal length. A site locator can be fabricated from an orthodontic wire and utilized to determine the insertion position of the screw in the bone (Fig. 4).

Figure 3: cone beam x-ray showing the locator

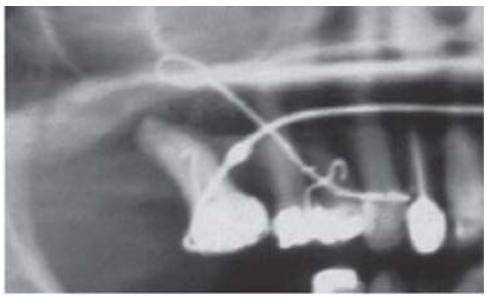
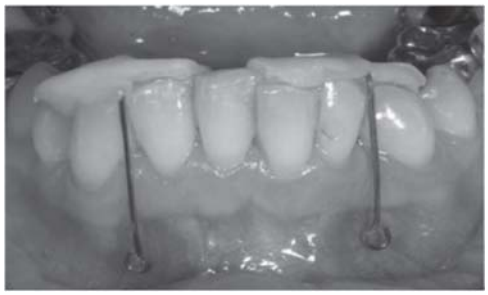


Figure 4: Implant locator

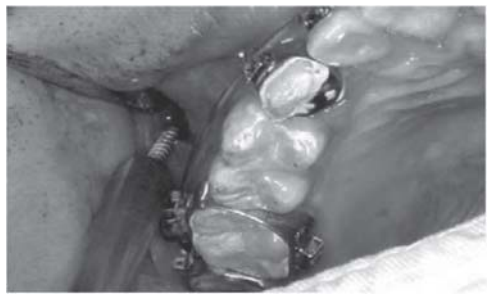


Surgical procedure

Topical anesthesia is recommended before infiltration anesthesia to reduce needle prick pain. Clinicians should not try to achieve profound anesthesia of the teeth, instead get numbness of soft tissue only. It is prudent for the teeth have some sensitivity, as the patient will complain of discomfort in the event of bone drill contacting the roots, then the drill can be redirected away from the roots[9]. Only one-fourth to one-third of a local anesthetic cartridge is needed for this type of anesthesia. Common sterilizing agents can be used to prepare intraoral & extraoral scrub for keeping the surgical area aseptic.

In Self-tapping method, a round bur (0.9mm) is used to make an indentation on the bony surface. Then pilot drilling is done to make a tunnel in the bone (Fig. 5). In order to avoid damages due to overheating,

Figure 5: Pilot drilling for self tapping implant implant



the drilling must be undertaken using the contra-angle hand piece (optimal 800 rpm, maximal 1500 rpm) and external cooling with a sterile, cooled, physiological saline solution (5°C/41 °F). The drilling should take place intermittently and without pressure so that the tip of the bur may cool down. Finally, miniscrew is loaded in the bone. In Self-drilling method, there is no pilot drilling and miniscrew is loaded directly into the bone.

Post surgical treatment

Patients should be given standard surgical post operative instructions emphasizing the importance of inflammation control and cautioned not to brush or touch the implant for a week. Ibuprofen or its equivalent is usually adequate for discomfort, and antibiotics are rarely necessary. A chlorhexidine rinse is usually prescribed for 7-14 days, but no other post surgical care is required. Patients with miniscrews should return to the orthodontic office as soon as possible for loading, preferably within one week. In theory, vector of force to stabilize miniscrews is critical to counter tissue, tongue and masticatory forces. Osseointegration is not expected, therefore mechanical stabilization is crucial.

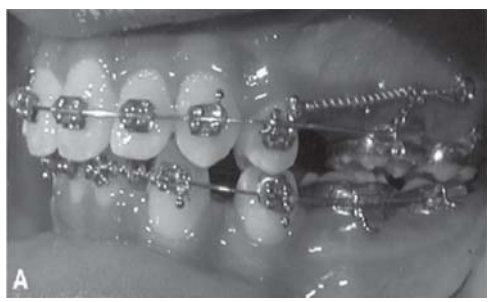
Miniscrew removal

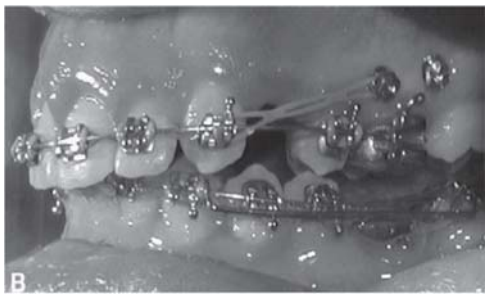
Fortunately, strong osseointegration does not occur between miniscrew and bone, and this simplifies the removal of these microscrews. Clinician can engage the miniscrew head with the driver and turn it in the opposite direction of the insertion that will easily remove it[10]. What is more, local anesthesia is not needed during this procedure. Patients may have some minor discomfort when the implant irritates the soft tissue while its removal, but this gives far less discomfort than an anesthetic needle-prick.

Clinical implications

1. Closure of Extraction Spaces: Loss of posterior anchorage during extraction space closure can exacerbate the curve of Spee and deepen the bite. Miniscrews provide reliable skeletal anchorage for anterior retraction in either arch, whether a single tooth at a time or en masse (Fig. 6).

Figure 6: Retraction of anterior teeth with implant as anchorage source.

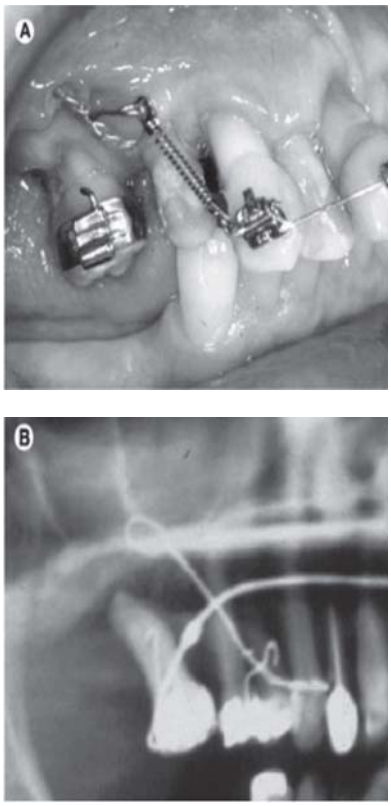




2. Symmetric Intrusion of Incisors: Many patients present with moderate - to - severe deep bites requiring pure intrusion of the anterior teeth to level the occlusal plane. Unless the deep bite is so extreme that absolute anchorage is needed, it may be inadvisable to place miniscrews simultaneously in both arches in young patients. In these cases, miniscrews can be used to reinforce conventional orthodontic mechanics.

3. Single tooth intrusion: a few patients often report with extruded tooth due to early loss of its antagonist. So, in order to correct its vertical position we plan for single tooth intrusion (Fig.7).

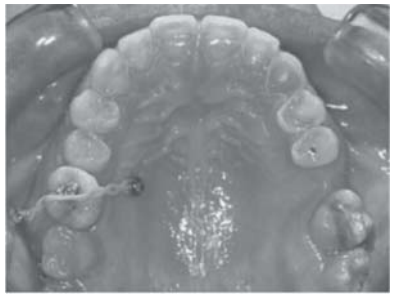
Figure 7: Intrusion of single tooth with implant.



4. Correction of Canted Occlusal Plane: A canted occlusal plane is often considered impossible to level with traditional orthodontic treatment. Miniscrews, on the other hand, provide skeletal anchorage for intrusion of the appropriate teeth on the canted side.

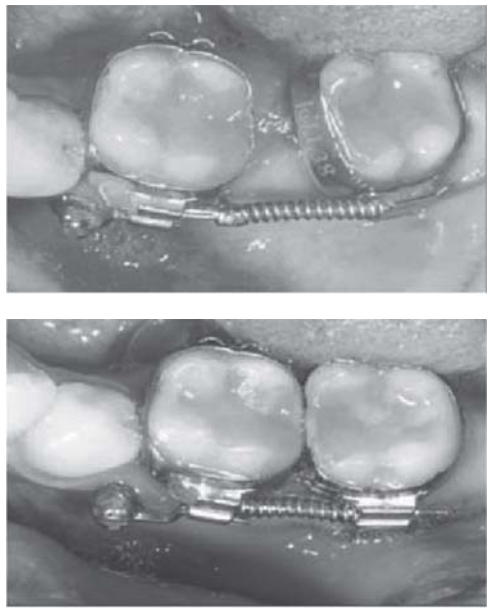
5. Molar Intrusion: Opinions have differed regarding the efficacy of orthodontic intrusion of posterior teeth. Although miniscrews can be a reliable source of anchorage, it is difficult to place them precisely in the narrow space between the roots of the first and second molars without interfering with the roots. In some cases, more than one screw might even be needed to withstand a relatively high intrusion force (Fig.8). Therefore, we suggest limiting the use of miniscrews to situations where simple intrusion of one or two molars is needed and here placement will be unproblematic.

Figure 8: Intrusion of molar with implant as anchorage source.



6. Molar Mesialization: Molars are often moved mesially in orthodontic treatment to close extraction spaces or edentulous spaces. Molar mesialization is not a simple movement and can lead to problems such as loss of anterior anchorage and molar tipping. Furthermore, if there is a knife-edge alveolar ridge in the space to be closed, alveolar bone may be lost. A miniscrew placed mesial to the space, at a height that will produce a force vector approximating the center of resistance of the molar, can be a valuable source of anchorage (Fig. 9). If the screw is inserted after the initial leveling and alignment has been completed, a full-size archwire can be used to prevent mesial crown tipping of the molar during space closure. Because mesial movement is usually slow, especially in the mandibular arch, no more than 2-3mm of molar mesialization should be attempted.

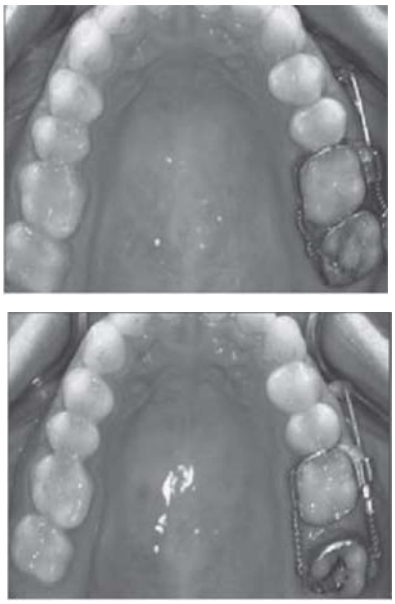
Figure 9: Mesialization of molar with implant as anchorage source.



7. Molar Distalization: Miniscrews + Distal Jet may be a solution. After the Distal Jet appliance has been placed and activated, palatal miniscrews are inserted between the roots of the first and second premolars, mesial to the activation locks attached to the anterior rests. The miniscrews block mesial movement of the appliance during distalization, thus preventing loss of anterior anchorage. Further compression of the Distal Jet's coil springs will move the locks distally, away from the miniscrews; during this phase, anchorage loss can be prevented by bonding light-cured composite between the screw heads and the locks. After molar distalization, the Distal Jet is converted to a passive retainer, and brackets are bonded to the teeth for completion of the Class II correction. Another option is to remove the miniscrew after molar distalization and replace it just mesial to the distalized

molar, where it will stabilize the molar while the remaining teeth are moved posteriorly (Fig.10).

Figure 10: Distalization of molar with implant as anchorage source.



Conclusion

Success of orthodontic treatment relies on anchorage control. So anchorage preparation is a very important part of orthodontic treatment. Prior to initiation of orthodontic therapy, it is essential to carefully assess the anchorage demands of an individual case so that appropriate treatment modalities can be executed. Various sources of anchorage have been used from the 17th century to till date. Every anchorage source has some advantages and some limitations. Anchorage preparation is different from patient to patient. Use of extraoral anchorage such as headgears requires full patient cooperation, but this cooperation is not seen especially in teenagers. With the introduction of implants in orthodontics, this problem has been solved. Implants do not require any patient cooperation, so we can get a good anchorage control in our patients. Implants provide absolute anchorage i.e. complete bone anchorage. Implants have revolutionized the field of anchorage in orthodontics. So by choosing a correct anchorage source we can get good results in orthodontic treatment.

References

1. Ottofy L : Standard Dental Dictionary. Chicago, Laird and Lee, Inc, 1923.
2. Gianelly A, Goldman H: Biological Basis of Orthodontics. Philadelphia, Lea And Febiger, 1971.
3. Marcotte M: Biomechanics In orthodontics. Toronto, BD Decker, 1990.
4. Burrstone CJ: Enmasse space closure, in Burstone CJ (ed): modern Edgewise Mechanics and the Segmented Arch Technique. Glendore, Ormco Corp, 1995, pp 50-60.
5. Ravindra Nanda, Andrew Kuhlberg, Flavio Uribe. Biomechanical basis of extraction space closure. Biomechanics and esthetic strategies in clinical orthodontics, 194-210.
6. Jae- Hyun Sung, Hee-Moon Kyung, Seong- Min Bae, Hyo-Sang Park, Oh- Won kwon, James A. Mcnamara, Jr. The history of skeletal anchorage in orthodontics. Microimplants in orthodontics, 7-14, 2006.
7. Costa A, Raffini m. Melsen B. Microscrew As orthodontic Anchorage. Int J Adult Orthod Orthognath Surg 1998; 13: 201-209.
8. Linkow LI. The endosseous blade implant and its use in orthodontics. Int. J Orthod 1969;18: 149-154
9. Gainsforth BL, Higley LB. A study of orthodontic anchorage Possibilities in basal bone. Am J Orthod Oral Surg 1975;31:406-417
10. Sherman Aj. Bone reaction to orthodontic Forces on vitreous carbon dental implants. Am J Orthod 1979; 76: 618-637.

A case report of suicidal death of a female prisoner consuming formalin in RIMS Hospital, Kadapa – How far hospital administration is responsible?

Ananda Kumar L*, Subba Reddy K, Obulesu l.C**, Reshma Sireesha l***, Sureswar Reddy M****, Krishna Prasad S*****

*Asst. Professor, Dept. of Forensic Medicine RIMS Medical College, Kadapa 516002, A.P., India, ** Professor & Head, Dept. of Forensic Medicine RIMS Medical College, Kadapa 516002, A.P., India, ***P.G. Student Kurnool Medical College, Kurnool 518001, A.P., India, ****Assistant Professor, Dept. of General Medicine and C.S.R.M.O RIMS Hospital/ Medical College, Kadapa 516002, A.P., India, ***** Asst. Professor, Dept. of Forensic Medicine, S.V.S Medical College, Mahabub Nagar 509001, A.P., India

Abstract

Formaldehyde is formed when methyl alcohol vapor is passed over a red hot spiral of platinum wire. It is a colorless gas, possessing a strong pungent, irritating odour. It is soluble in water a 40 percent solution being a pharmacopoeial preparation. In the present case a female prisoner aged about 28 years W/o Srinivasulu admitted in Psychiatric ward on 29/01/2010 at 1:50 AM by the orders of the Nandalur Court in Cr.No:3/2010 of Penagalur P.S U/s 29 Mental&Health act. A case was registered on the complaint given by her parents against the deceased as there was conflict in the family. She committed suicide by consuming formalin solution in the hospital

Keywords

Lunatic prisoner, Suicide by Formalin in the hospital, Post-Mortem findings.

Introduction

Formaldehyde is a colorless gas, possessing a strong pungent, irritating odour. It is soluble in water a 40 percent solution being a pharmacopeial preparation. Liquid formaldehyde is commercially known as formalin or formal. It is used a disinfectant, fixative for tissues, embalming and in the preparation of artificial ivory, crease resistant cloth celluloid horn and plastics.

It is also frequently used as a preservative for food, especially milk. Formalin solution is also used for the fumigation of Burns and ICU wards. 30 to 60 ml of formalin causes death with in hours.

Case history

A female lunatic prisoner patient aged about 28 years by name Sujatha was admitted in Psychiatric prisoner ward on 29/01/2010 at 1:50 AM by the court orders on the complaint given by her parents. She was treated at RIMS Hospital by the Physician and Psychiatrist under the supervision of C.S.RMO. She was referred to the Institute of Mental Health Hospital, Yerragadda, Hyderabad for further treatment at 11:00 A.M. on 4/2/2010. On 04/02/2010 at 4:30 P.M, while waiting for the arrival of the escort she went to bath room and consumed Formalin which was kept by the ward boy for the fumigation of Burns ward which was located by the side of the prisoners ward. She was noticed by the women police escort and brought to ICU ward where she succumbed within 30 minutes after consumption of Formalin solution. The ward boy who is responsible is suspended for his negligence after an enquiry by the RMO. No Police case was initiated against any employee of the Hospital

A case was registered U/s 176 Crpc in Cr.No. 14/10 in RIMS P.S Kadapa and on 05.02.2010 at 4:00 P.M the Inquest was conducted by the Sub- Divisional Magistrate, Rajampet and the team of doctors conducted Post-Mortem examination under the video coverage.

POST-MORTEM FINDINGS: No injuries found on the body.

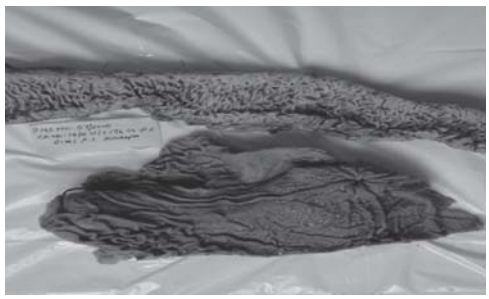
Externally cyanosis of finger nails and congestion of eyes and face present

The smell of formalin noticed on opening the body. Externally the appearance of stomach and intestines look like hard rubber tubes like in consistency muscle walls are thickened foldings of mucous membrane increased in size up to 1.5 to 2cm like raised projecting folds. The mucous membrane of the stomach is red inflamed and eroded with extravasation of blood smell of formalin odour is present. All internal organs are congested. Viscera preserved and sent for chemical analysis. The Regional Forensic Science Laboratory Vide Tox. No: 489/2010 dated 6/4/2010. Formaldehyde solution was found in the stomach and intestines but not found in kidney, liver and blood.

Cause of death: Death due to Formalin Poisoning.

Figure no 1:

Figure no 2:



References

1. Singhal's Toxicology at a glance S.K.Singhal M.D, LLB sixth edition, The National Book depot Opp: Wadia Children Hospital Parel, Mumbai-12.
2. The Essentials of Forensic Medicine and Toxicology Dr.K.S.Narayana Reddy Twenty sixth Edition – 2007.
3. Modi's Medical Jurisprudence and Toxicology Twenty third edition, Editor K.Mathinaren and Amrut K Patnaire.

Study of bilateral asymmetry of tibia in Vidarbha region of Maharashtra

Charulata Annaji Satpute*, Meena Meshram**

*Lecturer, Department of Anatomy, Indira Gandhi Government Medical College, Nagpur, **Professor, Department of Anatomy, Government Medical College, Nagpur

Abstract

In the present study 68 dissection hall cadavers 48 males and 20 females of known sex (from G.M.C., I.G.G.M.C., L.M.M.C. Nagpur) were taken. It is observed that right tibia shows differences in the length, although there is slightly greater difference ,in weight.The 't'test was applied and found that statistically there is no significant difference in length as well as in weight between the two sides of tibia.

Keywords

bilateral asymmetry,tibia.

Introduction

Bilateral asymmetry in its manifestations has long been in interesting but baffling problem. Probably right and left handedness was recognized first and an early record of left handed soldiers was found in the Bible,(Judges 3:15;20:16)[2]Asymmetry of paired dimensions has been recognized as a indicator of environmental stress.

The study of asymmetry in the use of hand, foot and eye has received much attention in more recent years(Schaeffer,1928)[18];(Leche 1933)[13];(Jantz,1964)[5].Differences in the time of ossification of the right and left bones of the hand, especially, have been noticed and studied(Flecker,1942)[3];(Noback,1944)[15];(Noback and Robertson,1951)[16];(Baer and Durkatz,1957)[1].

Asymmetry in weight and length of adult paired bones has been reported and a few of the records are: Ingalis (1931)[4]; Schultz (1937)[19]; Trotter and Gleser (1952)[20] and Jolicoeur (1963)[6].These studies give references to many of the earlier reports. Latimer H.B. and Lowrance E.W. (1965)[8] studied the bilateral asymmetry in weight and lengths of right and left bones of each pair of human bones and found that the long bones of upper extremity are heavier and longer on right side and bones of the lower extremity tend to be heavier and longer on left side thus suggesting the "cross symmetry".

There have been several reports of asymmetry in vertebrates other than man and some of these reports describe very few asymmetries with percentages over 50(Latimer, 1936[9];1937[10];1938[11];Latimer and Riley,1934[7] and Latimer and Wager,1941)[12].

(* Corresponding author email drcharusatpute@gmail.com Department of Anatomy, Indira Gandhi Government Medical College, Nagpur.)

Material and methods

The length of each bone along with their articular cartilage intact was measured in cms. on the osteometric board and the weight was taken in gms. Each parameter was analysed to find out the mean, standard deviation, coefficient of variation and standard error.

The average differences in length and weight for right and left tibia was calculated and recorded seperetely.The "t" test was applied to know whether the differences are significant or not.

Observations

From the above observations it is seen that the right tibia averages 3.37 Gms. heavier than the left tibia.

The "t"test was applied to know whether this difference in mean between the right and left side of tibia is significant or not.

$T = 0.32$

$P > 0.05$ N.S.

Thus statistically there are no significant differences in weight between right and left tibia.

Table 1: able showing weight in Gms. of each pair of Tibia in both sexes.

Measurements	Right tibia	Left tibia
No. of bones	68	68
Range	125-352	120-365
Mean	250.89	247.52
Standard deviation(S.D.)	58.27	61.89
Coefficient of varitation	23.22	25.00
Standard error	7.06	7.50

Disscussion

In the present study it is observed that the right tibia shows the least difference in the length, although there is slightly greater difference, in weight .The "t"test was applied and found that statistically there is no significant difference in length as well as in weight between the two sides of tibia.

There have been many studies of asymmetry both morphological and physiological .Thus Latimer H.B. and Lowrance ,(1965)[8] studied the bilateral asymmetry in length and weight of right and left side of tibia from Asian skeletons and reported that the right tibia is heavier and left tibia is longer.

ngalis (1931)[4] studied the 100 skeletons from the Todd collection and discussed the asymmetry of the paired bones. Trotter and Gleser (1952)[20] reported that the left tibia is longer and also started that all of the bones of lower extremity are longer on the left side.

Schultz (1937)[19] and Lowrance and Latimer (1957) [14]reported that there is greater asymmetry in the upper extremity and greater similarity in the lower extremity.

Jolicoeur (1963)[6] reported that the tibia is slightly but not significantly longer on the right side in male and on left side in female skeletons.

Prives (1960)[17] is of opinion that physical work also favours growth in length. Certain occupations and sports tend to have an unequal development of the upper extremities while the lower extremities are developed more equally. It is also shown that the muscle development is accompanied by a corresponding increase in size of their bony attachment. Thus the greater use of one upper extremity may be a factor in the larger asymmetries reported in the upper extremity compared with the lowers.

Conclusion

Statistically there is no significant difference in length as well as in weight of the tibia between two sides.

Bibliography

1) Baer M.J.and Durkatz (1957); Bilateral asymmetry in skeletal maturation of the hand and wrist. A roentgenographic analysis. Am.J.Phys.Anthrop. 15:181-196.
2) Bible ca 1200 B.C.: udges 3:15; 20:16 (Recordedca 600 B.C.)
3) Flecker H. (1942): Time of appearance and fusion of ossification

centers as observed by roentgenographic method. Am.J.Roent. 47:97-159.
4) Ingalis William N (1931): Observation on bone weight. American Journal of Anatomy, 48:45-98.
5) JantzR.Z. (1964): Some aspects of laterality among University of Kansas male students. Thesis for M.A.deg. University of Kansas, Lawrence.
6) Jolicoeur P.(1963): Bilateral asymmetry in limbs bones of Martes Americana and man. Rev.Can.Biol. 22:409-432.
7) Latimer H.B.and R.B.Riley (1934): Measurements of the skull and of some of the long bones of the muskrat (Ondatra zibethicus Cinnamominus) J.Morph. 56:203-212.
8) Latimer Homer B and Lowrance E.W. (1965): Bilateral asymmetry in weight and in length of human bones. Anat.Record, 152:217-224.
9) Latimer H.B.(1936): Weights and linear measurements of the adult cat. American Journal of Anatomy, 58:329-347.
10) Latimer H.B. (1937): Weights and liner dimensions of the skull and some of the long bones of theskunk (Mephitis mesomelas avia) J.Morph.60:379-391.
11) Latimer H.B.(1938): Weights and liner dimensions of the skull and of some of the long bones of the Red-tailed hawk (Buteo borealis borealis) Univ.of Kana Sci.Bull. 25:190-212.
12) Latimer H.B.and Wager (1941): Weights and liner dimensions of the skull and of some of the long bones of the Mall and duck (Anas Platyrhynchos) Univ.of Kans Sci.Bull. 27:5-18.
13) Leche S.M. (1933) Handedness and bimanual dermatoglyphic differences American Journal of Anatomy, 53:1-53.
14) Lowrance E.W. and Latimer Homer B. (1957): Weights linear measurements of 105 human skeletons from Asia. American Journal of Anatomy, 101:445-459
15) Noback C.R.(1944): The developmental anatomy of the human osseus skeleton during the embryonic, fetal and circumnatal periods. Anatomical Record, 88:91-125.
16) Noback C.R. and Robertson G.G. (1951): Sequences of appearance of ossification centers in the human skeleton during the first five prenatal months. American Journal of Anatomy, 89:1-28.
17) Prives M.G. (1960): Influence of labor and sport upon skeleton structure in man. Anatomical Record, 136:261.
18) Schaeffer A.A. (1928): Spiral movement in man. J.Morph and Physiol., 45:293-398.
19) Schultz A.H. (1937): Proportion variability and asymmetry of the long bones of the limbs and the clavicles in man and apes. Human Biol., 9:281-328.
20) Trotter Mildred and Gleser Goldine C. (1952): Estimation of stature from long bones of American Whites and Negroes. Am.J.Phys.Anthrop. Vol.10:463-514.

The use of digital C-arm fluoroscopy in the surgical removal of foreign bodies from maxillofacial region

Iqbal Ali*, Mohd Faisal**, Chetan Chandra***, Vikas Kumar****, Abu Amir*****

*HOD, Oral & Maxillofacial Surgery Department, Career PGIDSH, Lucknow, **Senior Lecturer in Oral & Maxillofacial Surgery Department, Career PGIDSH, Lucknow, ***Senior Lecturer in Periodontology and Implantology Department, Sardar Patel Dental College, Lucknow, ****Junior Resident in Oral & Maxillofacial Surgery Department, Career PGIDSH, Lucknow, *****Junior Resident in Oral & Maxillofacial Surgery Department, Career PGIDSH, Lucknow

Abstract

Foreign bodies are often encountered by oral and maxillofacial surgeons and may present a diagnostic challenge, due to many factors such as the size of the object, the difficult access and a close anatomical relationship of the foreign body to vital structures. A 23 year old male reported to the Oral & Maxillofacial Surgery Department with the chief complaint of pain and irritation on the left cheek region since 5-6 months. While giving the history the patient revealed that he had traumatic injury due to a road traffic accident eight months earlier. He had sustained multiple lacerations on the left side of the face which were primarily closed at that time. On examination, a scar was noticed on the left side of the face. A swelling was present on the left side of face at the lateral surface of the mandibular ramus. Panoramic Radiograph and Computed Tomography Scan revealed several well defined radio-opacities at the left side of the mandibular ramus region. A provisional diagnosis of foreign body embedded in soft tissue was considered. This article describes the use of the C-arm digital fluoroscope for retrieval of foreign bodies from maxillofacial region.

Keywords

Digital C-arm Fluoroscope, Foreign bodies, Left mandibular ramus region, Traumatic injury.

Introduction

Foreign bodies pose diagnostic difficulties as they often get cicatrized within living tissues and are missed during primary wound management.Tissue can be damaged by traumatic injury wounds, or altered by scaring after an operation that resulted in an iatrogenic foreign body . Inflammatory response in the tissues around a foreign body may add to the difficulties1,2 . There are many ways of detecting and localizing foreign bodies. Plain radiographs, computed tomograms (CT), magnetic resonance images (MRI) and ultrasound, digital C-Arm Fluoroscopy may be used, depending on their site and composition3,4.

Delayed removal presents with many difficulties due to factors such as nature and size of the object, difficult access, and close anatomical relationship of the foreign body to vital structures and scarring of tissues. They are a result of injuries or operations. Fragments of broken instruments can be left behind and entire teeth or their fragments can be displaced during extraction 4,5,6,7,8. Occasionally, foreign bodies may be retained for some time causing persistent and distressing symptoms 9,10 . The foreign body can often modify the regional anatomy.

Digital C-Arm is ubiquitous and is used frequently for removal of foreign bodies from maxillofacial region as it is readily available at most hospitals and trauma centres. Digital C-Arm is more reliable, offers real time imaging, with precise intraoperative localization of foreign body. The foreign body removal in the facial region poses a danger of damaging important anatomical structures. Even if the exact position is known from imaging data, the accurate reproduction of its position in the patient's body can be difficult if the foreign body is not adjacent to a definitive anatomical landmark. The search for a foreign body in a larger area rather than at a definite position increases the risk of damage to adjacent structures 1. The purpose of this report is to present a case of a foreign body in a patient's facial region caused by traumatic injury and calls for the attention of the importance of good primary care, primary exposure, clinical and radiographic evaluation at the instance itself and a probable referral to a maxillofacial specialist for adequate management and also to counter medicolegal issues that may be forthcoming.

Case Report

A 23 year old male reported to the Oral & Maxillofacial Surgery Department with the chief complaint of pain and irritation on the left cheek region since 5-6 months. While giving the history the patient revealed that he had traumatic injury due to a road traffic accident eight months earlier. He had sustained multiple lacerations on the left side of the face which were primarily closed at that time. Medical, family, and past dental history were non-contributory. On examination, a scar was noticed on the left side of the face. A swelling was present on the left side of face at the lateral surface of the mandibular ramus. The swelling was nodular, tender on palpation, and hard in consistency.

Panoramic Radiograph (Fig. 1) and Computed Tomography Scan (Fig. 3) revealed several well defined radio-opacities at the left side of the mandibular ramus region. A provisional diagnosis of foreign body embedded in soft tissue was considered. The foreign body was approached by the already formed scar and removed along the path of its insertion thus causing minimal trauma to the anatomical structure and preserving the aesthetics. Due to distortion of tissue planes by scar formation one glass piece was not accessible. At this point digital C-arm Fluoroscope was used to gain a guided access to that piece in the absence of a normal anatomical landmark in the vicinity of the foreign body; a hypodermic needle under fluoroscopic guidance was used to help in localization of the glass piece. After the foreign body's removal, the soft tissues were explored, washed copiously, and sutured with 5-0 monofilament suture (Fig . 3).

Discussion

The first step with a traumatized patient is the detailed analysis of the correct functionality and symmetry of both the face and neck, followed by an accurate, objective test and proper surgical cleansing of the lacerated-contused tissues. This evaluation must have the backup of aimed radiographic tests such as radiography, CT with axial and coronal projections, and Nuclear Magnetic Resonance in selected cases. MRI scans are not often used if the nature of foreign body is not known. Metallic foreign objects tend to get displaced under high

Fig 1. Panoramic radiograph showing foreign bodies in left side of the maxillofacial region

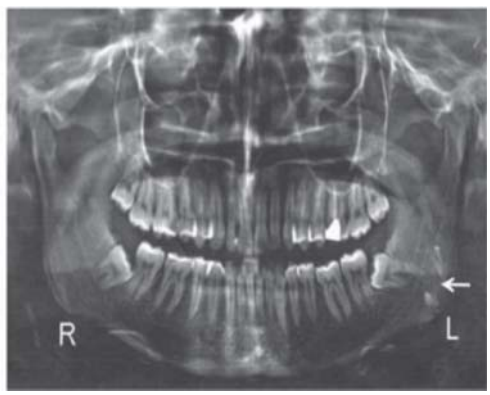
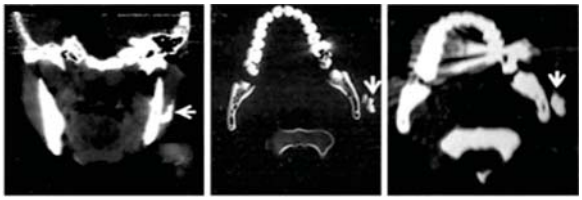


Fig. 2: Plain CT scan showing foreign bodies in left side of the maxillofacial region



magnetic fields. Digital ultrasound imaging has often been of removal of such foreign objects, however due to its limited availability and limited usefulness in locating objects within deeper tissue was not used.

The removal of the foreign bodies can be delayed due to an inappropriate diagnosis, post traumatic facial oedema or because of their asymptomatic behaviour. In the reported case, the patient delayed the removal ,due to inappropriate diagnosis during the primary management of the injury. Furthermore, despite the consequences of foreign bodies in the case, it remained asymptomatic for months.

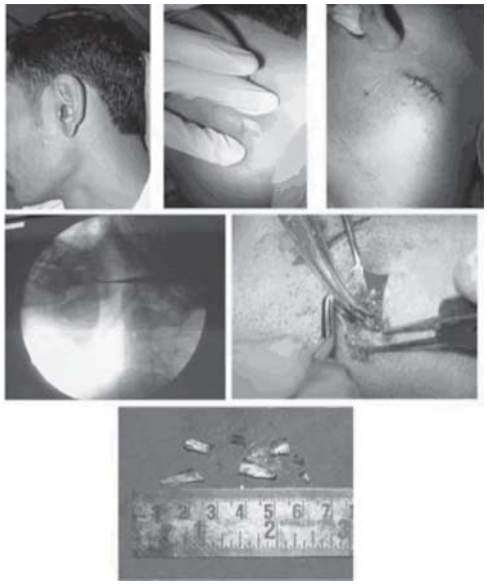
Fluoroscopic systems consist of an x-ray image intensifier connected to video cameras. Fluoroscopic technology allows for real-time radiographic visualization of the foreign body and affords the clinician the opportunity to precisely locate and fix the foreign body using markers. The necessary radiographic equipment for fluoroscopy is available in most of the hospital.

We recommend necessary clinical and radiographic evaluation and prompt referral to a maxillofacial specialist at the time of injury during primary trauma management not only for adequate management but also to circumvent any medicolegal issues that may be forthcoming. In the absence of this intraoperative fluoroscopic guidance may be used for retrieval of foreign bodies from the maxillofacial region.

References

1. Eggers G, Haag C, Hassfeld S. Image-guided removal of foreign bodies. Br J Oral Maxillofac Surg 2005:404–409.
2. Holmes P-J, Miller JR, Gutta R, Louis PJ. Intraoperative imaging techniques: A guide to retrieval of foreign bodies. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005;100: 614-618.
3. Krimmel M, Conelius CP, Stojadinovic S, Hoffman J, Reinert S. Wooden foreign bodies in facial injury: a radiological pitfall. Int J Oral Maxillofac Surg 2001; 30:445-447.

Fig. 3: Preoperative photograph of foreign body approached by the already formed scar; Removal along the path of its insertion; Sutured with 5-0 monofilament suture; Digital C-arm view just prior to foreign body removal; Fragments of glass pieces seen after removal.



4. Mahmood S, Lello GE. Tooth in the nasopharynx. Br J Oral Maxillofac Surg 2002; 40:448–449.
5. Paoli JR, Dekeister C. A tooth in the orbit. *Br J Oral Maxillofac Surg 2001; 39(4):327.*
6. Quayle AA. The significance of small wounds in the eyelids. Br J Oral Maxillofac Surg 1986; 24:17-21.
7. Thach AB, Ward TP, Dick II JSB, Bauman WC, Madigan WP, Goff MJ, Thordsen JE. Intraocular Foreign Body Injuries during Operation Iraqi Freedom. *Ophthalmology* 2005;112: 1829–1833.
8. Waldman LA. Facial cellulitis caused by unrecognized foreign body. *Oral Surg Oral Med Oral Pathol* 1979; 48(5):408-409.
9. Cameron M, Phillips B. Snookered Facial infection secondary to occult foreign body. Int J Oral Maxillofac Surg 2006; 35:373–375.
10. Khyani I A. M, Rahman N, Alam J. Foreign body in Submandibular region.J Coll Physicians Surg Pak 2007; 17: 626-8.

Myocardial infarction in a 22 year old male- A case report

Shankar M Bakkannavar[1], Francis N P Monteiro[2*], Prashantha Bhagavath[1], Kiran Yagain[1b], Yajnesh Kidiyoor[3], Pradeep Kumar G[4]

[1] Assistant Professor, Department of Forensic Medicine and Toxicology, Kasturba Medical College , Manipal, India [1b] Assistant Professor, Department of Pathology, Kasturba Medical College , Manipal, India, [2] Associate Professor, Department of Forensic Medicine and Toxicology, A.J. Institute of Medical Sciences , Mangalore, India, [3] Tutor, Department of Forensic Medicine and Toxicology, Kasturba Medical College , Manipal, India, [4] Professor & Head, Department of Forensic Medicine and Toxicology, Kasturba Medical College , Manipal, India

Abstract

Sudden unexpected death is a catastrophic complication of acute myocardial infarction. Every unexpected death has an actual or potential medico-legal significance, and in most countries such deaths come under the purview of a medico-legal investigative system. A forensic expert has the responsibility of establishing the cause of death in these unexpected fatalities. Sudden deaths in adults from presumably natural causes occur more frequently than is commonly thought of. Numerically they constitute a significant fraction of the total mortality in any sizeable community. Here in we report a case of a twenty two year old young male who was apparently healthy suddenly developed chest pain and was declared dead on arrival to the hospital. This created suspicion in the minds of the family members, who insisted on an autopsy. External examination did not reveal any significant findings except for bluish discoloration of nails. Gross examination of the heart revealed near total occlusion of the coronaries and a hyperaemic area over the heart due to congested epicardial capillaries. The heart was subjected for histopathological examination which revealed infarction changes thereby aiding the determination of cause of death.

Keywords

Sudden death; Acute myocardial infarction; Young adults; Histopathology.

Introduction

Forensic pathologists deal not only with criminal, accidental and suicidal deaths, but also with a wide range of natural deaths, especially, if they had occurred suddenly in apparently healthy individuals. WHO has predicted that by AD 2020 up to three-quarters of death in developing countries would result from non-communicable diseases and that coronary heart disease will top the list of killers.[1] Coronary artery disease, looming large as the new epidemic, is afflicting the Indians at a relatively younger age group.[2,3] The risk of coronary artery diseases is 3 to 4 times higher in Indian populace when compared to White American populace, 6 times higher than the Chinese and 20 times higher when compared to the Japanese populace.[4,5] The disease is accelerated, severe, extensive, and follows protracted and malignant course.[6,7] In some studies from India, the percentage of patients below the age of 45 years suffering from acute myocardial infarction is reported as high as 25-40%.[8] Coronary Atherosclerosis results from interaction of many factors, resulting ultimately in endothelial injury and inflammation, leading on to plaque formation. The best news about Atherosclerosis is that it is preventable.

We report a case of sudden unexpected death due to myocardial infarction in a 22 years old male.

Case history

An apparently healthy young man, aged 22 years and a smoker since 6 years, suddenly developed severe chest pain at 8 O'clock in the night after supper. Assuming it to be a case of gastric origin his spouse applied a local ointment. Next day at 6 O'clock in the morning he started vomiting and collapsed. The man was shifted to the hospital within an hour of this incident. He was declared dead on arrival and the body was subjected for a medico-legal autopsy on the same day. There was no history dyslipidemia or sudden death in the family.

External examination

The deceased was moderately built and nourished. There was bluish discoloration of nails. Body natural orifices were unremarkable. No external injuries were present on the body.

Internal examination

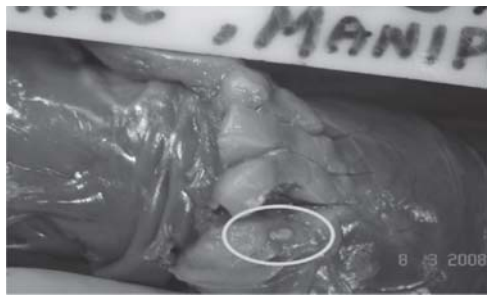
The heart weighed 230 grams. There was a hyperaemic area (measuring 3x2 cms) over the apex circumferentially due to congested epicardial capillaries. Coronaries were thick and had gritty feeling on cut section. Right coronary artery showed more than 50% narrowing of the lumen due to the atheromatous plaque deposition within the intima of the arterial wall. Left anterior descending and left circumflex coronary artery showed near total occlusion due to the atheromatous plaque deposition within the intima of the arterial wall with a thrombus in situ (Fig.1) Descending and ascending aorta showed intimal deposition of atheromatous plaques at places. Lungs and brain were oedematous. Examination of other organs and toxicological analysis were unremarkable.

Histopathology

Multiple sections from the heart were subjected for histopathological examination using Eosin & Haematoxylin stain. No significant changes in myocardium were evident except for an area of waviness of myocardial fibers corresponding to hyperemic area suggesting an early myocardial infarction. (Fig. 2) Microscopy of the sections from anterior wall showed patchy areas of vacuolar degeneration indicative of long standing ischemia. (Fig. 3) Sections from the left anterior descending and left circumflex coronary arteries showed more than 95% block of the lumen due to a large circumferential atheromatous plaque composed of a ruptured fibrous cap with superimposed fibrin thrombi. (Fig. 4) Cause of death was opined as to be due to acute myocardial infarction secondary to near total occlusion of the coronary artery.

Explanatory Legends

Figure 1: Cut section of the left anterior descending coronary artery showing near total occlusion of the lumen



Corresponding Address:
Dr Francis NP Monteiro
Associate Professor Department of Forensic Medicine & Toxicology
A.J. Institute of Medical Sciences Mangalore - 575004, India.
Phone No: +91-9448327389 (M)
Email- drfrancis@rediffmail.com

Figure 2: Microscopy of the myocardium showing waviness of myocardial fibers

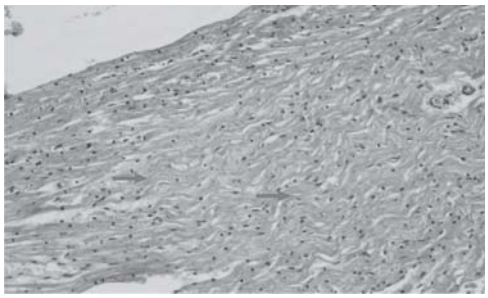
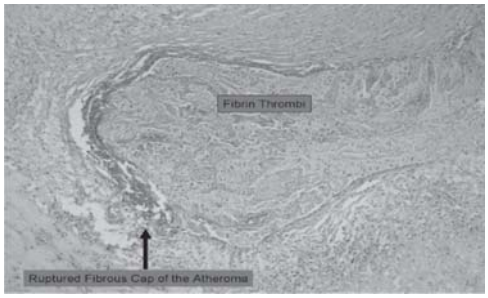


Figure 4: Microscopy of the section from the left anterior descending coronary artery showing more than 95% block of the lumen due to a large circumferential atheromatous plaque composed of a ruptured fibrous cap with superimposed fibrin thrombi.

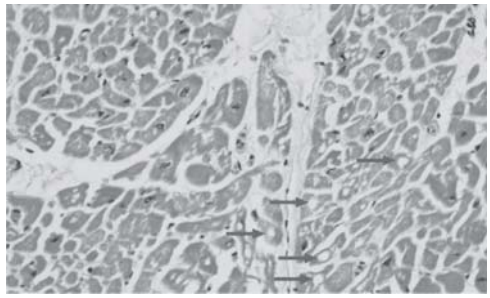


Discussion

Ischemic heart disease (IHD) is the generic designation for a group of closely related syndromes resulting from myocardial ischemia—an imbalance between the supply (perfusion) and demand of the heart for oxygenated blood. Ischemia comprises not only insufficiency of oxygen, but also reduced availability of nutrient substrates and inadequate removal of metabolites. Myocardial infarction (MI), the most important form of IHD, in which the duration and severity of ischemia is sufficient to cause death of heart muscle.[9] MI in young adults can be broadly divided into two groups, those with angiographically normal coronary arteries and those with coronary artery disease (CAD) of varying etiology.[10] The pathophysiology of MI in the presence of 'normal' coronary arteries remains unclear but can be explained on the basis of coronary artery thrombosis, embolisation, spasm or a combination of these processes. Coronary thrombosis can be seen in hypercoagulable states such as in the nephrotic syndrome, antiphospholipid syndrome and protein S, and factor XII deficiencies.[11,12,13,14,] Coronary embolisation is rare but has been reported with endocarditis usually affecting the aortic valve.[15] Coronary artery spasm causing myocardial infarction is recognized with both the recreational and therapeutic use of cocaine.[16,17]

In more than 90% of cases, the cause of myocardial ischemia is reduction in coronary blood flow due to atherosclerotic coronary arterial obstruction. Thus, IHD is often termed coronary artery disease (CAD) or coronary heart disease. In most cases, there is a long period (decades) of silent, slowly progressive, coronary atherosclerosis before these disorders become manifest. Thus, the syndromes of IHD are only the late manifestations of coronary atherosclerosis that probably began during childhood or adolescence.[9] "Premature CAD" is defined as CAD occurring before the age of 65 years in women and 55 years in men, while "coronary artery disease in young" is defined as CAD occurring before the age of 40 years and represents the most severe form of CAD.[18] Incidence of CAD in young has been reported to be as high as 16% among Asian Indians compared to only 2 – 5% in Western population.[6] High rates of CAD in Asian Indians have been shown to be

Figure 3: Microscopy of the section from the anterior wall of the heart showing patchy areas of vacuolar degeneration.



accompanied by a paradoxically low prevalence of conventional risk factors for CAD.[18] They include hypertension, diabetes mellitus, smoking, hyperlipidemia, tobacco consumption and central obesity at much younger age.[8] The only risk factor which was apparent in our case was 6 years history of smoking. Smoking increases the risk of CAD by 3 -5 times. In the first world countries, smoking has significantly decreased and socially looked down-upon. In contrast, in India, smoking is increasing particularly in the young age group. As the demand falls in the West, tobacco traders are dumping this atherogenic material in the Indian market. For Indians, tobacco remains a major risk factor as it is used in different forms.[8]

Early recognition of MI by pathologist can be difficult, particularly when the death has occurred within a few hours after the onset of symptoms. [9] In our case the duration of symptoms was less than 10 hours prior to death. Even though the coagulative necrosis is specific sign of early MI, we were able to demonstrate only waviness of myocardial fibers in the present case probably due to short duration between the onset of MI and death. This patient might had an episode of anginal pain 10 hours prior to death which culminated in a devastating MI. Another possibility is sudden disturbance in conduction system due to acute MI culminating in fatal ventricular arrhythmia. However near total blockade of coronaries ($>$ 95%, which is much above the critical narrowing of 70% cut off point) due to superimposed thrombi on a preexisting atheroma can be taken as a confirmatory evidence for MI as cause of death. This may be one of the very few case reports of MI at a young age (22 years), as per our literature search.

Conclusion

Coronary artery disease (CAD) that manifests at a younger age can have dreadful consequences for an individual, the family, and the society. Prevention of these deaths in young people is a moral responsibility of the nation. Of particular concern to India is not only the high burden of cardiovascular diseases (CVDs), but also the effects of these diseases on young individuals who constitute the productive workforce. There is a need to plan strategies for prevention and halting coronary atherosclerosis which is fast spreading as a malignant epidemic among the young. In the industrialized countries there is a continuing decline of CAD during the last three decades. This has been possibly by focusing on public education programmes for modifying the known risk factors and by targeting high risk individuals. This achievement of the industrialized nations must become a lesson and an inspiration for the physicians and the policy makers in India. This case report also highlights the need for utmost attention in cases of chest pain in young individuals.

Acknowledgement

We also wish to record our token of appreciation to Mrs. Poornima Bhagavath, Lecturer, Department of Chemistry, Manipal University, Manipal for proffering her technical expertise in the preparation of this manuscript.

References

1. Yeolekar ME, Coronary artery disease in Asian Indians. J Postgrad med 1998; 44: 26-8.
2. Mammi MVI, Pavithran K, Abdurahiman P, Pisharody R, Sugathan K. Acute myocardial infarction in north Kerala - A 20 year hospital based study. Indian Heart J 1991; 43 : 93-6;
3. Gupta R, Prakash H, Majumdar S, Sharma S, Gupta VP. Prevalence of coronary heart disease and coronary risk factors in an urban population of Rajasthan. Indian Heart J 1995; 47: 331-8.
4. Enas EA, Garg A, Davidson MA, Nair VM, Huet B, Yusuf S. Coronary heart disease and its risk factors in the first generation immigrant Asian Indians to the United States of America. Indian Heart J 1996; 48: 343-54;
5. Enas EA. High rates of CAD in Asian Indians in the United States despite intensive modification of life style. What next? Curr Sci 1998; 74.
6. Enas EA, Mehta JL. Malignant coronary artery disease in young Asians Indians: Thoughts on pathogenesis, prevention and treatment. Clin Cardiol 1995; 18: 131-5.
7. Goyal P, Kale SC, Chaudhry R, Chauhan S, Shah N. Association of common chronic infections with coronary artery disease in patients without any conventional risk factors; Indian J Med Res 2007; 125: 129-136
8. Rissam HS, Kishore S, Trehan N. Coronary artery disease in young Indians – The missing link; J Indian Acad Clin Med 2001; 2: 128-132
9. Frederick J. Schoen. Ischemic Heart Disease. In: Vinay kumar, Abul k. Abbas, Nelson Fausto, editors, Robbins and Cotran Pathologic Basis Of Diseases. 7th ed. Pennsylvania :Elsevier Saunders; 2005, p. 571-587
10. Osula S, Bell GM, Hornung RS; Acute myocardial infarction in young adults: causes and management; Post grad. Med. J. 2002; 78: 27-30
11. Fujimura O, Gulamhusein S. Acute myocardial infarction: Thrombotic complications of nephrotic syndrome; Can J Cardiol 1987; 3: 267–9.
12. Hamsten A, Norberg R, Bjorkholm M, de Faire U, Holm G. Antibodies to cardiolipin in young survivors of myocardial infarction: an association with recurrent cardiovascular events. Lancet 1986;1(8473):113–16.
13. Manzar KJ, Padder FA, Conrad AR, Freeman I, Jonas EA. Acute myocardial infarction with normal coronary artery: a case report and review of literature. Am J Med Sci 1997; 314:342–5.
14. Penny WJ, Colvin BT, Brooks N. Myocardial infarction with normal coronary arteries and factor XII deficiency. Br Heart J 1985; 53:230–4.
15. Agirbasli MA, Hansen DE, Byrd BF. Resolution of vegetations with anticoagulation after myocardial infarction in primary antiphospholipid syndrome. J Am Soc Echocardiogr 1997; 10:877–80.
16. Simpsons RW, Edwards WD. Pathogenesis of cocaine induced ischaemic heart disease. Autopsy findings in a 21-year old man. Arch Pathol Lab Med 1986; 110:479–84.
17. Ross GS, Bell J. Myocardial infarction associated with inappropriate use of cocaine for treating epistaxis. Am J Emerg Med 1992; 10:219–22.
18. Enas EA, Dhawan J, Petkar S. Coronary artery disease in Asian Indians: Lessons learnt and role of lipoprotein (a). Indian Heart J 1997; 49: 25-34.

Profile of poisoning cases at Belgaum, Karnataka: A cross sectional study

Gurudut K.S[1], Hareesh S.Gouda[2], Sunil C. Aramani[3], Manjula Bai K.H[4]

[1]Assistant Professor, Dept. of Forensic Medicine & Toxicology, Belgaum Institute of Medical Sciences, Belgaum, Karnataka, [2]Assistant Professor, Dept. of Forensic Medicine & Toxicology, J.N.Medical College, Belgaum, Karnataka, [3]Assistant Professor, Dept. of Forensic Medicine & Toxicology, J.N. Medical College, Belgaum, Karnataka, [4]Professor & Head, Dept. of Forensic Medicine & Toxicology, J.N.Medical College, Belgaum, Karnataka

Abstract

According to World Health Organisation, 3 million cases of acute poisoning with 2,20,000 deaths occur annually worldwide. Of these 90% of fatal poisoning occur in the developing countries particularly among agricultural workers. According to an estimate, the number of persons who suffer from effects of poisoning in India, are day by day increasing alarmingly. The present cross-sectional study was carried out to know the pattern of poisoning cases admitted to KLE's Dr.Prabhakar Kore Hospital and MRC, Belgaum,Karnataka, during the period from 01-10-2004 to 30-03-2004. Out of 150 cases of poisoning, maximum number of victims were in the age group 21-30 years (50.67%) and majority of the victims were males (70%). Most of the victims (80%) had used poisons with suicidal intention. Maximum cases (45%) were during the months February to May. Majority of the victims were agriculturists (72%). In our study, maximum number of cases were due to poisoning by agrochemicals (71.33%), followed by antidepressant and anti-epileptic drug consumption (13.33%). Mortality rate was 12.67%.

Keywords

Poison, Organophosphate, Organochlorine, Mortality, Suicide.

Introduction

Poisons have always been a source of fascination and curiosity for the mankind. Poisoning, both accidental and intentional, is a significant contributor to mortality and morbidity throughout the world. According to WHO, 3 million cases of acute poisoning with 2,20,000 deaths occur annually worldwide. Of these 90% of fatal poisoning occur in the developing countries particularly among agricultural workers.[1,2] According to an estimate, the number of persons who suffer from effects of poisoning in India, are day by day increasing alarmingly.[3] Sadly its heartwarming to note that people are killing themselves intentionally than the poisons being mishandled. This shows that the problem is getting worse with time as newer drugs and chemicals are developed in vast numbers. In this regard the exact scale of this problem in India still remains uncertain. It is reported that 1 to 1.5 million cases of poisoning occur every year of which nearly 50,000 people die. Thus making India, the country with highest incidence of poisoning in the world.[4, 5] But, whatever facts and figures depicted above, they are just tips of ice bergs. Because still a large number of poisoning cases go unnoticed or unreported. As per National Crime Records Bureau, India, reports if about 25,000 persons lose their lives due to poisoning, the reported number is shockingly about 2,000 only.[6] Poisoning is the 4th most common cause of mortality in rural India.[7] Type of poison used/ exposed has also changed a lot and depends on variety of factors like availability of poisons, socioeconomic status of population, religious and cultural influences, occupations and environmental factors.

Corresponding author:
Dr. Gurudut K.S, Assistant Professor, Dept. of Forensic Medicine & Toxicology, Belgaum Institute of Medical Sciences, Belgaum, Karnataka
Telephone: +919886643155, E mail: ksgurudutt@rediffmail.com

Methodology

The present cross-sectional study was carried out to know the pattern of poisoning cases admitted to KLE's Dr.Prabhakar Kore Hospital and MRC, Belgaum, Karnataka, during the period from 01-10-2004 to 30-03-2006, which were brought to the Hospital directly or as a referred case. Information about age of the victim and details of the poisoning were gathered from all possible sources like enquiring the victim's relatives and friends, police records along with direct conversation with the investigating officer, and hospital records. Hospitalized, cases were treated solely on signs and symptoms but were not subjected to confirmation by sending to Forensic Science Laboratory (FSL), however, in fatal cases the type of poison is confirmed by FSL. The so obtained data from all the above said sources was recorded in the proforma and analysed to fit into the objectives of this study.

Results

Out of 150 cases of poisoning, maximum number of victims were in the age group 21-30 years (76 cases; 50.67%), followed by 31-40 years (29 cases; 19.33%) and least (4 cases; 2.67%) in the age group of 51-60 years [Table 1]. Majority of the victims were males (105 cases; 70%) with male: female ratio being 2.3:1 [Table 1]. Maximum victims, 120 persons (80%) had used poisons with suicidal intention and 30 persons (20%) were accidentally exposed. Agrochemicals topped both the categories. None of the cases were homicidal [Table 2]. For assessment of seasonal variation, poisoning cases during one complete year from 01-01-2005 to 31-12-2005 were selected. There were 100 admissions of poisoning cases during this period. Maximum cases (45%) were in the summer months ie. February to May [Table 3]. Majority of the victims were agriculturists (108 cases; 72%). Yet again in both agriculturists and non-agriculturists agrochemicals were the most common agents abused as suicidal and accidental agents [Table 4]. In our study, maximum number of cases were due to poisoning by agrochemicals (107 cases; 71.33%), followed by antidepressant and anti-epileptic drug consumption (20 cases; 13.33%). Corrosives contributed for 4% (6 cases) and alcohols for 4.66% (7 cases). Animal bites contributed for 6% (9 cases) of the cases. A case of hydrocyanic acid was also admitted [Table 5]. Out of 150 cases, 19 victims died (12.67%) and of these 18 cases were suicidal and one was accidental poisoning which was due to scorpion sting. Among fatal cases once again agrochemicals topped the list (13 cases; 68.42%) [Table 6].

Discussion

In the present study, more than 50% victims were in the age group 21-30 years. Among the 19 expired cases, again maximum

Table 1: Age and Sex wise distribution of cases:

Age (years)	Male	Female	Total	
			Number	%
<10	04	03	07	04.67
11-20	12	05	17	11.33
21-30	56	20	76	50.67
31-40	21	08	29	19.33
41-50	10	07	17	11.33
51-60	02	02	04	02.67
Total	105(70%)	45(30%)	150	100

Table 2: Distribution of cases based on manner of poisoning:

Type of Poison	Suicidal		Accidental		Homicidal
	Number	%	Number	%	Number
Agrochemicals	95	79.16	12	40.00	00
Antidepressants & antiepileptic tablet	20	16.54	00	00.00	00
Corrosives	04	03.47	02	06.67	00
Alcohol	00	00.00	07	23.33	00
Hydrocyanic acid	01	00.83	00	00.00	
Snake Bite	00	00.00	08	26.67	00
Scorpion Sting	00	00.00	01	03.33	00
Total	120 (80%)	100	30 (20%)	100	00

Table 4: Distribution of cases based on occupation of victims:

Type of Poison	Agriculturists	Non-agriculturists
Agrochemicals	77	20
Antidepressants &	07	03
anti epileptic tablet	08	12
Corrosives	04	02
Alcohol	05	02
HCN	00	01
Snake Bite	06	02
Scorpion Sting	01	00
Total	108 (85.3%)	42 (14.7%)

Table 6: Profile of fatal poisoning cases:

Type of Poison	Number	%
1) Agrochemicals a) Organophosphates:11 b) Organochlorines: 02	13	68.42
2)Antidepressants & anti-epileptic tablet	0	00.00
3) Corrosives	3	15.80
4) Hydrocyanic acid	1	05.26
5) Alcohols a) Ethyl alcohol: 0 b) Methyl alcohol:1	1	05.26
6) Animal bites a) Snake bites: 0 b) Scorpion bites:1	1	05.26
Total	19	100

number of victims were in the age group of 21-30 years (57.9%). This result is similar to the results of the studies at, Berhampur, Orissa where the maximum number of cases were in age group of 21-30 years (40.5%),[8] District Civil Hospital, Beglaum, 20-29 years (40%),[9] University Medical Unit, Sri Lanka, 20-29 yeas (54%) [10] and in most of the other studies the maximum number of cases fall in the same age group.[11,12,13,14,15] The only wide contrast result was in the study done at Forensic Medicine Department of Leeds where the mean age of maximum cases was 51 yrs for females and 45 years for males.[16] The present study along with many other studies, gives the picture that its the youth between the ages of 20-29 years who are more prone for poisoning. In most of these youths, it is suicidal intent rather than accidental which is killing factor, which is a big loss to any society or nation. Reasons seem to be many, mental instability and inability to face adverse eventualities in life like unemployment in spite of being graduates, love failures, repeated failures in exams, failure of crops, family disputes, poverty and so on.

In our study, more than 2/3rd victims were males with male:female ratio being 2.3:1. These ratios are similar to that of the studies at, S.N. Hospital Agra, where M : F = 2.33:1,[17] Government Medical College, Jammu, M:F = 2.55 :1, [18] Rohtak, M:F = 2.18 : 1, [19] Government Medical College, Chandigarh, M:F = 2.5:1,[15] Government Wenlock

Table 3: Seasonal variation of cases:

Season	Number	Percentage
Summer [February to May]	45	45
Rainy [June to September]	28	28
Winter [October to January]	27	27
Total	100	100

Table 5: Profile of poisoning cases:

Type of poison	Number	Percentage
1) Agrochemicals a) Organophosphates:97 b) Organochlorines:10	107	71.33
2) Antidepressants and anti-epileptic tablet a) Diazepam:9 b) Barbiturates: 6 c) Alprazolam:3 d) Lorazepam:2	20	13.34
3) Corrosives a) Sulphuric acid:3 b) Nitric acid:2 c) Hydrochloric acid:1	6	04.00
4) Alcohol a) Ethyl alcohol:6 b) Methly alcohol:1	7	04.66
5) Animal bites a) Snake bites:8 b) Scorpion bites:1	9	06.00
6) Hydrocyanic acid	1	00.67
Total	150	100

Hospital, Mangalore, M:F = 2.3:1,[20] Civil Hospital, Belgaum, M:F = 2.8:1,[9] University Medical Unit, Srilanka, 2.5: 1 [10] and in most of the other studies male preponderance is evident.[3,12,13,14] The results in contrast to the present study was observed from the studies at, Southern District, Srilanka where M: F=1:1, [21] Department of Forensic Medicine Leeds where females outnumbered males, M:F = 1:1.03[16] Khonkaen Hospital, Thailand, M:F = 1:1.2.[22] It is evident from present study and most other studies, that males are doubly (or even more) prone for poisoning. Females are well guarded from adversities of life, starting from childhood up to old age, as daughter by parents, as wife by husband and as mother by son. The Indian society, traditionally and culturally, is sympathetic to women which boosts their morality and self-confidence in life. Man being the bread earner of the family in most cases, all transactions go in his name. If failed to fulfill the basic requirements for the family, due to frustration they end their lives.

In the present study, 80% cases were suicidal in nature 20% cases accidental. None were homicidal. This results are similar to results of studies at Office of Judicial Medical Officer, Colombo, where percentage of suicidal cases were 77.7%, [23] Government Medical College, Jammu – 84.1%,[13] Government Medical College, Chandigarh – 89.1%, [15] Rural Hospital, Trichy – 75.2%,[24] Medico-legal Institute, Colombo - 75%,[25] Post-graduate Institute, Chandigarh – 71.8%[12] and in most of other studies poison has been consumed mainly with suicidal intention.[3,14,18,19,20] It is evident from the present study as well as from other studies quoted, that in most of the poisoning cases, suicide is the main intention, in both developing and developed countries. India recorded a 31.6% rise in number of suicide cases between 1993 (84,244 cases) and 2003 (1,10,851 cases) and the most common method chosen for suicide was by consumption of poison in general and agrochemicals in particular.[26] Agrochemicals are the most commonly used agents in developing countries like India, Srilanka and Bangladesh. This can be attributed to the fact that, even for trivial problems, people have found suicide as best solution by agrochemicals which are easily available and which could be easily consumed.

In this study, majority of the victims were from agricultural

community. This results tally with the results of studies conducted at District Civil Hospital, Belgaum,[9] which is a different hospital in the same region of the present study, and B.K. Hospital, Faridabad, Haryana.[3] In most of the other studies, there have been no comments on this aspect of occupation. It is evident from the present and two other studies, that agriculturists are more prone for suicidal poisoning. The reasons as gathered orally, from victims and relatives seem to be manifold, like, repeated crop failure, unable to return the loans taken for the sake of agriculture, ever increasing debt and interest of loans, lack of even basic needs for survival back home, no knowledge of other jobs etc. Thus frustrated and dejected, they end their lives. In the present study, maximum number of cases were observed in summer season. This is similar to the study done at Berhampur, Orissa.[8] This seasonal variation with summer season dominance can be attributed to agriculture and rain. In summer there is scarcity of water for agriculture. In India rain is the main source of water for agriculture and failure of adequate rains has become a common phenomenon in most parts of the country with some exceptions. This drought situation is highly unsuited for cultivation. Thus those families which are totally dependent on agriculture and rains for agriculture, face the worst threat for basic needs. Finally instead of starving to death, they cling on to this method of suicide. Obviously such people use agrochemicals present at home.

In this study, maximum number of cases were due to agrochemicals (71.33%). This result is similar to the results of the studies conducted at university medical unit at Perideniya, Srilanka where it was 65.36%,[10] Judicial Medical Officer Office, Colombo-81%, [23] District Civil Hospital, Belgaum – 62.1%, [9] and many other studies had the result of favor of agrochemicals with varying percentages.[3,17,18,19] But this result of agrochemical dominancy is in contrast with the results of the studies conducted at Department of Forensic Medicine, Leeds where the most common type of poisoning was carbon monoxide (90% in males and 70% in females), [16] at Rural Hospital, Trichy, it was vegetable poisons (87.8%),[24] at Referral Hospitals, Zimbabwe, it was traditional medicines (22.95), [27] and at Khonkara Hospital, Thailand, it was animal poisoning. [22]

Among 107 cases of agrochemicals, maximum number of cases were due to organophosphorous compounds (64.67%). This result is similar to studies conducted at, Office of Judicial Medical Officer, Colombo (57.6%),[23] District Civil Hospital, Belgaum (60.4%),[9] Medico-legal Institute, Colombo (57.6%),[25] University Medical Unit at Perideniya (51%)[10] and also in certain other studies organophosphorous was the commonest agrochemical abused.[8]

Agrochemicals in general and organophosphorous in particular are the agents commonly abused, as seen in the present study. This can be attributed to a number of factors, like, easy availability as they are sold in open market without strict vigil and also much cheaper. The occupation of most victims being agriculture, these chemicals are almost always present in home and readily procurable. These can be easily consumed orally. Another thing that was noticed, upon inquiring the hospitalized victims, was they were sure of mortality due to these compounds as they have seen many die the same way in their vicinity. Thus people are more knowledgeable about agrochemicals than any other poison. The contrast results can be attributed to place of study, people's knowledge about poisons, social practices and environment in which they reside. As in Leeds and Germany, the people are sophisticated, more knowledgeable, most own cars and thus carbon monoxide was the common poison abused. Trichy being a place surrounded by dense forests and people being well versed with the different plant products, vegetable poisons were maximum. Zimbabwe being a backward underdeveloped country, old traditional ways of treating patients still in practice, these traditional medicines have contributed the most.

In this study, the overall mortality due to poisoning was 12.67%. This result is similar to the results of the studies at, S. N. Hospital Agra (11.5%),[17] Berhampur, Orissa (20.3%),[8] PGI, Chandigarh (17.30%),[12] University Medical Unit, Sri Lanka (16%),[10] Government Wenlock Hospital, Mangalore (15.7%)[20] and Referral Hospitals, Zimbabwe (15%).[27] The results of the present study are in contrast with the results of studies done at Britain (0.712%)[28] and U.S (0.101%)[29] where mortality rates were very low. Whereas, mortality in studies at District Civil Hospital, Belgaum (30.7%)[9] and Medical College Hospital, Rohtak (35.82%)[14] were higher than the present study.

In the present study, out of 19 cases expired, maximum (67.43%) were due to agrochemicals (organophosphates-11 & organochlorines-2). This is similar to results at District Civil Hospital, Belgaum (72%),[9] Medcial College, Rohtak (67.6%),[14] and Shri. Vasantrao Naik Medical College, Yavatmal (55.4%).[29] In many other studies the agents causing maximum deaths were Agrochemicals (either organophosphates or aluminium phosphide) with varying percentages.[8,19] The reasons behind agrochemicals becoming main causative agents can again be attributed to the same reasons as quoted previously. So these agents which are meant to protect crops, are unfortunately used by humans to kill themselves.

Conclusion

Poisoning has become a major cause for morbidity and mortality in India. Poisoning by agrochemicals has become practically inevitable, because modern day agriculture is unthinkable without the use of agrochemicals. The abundant availability of pesticides also cannot be cut off in India as annual losses due to pests are very high. Most of the cases of poisoning in India are suicidal, particularly in males. Thus basically the reasons behind this suicidal intent like poverty, unemployment, immaturity to deal with adversities of life, financial insecurity etc have to be addressed. This can be achieved to some extent by educating people about the importance of self-employment, small scale industries, new policies of government etc. India being agriculture based country, the problems faced by agriculturists needs special attention, which are like scarcity of water, power cuts, financial burden of loans etc. These can be tackled by educating them about newer methods of agriculture, proper storage of water, schemes like crop insurance in case of crop failure, subsidies by the government, avoiding middle men (brokerage) for selling their products. In addition to the above, implementation of pension system for farmers especially who are below poverty line, facilities of free education and food for children of farmers so that they gain knowledge for further improvement in agriculture or self-employment; frequent awareness campaigns about scientific methods of agriculture by agricultural officers, concession in expenditure towards the transportation of agricultural products, etc. should also be made. Incidence of mortality due to poisoning can effectively be reduced by the establishment of Poison Control Centre at every referral or tertiary level hospitals for the immediate diagnosis and successful management of cases of poisoning.

Reference

1. Reddy KSN. The essentials of forensic medicine and toxicology. 24th ed. Hyderabad: K Sugunadevi; 2005.
2. Kumar A, Vij K. Trends of poisoning in Chandigarh – A six year autopsy study. Journal of Forensic Medicine and Toxicology 2001 Jan-Jun; 18(1):8-10.
3. Bharadwaj DN, Dogra TD, Singh B, Reddiah VP, Kulshrestha S. Status of poisoning in Faridabad district of Haryana. Medicolegal Update 2004; 4(3):79-83.
4. Dudani AT. Taking the sting out of poisons. The Economic Times 1995 November 16; 6.
5. Aggarwal P, Murthy OP. Approach to manage a patient with poisoning – proceeding of national workshop on practical and emergency toxicology 1998a; 1:25-31.
6. Gururaj G. Injuries in India – A national perspective. Available from: http://www.whoindia.org
7. Shankar A. Handbook of Poisonings. 2nd ed. Mumbai : Bhalani Publishing House; 2005.
8. Dash SK, Raju AS, Mohanty MK, Patnaik KK, Mohanty S.

Sociodemographic profile of poisoning cases. Journal of Indian Academy of Forensic Medicine 2005; 27(3).
9. Jirli PS. A cross sectional study of poisoning cases admitted and autopsied at district hospital, Belgaum (unpublished Doctoral Dissertation, Rajiv Gandhi University of Health Sciences, Bangalore 2001), p.54-65.
10. Senanayake N, Karalliedde L. Pattern of acute poisoning in a medical unit in central Sri Lanka. Forensic Science International 1988; 36:101-104.
11. Singh S, Wig N, Chaudhary D, Sood NK, Sharma BK. Changing pattern of acute poisoning in adults – experience of a large north-west Indian hospital. Journal of Association of Physicians of India 1997; 45(3):194-196.
12. Singh S, Sharma BK, Wahi PL, Chung KS. Spectrum of acute poisoning in adults (10 year experience). Journal of Association of Physicians of India 1984; 32(7):561-563.
13. Sharma BR, Dassari H, Vij K. Poisoning in northern India – Changing trends, causes and prevention thereof. Medicine Science and the Law 2002; 42(3):251-257.
14. Siwach SB, Gupta A. The profile of acute poisonings in Haryana – Rohtak study. Journal of Association of Physicians of India 1995; 43(11):756-759.
15. Vij K, Sharma BR, Dassari H. Poisoning in northern India – Changing trends, causes and prevention thereof. Med Sci Law 2002; 42(3):251-257.
16. Crowe MTI. Trends in fatal poisonings in Leeds, 1977 to 1987. Medicine Science and the Law 1989; 29(2):124-129.
17. Tandon SK, Qureshi GV, Pandey DN, Agarwal A. A profile of poisoning cases admitted in SN Medical College and Hospital, Agra. Journal of Forensic Medicine and Toxicology 1996; 13(1&2):10-12.
18. Sharma BR. Trends of poisons/ drugs abused in Jammu. Journal of Forensic Medicine and Toxicology 1996; 13(1&2):7-9.
19. Dhattarwal SK, Dalal SS. Profile of deaths due to poisoning in Rohtak, Haryana in the year 1995. Journal of Forensic Medicine and Toxicology 1997; 14(1):51.
20. Unnikrishnan B, Singh B, Rajeev A. Trends of acute poisoning in South Karnataka. Katmandu University Medical Journal 2005 (Cited 2006 July 13); 3(2):149-154. Available from: http://www.kumj.com.np/past/vol3/issue10/145-154.
21. Islam HN, Islam N. Retrospective study of 273 deaths due to poisoning at Sir Salimullah Medical College from 1988 to 1997. Legal Medicine 2003; 5(1):129-131.
22. Chirasirisap K. A study of major causes and types of poisoning in Khonkaen, Thailand. Vet Hum Toxicology 1992; 34(6):489-492.
23. Alwis LBL, Salgado MSL. Agrochemical poisoning in Sri Lanka. Forensic Science International 1988; 36:81-89.
24. Aleem MA, Paramasivam M. Spectrum of acute poisoning in villagers. Journal of Association of Physicians of India 1993; 41(12):859.
25. Alwis LB, Salgado MS. Agrochemical poisoning in Sri Lanka. Forensic Science International 1988; 36(1-2):81-89.
26. 31.6% rise in number of suicide cases between 1993 and 2003. The Hindu 2006 July 12; 6.
27. Nhachi CF, Kasilo N. The pattern of poisoning in urban Zimbabwe. Journal of Applied Toxicology 1992; 12(6):435-438.
28. Eddleston M, Phillips M. Self-poisoning with pesticides. British Medical Journal 2004; 328:42-44.

An analysis of 188 cases of fall from height at Belgaum, Karnataka

Hareesh S.Gouda*, Ajay Kumar T.S**

*Assistant Professor, Dept. of Forensic Medicine & Toxicology, J.N.Medical College, Belgaum, Karnataka, India, **Post Graduate, Dept. of Forensic Medicine & Toxicology, J.N.Medical College, Belgaum, Karnataka, India

Abstract

Fall is an event which results in a person coming to rest inadvertently, on the ground or floor or other lower level as per World Health Organization. As per National Crimes Record Bureau of India, fall from height accounted for 2.6% of accidental deaths and for 2.8% of accidental deaths due to un-natural causes in India in the year 2008. In this study, 188 cases of fall from height admitted to the KLE's Dr.Prabhakar Kore Hospital & MRC, Belgaum, Karnataka, during the period from 01-01-2009 to 31-12-2009 were analyzed. Males outnumbered females with ratio being 3:1. Majority of the victims were in the age group 21-30 years (25%) and more than 50% victims were in the earning age group of 21-50 years. Victims from urban areas (55.9%) were more than rural (44.1%). Most of the falls (41%) occurred during the afternoon and evening hours and during summer months (36.1%) with maximum cases in the month of April (11.8%). More than 50% falls occurred at home. Majority of the victims fell from the staircase (25%), followed by tree (12.8%). Head and face was the most commonly injured region (37.5%). Mortality rate was 10.1% and in majority of the cases cause of death was head injury (47.4%).

Keywords

Fall from height, staircase, mortality, cause of death, head injury.

Introduction

Fall is an event which results in a person coming to rest inadvertently, on the ground or floor or other lower level as per World Health Organization.[1] falls may be accidental, suicidal or homicidal, however, among these accidental falls are encountered more commonly. Morbidity and mortality due to fall from height puts significant financial burden on the individual and his family and health care system. Also, the victims and their family face mental as well physical stress. Most of the incidents of fall are preventable. Constant supervision of the children by parents and teachers, safety measures at workplace and apart from these, importantly the alertness of the people during work or day to day activities will minimize such events to greater extent. As per National Crimes Record Bureau of India, fall from height accounted for 2.6% of accidental deaths and for 2.8% of accidental deaths due to un-natural causes in India in the year 2008.[2]

Methodology

All the cases of fall from height admitted to the KLE's Dr.Prabhakar Kore Hospital & MRC, Belgaum, Karnataka, during the period from 01-01-2009 to 31-12-2009 were included in the study. This cross-sectional study was analyzed with respect to the age, sex, place of residence (urban/ rural), site of accident (home/ workplace/ school or play ground), place and time of fall, injuries sustained, cause of death in fatal falls and manner of fall (accidental/ suicidal/ homicidal). Cases of falls from animals, vehicles, burning buildings, falls into water and machinery were excluded from the study. Data required for the study were obtained by direct interrogation with the patients and/ or their parents or relatives, hospital records and autopsy reports. Patients were examined and injuries sustained were recorded. All the data collected are analyzed with respect to the objectives of the study.

Corresponding author:
Dr. Hareesh .S.Gouda
Assistant Professor, Dept. of Forensic Medicine & Toxicology, J.N. Medical College, Belgaum-590010, Karnataka State, INDIA
Telephone: +919620237977
E mail: hareeshfmt@rediffmail.com

Results

During the study period, total of 188 cases of fall from height were admitted to the KLE's Dr.Prabhakar Kore Hospital & MRC, Belgaum. All the cases were accidental. Number of victims died was 19, accounting for 10.1% mortality rate. Cases involving males (142 cases; 75.5%) were more than females (46 cases; 24.5%) [Table 1]. Majority of the victims were in the age group 21-30 years (47 cases; 25%) followed by 31-40 years (36 cases; 19.1%). Eighty six percent victims were below 50 years and more than 50% victims were in the earning age group 21-50 years [Table 1]. Most of the falls (77 cases; 41%) occurred during the afternoon and evening hours (12 noon to 6 pm period) [Table 2]. Maximum number of falls occurred at home (96 cases; 51.1%) followed by workplace (77 cases; 40.9%) [Table 3]. Majority of falls occurred during summer months i.e February to May (68 cases; 36.1%) with maximum cases in the month of April (21 cases; 11.8%) [Table 4]. Majority of the falls were from staircase (47 cases; 25%), followed by tree (24 cases; 12.8%) and ladder (19case; 10.1%) [Table

Table 1: Age and Sex wise distribution of cases:

Age (Years)	Male	Female	Total	
			Number	Percentage
d" 10	15	12	27	14.4
11-20	19	07	26	13.8
21-30	38	09	47	25.0
31-40	28	08	36	19.1
41-50	20	04	24	12.8
51-60	12	00	12	06.4
61-70	04	03	07	03.7
71-80	04	02	06	03.2
81-90	01	01	02	01.1
> 90	01	00	01	00.5
Total	142 (75.5%)	46 (24.5%)	188	100

Table 2: Distribution based on Time of fall:

Time	Number	Percentage
6 am-12 noon	63	33.5
12 noon-6 pm	77	41.0
6pm-12 mid night	43	22.9
12 midnight-6 am	05	02.6
Total	188	100

Table 3: Distribution based on site of fall:

Site of Fall	Number	Percentage
Home	96	51.1
Workplace	77	40.9
School/ Playground	15	08.0
Total	188	100

Table 4: Month wise distribution of cases:

Month	Number	Percentage
January	13	06.9
February	14	07.4
March	16	08.5
April	21	11.2
May	17	09.0
June	15	08.0
July	20	10.7
August	15	08.0
September	15	08.0
October	15	08.0
November	10	05.3
December	17	09.0
Total	188	100

Table 5: Distribution based on place of fall

Place	Number	Percentage
Rooftop	14	07.5
Staircase	47	25.0
Balcony	08	04.3
Cot/ Crib	06	03.2
Chair/ Furniture/ Playing equipment	06	03.2
Window	01	00.5
Attic	04	02.1
Wall/ Compound	06	03.2
Ladder	19	10.1
Tree	24	12.8
Building	15	07.9
Electric Pole	11	05.9
Fall from vehicle while repairing/ loading	13	06.9
Others	14	07.4
Total	188	100

Table 6: Distribution of cases based on Body region involved:

Body Region	Number	Percentage
Head & Face	69	37.5
Neck	07	03.5
Thorax	13	06.8
Abdomen & Pelvis	09	04.6
Extremity	34	18.0
More than one region involved· • Head&faceandExtremity:37· • Thorax and Abdomen:02· • Thorax and Extremity:06· • Abdomen and Extremity:04 · • Head& face and Abdomen02· • Head& face and Thorax:05	56	29.6
Total	188	100

Table 7: Distribution based on residence:

Residence	Number	Percentage
Urban	105	55.9
Rural	83	44.1
Total	188	100

Table 8: Distribution of cases based on Cause of Death:

Cause of death	Number	Percentage
Head Injury	09	47.4
Spinal injury	06	31.5
Thoracic Injury	01	05.3
Abdominal injury	01	05.3
Injury to extremity	02	10.5
Total	19	100

5]. Head and face was the most commonly injured region (69 cases; 37.5%), followed by involvement of more than one body regions (56 cases; 29.6%) [Table 6]. Victims from urban areas were 105 (55.9%) and rural areas 83 (44.1%) [Table 7]. Out of the total 188 victims, 19 (10.1%) died and in majority of the cases (9 cases; 47.4%) cause of death was head injury [Table 8].

Discussion

Falls from height is one of the common causes of morbidity and mortality in all age groups. In this study, all falls were accidental and this is similar to the result of the study by Osifo OD et al. More than 50% victims were in the earning age group of 21-50 years. People of this age group are more active and go out for the work; hence, they are more prone for accidental fall. However, in the similar study conducted by Al B et al. [3], 83.5% victims were below 20 years. In our study, males had a higher rate of fall from height than females and this is consistent with the result of the studies by Al B et al. [3] and Osifo OD et al.[4]

In the present study, about 75% of falls occurred during the day time i.e from 6 am to 6 pm. This could be due to the fact that the people are more active during this period, adults at work, children playing etc. In this study, we observed no significant difference in the incidence of falls at home and workplace, which suggests that falls can occur anywhere.

In the present study, majority of the falls occurred during the summer season i.e February to May, followed by rainy season June to September. Children have contributed significantly to the number of cases of fall in the summer. This could be attributed to the fact that children are more involved in playing during these months of school vacation. Higher number of cases in the rainy season could be due to the slippery outdoors. In the study done by Osifo OD et al., [4] peak incidence of falls was between March and May. In this study, maximum number of falls were from staircase followed by tree and building. However, in the study by Osifo OD et al.,[4] falls from moving vehicles, top of the buildings and treetops were common.

In our study, head and face was the most commonly injured body region. This could be due to the reason that our hospital is a tertiary level referral hospital and cases of head injury are referred from neighboring districts of Karnataka, Goa and Maharashtra. Conversely, in the study by Hahn MP et al. the most common injuries were fractures of the thoracic and lumbar spine (83%) with a preference for the thoraco lumbar junction.[5] Cranio-cerebral injury was the most common injury in fatal falls in this study and similar was the result of the study by Al B et al.[3]

Conclusion

Fall from height occur in the individuals of all the age groups, however, the cause for the falls may vary. Burden due to the falls can be reduced by preventing the occurrence of falls. Paediatric falls can be minimized by the constant supervision of the children by parents and teachers; falls in adults can be reduced by education and use of safety measures at workplace, moreover, the young active risk taking adults require education regarding the ill effects of falls and should ask them not to involve in any activity and acts which have the possibility of sustain injuries due to fall; elderly people should be very cautious during their day to day activities as they are more prone to fall during their routine work at home because of their physical condition.

Reference

1. Peden M, Oyegbite K, Ozanne-Smith J, Hyder AA, Branche C, Rahman AKMF et al. World Report on Child injury prevention-WHO & Unicef. Geneva: WHO Press; 2008:101-121.

2. Accidental Deaths in India. National Crimes Record Bureau - Ministry of Home Affairs. [Cited 2010 May 13]. Available from: http://ncrb.nic.in/ADSI2008/accidental-deaths-08.pdf.
3. Al B, Yildirim C, Coban S. Falls from heights in and around the City of Batman. Ulus Travma Acil Cerrahi Derg 2009; 15(2):141-7.
4. Osifo OD, Iribhogbe P, Thomas HI. Falls from heights: Epidemiology and pattern of injury at the accident and emergency centre of the University of Benin Teaching Hospital. Injury 2010; 41(5): 544-7.
5. Hahn MP, Richter D, Ostermann PA, Muhr G. Injury pattern after fall from great height – an analysis of 101 cases. Unfallchirurg 1995; 98(12): 609-13.

Adenomatoid odontogenic tumour of maxilla – A case report

Kamala R.*, Sunita Srivastava**

*Prof. and HOD, **Post Graduate, Department of Oral Medicine and Radiology, Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow.

Abstract

Adenomatoid odontogenic tumour (AOT) is a benign epithelial odontogenic tumour that accounts for 3 - 7% of all odontogenic tumours and only 0.1% of tumours and cysts of jaw. In 1907, Dreibladt first described this rare tumour as pseudoadenoma adamantinum.[1] Clinically it present as a slow growing, painless mass found in maxilla .More frequently seen in females and has a peak incidence in the second decade of life. There are three variants of AOT: follicular, Extrafollicular and peripheral type.[2] Irrespective of the type they show similar histological appearance with gland like structures, calcifying areas and amyloid like material .

A case report of adenomatoid odontogenic tumour of the maxilla, in a 20 year old male patient .

Keywords

Adenomatoid odontogenic tumour; Maxilla, extrafollicular, ductlike structure

Introduction

The adenomatoid odontogenic tumour is an uncommon histologic type of odontogenic tumour which is characterized by the formation of ductlike structures by the epithelial component of the lesion. [3]

In 1907, Dreiblast first described this rare tumor as 'pseudo - adenoma adamantinum'. ·Later, it was labeled as 'epithelial tumor' by Stafne in 1948 and was credited for recognizing the AOT as a separate entity. It was also termed as adenoameloblastoma by [Thoma 1955, Bemier and Tick, 1956, Gorlin and Choudary (1958), Waldron (1959), Topazian (1960), Shira (1961)];[4,5] AOT has been formerly reported in the literature under varying designations such as : Adenoameloblastoma, which was thought to be a subtype of ameloblastoma

In 1969 Philipsen and Birn fully characterized AOT as a distinct clinical and histopathologic benign tumour of odontogenic origin and presented a review based on 76 cases of AOT, which showed the tumour to be an entity clearly distinguishable from solid or multicystic ('classical') ameloblastoma. They introduced the term adenomatoid odontogenic tumour, to be adopted by the WHO in their "Histological Typing of Odontogenic Tumours, Jaw Cysts and Allied Lesions" and believed that the lesion is not a neoplasm and it is now the generally accepted nomenclature.[6]

Case Report

A 20 year-old male patient reported to the Department of oral medicine and radiology of Sardar patel Post Graduate institute of Dental and Medical Science., Lucknow with a complaint of swelling in the left side of upper jaw since one year. The swelling was insidious in onset which gradually increased to the present size. Initially swelling was painless but later developed pain. Pain was gradual in onset, dull, localized & intermittent in nature .There was no history of trauma and not associated with, fever, numbness or pus discharge.

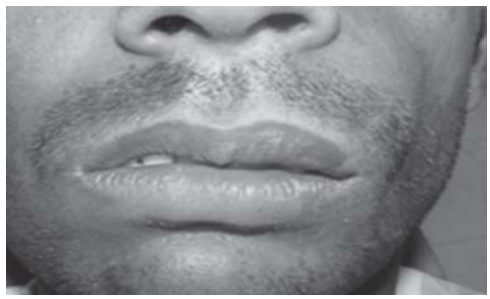
Corresponding Author:
Dr.Kamala.Rawson
Prof. and HOD, Department of Oral Medicine and Radiology, Sardar Patel Post Graduate Institute of Dental and Medical Sciences Lucknow, UP. (E-mail ID- kamalarawson@yahoo.com)

The patient medical history and family history were non-contributory. On general Examination gait was normal, moderately built and nourished with all the vital signs within the normal limits.

Extraoral Examination revealed a single localized swelling in the left middle third of face involving body of maxilla causing facial asymmetry, measuring 4 x 3 cm, oval in shape and superiorly extended from the infraorbital margin to the left lip commissure inferiorly, medially extended from left ala of the nose laterally to the zygomatic arch causing obliteration of nasolabial fold, with well defined margins. Skin overlying the swelling was normal with no surface pulsation. On palpation there was no local rise in temperature, non tender, hard in consistency, not freely movable.(fig 1)

Intraoral Examination revealed a solitary swelling seen in left maxillary alveolus and palatal mucosa. Measuring 4x 2 cm on labial and 2x3cm

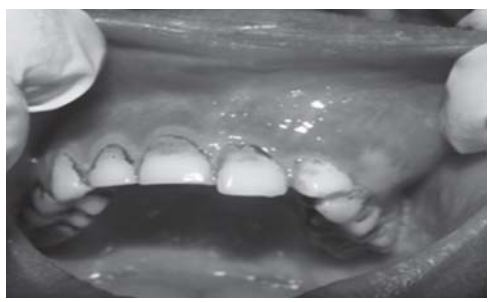
Fig. 1: Extra oral photograph showing facial asymmetry due to swelling in left middle third of face



on palatal side, extending from mesial of 21 to distal of 25 anterioposteriorly with obliteration of labial vestibule and extending palatally from left palatal rugae up to midpalatal suture involving 21 to 24 with well defined borders, and overlying mucosa was normal. On palpation inspectory findings were confirmed, swelling was non tender, hard in consistency and bicortical expansion was seen. Fixed to the underlying structure. Hard tissue examination revealed mobility grade I in relation 24, grade II in relation 23. Extrusion – of 21 and displacement – 22, 23.Pulp vitality test revealed nonvital 22 & 23. On aspiration straw coloured fluid was obtained. (fig 2)

Based on the History ,clinical examination a provisional diagnosis of lateral periodontal cyst was included and a clinical differential diagnosis

fig 2: Intraoral photograph showing a large swelling in left maxillary alveolus causing obliteration of labial sulcus causing extrusion of 21 and displacement of 22

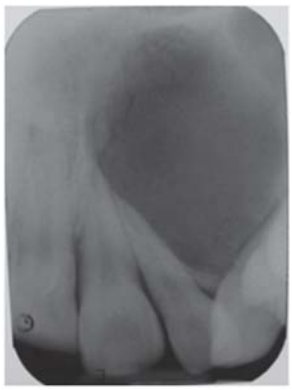


of adenomatoid odontogenic tumor, Calcifying epithelial odontogenic cyst, unicystic ameloblastoma, Dentigerous cyst were included.

Intraoral periapical radiograph in relation to 21, 22, 23 region revealed a large, well-circumscribed unilocular radiolucency between 22 and 23. The radiolucency is inverted pear shaped, with sclerotic border measuring 3cm in diameter with displacement of 22, 23, 24 had no significant root resorption. Small flecks of calcification seen at the periphery of radiolucent lesion. Maxillary occlusal radiograph revealed a unilocular radiolucency extending from 21 to 26 with bicortical expansion with thinning of buccal and lingual cortices. Panoramic view revealed a unilocular radiolucency extending from from 22 to 25.There is no root resorption of 23,24 and displacement of 21, 22 ,23 and 24. (fig 3)

Blood investigations were carried out and were within normal limit. The patient was admitted and treated under general anaesthesia.

Fig 3: Intraoral periapical radiograph showing a large, well-circumscribed unilocular radiolucency between 22 and 23



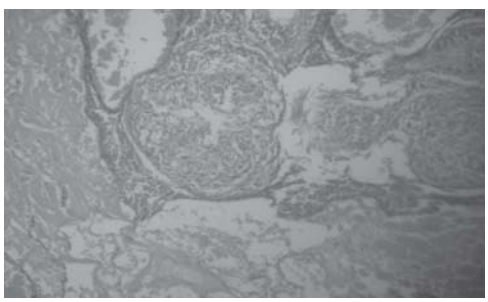
The lesion was surgically enucleated in total with extraction of 22, 23. The gross specimen with teeth (fig 4) .

Histopathologically the section revealed epithelial proliferation in forms of solid strands resembling rosette pattern. Cells were small dark

Fig 4: Enucleated specimen with extracted 22,23

polygonal in appearance interspersed with cuboidal and columnar lining cells.Intervining areas of abundant extracellular eosinophillic

fig 5: Photomicrograph showing rosette pattern (10x magnification)



material and extravasated RBCs were seen.with a very sparse connective tissue stroma (fig 5). Based on the histopathological findings the case was diagnosed as Adenomatoid odontogenic tumour.

Discussion

Adenomatoid odontogenic tumour is a rare tumor that comprises only 0.1% of tumours and cysts of jaw and 3% of all odontogenic tumours of jaw.

It is hypothesized that AOT develops from the enamel organ, dental lamina, reduced enamel epithelium or their remanants.[2]Although it is generally believed that AOT is a hamartoma rather than a neoplasm, the lesion sometimes exhibits aggressive behaviour ,such as becoming unusually large.

There are three clinicopathologic variants of AOT, namely intra-osseous follicular *(70%,* dentigerous type), intra-osseous extra follicular (26%), and peripheral (4%, extra-osseous) all with identical histology. The follicular is a central intraosseous lesion associated with follicular relationship with an impacted tooth it does not attach to C.E.J, but surrounds a greater part of tooth, while extra follicular is intraosseous. AOT has no relation with an unerupted tooth, it is often located between, above, or superimposed upon the roots of adjacent teeth. Unerupted permanent canine are the teeth most often involved in AOT.[2]

Clinically, AOT presents as a slowly growing, painless mass 2/3rd of the tumours are diagnosed in the second decade of life and more than half the cases are found between 13 -19 years of age.[6]Female to male ratio is approximately 2:1.[2,7]In the present case ,a slow growing ,asymptomatic swelling occurred in the maxillary anterior jaw region in a 20 year old male associated with no missing teeth.53% of AOT occurs in anterior maxilla, 9% in maxillary premolar region, 2% in maxillary molar region, 27% in mandibular anterior region, 7% in mandibular premolar region, and 25 in mandibular molar region.[8]

The extra-follicular variant, presents as residual, globulomaxillary, radicular, or lateral periodontal cyst and not associated with impacted tooth.[9]

AOT appears as a unilocular radiolucent lesion with well demarcated, corticated or sclerotic border .Radiopacities are found in 2/3rd of cases and internal structure ranges from completely radiolucent to faint radiopaque foci to dense clusters of ill-defined radiopacities appearing as small pebbles which is similar to our case. IOPA may be required to demonstrate the calcifications within the lesion, which may not be seen on panoramic radiographs.[10]

In maxillary occlusal view expansion of buccal cortex is seen in maxillary as well as mandibular lesion.[10]

Nomura and co-workers found that displacement of neighboring teeth due to tumour expansion is much more common than root resorptions. The panoramic view in this case exactly coincides with the above mentioned features of an Adenomatoid Odontogenic tumor[11]

According to Toller if the protein level in a cyst fluid is 5.0g/100ml and over, then the cyst epithelium is likely to be non- keratinized. A cystic cavity if, present in AOT is always lined by non-keratinized stratified squamous epithelium therefore the above-present case having protein content 8.0g/ml is in accordance with Toller's guidelines.[12].

Treatment and follow-up

All variants of AOT shows a benign biologic behavior and since they are usually well encapsulated,conservative surgical excision and curettage has proven the treatment of choice.Recurrence is extremely rare.

Conclusion

A case of Extrafollicular AOT in a 20 year old male patient is reported,Though it is a common site in the anterior maxilla ,it should be added to the differential diagnosis of cysts of the jaw.Although histopathology provides the confirmation,an ardent search of radiographic clues will help one to arrive at a more accurate diagnosis.

Acknowledgement: I would like to thank Dr. Bastian, Prof. and Head and the staff of Oral and Maxillofacial Pathology, SPPGIDMS, Lucknow for their valuable contribution.

References

1. Bhaskar S N ,Washington(1964):Adenoblastoma;Its histogenesis & report of 15 new cases, J Oral Surg 22;218 – 26
2. Philipsen H.P., P.A. Reichart, K.H. Zhang, H. Nikai and Q.X. Yu, Adenomatoid odontogenic tumor: biologic profile based on 499 cases, J Oral Pathol Med 20 (1991), pp. 149–158.
3. A text book of Oral Pathology; 4th edition. William G.Shafer, Maynard K. Hine, Barnet M. Levy. W. B. Saunders Company. 1999.
4. Daniel L M,Cherrick H M (1980);Oral and Maxillofacial surgery – Vol II ,C.V Mosby,St.louis.
5. Tchertkoff V J A Daino (1969):Ameloblastic adenomatoid odontogenic tumour,oral Surg Oral med Oral Pathol ,127:72 -82
6. Philipsen H.P., Reichart P.A (1998): adenomatoid odontogenic tumour:facts and figures oral oncol,35:35:125 -31.
7. S.C. White and M.J. Pharoah, Oral radiology: principles and interpretation (5th ed), Mosby, St. Louis (2004), p. 395 502.
8. B.W. Neville, D.D. Damm, C.M. Allen and J.E. Bouquot, Oral and Maxillofacial Pathology (2nd ed), WB Saunders, Philadelphia (2002), pp. 621–623.
9. Alice E .Curran,Edward J.Miller,Valerie A .Murrah, Adenomatoid odontogenic tumour presenting as periapical disease,Oral surgery Oral med 1997 :84:557 -60.
10. Langlias R.P,Langland O.E,Nortje C.J:Diagnostic Imaging of the jaws.William and Wilkin 1995:pg30,31,pg.312 -315.
11. N.K. Wood and P.W. Goaz, Editors, K. Wood and I.M. Kuc, Pericoronal radiolucencies. Differential diagnosis of oral and maxillofacial lesions (5th ed), Mosby Yearbook Inc, St. Louis .
12. Adenomatoid odontogenic tumour-:Asha V,Sujatha D,Anuradha Pai,K.S.Ganapathy. JIAOMR 2007,19:04,523 -528.

Cyber crime - A Review

Satish N.T*, Dayananda R**, Harish.s***

*Assistant Professor, **Post Graduate, ***Professor & Head, Department of Forensic Medicine, M.S.Ramaiah Medical College, Bangalore

Abstract

Crime is a social and economic phenomenon, and is old as human society, crime is a legal concept and has the sanction of law. Crime/ offence is a legal wrong that can be followed by criminal proceedings which may result into punishment or acquittal. The cyber crime is perhaps the most complicated problem in the cyber world, apparently there is no distinction between cyber and conventional crime but on deep introspection, there is a fine line of demarcation between conventional and cyber crime , demarcation being the medium[1]. Cyber crime mainly consists of unauthorized access to computer system, data alteration, data distribution and theft of intellect property, the intention may be to commit a financial fraud or steal sensitive data. In cases of cyber-terrorism, the intention is to damage computer system for disrupting telecommunication, railway, power supply and critical infrastructure concerned to defense. The transnational characteristic of cyber crime and minimal prosecution due to the lack of evidence, the need of the hour is to create awareness among the citizen, law enforcement agencies and other professionals. The lack of knowledge, law, regarding cyber crime is a final nail in the coffins. So, here an effort is being made to portray the basics of cyber crime.

Introduction

Computers have entered the nook and corner of our life. There is hardly any profession where computers are not used. From white collar criminal to terrorist organisation and from teenagers to adults all of them use computers. The new generation is growing up with computers and its application in criminal activities is on rise.

Cyber crime is a misnomer, term has no where has been defined in any statue/act passed or enacted by Indian parliament, this crime is not radically different from conventional crime. Cyber crime is the latest and perhaps the most complicated problem in the cyber world. Cyber crime is said to be those species, of which, genus is the conventional crime, and where either the computer is an object or subject of conduct constituting crime.

Conventional crimes like forgery, extortion, kidnapping etc are being committed with the help of computers. Most importantly monetary transactions are moving on to the internet.

Definition

"All crimes performed or resorted to, by abuse of electronic media or otherwise, with the purpose of influencing the functionary of computer systems."

"Any criminal activity that has a computer either as an instrumentality, target or a means for perpetuating further crimes comes in the ambit of cyber crime"

Computer crime is any crime where

Computer is a target
Computer is a tool of crime
Computer is incidental to crime[2]

Address for correspondence:
Dr. N T Satish
Assistant professor, Department of Forensic Medicine, M.S.Ramaiah medical college, M S R Nagar, M S R I T post, Bangalore 560054.
Email: nsatish302@gmail.com,
Mob: 9945791560

Classification

Type I cyber crime
Type II cyber crime

Type I cyber crime has the following characters.

1. It is generally a singular or discrete; event from the perspective of the victim.
2. It often is facilitated by the introduction of crime ware programs such are key stroke loggers, viruses, root kits or Trojan hisses into the user's computer system.
3. The introduction can, but not necessarily be facilitated by vulnerabilities.
   1. The users goes online to perform a task i.e. access www, or read reply to email.
   2. User takes action which then allows the criminal access to information centering personal information on look a like site (or) clicks on some objects resulting in the downloading of a Trojan or key stroke.
   3. This information is used by the attacker.
   4. The user becomes aware of crime, this is the single event from perspective of
      the user. This usually occurs much later in the life cycle of cyber crime.

Type II crime has following characters.

1. User (a) goes online to see what she can find about I lama farming.
2. User (a) decides to participate in online forum about I lama farming.
3. User (b) sees user (a), watches her participating in the farming for several days, responds to the same of her comments.
4. User (b) then sends a request for private chat using a common Instant messaging client.
5. User (a) being familiar with user (b) via the online forum, responds positively and the begin to chat daily as well as participate in the forum. This is a period known as instilling trust.
6. After several interactions user (a) reveals that she is single likes farming, has a quarter of a million dollars available to start. O farm and that she likes to go to concerts. She tells her real name is Jenny.
7. User (b) asks user(a) to meet in person and go to concert.
8. User (a) becomes suspicious when user (b) doesn't give his contact information other than online information and she refuses.
9. User (b) becomes irrational and begins to port false claims against user (a) in the online forums, accessing her of fraud, and of being there to pick up men, not to find other interested in farming. He ports her home number. He also goes onto other posing as user (a) and leaves message asking for dates leaving her real phone number and real name.[2]

Comparison between traditional criminal technique and cyber crime[3]

Terminologies

1. Hacking: Hacking in simple terms meaning illegal intrusion into a computer system without the permission of the computer owner/

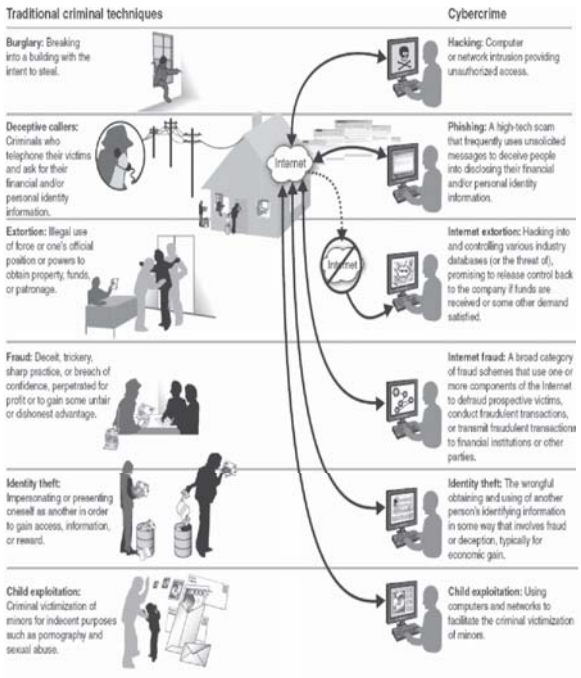


Fig. 1:

Fig. 2:

user.

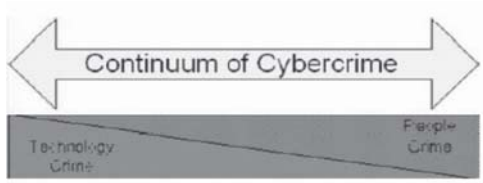
2. Denial of service attack: This is an act by the criminal, who floods the bandwidth of the victim's network or fills his e-mail box with spam mail depriving him of the services he is entitled to access or provide.
3. Virus dissemination: Malicious software that attaches itself to other software.
4. Software privacy: Theft of software through the illegal copying of genuine programs or the counterfeiting and distribution of products intended to pass for the original.
5. Pornography: Pornography is the first consistently successful e-commerce product. Deceptive marketing tactics and mouse trapping technologies Pornography encourage customers to access their websites. Anybody including children can log on to the internet and access websites with pornographic contents with a click of mouse. Publishing, transmitting any material in electronic form which is lascivious or appeals to the prurient interest is an offence under the provision of section 67 of I.T. Act-2000.(4)
6. IRC crime: Internet Relay Chat (IRC) servers have chat rooms in which people from anywhere the world can come together and chat with each other. Criminals use it for meeting co-inspirators.
7. Credit card fraud: If electronic transactions are not secured the credit card numbers can be stolen by the hackers who can misuse this card by impersonating the credit card owner.(photo)
8 Net extortion: Copying the company's confidential data in order to extort said company for huge amount.
9 Phishing: It is technique of pulling out confidential information from the bank/financial institutional bank/financial institutional account holders by deceptive means.
10 Spoofing: Getting one computer on a network to pretend to have the identity of another computer, usually one with special access privileges , so as to obtain access to the other computers on the network.
11 Cyber stalking: The Criminal follows the victim by sending emails, entering the chat rooms frequently.
12 Threatening: The Criminal sends threatening email or comes in contact in chat rooms with victim.
13 Salami attack: In such crime criminal makes insignificant changes in such a manner that such changes would go unnoticed.[4]
14 Replacing ads the below (fig 1) shows *CNN* website as seen by an infected user (on Janu-ary 5, 2009, Monday). Everything on it looks normal, except perhaps for the Vimax pills ad. The nature of this ad makes it somewhat un-usual that it is being displayed on a mainstream news website. In fact, the Vimax pills ad is not what CNN intended to show to its visitors (fig 2) . The ad should instead show a car for sale.[5]

Discussion

Crime involving computers as a communication tool, a storage device or as target itself is cyber crime, Storing pornographic material and physically damaging computer are examples of the later two mechanisms. Illegal distribution of racist or sexually explicit materials, intellectual property theft, stealing credit card numbers, and launching computer viruses are all examples of cybercrimes effected over the internet: The internet is heavily dependent on the public telecommunications infrastructure.

Cybercrime really presents a continuum ranging from crime which is almost entirely technological in nature and crime which is really, at its core, entirely people-related . Consider, for example, a fraud carried out via e-mail where the user is directly and simply asked to send money to a particular physical address in return for some service which never materializes. At its core, this fraud would work via regular paper mail or telephone.

The interconnected networks of the internet have enabled unprecedented economic opportunities and linked population around



the globe in a way never before possible. The benefits of the internet, however, are being masked by exploiting its capabilities to cause the detriment effect.

Profile of cyber criminal

Disgruntled employee, Teenagers, Political hacktivist, Professional hackers, Business rival, Ex boy friend, Divorced husband etc.

Victims

Gullible, Desperados and greedy people, Unskilled and inexperienced people, Unlucky people.

Efforts to address cybercrime follow the same basic process as efforts to address traditional crime. This basic process is one of protection, detection, investigation, and prosecution.

To protect networks and information against cybercrime, organizations and individuals implement cyber security techniques such as access controls (passwords) and firewalls. In addition, they use monitoring devices or intrusion detection systems to detect incidents that could potentially be criminal intrusions. When a suspected cybercrime is detected, organizations and individuals must decide what action to pursue. Depending on the severity of the incident, the level of evidence, and their comfort with revealing the incident, they may or not report it to law enforcement.[6]

Based on various studies and expert opinion, the direct economic impact from cybercrime is estimated to be in the billions of dollars. The estimated losses associated with particular crimes include $49.3 billion in 2006.There is concern about threats that nation-states and terrorists pose to our national security through attacks on our computer-reliant critical infrastructures and theft of our sensitive information. According to FBI testimony, terrorist organizations have used cybercrime to raise money to fund their activities. However, despite the reported loss of money and information and known threats from our nation's adversaries, there remains a lack of understanding about the true magnitude of cybercrime and its impact because Organizations and individuals do not always detect cybercrimes. The effectiveness of the systems put in place to audit and monitor systems, including intrusion detection systems, intrusion protection systems, security event correlation tools, and computer forensics tools, have limitations that impact their ability to detect a crime.

Crime ware

The software used in Cybercrime is referred to as crime ware .It is defined as software that is:

1. used (directly or indirectly) in the commission of the criminal act;
2. and not generally regarded as a desirable software application from the perspective of the computer user;
3. and not involuntarily enabling the crime.[2]

Strategy for tackling cyber crime

A comprehensive strategy to tackle the emerging problems of cyber crime should have following components:

1. Emphasis on adequate in built security features in computer system.
2. Establishing legal frame work and evolving investigative techniques to gather evidence.
3. International co-operation.

Legal Framework

In United States and United kingdom

1. Computer Fraud and Abuse Act,1986,
2. Intellectual property act 1986
3. Computer misuse act 1991,
4. Telecommunication act 1996.
5. Electronic fraud act 1996,
6. Digital signature registration 1996,
7. Electronic communication privacy[7]

In India

The Information and technology act (ITA-2000) was notified as an Indian parliament act on 17 October 2000.following issues were addressed

1. Legal recognition of electronic documents.
2. Legal recognition of digital signatures
3. Offences and contraventions.
4. Justice dispensation system for cyber crimes.

The bill was amended in 2006 and re-amended in 2008; finally the bill was accepted with a new name as information technology (amendment) bill 2008.[8]

Prevention of cyber crime

Prevention is always better than cure. It is always better to take certain precaution while operating the net. A should make them his part of cyber life. The Mumbai Police Cyber crime Cell, advocates the 5P mantra for online security: ***Precaution, Prevention, Protection, Preservation and Perseverance.*** A citizen should keep in mind the following things.

1 to prevent cyber stalking avoid disclosing any information pertaining to oneself. This is as good as disclosing your identity to strangers in public place.
2. always avoid sending any photograph online particularly to strangers and chat friends as there have been incidents of misuse of the photographs.
3. always use latest and up date anti virus software to guard against virus attacks.
4. always keep back up volumes so that one may not suffer data loss in case of virus contamination
5. never send your credit card number to any site that is not secured, to guard against frauds.
6. always keep a watch on the sites that your children are accessing to prevent any kind of harassment or depravation in children.
7. it is better to use a security programme that gives control over the cookies and send information back to the site as leaving the cookies unguarded might prove fatal.
8. web site owners should watch traffic and check any irregularity on the site. Putting host-based intrusion detection devices on servers may do this.
9. use of firewalls may be beneficial.
10. web servers running public sites must be physically separate protected from internal corporate network.[4]

Conclusion

Cyber crime is a vast and global issue in a a boundary-less world. This is an attempt to address the definition, classification and few basic concepts of cyber crime. This understanding is of critical importance, as organizations tasked with defending populations against Cybercrime must begin to consider all the crimes within this continuum, and designate appropriate resources to prevent, defend against, and investigate Cybercrime. This is especially important as new laws which address "Cybercrime" begin to take effect at a national and State level[5]

The situation with Cybercrime and crime ware is rapidly evolving. By attempting to view the problem more inclusively, it should be possible to foresee new developments and take steps toward remediation rapidly. Narrowing or ignoring the problems will create the perfect environment for the Cybercriminal to flourish, undermining the perceived stability and reliability of electronic systems worldwide. In this era of terrorism, many terrorist organization hacking into the vital websites particularly defence home ministry has been increasing , which is a real threat to the nation sovereignty .

Although surveys and studies show that the nation potentially loses both billions of dollars annually and sensitive information as a result of cybercrime, definitive data on the amount of cybercrime is not available. When a cybercrime is detected, entities and individuals can choose to report it to law enforcement or not. They weigh the

cost and impact of the incident with the time and effort needed to support an investigation and prosecution.

There is rapid evolution of technology and cyber crime techniques hence the law enforcement agencies must continuously upgrade technical equipment and software tools which are expensive. For example, in order for investigators to perform cyber forensic examinations and gather the evidence required to support a prosecution, the examiners and investigators must, in some cases, store and analyze huge amounts of digital data. However, according to law enforcement officials, state and local law enforcement agencies do not always have the resources to obtain the equipment necessary to analyze large amounts of data.

A major challenge is educating the public in how to recognize cybercrime when it is occurring. Criminals prey on people's ignorance and susceptibility to ruses. For example, attackers create e-mail and Web sites that appear legitimate, often copying images and layouts of actual Web sites. Some cybercrime techniques also take advantage of combinations of vulnerabilities. Despite efforts by public and private entities to raise awareness about the importance of information security and the techniques used by criminals, users continue to not understand the need for protecting their personal information and to recognize unusual requests that could be criminal activity. The types of cybercrime that the media highlight, such as child pornography cases and major companies being hacked, do not tend to undermine people's trust in the Internet. There are numerous steps being taken to improve security of information systems and raise user awareness.

Following the 9/11 attacks there is further momentum by raising the spectra of cyber attacks on critical infrastructures, facilities, financial, defence institutions and other government systems. There is a need to evolve mechanisms for legal co operation by reaching at international agreements on common definitions; issues concerning to jurisdiction and procedural law and extradition etc. policy makers need to concentrate on these issues

1. Organizational structure of computer crime investigation teams.
2. Training of investigators and prosecutor.
3. Forensic experts specializing in these areas.
4. Evolution of legal tools.
5. Co-operation between police investigation agencies and industry.[7]

References

1. Cyber Crime by Parathasarthi pati(superident police.Cyber crime investigation cell 5th floor, block no 3,CGO complex Lodi road New Delhi-110003.
2. SarahGordon,RichardFord journal of Comput Virol(2006)2;13-20.
3. *Cybercrime: Public and Private Entities Face Challenges in Addressing Cyber Threats*, U.S. Government Accountability Office, GAO-07-705, June 2007, http://www.gao.gov/new.items/d07705.pdf.
4. Cyber crime awareness cyber crime investigation cell, crime branch CID Mumbai http//www.cybercellmumbai.com accessed on 10 2 2010
5. Ben April, Feike Hacque bord and Rainer Linte, A Cyber hub http;//www.trendmicro.com accessed on 15.3.2010
6. ITUtool kit for cyber crime legislation, http// itu.int/ITU-D/cyb/cyber security/legislation.htmn
7. Balwinder singh Cyber crime-Anew challenge for the police central vigilance commission,G O I New
8. Amendments to I T act 2000 ,http;//cyber laws.net/itamendments/tol1.html accessed on 25-1-2010.

Primary squamous cell carcinoma of the gingiva: A case report

Nidha Gaba*, Pramod G.V.**, Ashok L***, D.S Mehta****

*Postgraduate Student, **MDS Reader, ***MDS Professor and Head Department of Oral Medicine and Diagnostic Radiology, ****MDS Professor and Head Department of Periodontics Bapuji Dental College and Hospital, Davangere 577004, Karnataka, India

Abstract

Background

Squamous cell carcinoma is the most common malignancy involving the head and neck region. Primary gingival squamous is a subset of oral squamous cell carcinoma but it exhibits numerous attributes different from other oral carcinomas. It can present as a variety of different lesions distinguishing itself as a unique entity. We report a case of primary squamous cell carcinoma occurring on the gingiva.

Methods

A 40 year old male patient presented with the complaint of growth in the right lower gum region associated with pain since two months. His personal history revealed he was a gutka chewer and chews 5 packets of gutka per day since past 5 years. On intraoral examination, blanching of lower labial mucosa, right and left buccal mucosa and soft palate was evident. On palpation fibrous bands could be felt. An ulceroproliferative growth was evident both labially and lingually involving marginal, interdental and attached gingiva on the right side of the mandible in relation to #31,#41,#42,#43,#44,#45 &#46. A provisional diagnosis of a malignant growth involving the labial and lingual gingiva and alveolar mucosa on the right side of mandibular anterior region and oral submucous fibrosis was made.

Results

Radical Neck dissection, wide tumor excision and segmental mandibulectomy extending from distal aspect of # 35to distal aspect of #48 was carried out under general anaesthesia.Mandibular reconstruction was done using a recon plate.

Conclusion

This is a case of primary squamous cell carcinoma occurring on the gingiva. This report demonstrates that even though oral cancers involving the periodontium are a relatively rare occurrence, they should not be overlooked and vigilant oral examination should be carried out. It also highlights importance of utilizing histopathological examination to confirm diagnosis of the suspicious lesions.

Keywords

Squamous cell carcinoma,Oral Squamous cell carcinoma, Primary Squamous cell carcinoma of gingiva, Radical Neck dissection.

Introduction

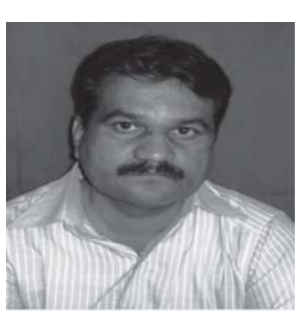
Squamous cell carcinoma is the most common malignancy involving the head and neck region. Primary gingival squamous cell carcinoma is a rare insidious neoplasm which arises from the keratinized part of oral mucosa. Despite abundant of published data on squamous cell carcinoma, very little is known about gingival squamous cell carcinoma specifically. It is mainly due to a very few cases reported in the literature. The exact etiology of the disease is unknown. However few predisposing factors such as exposure to tobacco and alcohol have been associated with it. The incidence of Oral Squamous Cell Carcinoma (OSCC) of gingival has been found to occur 3 times more in smokers as compared to non smoker individuals. [3] Striking feature of gingival squamous cell carcinoma is the clinical variants in comparison to the routine features of squamous cell carcinoma of the other sites in oral cavity. [1] Clinically it can lead to misdiagnosis as it can mimic a number of other benign conditions occurring in the oral cavity. Lymph node metastasis has been reported with the incidence of 45.71%. [1] Panoramic radiographs are the most common imaging modality. Computed Tomography (CT) scan, Magnetic Resonance Imaging (MRI) and bone Scintigraphy have been found to be highly sensitive imaging modalities.[12] Histologically,most of the gingival oral squamous cell carcinomas are moderately differentiated or well differentiated. [14] Standard therapy is based on surgery with radical neck dissection and radiation therapy.[16] Postoperative prognosis is fair and distant metastasis is rare.

Case description and results

A 40 year old male patient (figure -1) came to the Department of Oral Medicine and Radiology, Bapuji Dental College and Hospital with the complaint of growth in the right lower gum region associated with pain since two months. The growth was first noticed by him as a small sized roughened area which gradually increased over a period of one month. Pain started one month back. It was insidious in onset, gradually progressive and radiated to right submandibular region. His personal history revealed he was a gutka chewer and chews 5 packets of gutkha per day since past 5 years. He used to keep it in the lower anterior labial vestibular region for approximately 2 minutes before spitting it.He also used to chew arecanut three to four times a day since past 10 years.

General physical examination revealed he was moderately build and nourished and all the vital signs were within the normal limits. Solitary right submandibular lymph node was palpable, measuring approximately 1 X 1 cms in size, oval in shape, hard in consistency, non tender and fixed to overlying structures. Mouth opening of 2.1 cms was present. On intraoral examination, blanching of lower labial mucosa, right and left buccal mucosa and soft palate was evident. On palpation fibrous bands could be felt running in a vertical direction in right and left buccal mucosa and in a circular direction in the lower labial mucosal region.Restricted protrusive and lateral movements of tongue were also evident.

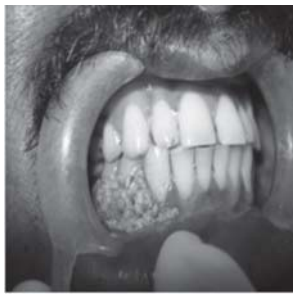
Figure 1: Pre-operative view of the patient



Corresponding Author:
Dr. NIDHA GABA
Postgraduate Student, Department of Oral Medicine and Diagnostic Radiology, Bapuji Dental College and Hospital, Davangere-577004, Karnataka, India
E Mail ID-nidha24@yahoo.com
Phone no.-09902100212

An ulceroproliferative growth measuring approximately 5X3 cms in size and involving marginal, interdental and attached gingiva labially was evident on the right side of the mandible.(figure-2) Anteriorly it extended from the distal aspect of #42 to posteriorly mesial aspect of #46. Superiorly, it extended from the marginal gingiva in relation to above mentioned teeth and inferiorly involved the whole depth of vestibule irt these teeth. The surface over the growth appeared granular giving it a cauliflower like appearance. Yellowish white necrotic slough and debris was evident on the surface interspersed with erythematous areas. The margins of the lesion appeared to be everted.On palpation all inspectory findings were confirmed. The surface appeared rough and granular, firm in consistency and tender on palpation.Induration was present on the inferior aspect of the growth.

Figure 2: Ulceroproliferative growth on the gingiva on labial aspect of right mandibular quadrant



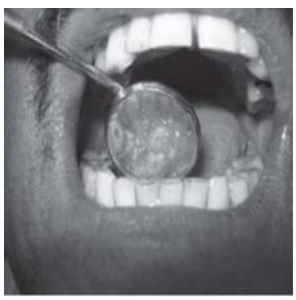
Exactly similar type of growth was present on the lower lingual aspect measuring approximately 4 X 3 cms in size (figure-3). It extended from the mesial aspect of #31 to posteriorly mesial aspect of #46.The surface over the lesion appeared granular giving a cauliflower like appearance. The margins of the growth were seen to be everted.On palpation,similar to the labial growth the surface over the lingual growth was rough and granular in texture.Induration was evident on the inferior aspect. The lesion was firm in consistency and tender on palpation.Generalized gingival inflammation was present.

Hard tissue examination revealed a normal compliment of teeth. Midline diastema was present between # 11and #21.Root stump was present in relation to #24 . Partially erupted # 48 was present and grade 1 mobility was present irt # 41, 42, 44 and 45. Generalized plaque deposits were also present.

Considering the history and the clinical features, a provisional diagnosis of a malignant growth involving the labial and lingual gingiva and alveolar mucosa on the right side of mandibular anterior region and oral submucous fibrosis was made.

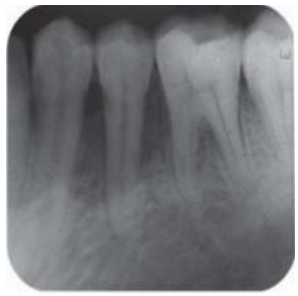
The differential diagnoses considered for the growth were verrcous Leukoplakia and verrucous carcinoma. Localized scleroderma and anemic stomatitis were also considered possible differential diagnosis for the generalized fibrotic oral mucosa. However they were ruled out due to lack of systemic involvement and normal blood picture.

Figure 3: Ulceroproliferative growth on the gingiva on lingual aspect of right mandibular quadrant



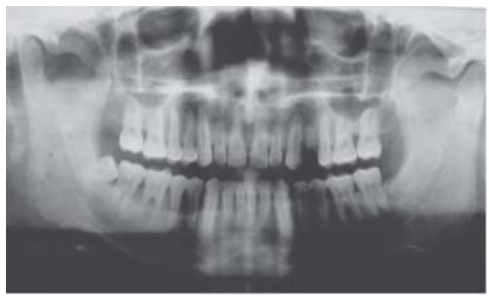
IOPA radiograph in relation to #44, #45 and #46 region was taken (figure-4) .It revealed erosion of the underlying bone. An ill-defined type of radiolucency was evident extending from the alveolar crestal of #44, 45 and 46 up to the middle third of the root surface irt these teeth. The borders were ill-defined. There was associated loss of lamina dura # 44, #45,#46.Mild root resorption was evident in the apical third of #44.

Figure 4: IOPA radiograph in relation to right mandibular first and second premolars and first molar showing erosion of the underlying bone



Orthopantomograph was made to delineate the complete extent of underlying bone involvement. It revealed normal complement of teeth (figure-5).Root stump was present in relation to #24.Erosion of underlying alveolar crestal bone which extended anteriorly from the distal aspect of # 42 to posteriorly mesial aspect of #46 was evident. An ill-defined radiolucency was present measuring roughly 4 X 2 cms in size extending superiorly from the crestal region and inferiorly up to the middle third of root surface of #45.The internal structure appeared to be completely homogenous. There was no displacement of the associated teeth. Chest radiograph was also taken. It revealed no metastatic lesions.

Figure 5: OPG radiograph revealing destruction of the underlying bone extending from the distal aspect of 42 to posteriorly mesial aspect of 46.



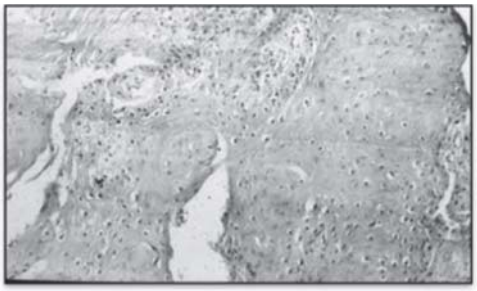
Ultrasonographic examination of the neck region was done but no definite evidence of lymph node metastatis was revealed.

Histology

The patient was subsequently referred to the Dept. of Oral and Maxillofacial Surgery of our college wherein an incisional biopsy was done from both labial and lingual gingival aspects. Histopatholgical report revealed oral mucosa with infiltrating squamous epithelial cells (figure-6). The cells were large, polygonal with acidophilic cytoplasm. Nuclei were large with vesicular nucleoli. Individual cell keratinization was evident. The histological report was suggestive of infiltrating poorly keratinizing squamous cell carcinoma of gingiva.

Radical Neck dissection, wide tumor excision and segmental mandibulectomy extending from distal aspect of left mandibular second premolar to distal aspect of right mandibular third molar was carried

Figure 6: H & E section revealing infiltrating squamous epithelial cells. The cells are large, polygonal with acidophilic cytoplasm. Nuclei are large with vesicular nucleoli. Individual cell keratinization ,with keratin pearl formation is evident.(H& E stain x10).



8 Figure: Excised specimen

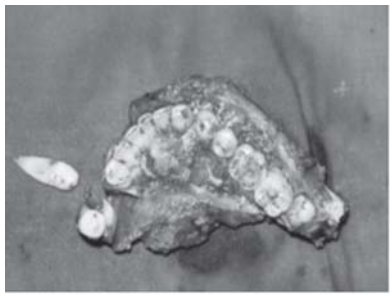


Figure 10: Post operative view of the patient

out under general anaesthesia (figure-7,8).Mandibular reconstruction was done using a recon plate.

The patient reported with a marked improvement in function and esthetics. The patient is currently under follow-up.

Discussion

Oral squamous cell carcinoma (OSCC) presents less than 3% of all malignant carcinomas. It is the 6th most common cancer in males and 12th most common in females. Oral squamous cell carcinoma accounts for 94% of all oral malignancies. Most common sites of occurrence are ventral, lateral aspects of tongue, floor of mouth, buccal mucosa and very less on gingiva. Smoker et al reported that squamous cell carcinoma of gingival accounts for fewer than 10% of all oral cavity cases.[1] In1941, a classical report on oral squamous cell carcinoma of gingiva was published. Till than only few cases have been reported in the literature.[2]

In a recent review OSCC is reported to occur with an incidence of 6.3% of all oral cavity carcinomas.[2]

The exact etiology of OSCC is unknown. Arecanut chewing is related to high risk of oral squamous cell carcinoma of gingiva and oral submucous fibrosis concomitantly as was evident in our reported case.Arecanut has been ranked as a group 1 carcinogen to human

Figure 7: Intraoperative photograph showing segmental mandbulectomy.

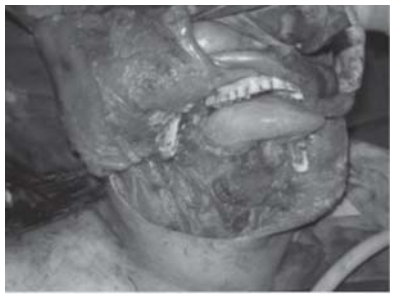
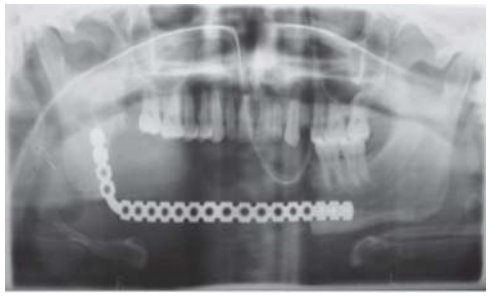


Figure 9: Postoperative OPG radiograph showing recon plate reconstruction



fibroblast matrix mettaloproteinases (MMP-2). Altered MMP-2 which is increased in the saliva of arecanut chewers which facilitates epithelial oncogenesis.[4]

Schwartz et al reported that tumorogenesis is not only dependent on type and level of carcinogen but also genetic sensitivity of an individual to mutagen induced chromosomal damage.[5] In younger age groups the development of OSCC of gingival is also linked with many hereditary conditions such as familial genodermatosos and chromosomal instability syndrome etc. Association with Fanconi's anemia and Xeroderma Pigmentosa has also been reported.[6]The role of HPV virus in the pathogenesis appears to be controversial-53 expression and cyclin –D1 polymorphism has been documented in the patients with no identifiable risk factors.[5]

The age of occurrence is 35-65 years. It has been found to be 12% more in the age groups more than 65 years. However it has been reported in the younger age group less than 40 years with the incidence of 0.4-6%.Four cases have been reported to occur in adolescence individuals.[2,5]

The incidence rate in females is more than males. It was found to be 8.5:3.3 for females: males per 100,000 individuals.Brasch et al reported that percentage of oral squamous cell carcinoma of gingiva in females was more than males.[17]

It arises from the keratinized mucosa and is more common in the mandible (89%) than in the maxilla (77.4%).[7,2] In the maxilla involvement of the maxillary antrum is frequent. It can also spread and involve vital structures such as facial and neck spaces and can lead to poor prognosis. Buccal space is involved in 42% of mandibular gingival carcinoma and 47% in case of maxillary gingival carcinoma.[1] Masticatory space is the second most common site of involvement.[8]

Gingival oral squamous cell carcinoma presents as a variety of different lesions distinguishing itself as a unique entity. Numerous reports document that gingival OSCC either mimics as another lesion or rapidly progresses from the previous lesion.[1] Usually it starts as an asymptomatic lesion. It can present as an intraoral mass, swelling, ulceration, and pain, mobility of teeth or unhealed extraction sites.[2]

Clinically it can appear as a white, nodular, exophytic sometimes granular or papillary appearing proliferation on gingiva as in our

reported case. It can mimic localized periodontal disease or an inflammatory condition such as osteomyelitis, especially when the oral bacteria secondarily infect the tumor.[5]

Fatahzadeh et al in 2004 documented a case of gingival squamous cell carcinoma in a 58 year old patient on mandibular gingiva which originated as lichen planus and progressed to gingival squamous cell carcinoma. Heller et al reported another case that presented as an endo-perio lesion.[1]

Clinically the lesion may mimic a wide array of conditions such as benign epithelial hyperplasia, squamous pappiloma, verruca vulgaris. Exophytic presentation can mimic Pyogenic granuloma,peripheral ossifying fibroma or a peripheral giant cell granuloma.Inflammatory lesion can mimic parulis or an abcess.Granular appearance can appear as foreign body reaction, deep fungal infection or oral manifestations of tuberculosis.[5,3]

Level I, II, III, IV lymph node all have been reported to be involved in distant metastasis.[9] Nodal metastasis has been found in 55% cases from the upper gingival buccal complex and 33% from the lower gingival buccal complex.[10] Retropharyngeal lymph nodes involvement has also been reported from the carcinoma of the upper gingiva.[11]

Gingival oral squamous cell carcinoma can involve the underlying bone from any direction producing a radiolucency that is polymorphous and irregular in outline. Three patterns of bone destruction have been described i.e. erosive, invasive and mixed. Invasive pattern occurs in more than half of the cases and was evident in our case. It is characterized by an ill defined, non corticated border. Erosion has smooth borders but no evident cortex.[12]

The internal structure can be completely radiolucent or occasionally small islands of normal trabaculae are visible within the central radiolucency. Evidence of invasion can appear as widening of periodontal space with the loss of lamina dura.Teeth can appear to float in the radiolucency. Sometimes it can cause destruction of the corticated borders of maxillary antrum and inferior alveolar canal.[13]

Histologically, most of the gingival oral squamous cell carcinomas are moderately or well differentiated with abundant cytoplasm, altered nuclear cytoplasmic ratio and keratin pearl formation.[14]

T-classification proposed by UICC(International Union of Cancer Control) defines T_1, T_2, T_3 according to superficial structure involvement and T_4 involving the adjacent structures. Gingival oral squamous cell carcinoma and carcinoma of alveolus are most commonly classified as T_4 because of the anatomic characteristics. However several other studies have shown that a modified T classification based on inferior alveolar nerve involvement should be followed for the mandibular carcinomas and SNF (sinus, nasal floor) classification should be followed for the mandibular carcinomas.[15]

Treatment of gingival carcinoma should provide maximum probability of cure and maintainenece of good life. It is reported that surgery should be based on clinical, radiological and histopathological findings. On a panoramic radiograph if the bone defect is above the canal or invasive defect is confine to the superficial area or no bone involvement is seen then marginal resection is indicated. Segmental resection is indicated if the bone involvement is more.[16]

Post operative prognosis is good. Distant metastasis is rare. Overall survival rate is 5 years which is comparable to oral squamous cell carcinomas of other sites.

Therefore although Gingival oral squamous cell carcinoma is a subset of oral squamous cell carcinoma but it exhibits numerous attributes different from other oral carcinomas. This case in particular reinforces the need for vigilant clinical examination, necessary investigations and proper treatment for such a rare entity.

References

1. Yoon TY, Bhattacharyya I, Katz J, Towle HJ, Islam MN. Squamous cell carcinoma of the gingiva presenting as localized periodontal disease. Quintessence Int. 2007;38:97-102.
2. Seoane J, Pablo I, Centelles V, Trevor F,Walsh, Jose L,Cedrun L, Vazquez I. Gingival Squamous Cell Carcinoma:Diagnostic Delay or Rapid Invasion? J Periodontol 2006;77:1229-1233.
3. Khan SM, Gossweiler MK, Zunt SL, Edwards MD, Blanchard SB. Papillary squamous cell carcinoma presenting on the gingiva.J Periodontol 2005;76:2316-21.
4. Lu HH, Liu CJ, Liu TY, Kao SY, Lin SC, Chang KW. Areca-treated fibroblasts enhance tumorigenesis of oral epithelial cells. J Dent Res 2008;87:1069-74.
5. Isharif MJ, Jiang WA, Zhao Y, Shan Z, Chen X. Gingival squamous cell carcinoma in young patients: Report of a case and review of the literature.Oral Surg Oral Med Oral Pathol Oral Radiol Endod.2009;107:92-99.
6. Binahmed A, Charles M,Campisi P, Forte V,Carmichael RP, Sándor GK.Primary squamous cell carcinoma of the maxillary alveolus in a 10-year-old girl.J Can Dent Assoc.2007 ;73:715-8.
7. Rautava J, Luukkaa M, Heikinheimo K, Alin J, Grenman R, Happonen RP. Squamous cell carcinomas arising from different types of oral epithelia differ in their tumor and patient characteristics and survival.Oral Oncol 2007;43:911-919 Epub 2007 Jan.
8. Kimura Y, Sumi M, Sumi T, Ariji Y, Ariji, E and Nakamura T. Deep Extension from carcinoma arising from the gingiva:CT and MR imaging features. Am J Neuroradiol 2002; 23:468–472.
9. Cady B,CatlinD. Epidermioid carcinoma of the gum:A 20-year survey. Cancer 1969;23:551-569.
10. Pathak KA, Mathur N, Talole S, Deshpande MS, Chaturvedi P, Pai PS, Chaukar DA, D'Cruz AK.Squamous cell carcinoma of the superior gingival-buccal complex.Oral Oncol 2007 ;43:774-9. Epub 2007 Feb.
11. Yukinori Kimura, Tomomi Hanazawa, Tsukasa Sano, and Tomohiro Okano.Lateral retropharyngeal node metastasis from carcinoma of the upper gingiva and maxillary sinus .Am J Neuroradiol 1998;19:1221–1224.
12. E Nakayama et al. A study of the association between the prognosis of carcinoma of the mandibular gingiva and the pattern of bone destruction on computed tomography. Dentomaxillofac Radiol 2000; 29:163 -169.
13. Robert E Wood .Malignant diseases of the jaws.In:White SC, Pharoah MJ. Oral Radiology Principles and Interpretation. ed 5. Philadelphia : Mosby,2004:459-463.
14. Epithelial pathology.In:Neville BW, Damm DD, Allen CM, Bouqut JE. Oral and Maxillofacial Pathology ed 2 Philadelphia: Saunders ,2004:362-363.
15. Sasaki T, Imai Y, Fujibayashi T. New proposal for T classification of gingival carcinomas arising in the maxilla. Int J Oral Maxillofac Surg. 2004;33:349-52.
16. Nomura T, Shibahara T, Cui NH, Noma H.Patterns of mandibular invasion by gingival squamous cell carcinoma.J Oral Maxillofac Surg. 2005;63:1489-93.
17. Barasch et al.Squamous cell carcinoma of the gingiva.A case series analysis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod.1995;80:183-187.

Xerostomia: A review

Poornima R*, Rajeshwari G. Annigeri**, Ashok L.***

*Assistant Professor **Professor and Head, Dept. of Oral Medicine and Radiology College of Dental Sciences, Davangere 577004, Karnataka, India, ***Professor and Head, Dept. of Oral Medicine and Radiology, Bapuji Dental College and Hospital, Davangere, Karnataka 577004

Abstract

Saliva is a unique biologic fluid. Saliva is one of the most complex but versatile and important body fluids and contains a number of systems which serve a wide spectrum of physiological needs. Saliva is principle defense factor of the mouth. Similarly to any fluid in the body even saliva can have eccentric variations, which either can be increase or decrease in levels that can be physiologic or pathologic disparities. Persistent alteration in the normal flow of saliva into the oral cavity is of considerable significance to the integrity of the oral tissues. Although not life threatening but result in overall reduction in the quality of life.

Saliva plays a key role in the maintenance of oral health. Xerostomia or reduced salivary flow may be defined as subjective sensation of dryness of the oral mucous membrane with objective evidence of significantly decreased salivary flow. It is not a disease but an early symptom of several morbid systemic conditions with important implications for the medical and dental management of patients.

Key words

Saliva, Xerostomia, Hyposecretion

Introduction

Nature has bestowed saliva "the natural source" with many functional capabilities which plays an important role in maintaining well-being of mouth. Saliva plays major role in protection of oral tissues, digestion, lubrication and speech.

The total salivary flow produced during 24 hr period is about 1000 to 1500 ml. The mean resting flow rate for whole saliva is approximately 0.4ml per minute. Saliva is dilute fluid over 99% being made up of water and consists of organic and inorganic constituents. Composition of saliva is influenced by flow rate and its biological, environmental factors.

Xerostomia

Xerostomia is a common clinical complaint that predisposes individuals to oral diseases and considerable discomfort that may manifest as increased incidence of caries, susceptibility to oral candidiasis, altered taste sensation, glossodynia and numerous other problems. Xerostomia may occur with the use of medication; as a complication of connective tissue and autoimmune diseases; with radiation to the head and neck region; or with a number of other systemic or local conditions. Investigations for xerostomia include a thorough clinical and oral examination, salivary flow rate estimation, radiologic and histopathologic examination of the salivary glands. Clinical laboratory tests are also indicated to help in the diagnosis. Patient education plays a vital role in the management of xerostomia. The general approach to treatment consists of palliative treatment for the relief of symptoms and prevention of oral complications. Relief of symptoms may be achieved by paying rigorous attention to personal oral hygiene; diet counseling; drug substitution/dose modification.

Corresponding Author:
Dr.Poornima.R.
Assistant Professor, Department of Oral Medicine and Radiology Bapuji Dental College and Hospital Davangere Karnataka-577004
E mail: drpoornima_omr@yahoo.co.in

Salivary flow may be enhanced by using medication or by gustatory stimulation. Artificial saliva substitutes may be used for relieving symptoms. Alternative therapy like acupuncture may be sought if conventional treatment has failed to offer relief. Complications like increased incidence of dental caries, candidiasis, and difficulty in using dentures, usually associated with xerostomia can be managed by giving rigorous attention to personal and professional oral hygiene measures; strict adherence to non cariogenic diet; placement of sealants and topical fluorides; and anti fungal therapy.

Definition

Xerostomia may be defined as a subjective sensation of dryness of the oral mucous membrane with objective evidence of significantly decreased salivary flow.

Sreebny (1988) defined Xerostomia as the subjective feeling of oral dryness and it is the result of salivary gland hypofunction. This symptom is more common in ageing populations, but is not caused by ageing.[1](Endnotes)

[1]Sreebny LM and Anthony Valdini. Xerostomia. Part I: relationship to other oral symptoms and salivary gland hypofunction. Oral Surg Oral Med Oral Pathol 1988; 66: 451-8.

Historical review

More than one hundred years ago, Barley (1868)[2] defined "dry mouth" as a condition with clear evidence of dryness of the oral mucosa, obliteration of the salivary duct orifices, and/or glossitis. Bahn (1972) Conger (1973) Sreebny and Broich (1989) Wolff (1990) –told that Xerostomia is associated with caries, periodontal disease, mucositis, angular chelitis, disturbed oral sensation, altered taste. Niinimaa et al (1981) showed that Mouth breathers have xerostomia. Syrjanen (1982) showed that autoimmune diseases such as Sjogren's syndrome are associated with xerostomia. Schubert and Izutsu (1987) showed that radiation to head and neck region causes xerostomia. Tandler et al (1987) showed that diseases affecting salivary glands directly will cause xerostomia. Sreebny et al (1987) showed that patients with mental depression will have xerostomia. Sreebny & valdini (1988) [1] told that xerostomia is associated with difficulty in speech, swallowing. Sreebny et al (1989) showed that xerostomia causes dryness of throat, skin and eyes.

Rhodus and Brown (1990) showed that malnutrition in elderly causes xerostomia. McDonald & Maino (1991) showed that elderly patients will have xerostomia. Navazesh et al (1992) [3]established clinical criteria for salivary gland hypofunction:

1. Dryness of lips
2. Dryness of buccal mucosa
3. Absence of saliva produced by gland palpation.
4. Total DMFT

Sreebny et al (1992) [4] showed that diabetes causes xerostomia due to poor glycemic control and direct metabolic effects on salivary glands and that xerostomia causes mucosal soreness, gingivitis, chelitis, fissuring of tongue, infection of salivary glands, difficulty in chewing, speaking and swallowing and intolerance to dentures.

Etiology factors

- Physiological causes include
  - o Aging, Anxiety & Depression, Fear, excitement & stress,

Increased body temperature, Dehydration, Physical exercise, Mouth breathing

- Developmental causes
  - Salivary gland aplasia/ agenesis/duct atresia, Hereditary ectodermal dysplasia
- Salivary gland disorders
  - Obstructive –sialolithiasis
  - Infections
    - Paramyxo virus, CMV, HIV, HCV
- Immunological
  - Sjogrens syndrome, Mikulicz disease, Sarcoidosis
- Drug induced
- adiation induced
- Metabolic disorders
  - Diabetes mellitus, End stage renal disease
- Nutritional disorders
  - Multivitamin deficiency
  - Protein energy malnutrition
- Others
  - Oral candidiasis
  - Burning mouth syndrome

Local and systemic factors associated with xerostomia [5]

Sialadenitis and obstruction

The most common inflammatory disorders of the major salivary glands are caused by viral and bacterial infections. The viruses which are known to cause sialadenitis are Paramyxo virus, Cytomegalovirus virus, HIV, Hepatitis C, Echo viruses, Epstein Barr Virus, Parainfluenza virus .[6] The most common is mumps which causes painful, bilateral parotid swelling. Bacterial infections may be associated with duct obstruction due to sialolithiasis (stones), fistulas, mucocele or neoplastic infiltration. Inflammatory disorders of the major glands are characterized by painful swelling of the affected gland with alteration in salivary secretion rate and character. Allergic sialadenitis is a self-limiting enlargement of the salivary glands associated with exposure to allergens including various drugs. The disease is characterized by acute enlargement of the salivary gland often accompanied by itching over the gland.

Ionizing radiation

The rapid onset of xerostomia or hypofunction of salivary glands is one of the most common side effects and a major cause of morbidity in patients undergoing radiation treatment for cancer of the head and neck.

The severity of hypofunction of the salivary glands is directly related to the radiation dose, dose rate and amount of salivary tissue irradiated.

The radiation dose is the amount of radiation delivered to a specific field, while the dose rate is the time interval during which the dose is administered. It has been reported that irreversible damage to the salivary glands and permanent hyposalivation frequently occur after a radiation dose of 4000 cGy is delivered directly to the gland.[7]

This radiation in effect disrupts the electron orbital structure of tissue atoms, which subsequently damages individual cells and tissues. Normal tissue functions depend primarily on cell integrity and viability, as well as on the ability of the cells to replace and organize. Cells are most vulnerable to injury when they are in the process of dividing and multiplying.

Radiation induced DNA damage are responsible for mitotic delays and replication linked cell deaths. Often the serous cells undergo interphase cell deaths after irradiation. The cell membrane is a primary target for ionizing radiation.

The serous parotid glands react to radiation rapidly and widely irreversibly, the mucous palatal glands show less sensitivity or a better tolerance against the damaging effect of radiation. The mucous glands have the potential to recover partially and to perform nonstimulated basic secretion for some month after radiation therapy.[8]

The major salivary glands are at times unavoidably exposed to 20-30 Gy during radiotherapy for cancer in the oral cavity or oropharynx. The parenchymal component of the parotid salivary gland is more radiosensitive when compared to submandibular/sublingual glands. A marked and progressive loss of salivary secretion is seen in first few weeks after initiation of radiation therapy. The extent of reduced flow is dose dependent and reaches essentially zero at 60Gy. The mouth become dry and tender and swallowing is difficult and painful because the residual saliva loses its normal lubricating properties. The small volume of viscous saliva that is secreted has a pH value of 1 unit below normal (an average of 5.5 in irradiated patient compared with 6.5 in unexposed individual) this pH is low enough to initiate decalcification of normal enamel. In addition the buffering capacity of saliva fall as much as 44% during radiation therapy. If some portions of the major salivary gland have been spared, dryness of mouth usually subsides in 6 to 12 months because of compensatory hypertrophy of residual salivary gland tissue. Reduced salivary flow that persists beyond a year is unlikely to show significant recovery.

Mossman and colleagues reported 92% of patients who underwent radiotherapy that included the parotid gland experienced severe, chronic xerostomia and this severity persisted even for 6 to 7 years after treatment. For the remaining 8% patients dryness of mouth resulting from decreased salivation may be only a temporary phenomenon that only lasts for just few weeks or months.[9]

It is been reported that after just 5 radiation treatments at a dose of 200cGy per day, the salivary rate decreased by up to 57% and that salivation can be reduced as much as 93% when all the major salivary glands are irradiated. The best way to estimate the degree of salivary impairment or dysfunction caused by radiation therapy is to determine the field in which the radiation beam will pass or has passed and the volume of gland tissue exposed.[8]

The threshold dose of radiation for irreversible damage of parotid glands were seen after application of 10 to 20 Gy in 1.8 to 2.0 Gy daily fractions, found that decrease of parotid secretion to 40 % of the original values after irradiation and an increase to 72% 18 months after radiotherapy.[7]

Sjogren's syndrome

Sjogren stated that "the whole symptom complex rarely occurs in one patient. As a rule only one or two of the associated symptoms occur, with no fixed order of appearance".

Sicca complex-is when a patient appears with only the effects of decreased lacrimal and salivary gland secretion and without a systemic autoimmune disease.[2] The female to male ratio for the disease is 9:1 and up to one third of all patients with rheumatoid arthritis may have sjogrens syndrome. Criteria (European criteria) used at the university of Minnesota in 1999 for the diagnosis of Sjogrens syndrome includes Oral symptom one among other criteria's which include of dry mouth daily $>$ 3 months, Swollen salivary glands, Needs fluids to swallow food. [10]

Diabetes mellitus [4]

Diabetes mellitus is a term used to describe a group of metabolic disorders that share the common characteristics of glucose intolerance associated with an alteration in insulin secretion or action. The classic symptoms are weight loss, fatigue, polyuria and polydypsia. Bilateral parotid enlargement is not an uncommon finding and is usually associated with a moderate to severe diabetic condition. Xerostomia is a commonly reported symptom and is associated with a decrease in resting and stimulated flow rates.

Dryness of the mouth as a feature of uncontrolled diabetes was first described in 1942 by Sheppard.[11] The xerostomia may be a consequence of dehydration, although long standing oral dryness may be due to microvascular disease and neuropathy affecting the

major salivary glands. In addition the xerostomia may be due to the concomitant drug therapy (antihypertensives, diuretics, anxiolytics or antidepressants).[12]

Prolonged xerostomia predisposes to local accumulation of plaque and debris and may contribute to the development of opportunistic oral infections and liability to dental caries, periodontal disease), altered taste, oral malodour and oral mucosal soreness.[13]

Xerostomia in type I DM seems to be dependent upon glucose control. Whereas in type 2 DM, salivary secretion seems to be particularly influenced by xerogenic drugs and autonomic neuropathy.[14, 15]

Xerostomia in association with systemic diseases of significance are mobility impairment, Cardiovascular disease, Osteoporosis, Dementia, Arthritis, Degenerative arthritis, Conjunctivitis, Depression, Hypothyroidism, Parkinson's disease, Insomnia, Chronic leukemia and Multiple sclerosis. [16]

Drugs and xerostomia

Xerostomia is a side effect of many commonly prescribed medications. It is a potentially reversible state. A review of the 200 most frequently prescribed drugs showed the most frequent oral adverse drug reactions (ADRs) to be dry mouth (80.5%), dysgeusia (47.5%), and stomatitis (33.9%).[17]

How drugs cause xerostomia?[18]

1. Drugs may act at M3- muscarinic receptors which mediate parasympathetic cholinergic neurotransmission to salivary glands.
2. Some may act at alfa 1A, beta 1, M3 & H2 receptors mediate exocytosis via cAMP protein kinase A pathway and some act by K1 & M3R via another pathway.
3. GABA and benzodiazepines act by decreasing in fluid secretion and amylase release.

The following are common group of drugs causing xerostomia [18]

1. Analgesics & Psychotherapeutic
   - Meperidine hydrochloride
   - Alprazolam
   - Diazepam
   - Triazolam
   - Tricyclics antidepressants
2. Anorectic
   - Methamphetamine hydrochloride (amphetamine)
   - Phendimetrazine tartrate (non amphetamine)
3. Antiacne preparation
   - Isotretinoin
4. Antiarthritic
   - Piroxicam
5. Anticholinergic; antispasmodic (gastrointestinal)
   - Atropine sulfate
   - Clidinium bromide
   - Dicyclomine hydrochloride
   - Glycopyrrolate
   - Hyoscyamine sulfate
   - Propantheline bromide
   - Combination drugs
6. Antidiarrheal
   - Diphenoxylate hydrochloride and atropine
7. Antihistaminic
   - Diphenhydramine hydrochloride (Benadryl)
   - Bromopheniramine maleate
   - Combination drugs

9. Antihypertensives
   - Clonidine hydrochloride
   - Prazosin hydrochloride
10. Antihypertensives and diuretics
   - Chlonidine hydrochloride and chlorthalidone
   - Nadolol and bendroflumethiazide
   - Propanolol hydrochloride and hydrochlorthiazide
11. Antiparkinsonism drugs
   - Biperiden hydrochloride and biperiden lactate
   - Benztropine mesylate
12. Antipsychotic
   - Lithium carbonate
   - Thioridazine hydrochloride
   - Trifluoperazine
13. Diuretics
   - Chlorothiazide
   - Hydrochlorothiazide
   - Triamtereine and hydrochlorothiazide

Clinical features of xerostomia

Prevalence in general population is estimated to range between 10% and 29% more frequently in females than in men. [19, 20, 21, 22] An estimated 120 million people per year obtains prescription medications among them the prevalence of xerostomia or the subjective complaint of dry mouth has been reported to be 29% in an adult population. [23] Incidence is high in older adults ranging from 14-18%. [24, 25, 26] 27-37% in elderly population. [27] Incidence of dry mouth was found to be increasing with medication usage.[28, 29]

Age and medication seem to play a more important role in individuals with objective evidence of hyposalivation, while female gender and psychological factors are important in individuals with subjective oral dryness. [30]

In a large survey of 3311 evaluable questionnaires, 21.3% of men and 27.3% of women reported dry mouth, with women statistically reporting higher prevalence of dry mouth than men. [31]

Dry mouth is significantly age-related, and there is a strong co-morbidity between reported prevalence of dry mouth and ongoing pharmacotherapy. Unstimulated salivary flow rates were lower among older persons who were female or taking antidepressants, and higher among smokers or people who were taking hypolipidemic drugs.[32]

Hence it is clear that medication is a better predictor of risk status for dry mouth than either age or gender. [33]

A study was conducted to know the association between xerostomia and health status indicators in elderly patients. They have found out that xerostomia did not have a significant impact on chewing capacity, morale/stress but it contributed to the variability of the oral health related quality of life. They concluded that xerostomia has a significant and negative impact on the quality of life of elderly individuals though oral function may be less affected. [34]

Oral symptoms associated with xerostomia

a) Principal symptoms

- Dry mouth (xerostomia), ropy saliva (Spinn barkeit increases i.e. saliva can be drawn into long thin threads)
- Thirst
- Difficulty in swallowing (dysphagia)
- Difficulty in speaking (dysphonia)
- Difficulty in eating dry foods
- Need to sip water frequently at meals
- Difficulty in wearing dentures
- Frequent measures to keep mouth moist
- Frequent pain and irritation from the throat, simulating tonsillitis

b) Other symptoms

- Burning, tingling sensation, especially on the tongue
- Abnormal taste sensation (dysgeusia)
- Keeps fluids at bed side at night
- Fissures, sores at corner of lips

Nonoral symptoms associated with xerostomia

- Blurred vision
- Dry eyes
- Burning eyes
- Sandy, gritty eyes
- Use eye drops
- Dry throat

To summarize symptoms frequently associated with xerostomia [1]

Oral	Systemic
Saliva: decrease in amount, foamy, viscous, ropy (increase in 'spinnbarkeit') **Lips:** dry, cracked, fissured (cheilosis)Tongue: burning (glossopyrosis), pain (glossodynia) **Buccal mucosa**: dry**Salivary glands**: swelling, pain **Thirst**: frequent ingestion of fluids, especially while eating; keep water at bedside **Mastication**: difficulty with eating dry foods; difficulty with the use of a denture **Swallowing** difficulty (dysphagia) **Speech**: difficulty (dysphonia) **Taste**: alteration (dysgeusia)	**Throat:** dryness, hoarseness, persistent dry cough **Nose**: dryness, frequent crust formation, decrease in olfactory acuity **Eyes**: dryness, burning, itching, gritty sensation, feeling that the lids stick together, blurred vision, sensitivity to light **Skin**: dryness, butterfly rash, vasculitis **Joints**: arthritis, pain, swelling, stiffness **GI tract**: constipation **Vagina**: dryness, burning, itching, history of recurrent fungal infections, dyspareunia **General symptoms**: fatigue, weakness, generalized aching, weight loss, depression.

- Dry skin
- Breathe through mouth
- Dry nose
- Change, sense of smell

Symptoms found in females only are

- Vaginal itching
- ·History of vaginal fungal infections
- Vaginal burning
- Vaginal dryness

To summarize symptoms frequently associated with xerostomia [35]

Clinical evaluation [36]

Initial visit: Each patient is asked a standardized series of questions concerning impressions of oral dryness and oral functions with multiple, specified answers. The responses were recorded and entered on coded forms for data analysis.

These are the few examples

1. Does your mouth feel dry at night or on awakening?
2. Does your mouth feel dry at other times of the day?
3. Do you keep a glass of water by your bed?
4. Do you sip liquids to aid in swallowing dry foods?
5. Does your mouth feel dry when eating a meal?
6. Do you have difficulties swallowing any foods?
7. Do you chew gum daily to relieve oral dryness?
8. Do you use hard candies or mints daily to relieve oral dryness?
9. Does the amount of saliva in your mouth seem to be too little, too much, or you don't notice it?

Mirror test [37]-a dental mirror sliding test is used –back of a mouth mirror is drawn along the right and left side buccal mucosa and the friction is registered according to a three-point scale-

Grade I: no obvious friction, i.e. the mouth mirror slides easily along the buccal mucosa

Grade II: some friction is registered

Grade III: high friction, i.e. the mouth mirror is almost stuck to the mucosa.

Lip dryness [28] – Dryness and cracking of the corners and/or the vermillion borders of the lips scored as

0 –normal

1-dry vermillion border

2-dry, chapped and/or fissured tissue

3- angular chelitis, redness or fissuring at the commissure with lesions of traumatic origin excluded the dryness and fissuring are scored even if present unilaterally.

Wafer test [38] - a semi quantitative test to screen for xerostomia. Methodology –time taken for dissolution of wafer is noted in minutes. Noted values are

2.8±2.1 minutes-healthy group

3.3±1.5minutes-connective tissues diseases

9.2±3.9 minutes-primary Sjogren's syndrome

Cut off value was taken as 4 minutes. Concluded that wafer test is valid and reliable to identify subjects with xerostomia.

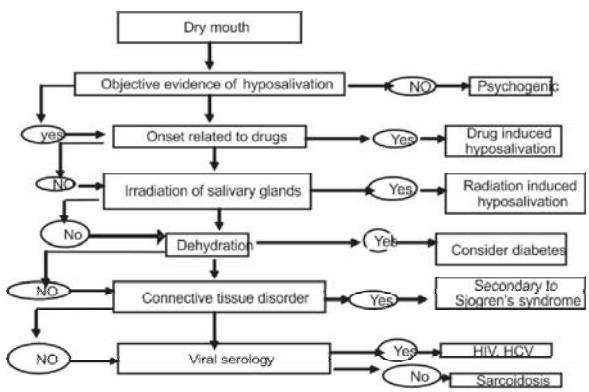
Electrical methods used to assess the mucosal dryness are Resistance, Capacitance and Polarization resistance [25]

Clinical tests for sjogrens syndrome [39] Schirmer's tests, Occular staining and Tear lysozyme and IgG levels

Clinical Method To Assess The Dryness Of Oral Mucosa Screening tests: Characteristics of whole saliva [35]

Function	Healthy subjects	Patients with salivary gland hypofunction
Appearance	Serous; slightly foamy; opalescent	Viscous; foamy
Unstimulated flow rate	0.3-0.4ml/min	Decreased
Stimulated flow rate	1-2ml/min	Decreased
pH:unstimulated saliva	6.5-6.0	Decreased
Buffer capacity (stimulated saliva)	5.75-6.5	Decreased
Lactobacillus index	< 1,00,000 cpu/ml (in 55% of population	Increased
Yeast index	<1,00,000 cpu/ml (in 90% of population)	Increased

Flowchart for sequential diagnosis of patients with dry mouth



How much saliva is enough for avoidance of xerostomia? [40]

Xerostomia, the subjective sensation of dry mouth, occurs when the salivary flow rate is less than the rate of fluid loss from the mouth by evaporation and by absorption of water through the oral mucosa. Evaporation can only occur during mouth-breathing but could reach a maximum rate of about 0.21ml/min at rest. Water absorption through the mucosa can occur because saliva has one sixth the osmotic pressure of extracellular fluid, thus creating a water gradient across the mucosa. The maximum absorption rate is calculated to be about 0.19ml/min, declining to zero as the saliva reaches isotonicity. Thus, xerostomia appears to be due, not to a complete absence of oral fluid, but to localized areas of mucosal dryness, notably in the palate. Unstimulated salivary flow rates >0.1-0.3ml/min may be necessary to avoid xerostomia.

Investigations for xerostomia

Clinical investigations [3]

1. History, oral examinations and salivary flow tests

It is important that a through clinical, medical and drug history be elicited prior to examination of the oral cavity. While a range of investigations have been established in the assessment of patients with salivary gland dysfunction, most clinicians accept that a detailed history is of paramount importance in establishing a diagnosis. Certain elements of the medical history such as prior radiation therapy, diagnosed systemic disease or the prescribed use of xerostomic drugs may suggest the cause.

A qualitative assessment of the salivary gland dysfunction may be obtained from information related to difficulties with masticatory or gustatory function. A thorough oral examination is required. This should include assessment of the oral mucosa, palpation of the major salivary glands and inspection of the duct apertures. The periodontal status and caries status should be accurately documented.

Salivary flow rate tests provide a semi-quantitative assessment of salivary function in a clinical setting. This may include collection of whole saliva.

Measurement of salivary flow rates, or sialometry, appeals as a marker of salivary function. However, its value as a diagnostic aid has been limited due to the very wide range of values found in healthy individuals. Attempts have been made to collect resting flows of saliva as well as maximally stimulated flows. At present, the results may support a particular diagnosis, but some clinicians prefer to use these as a marker for progress in an individual.

2. Radiography and imaging [41, 42, 2]

Interest in salivary gland imaging has developed considerably in recent years, by technological advances.

Includes

- Plain film radiography
  - o Intraoral views
  - Intraoral buccal IOPA technique
  - Intraocclusal films- cross-sectional and oblique view
  - o Extraoral radiography
  - Panoramic radiography
  - AP view/rotated PA view
  - Lateral projection of mandible
  - True lateral skull
  - Conventional Sialography
  - Ultrasound
  - Computed tomography
  - Magnetic resonance imaging
  - Radioisotope imaging- Scintigraphy

3. Labial gland biopsy [39]

Labial gland biopsy is a diagnostic measure used for the histological assessment of salivary hypofunction. The technique involves a 15-20 mm superficial incision in the mucosa of the lower lip extending from the midline towards the commissure and midway between the sulcus and the vermillion border. An area is chosen preferably with palpable gland tissue and following incision, the mucosa is everted brining the glands towards the surface. Careful blunt dissection is necessary to avoid damage to local sensory nerves. At least five glands are required for a representative sample.

4. Laboratory Investigations [39]

4. (A) Sialochemistry:

	Normal	Xerostomia /Sjogren's syndrome
Flow rate (ml/min. per gland)	0.58 ±0.07	0.17±0.03
Electrolytes (meq/lt)		
Sodium	23±3	65±5
Chlorine	23±3	64±4
Phosphorous	6.3±0.7	2.3±0.3
Potassium	22±1	20±1
Urea (mg/100ml)	10.5±0.9	9.8±1.1
Proteins (mg/100ml)	3.6±0.5	5.8±0.7
IgA	0.6±0.5	1.0±0.5
IgG	1.2±0.3	1.0±0.5
Albumin	00.1±00.1	00.1±00.1

B. Hematology

In the presence of significant xerostomia of indeterminable origin, blood tests are indicated. These may include a full blood examination, antinuclear antibodies or other specific tests if specific diseases are suspected. Serology is of value in the diagnosis of inflammatory exocrinopathy and connective tissue disorders (ESR, anti-Ro [SS-A], anti-La [SS-B], anti RNP, rheumatoid factor, antinuclear antibody, anti-double-stranded DNA, anti-centromere and Sel 70). Serum angiotensin converting enzyme levels, as well as the ESR, may be elevated in sarcoidosis, particularly in the active phases of the disorder. Plasma glucose levels are used in the diagnosis of diabetes mellitus; fructosamine and glycosylated hemoglobin concentrations are also used to evaluate degrees of its control.

Treatment of xerostomia falls into 4 categories

1. Preventive therapy
2. Symptomatic treatment
3. Local or topical stimulation
4. Systemic salivary stimulation

1. Preventive Treatment:

- *Topical fluorides*: given to prevent dental caries. It is given in the form of mouthrinses, brush on forms or by use of custom trays. 0.4% stannous fluoride or 1.1%sodium fluoride is given.
- Maintenance of oral hygiene: visit dentist once in 4 months.
- ince xerostomia cause demineralization of teeth and loss of tooth structure use remineralizing solutions.
- Candidiasis is more in xerostomia patients. It is due to Acidic and anaerobic climate and poor oral hygiene. So Candidiasis should be treated.

Treatment Of Candidiasis:

√ Nystatin - 5,00,000 U – Adults
- 1,00,000 U – children - 4 times daily for 7 -21 days

√ Amphotericin - oral suspension- 100mg/ml
- Lozenges – 10 mg

√ Clotrimazole – 10 mg

√ Fluconazole – 50 mg for 10 days

√ Ketaconazole- 200mg/day.

Summary for management of xerostomia: [9]

Moisture and lubrication (continuous, as needed)	
General·	**Specific**
drink (sip water, liquids)· use sugarless candy or gum· avoid ethanol· avoid tobacco· avoid coffee, tea and other caffeinated beverages	Oral balance (especially at night)Pilocarpine hydrochloride 2% (Salagen 5mg, 3 times daily)Mouthkote (artificial saliva)Optimoist Salagen 5mg, 3 times daily)Mouthkote (artificial saliva)Optimoist (artificial saliva)Salivart (artificial saliva)Sodium carboxymethyl cellulose 0.5% solution
Soft tissue lesions and soreness (treatment and maintenance)	
General	**Specific**
Oral Balance Biotene mouthwash	Benadryl 25mg/10ml + Maalox 64mL +Nystatin 1,00,000 U/ml elixir (Carafate, optional)(Lidocaine, 2% optional for I acute lesions)Decadron 0.5mg/5ml elixir (for acute lesions)Triamcinolone 0.1% [(in orabase) for acute lesions] Orabase –HCA (for acute lesions)Mycelex 60mg troches (for candidiasis)Mycolog II ointment (lips and tongue)
Prevention of caries and periodontal disease (continuous)	
General	**Specific**
• meticulous perioral hygiene· • avoid acidic foods· • regular hygiene and prophylaxis recalls· • sodium bicarbonate rinses (optional)· • halitosis (retardex)	Biotene toothpaste (neutral sodium fluoride 1.0% trays) Neutral sodium fluoride 1.0% applied in trays 2 times daily (Prevident 5000ppm)Peridex (chlorhexidine gluconate)Waterpik

√ Itraconazole- 100 -200 mg/ day – for 2 weeks.
√ Flucytosine – 50 – 150 mg/kg/ day – 4 times daily.

2. Symptomatic treatment:

- Frequent sipping of water to moisten the oral cavity, hydrate the mucosa and clear debris from mouth.
- Use of water with meals can make chewing and forming food bolus easier will ease swallowing and improve taste perception.
- Use of room humidifiers particularly at night may lessen discomfort.
- Avoid alcohol, smoking, dry foods.
- Use of moisturizing creams.
- Use of oral rinses and gels.

Saliva substitutes: Contain sodium carboxy methyl cellulose. All are available in spray form except Oral Balance which is available in gel form. Saliva substitutes available in market are Glandosone, Luborant, Oral Balance, Saliva orthana, Salivace, Saliveze.

3. Local Salivary Stimulation:

√ Chewing gums and mints
√ An **electronic device** [35]- very low voltage electrical charge which is applied to the tongue and palate, the SALITRON (Biosonics Inc., Philadelphia PA) has been used to stimulate the flow of saliva in patients with Sjogren's syndrome. Those who advocate its use claim that it stimulates flow by augumenting the normal physiologic salivary reflexes.

4. Systemic salivary stimulation:

- Bromhexine: a mucolytic agent. Mechanism of action for saliva stimulation is unknown. It stimulates lacrimal function in patients with Sjogren's syndrome.
- Bethanechol: Stimulates parasympathetic nervous system. Given in the dose of 75-200 mg/day
- Anetholetrithione – It is a mucolytic agent. Act by stimulating muscarinic receptors. Given in dosage of 75 mg tid. Side effects include Abdominal discomfort, flatulence.
- Pilocarpine HCl: It is a parasympathomometic drug functioning as a muscarinic cholinergic agonist. Given in dose of 5.0 -7.5 mg tid. Duration of action is 2 to 3 hrs. Side effects include sweating, Hot flashes, increase in urinary frequency, diarrhoea, blurred vision.
- Cevimeline HCl: It is a parasympathomometic drug. Targets muscarinic receptors of salivary and lacrimal glands. Given in dosage of 30 mg tid. It should be used with caution in patients who have history of glaucoma, CVS, respiratory and gall bladder disease and patients who use various medications.
- Yohimbine: It is a alpha adrenergic antagonist which indirectly results in increase of cholinergic activity peripherally. Given in dose of 6 mg tid for 5 days
- IFN – alpha: Given in Lozenges – 150 IU tid for 12 wks.

Newer treatment modalities for xerostomia [43]

a) Acupuncture:

Use of acupuncture treatment here 21 patients with severe xerostomia were selected 11 were treated with acupuncture and 10 patients received placebo acupuncture. Six to eight points that were choosen among local (stomach channel) and distal points (hand, pericardium channel), and two to four points were choosen from auricular points according to traditional Chinese medicine. The needles used were Chinese (Hwa To Brand; diameter 0.32mm, length 25mm) made of stainless steel, and autoclaved before use. Insertions were done to depths between 0.5 and 2.0 cm after the usual skin sterilizing procedures for 20 minutes twice a week for six weeks, and the interval between the two acupuncture series was 7-10 days. Those patients who received acupuncture treatment showed increased salivary flow rates during and after the acupuncture treatment. The improved salivary values persisted during the observation year, whereas the patients who received placebo acupuncture showed some improvement of salivary flow rates only during the actual treatment. The results of the present study indicated that acupuncture may be a useful adjunct for the stimulation of salivary flow in patients with xerostomia.

b) Acupuncture like TENS therapy:

It relies on low voltage electrical stimulation of the acupuncture trigger points instead of using needles. Uses frequency of 1-4Hz, pulse duration of 150-250 micro seconds, amplitude of 30-80 milli amperes used for twice a week for six weeks.

c) Gamma linoleic acid:

Although its mechanism of action is not clearly understood, gamma linoleic acid (evening primrose oil) 2000 units daily orally for a minimum of 6 weeks has been recommended for xerostomia patients. This is believed to stimulate parotid and submandibular gland salivary flow.

Conclusion

In conclusion health care professionals like dental practitioners are often the first health professionals to observe the oral changes associated with secretory salivary gland function and therefore it is imperative that they recognize the symptoms and signs of salivary dysfunction and are able to assist in diagnosing the cause and treating the oral sequelae.

Dentists can play a vital role in identifying patients at risk for developing salivary dysfunction and people suffering from salivary dysfunctions and should provide appropriate preventive and interventive techniques that will help to preserve a person's health, function, and quality of life. Thereby decreasing the morbidity associated with salivary dysfunctions like Xerostomia.

References

1. Sreebny LM and Anthony Valdini. Xerostomia. Part I: relationship to other oral symptoms and salivary gland hypofunction. Oral Surg Oral Med Oral Pathol 1988; 66: 451-8.
2. Robin M. Rankow. Irving M.Polayes; Diseases of the Salivary Glands; W.B.Saunders Company
3. Navazesh, Christensen, Brightman et al. Clinical criteria for the diagnosis of salivary gland hypofunction. J Dent Res 1992; 71(7); 1363-69.
4. Sreebny LM. Xerostomia and Diabetes mellitus. Jr Diabetes Care 1992; 15(7); 900-04.
5. David N Crockett. Xerostomia: the missing Diagnosis. Australian Dental Journal 1993; 38(2); 114-8.
6. Grisius MM, Fox PC. Salivary gland disease, In:Greenberg MS, GlickM, editors. Burket's Oral Medicine Diagnosis and Treatment. 10ed. Elsevier India. BC Decker Inc 2003. p 235-270.
7. Carl W. Managing the oral manifestations of cancer treatment. Part I: head and neck radiation treatment. Compend Contin Edu Dent 1988; 9(4); 306-18.
8. Niedermeier W, Matthaeus C, Meyer C. Radiation induced hyposalivation and its treatment with oral pilocarpine Oral Surg Oral Med Oral Pathol 1998;86(5);541-9
9. Arun K Garg, Mauricio Malo. Manifestations and treatment of xerostomia and associated oral effects secondary to head and neck radiation treatment. J Am Den Assoc 1997; 128;1128-31
10 Nelson L.Rhodus. Sjogren's Syndrome. Quitessence Int 1999;30;689-99
11. Manfredi M, MJ McCullough, P Vescovi et al. Update on diabetes mellitus and related oral diseases. Oral diseases 2004;10;187-200
12. Sharon A, Ben Aryeh H, Itzhak B et al. Salivary composition in diabetic patients J Oral Med 1985;40;23-26
13. Rees TD. The diabetic dental patient. Dent Clin North America 1994;38;447-463
14. Conner S, Iranpour B, Mills J. Alteration in parotid salivary flow in diabetes mellitus. Oral Surg Oral Med Oral Pathol 1970;30;55-59
15. Meurman JH, Collin HL, Nisken L et al. Saliva in non-insulin-dependent diabetic patients and control subjects: the role of the autonomic nervous system. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86;69-76
16. Rigmor E Persson, Kenneth, Edmond et al. Differences in salivary flow rates in elderly subjects using xerostomatic medications. Oral Surg Oral Med Oral Pathol 1991;72;42-6
17. Smith RG, Burtner AP. Oral side-effects of the most fre-quently prescribed drugs. Spec Care Dentist.1994; 14:96-102.
18. Scully C. Adverse drug reactions in the orofacial region. Crit Rev Oral Biol Med 2004 ;15(4);221-39
19. Sreebny LM, Valdini A; Xerostomia: a neglected symptom. Arch Intern Med 1988;147;1333-335
20. Billings RJ, Proskin HM. Xerostomia and associated factors in a community-dwelling adult population. Community Dent Oral Epidemiol 1996;24;312-16
21. Nederfors T, Isaksson R et al. Prevalence of perceived symptoms of dry mouth in an adult Swedish population-relation of age, sex and pharmacotherapy. Community Dent Oral Epidemiol 1997;25;211-216
22. Pujol T, Coma M et al. Acta Primaria 1998;21;225-28
23. Ava, Jonathan, Bethesda et al. A characterization of major salivary gland flow rates in the presence of medications and systemic diseases. Oral Surg Oral Med Oral Pathol 1993;76;301-6
24. Locker D. Subjective reports of oral dryness in an older adult population. Community Dent Oral Epidemiol 1993;21;165-68
25. Thomson WM, Williams SM. Oral Surg Oral Med Oral pathol Oral radiol Endod 2000;89;46-50
26. Nayak L, Wolff A. Oral Biosci Med 2004;1;283-89
27. Ben-Arych H, Miron D et al. Xerostomia in the elderly: prevalence, diagnosis, complications and treatment. Gerodontology 1985;4;77-82
28. Sreebny LM, Schwartz SS.A reference guide to drugs and dry mouth. 2nd edition. Gerodontology 1997; 14;33-47
29. Porter SR, Scully C. Oral Surg Oral Med Oral pathol Oral radiol Endod 2004;97;28-46
30. Bergdahl M, Bergdahl J. Low unstimulated salivary flow and subjective oral dryness: association with medication, anxi-ety, depression, and stress. J Dent Res 2000; 79:1652-1658.
31. Nederfors T. Xerostomia: prevalence and pharmacotherapy. With special reference to beta-adrenoceptor antagonists. Swed Dent J 1996; 116(Suppl):1-70.
32. Thomson WM, Chalmers JM, Spencer AJ, Slade GD. Medication and dry mouth: findings from a cohort study of older people. J Public Health Dent 2000; 60:12-20.
33. Field EA, Fear S, Higham SM, Ireland RS, Rostron J, Willetts RM, et al. Age and medication are significant risk factors for xerostomia in an English population, attending general dental practice. Gerodontology 2001; 18:21-24.
34. David W. Matear, Locker D, Stephen M et al; Associations between xerostomia and health status indicators in the elderly. Jr of Royal Society for the promotion of health 2006; 126(2); 79-85.
35. W.M. Edgar D. DSC, PhD Saliva: its secretion, composition and functions, Br Dent J 1992; 172: 305-312.
36. Philip C Fox, Busch, Baum. Subjective reports of xerostomia and objective measures of salivary gland performance. JADA 1987;115;581-584
37. Vincent Henricsson, Alvar Svensson. Evaluation of some electrical methods for objective assessment of oral mucosal dryness. Scand J Dent Res 1990;98;520-8
38. Sanchez GE, Aguirre G et al. The Wafer Test: a semi quantitative test to screen for xerostomia. Rheumatology 2002;41;381-389
39. Norman. Color atlas and text of the salivary glands- Diseases, Disorders and surgery; Mosby Wolfe Publications 1995 page 49-55.
40. Dawes C. How much Saliva is enough for avoidance of Xerostomia? Caries Res 2004; 38; 236-240
41. Stuart C. White, Micheal J.Pharoah. Oral Radiology: principles and interpretation 5th edition 2004 Mosby pg 660
42. Eric Whaites. Essentials of Dental Radiography and Radiology 2nd edition 1998 Churchill Livingstone publication Pg 384-94
43. Blom, Dawidson et al. The effect of acupuncture on salivary flow rates in patients with xerostomia. Oral Surg Oral Med Oral Pathol 1992;73;293-8.

Sialorrhea: A review

Poornima R*, Rajeshwari G. Annigeri**, Ashok L.***

*Assistant Professor **Professor and Head, Dept. of Oral Medicine and Radiology College of Dental Sciences, Davangere 577004, Karnataka, India, ***Professor and Head, Dept. of Oral Medicine and Radiology Bapuji Dental College and Hospital, Davangere, Karnataka 577004

Abstract

Saliva is a unique biologic fluid. Saliva is one of the most complex but versatile and important body fluids and contains a number of systems which serve a wide spectrum of physiological needs. Saliva is principle defense factor of the mouth. Similarly to any fluid in body even saliva can have eccentric variations, which either can be increase or decrease in levels this can be physiologic or pathologic disparities. Persistent alteration in the normal flow of saliva into the oral cavity is of considerable significance to the integrity of the oral and dental tissues.

Sialorrhea (drooling or excessive salivation) is a common problem in neurologically impaired children (i.e., those with mental retardation or cerebral palsy) and in adults who have Parkinson's disease or have had a stroke. It is most commonly caused by poor oral and facial muscle control. It causes a range of physical and psychosocial complications, including perioral chapping, dehydration, odor, and social stigmatization, that can be devastating for patients and their families.

Keywords

Saliva, Sialorrhea, Drooling, Hypersecretion, Ptyalism

Introduction

Hypersalivation or sialorrhea is a rare condition associated with excess production of saliva, inability to retain saliva within the mouth, or problems with swallowing. Sialorrhea is an important clinical, social, and emotional issue, which contributes to poor quality of life and career burden.[1] Salivary gland secretion is controlled by the autonomic nervous system, mediated by adrenergic and cholinergic nerve endings, but primarily under parasympathetic cholinergic control. The major salivary glands (the paired parotid, submandibular, and sublingual) are responsible for 95% of the 1.5 L of saliva secreted daily. In the unstimulated (basal) state, 70% of saliva is secreted by the submandibular salivary glands.[2]

Sialorrhea

Sialorrhea is defined as saliva emanating beyond the lip margins.[3] The physical and psychosocial complications (associated with sialorrhoea) range from mild and inconvenient symptoms to severe problems that can have a significant negative impact on quality of life.

Drooling (also known as ptyalism or sialorrhea)

Drooling is the unintentional loss of saliva from the mouth. It is an indication of an upset in the co-ordinated control mechanism of oro-facial and palato-lingual musculature leading to excessive pooling of saliva in the anterior mouth and resultant unintentional loss of saliva from the mouth. [4] This commonly refers to anterior drooling and should be distinguished from posterior drooling, in which saliva spills over the tongue through the faucial isthmus. It is a significant disability for a large number of pediatric and adult patients with cerebral palsy and for a smaller number of patients with other types of neurologic or cognitive impairment.

Drooling is a normal phenomenon in children prior to the development of oral neuromuscular control till the age 18-24 months. However, drooling after age 4 years is uniformly considered abnormal. Children with neurologic impairment may be slow to mature their oral neuromuscular control and may continue to improve their control until approximately 6 years of age, which prompts physicians to delay any aggressive intervention until that time.

The actual prevalence rate for patients is unknown. However, 0.5-0.7% of all children born are diagnosed with cerebral palsy. It is stigmatizing and the prevalence in neurological diseases is high.

Sialorrhea occurs in about 50% of the patients with Amyotrophic lateral sclerosis and 20% of the victims need continuous saliva elimination, which has prevalence of about 70% in Parkinson disease, and between 10 and 80% in patients with cerebral palsy. The prevalence of sialorrhea in these affections is high, with impairment of social integration and difficulties to perform oral motor activities during feeding and speech, with repercussions in quality of life. [5]

From 10-37% of patients with cerebral palsy have been reported to have difficulty with drooling because of neurologic impairment. Reportedly, 10% of Swedish, 37% of Belgian, and 13% of Indian children with cerebral palsy have severe drooling. However, this is not an affliction that is particular to any specific ethnic background. Most of the patients requiring help for drooling belong to this group.[5]

Etiology for sialorrhea [6]

1) Neuromuscular/sensory dysfunction
- Mental retardation
- Cerebral palsy
- Parkinson's disease
- Pseudobulbar palsy
- Stroke

2) Hypersecretion
- Inflammation (teething, dental caries, oral-cavity infection, rabies)
- Drug side effects (tranquillizers, anticonvulsants)
- Gastro-oesophageal reflux
- Toxins (e.g. mercury, lead, arsenic)

3) Anatomic
- Macroglossia
- Oral incompetence
- Malocclusions
- Orthodontic condition
- Head and neck (H&N) surgical defect ("Andy Gump"deformity caused by resection of part of mandible which in turn causes drooping of cheek and decreased vertical height)

Etiology of drooling [7]

1) Direct causes
- Cerebral plasy
- Motor neuron damage
- Cerebrovascular accidents
- Parkinsonism
- Congenital suprabulbar palsy
- Major resection of the oropharynx

Corresponding Author:
Dr.Poornima.R
Assistant Professor, Department of Oral Medicine and Radiology Bapuji Dental College and Hospital Davangere Karnataka-577004
E mail: drpoornima_omr@yahoo.co.in

2) Indirect causes

- Nasal obstruction
- Malocclusion
- Tongue thrusting
- Constant open mouth and poor lip control
- Hypoactive gag reflex
- Gastro-oesophageal reflux
- Head posture and sitting position
- Concentration on a task

Pathophysiology

Drooling may be a result of hypersecretion (primary sialorrhea) of the salivary glands but more commonly is due to impaired neuromuscular control with dysfunctional voluntary oral motor activity that leads to an overflow of saliva from the mouth (secondary sialorrhea). Patients often have inefficient and infrequent swallowing, which further compounds the problem. Furthermore, problems with positioning due to poor head control and decreased neck strength magnify the effects. An enlarged tongue or tongue thrusting with poor control can contribute to the problem. Finally, dental caries and infection and diseased gingival tissues with gingivitis can markedly increase drooling.

Healthy subjects secrete from 1000 to 1500ml saliva within 24 hours. Many neurological diseases progress with difficulties in the oral motor control. When the production of saliva exceeds the subjects' skill to transport it from the mouth to the stomach, stasis, extraoral leak and aspiration may take place, in addition to concomitant difficulty of mastication and articulation.

Some people with drooling problems are at increased risk of inhaling saliva, food, or fluids into the lungs. However, this is unlikely to cause harm, unless the body's normal reflex mechanisms (such as gagging and coughing) are also impaired.

Isolated drooling in infants and toddlers is normal and is unlikely to be a sign of either disease or complications. It may be associated with teething. Drooling in infants and young children may be exacerbated by upper respiratory infections and nasal allergies. [8]

Hypersecretion is a rare cause of drooling. Most often, this occurs as an adverse effect of medications such as tranquilizers, anticonvulsants, and anticholinesterases that increase activity at the muscarinic receptors of the secretomotor pathway and result in hypersecretion.

Any impairment of the oral phase of deglutition secondary to neuromuscular disorders, trauma, surgical resection, or facial nerve paralysis can result in spillage of saliva from the oral cavity. The majority of patients who drool have impaired oral neuromuscular control due to cerebral palsy or severe mental retardation.

Drooling associated with fever or trouble swallowing may be a sign of a more serious disease including: [8]

- Retropharyngeal abscess
- Peritonsillar abscess
- Tonsilitis
- Mononucleosis
- Streptococcal throat infection
- Parkinson's disease

A sudden onset of drooling may indicate poisoning (especially by pesticides) or reaction to snake or insect venom. Excess Capsaicin can cause drooling as well, an example being the ingestion of particularly high Scoville Unit chili peppers.

Another form of ptyalism is associated with pregnancy, most common in women with a condition known as Hyperemesis Gravidarium, or uncontrollable and frequent nausea and vomiting during pregnancy which is far worse than typical "morning sickness". With Hyperemesis, ptyalism is a side-effect, which is a natural response to uncontrollable vomiting. With normal vomiting, salivary glands are stimulated to lubricate the esophagus and mouth to aid in expelling of stomach contents.

In the rare cases of genuine increased salivation, for reasons other than mucosal irritation, the patient becomes aware of the need to swallow more frequently and may experience saliva leaking from the mouth overnight. Those with an obsessive problem have no physical symptoms but may swallow compulsively at frequent intervals and are even inclined to accentuate this procedure, a pattern that causes some distress.

Infants and the mentally impaired are often affected as a result of diminished neural control and difficulty in swallowing. Foreign bodies, inflammatory lesions, Parkinson's disease, rabies, heavy metal ingestion (lead and mercury), and drug induced ptyalism are other causes of hypersecretion.

Drug commonly implicated are parasympathomimetic agents (e.g. neostigimine and pilocarpine), and the antitubercular drug Ethionamide, ammonium complexes and heavy metals (ie. bromide, iodine's, mercurial salts). Persons living in substandard conditions or in close proximity to automobile battery storage areas or lead paint can be exposed to extremely high levels of lead in their surroundings. These persons often exhibit neurologic deficits concurrent with sialorrhea and other oral findings.

The reason for excessive drooling seems to be related to
(1) lack of awareness of the build-up of saliva in the mouth,
(2) infrequent swallowing, and
(3) inefficient swallowing.

Clinical features

Excessive salivation is most evident as pooling of saliva in the floor of the mouth and/or drooling. Chronic drooling increases the permeability of the perioral skin and thereby reduces the effectiveness of the epithelial barrier. Opportunistic infections become more likely. Under these circumstances, candidal infection of the lips (monilial chelitis or angular chelitis) may occur. The infected epidermal surface appears red and inflamed and eventually ulcerated. When hypersalivation is secondary to lead poisoning, patients also have a dark gray linear pigmentation of the attached gingiva.

Diagnosis

I. Clinical History

A thorough history is invaluable prior to treatment. Make an assessment of the severity and frequency of drooling, and enquire about the effect on the quality of life for the patient and family. Importantly, identify factors contributing to drooling. Caregivers or parents can assist in assessing the characteristics of drooling, such as peak time of day, changes in volume with specific activities, consistency of saliva (ie, thick, mucinous, watery), and the frequency of drooling.

Quantitative measurements can be difficult, but classification schemes have been developed to give a general idea of the magnitude of the problem. Multiple classification schemes have been used by different authors to report the severity of drooling.

System for Assessment of Frequency and Severity of Drooling [9]

Some specific points to address when assessing the magnitude of the problem with care givers include the following:

Drooling	Points
Severity	
Dry (never drools)	1
Mild (wet lips only)	2
Moderate (wet lips and chin)	3
Severe (clothing becomes damp)	4
Profuse (clothing, hands, tray, objects become wet)	5
Frequency	
Never drools	1
Occasionally drools	2
Frequently drools	3
Constantly drools	4

- Number of bib or clothing changes per day
- Difficulties with keyboards or other communication devices
- Severity of perioral skin laceration and infections.

The system used by Wilkie and Brody [10] to classify the results of drooling procedures is as follows:

- Excellent - Normal salivary control
- Good - Slight loss of saliva with or without dried froth on the lips
- Fair - Improved, but with significant residual saliva loss or with thickened, offensive, brown, gummy froth
- Poor - Failure to control or too dry

Other clinical factors that could contribute to spillage of oral contents should be explored while taking the patient's history. Nasal obstruction with chronic mouth breathing can exacerbate drooling. The most common cause of obstruction is adenoid hypertrophy, anterior obstruction of the nose due to other causes, such as allergic rhinitis should also be considered. Malocclusion, gingivitis, and dental caries can contribute to drooling and should be addressed by a pediatric dentist at the outset of the evaluation.

II. Physical examination [5]

Perform a thorough head and neck examination. Give special consideration to those anatomic factors that could contribute to or exacerbate drooling so that these issues can be addressed prior to surgical intervention. Some key points to evaluate during the physical examination include the following:

- Head position and control
- Condition of perioral skin
- Tongue size and control and the presence of thrusting behaviors
- Tonsil and adenoid size
- Occlusion: Malocclusion, particularly an open bite deformity, is a common finding in patients with cerebral palsy. This can make proper oral hygiene very difficult. Open bite deformities can prohibit closing of the mouth and can mimic nasal obstruction in these patients.
- Dentition: Caries may be noted.
- Gingival tissues
- Gag reflex and intraoral tactile sensitivity
- Presence of mouth breathing
- Nasal obstruction and the appearance of tissues upon anterior rhinoscopy
- Swallowing efficiency: Determine this by observation, barium swallow, or fiber optic endoscopic evaluation of swallowing.
- Neurologic examination: Pay particular attention to cranial nerve examination findings.

III. Drugs-related Hypersalivation [11]

Most common drugs which have been implicated for causing sialorrhea are listed in the following table

Alprazolam	Guanethidine	Mefenamic acid	Tacrine
Amiodarone	Haloperidol	Mercurials	Tobramycin
Buprenorphine	Imipenem	Nicardipine	Triptorelin
Buspirone	Iodides	Niridazole	Venlafaxine
Clonazepam	Kanamycin	Pentoxifylline	Zaleplon
Diazoxide	Ketamine	Remoxipride	
Ethionamide	Lamotrigine	Risperidone	
Gentamicin	L-dopa	Rivastigmine	

IV. Lab Studies:[8]

- Salivary flow rate (ml/min): increase in weight of dental rolls/time of collection The absorbent dental rolls can be kept directly at the orifices of large salivary glands
- Drooling Quotient : 40 observations in 10 minutes (every 15 minutes) DQ% = 100 x number of drooling episodes/40
- Teacher Drooling Scale: 1-5
  1= no drooling
  3= occasional drooling
  5= constantly wet saliva leaking on clothes and furniture

V. Imaging Studies:[8]

- Lateral neck film: Adenoid hypertrophy can be confirmed if the patient has a history of nasal obstruction. Complete the adenoidectomy prior to further surgical intervention to neutralize the effects of mouth breathing on drooling.
- Modified barium swallow: Some authors recommend performing this study helps to rule out the contraindications to surgical therapy, including esophageal motility disorders, esophageal spasm, or aspiration.
- Radiosialography: For some authors and for research purposes, these scans using radioisotope are useful for evaluating the secretory function of the salivary gland when assessing the success of surgical therapy.

VI. Other Tests:

- Audiogram: Perform this study on patients being considered for tympanic neurectomy or chorda tympani nerve section because unilateral hearing impairment is a contraindication because of the risk of hearing loss associated with the procedures.
- Flexible nasopharyngoscopy: This is an alternative method to assess the amount of adenoid tissue if the patient has findings suggestive of nasal obstruction.

Management of Sialorrhea

Treatment of excessive drooling aimed at:

(1) increasing awareness of the mouth and its functions,
(2) increasing frequency of swallowing,
(3) increasing swallowing skill.

The methods available for the management of drooling [12]

- **Non- surgical methods**
  - Bio-feed back techniques
  - Bio-functional appliances
  - Physiotherapy
  - Behavioural therapy
  - Drug therapy – Scopolamine, Glycopyrrolate, Botox
  - Radiotherapy
- **Surgical methods**
  - severance of parasympathetic supply of salivary glands
  - salivary gland duct relocation/rerouting (retropositioning)
  - salivary gland duct ligation
  - salivary gland excision

I. Home care

Care for drooling due to teething includes good oral hygiene. Ice pops or other cold objects (e.g., frozen bagels) may be helpful. Care must be taken to avoid choking when a child uses any of these objects. Drooling also is common in children with neurological disorders and those with undiagnosed developmental delay.

II. Medical therapy

Treatment of patients with drooling problems has been successful at some centers using a team approach, including an otolaryngologist, pediatric dentist, speech pathologist, and physical therapist.

Aggressive medical management prior to considering surgical intervention is recommended. Medical management is directed towards correcting the oral motor dysfunction and decreasing the secretory volume of salivary glands. Physiotherapy and speech therapy may improve the handling of saliva, although these alone tend to be insufficient for all but lesser afflicted individuals. The second approach is to reduce salivation pharmacologically, with constant moisture at

Drugs In Treatment Of Sialorrhea [8]

Agent	How supplied	Dosage	Side effects
Glycopyrrolate	Scored tablets,1 or 2 mg	**Adults:** Start at 0.5 mg orally, one to three times daily;titrate to effectiveness and tolerability **Children:** 0.04 mg per kg per dose orally, two to three times daily; titrate to effectivenessand tolerability	Constipation, Excessive oraldryness, urinary retention, blurred vision, hyperactivity, irritability
Scopolamine Patch	1.5 mg Transderm Scop)	Apply patch every day	Pruritus at patch site, urinaryretention, i rritability, blurredvision, dizziness,
Botulinumtoxin A	Vial, 100 Uper vial	Under ultrasound guidance, injections of 10 to 40 units into each submandibular and parotid gland	glaucoma Pain at injection site, excessive oral dryness

the commissures of the mouth and over the skin of the chin, the epithelium becomes macerated and is vulnerable to secondary, opportunistic infection. In order to control this, it may be useful to apply regularly a coating of waterproofing, emollient wax over the area. A combination of beeswax and purified orange roughy oil (a liquid wax) is helpful. Jojoba oil would be an alternative to the fish oil. Topical antifungals, such as nystatin or miconazole ointment, or antibacterials, such as mupirocin ointment, are used as appropriate.

III. Nonsurgical methods for treating drooling problems include oral motor therapy [13]

Oral motor programs aim to develop oral skills such as sucking, lip closure, and tongue and jaw movement. The speech therapist plays a crucial role in evaluating the existing oral motor skills of the patients. Positive results were achieved in cerebral palsied children participating in a training program using mirrors, games, relative competitiveness and praise reinforcement by the physiotherapist, combined with a "chin cup" appliance to apply pressure on the chin to achieve an appropriate anterior oral seal.

Patients with cerebral palsy are often affected by varying degrees of physical disability, including lack of muscle tone affecting head position and oral dysfunction, which causes the initiation of swallowing to be uncoordinated and inefficient. Exercises are used to attempt to normalize muscle tone, stabilize body and head position, promote jaw stability and lip closure, decrease tongue thrust, increase oral sensation, and promote swallowing. The speech pathologist, physical therapist, and occupational therapist administer this mode of medical therapy.[14]

Behavioral modification via biofeedback [15]

Biofeedback techniques condition the patient to swallow at the sound of an auditory stimulus. A successful training program was conducted using an auditory electromyography (EMG) feedback with electrodes placed on the orbicularis oris muscle. It was considered that patients must be old enough, have relatively good intellectual functions, be auditory-prompted, be reasonably well motivated, present only a moderate drooling problem, and not become oblivious to the auditory device or its signal.

Verbal and auditory cues are used to attempt to increase the frequency and efficiency of swallowing. Several methods, including reward, overcorrection, and punishment, are used by caregivers to initiate swallowing. External devices that deliver timed auditory cues to swallow are also used.

Several authors note that the success of therapy is dependent on the patient's cognitive level of function and ability to concentrate. Hence, patient-to-patient variability of results is considerable. Also, success is variable according to the task being completed by the individual patient when the cue is delivered. Consequently, extrapolation of results to patient's daily environment is difficult. Regression of therapy has been shown to occur at varying times following training sessions. Repeat therapy is often required for reinforcement.

Orofacial regulation therapy: [16]

A functional appliance, employed according to the principles of Castillo Morales, has been used successfully in the management of drooling. It consists of an acrylic palatal plate with vestibular and lingual stimulators. The vestibular stimulator, formed by varying the depth of the ridge, stimulates the lip seal. The lingual stimulator is a median button with a central hole that induces sucking, and subsequently, tongue retrusion. This appliance is very helpful for patients with hypotonic perioral musculature and protruding tongue as commonly seen in Down's and Moebius syndrome. [17]

Radiotherapy: [18]

Radiation to the salivary glands is a reason-able treatment option in elderly patients who are not candidates for surgery and cannot tolerate medical therapy. Radiation produces xerostomia that may last months to years. The dose may be titrated to reach the desired effect, and treatment can be repeated as necessary. Malignancies induced by radiation therapy typically do not occur until 10 to 15 years after treatment and, therefore, are less of a concern in patients who are elderly and debilitated. The authors concluded that the desired response, with minimal discomfort, can be expected with five 4 Gy fractions to a total dose of 20 Gy using 9-18 MeV electrons prescribed to the 100% iso dose, encompassing both the parotid and submandibular glands with ipsilateral fields. Low doses of direct irradiation therapy to the parotid glands have been used to control drooling with impressive results.[19]

Botox: (Botulinum toxin type A)[3, 20]

Neurobotulinum toxin serotype A (TBA) has drastically changed the treatment of a broad range of autonomous hypersecretory alterations such as focal hyperhidrosis (axillary perspiration, sweating of the palms, or gustative perspiration), sialorrhea, pathological lacrimation, and rhinorrhea .

Its application as treatment for sialorrhea was first proposed in 1997 by **Bushara**, in the form of an injection into the parotid glands of patients with amyotrophic lateral sclerosis and other neurological diseases. This toxin is produced by a gram-negative anaerobic bacterium, Clostridium botulinum. Its action is based on the inhibition of acetylcholine release at presynaptic level. The toxin acts upon the cholinergic nerve endings, causing proteolysis of SNAP-25 (synaptosomal associated protein, implicated in synaptic vesicle fusion with the presynaptic membrane), thus resulting in local chemical denervation and the loss of neuronal activity in the target organ.[21]

Mechanism of action

- Acts by blocking the release of a chemical transmitter called acetylcholine from the nerve terminals.(Acetylcholine is released by the nerves connecting the brain to the salivary gland resulting in the production of saliva)
- Injection of salivary gland with BOTOX blocks the signals to produce saliva and excessive salivation is improved.

Iv. Surgical management of drooling [22]

In most cases, surgical intervention should be instituted following the failure of at least 6 months of more conservative therapy. Surgery is best delayed until the patient is aged 6 years or older in order to allow time for complete maturation of oral motor function and coordination.

Indications for surgery include

(1) Persistent drooling following at least 6 months of conservative therapy and
(2) Moderate to profuse drooling in a patient whose cognitive function precludes participation with conservative oral and physical therapy.

Contraindications

1. Patients who are at high risk for surgery because of other medical concerns.
2. Tympanic neurectomy and chorda tympani nerve sections are contraindicated in patients with unilateral hearing loss because of the small risk of hearing loss associated with these procedures.
3. Posterior rerouting of the submandibular or parotid ducts is controversial in patients who have difficulties with chronic aspiration due to their neurologic status. Associated conditions may include esophageal motility disorders, esophageal spasm, or aspiration. Some authors believe this procedure puts the patient at increased risk of aspiration because of the increased burden of secretions in the hypopharynx. Other authors have demonstrated no increased difficulty with aspiration in this patient population.
4. In patients with athetoid disorders with constant tongue thrusting, surgical procedures to correct drooling may result in an unpleasant, thick, discolored, malodorous residue being deposited on the teeth and lips. This may prove to be more offensive than the constant, watery drooling.

Oral manifestation of sialorrhea and considerations

Effect of the management of drooling on oral health has become a well established fact that a normal flow of saliva is paramount to oral and dental health.

Patients with sialorrhea have excess saliva of normal consistency because saliva pools in the floor of mouth, it may interfere with dental treatment that requires a dry operating field. Under these circumstances a rubber dam and salivary ejector should be used to complete routine restorative procedures. In addition, an upright chair position is more conducive to airway management. In addition to the complications a pronounced reduction in salivary flow (temporary or permanent) could result in rampant caries, rapid tooth destruction, dryness and cracking of the lips, fissuring of commisures, burning sensation of the mucous membranes, crusting of the tongue and palate and occasionally paraesthesia of the tongue or mucous membranes. Sialorrhea is also present as an early symptom in patients suffering from Acute necrotizing ulcerative gingivitis (ANUG) but the mechanism is not understood.

Individually designed dental preventive measures should be carried out in such patients, to minimize the adverse effects of treatment. Includes appropriate use of fluorides, oral hygiene measures, dietary advice, fissure sealants and regular professional scaling and polishing. Careful monitoring for oral complications following such treatment is essential.[23]

Conclusion

Sialorrhoea is an important clinical, social, and emotional issue, which contributes to poor quality of life and career burden. It is associated with a wide range of disorders. Drooling may be a result of hypersecretion (primary sialorrhea) of the salivary glands but more commonly is due to impaired neuromuscular control with dysfunctional voluntary oral motor activity that leads to an overflow of saliva from the mouth (secondary sialorrhea).

A multidisciplinary team is indispensable for appropriate assessment and management of drooling. Any aggravating problem, like significant dental disease, abnormal head position leading to abnormal salivary flow with gravity, severe malocclusion, airway obstruction, or certain drug effects, must be recognized and treated or relieved. In cases of drooling, a series of considerations are required which, in some centers, are addressed by a team which includes Physiotherapy, speech therapy, pharmaco therapy, Surgical management. Newer treatment modalities like photocoagulation of salivary ducts, tongue acupuncture are being tried. However, the technical skill and experience of the practitioners is a marked obstacle for such procedures.

In conclusion health care professionals like dental practitioners are often the first health professionals to observe the oral changes associated with secretory salivary gland function and therefore it is imperative that they recognize the symptoms and signs of salivary dysfunction and are able to assist in diagnosing the cause and treating the oral sequelae. Dentists can play a vital role in identifying patients at risk for developing salivary dysfunction and people suffering from salivary dysfunctions and should provide appropriate preventive and interventive techniques that will help to preserve a person's health, function, and quality of life. Thereby decreasing the morbidity associated with salivary dysfunctions like Sialorrhea.

References

1. Jean-Paul Meningaud et al. Drooling of saliva: a review of the etiology and management options. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101;48-57
2. Siebert G, Mielke A, Hanbrich J, Chilla R. Physiology and Biochemistry. In: Seifert G, et al (editors). Diseases of the salivary

Advantages and Disadvantages of Surgical Therapies for Sialorrhea [10]

Surgical therapy	Advantages	Disadvantages
Submandibular duct relocation	No external scarLow incidence of ranula with sublingual gland excision	Duct relocation is an uncommon procedurePot ential for anterior dental cariesWithout sublingual gland excision, patient may develop ranula Potential for aspiration
Submandibular gland excision	Very good control of sialorrhea Commonly performed procedure	External scarPotential for dental caries
Parotid duct relocation	Redirects flow in the stimulated state	Risk of sialocelePotential for aspiration Relocation is uncommon procedure
Parotid duct ligation	Simple, fast procedureDecreases flow in the stimulated state	Risk of sialocele
Transtympanic neurectomy	Technically easy, fast procedureDoes not require general anesthesia Useful in elderly patients	Predictable return of salivary function. Requires multiple procedures

glands. New York: George Thime; 1986,28–45.
3. Subhaschandra Shetty, Patrick Dawes, Dean Ruske, Mohannad Al-qudah, Brett Lyons. Botulinum toxin type-A (Botox-A) injections for treatment of sialorrhoea in adults: a New Zealand study. Journal of the New Zealand Medical Association, 18-August-2006 Vol 119:1240-43
4 . Cotton RT, Richardson MA. The effect of submandibular duct rereouting th the treatment sialorrhoea in children. Otolaryngology – head and neck surgery 1981;89:535-41
5. Gies R, Neumann M, Werner E, Riemann M, Beck I, Puls I, Reiners C, Toyka KV. Injections of botulinum toxin A into salivary glands improve sialorrhoea in amyotrophic lateral sclerosis. J Neurol Neurosurg Psychiatry 2000; 69: 121-3.
6. Neil G. Hockstein, Daniel S. Samadi, Kristin Gendron, Steven D. Handler. Sialorrhea: A Management Challenge. Am Fam Physician 2004;69:2628-34.
7. Hussein, AE Kershaw, JF Tahmassebi et al. The management of drooling in children and patients with mental and physical disabilities- a literature review. Int Jr of Pediat Dent 1998;8:3-11
8. http://en. Sialorrhoea. wikipedia.org
9. Thomas-Stonell N, Greenberg J. Three treatment approaches and clinical factors in the reduction of drooling. Dysphagia 1988;3:73-8.
10. Wilkie TF, Brody GS. The surgical treatment of drooling. A ten-year review. Plast Reconstr Surg. Jun 1977; 59(6):791-7.
11. Scully C. Adverse drug reactions in the orofacial region. Crit Rev Oral Biol Med 2004;15(4);221-39
12. Blasco PA, Allaire JH and participants of the consortium on drooling: drooling in the development disabled: management practices and recommendations. Developmental medicine and child neurology 1992;34:849-62
13. Harris MM, Dignam PF. A non-surgical method of reducing drooling in cerebral palsied patients. Dev Med child neurol 1980;22;293-9
14. Blasco PA, Allaire JH. Drooling in the developmentally disabled: management practices and recommendations. Consortium on Drooling. Dev Med Child Neurol. Oct 1992;34(10):849-62.
15. Koheli R, Sochaniwsky A et al. Biofeedback techniques and behavior modification in the conservative remediation of drooling by children with cerebral palsy. Dev Med Child Neurol 1987;29;19-26
16. Fischer-Brandies H, Avalle C, Limbrock GJ. Therapy of orofacial dysfunctions in cerebral palsy according to Castillo-Morales: first results of a new treatment concept. Eur J Orthod 1987;9:139-43
17. Hoyer H, Limbrock GJ. Orofacial regulation therapy in children with down syndrome, using the method and appliance of Castillo-Morales. ASDC J Dent Child 1990;57;442-44
18. Borg M, Hirst F. The role of radiation therapy in the management of sialorrhea. Int J Radiat Oncol Biol Phys. 1998; 41:1113–9.
19. Robinson AC, Khonery GG, Robinson DM. role of irradiation in the suppression of parotid secretions. Jrnl of Laryngo and Otol 1989;103:594-95
20. Fuster-Torres MA, Berini-Aytés L, Gay-Escoda C. Salivary gland application of botulinum toxin for the treatment of sialorrhea. Med Oral Pathol Oral Cir Buccal. 2007 Nov 1; 12(7):E511-7.
21. Bushara KO. Sialorrhea in amyotrophic lateral sclerosis: a hypothesis of a new treatment—botulinum toxin A injections of the parotid glands. Med Hypotheses. 1997 Apr; 48(4):337-9.
22. Robin M. Rankow. Irving M.Polayes; Diseases of the Salivary Glands; W.B.Saunders Company
23. Mc Donald RE, Avery DR, Stooky GK. Dental caries in the child and adolescent In: Mc Donald RE, Avery DR.Dentistry for the child and adolescent, 6th edition. St Louis: Mosby 1994;216-55

Clinico-medicolegal study of aluminium phosphide poisoning

Puneet Khurana*, J.S.Dalal**, A. S. Multani***, H.R. Tejpal ****

*Assistant Professor, Department of Forensic Medicine, Christian Medical College, Ludhiana, **Prof. & Head, Department of Forensic Medicine, Government Medical College, Patiala,Punjab, ***Ex. Prof. & Head, Medicine Department, Government Medical College, Amritsar, ****Additional Professor, Department of Forensic Medicine, Government Medical College, Amritsar

Abstract

Aluminium phosphide, an ideal fumigant, freely available over counter is a suicidal agent very toxic to heart, lungs, g.i.t and other organs. The present study of 50 cases of aluminium phosphide poisoning showed male preponderance, involvement of younger generation, short mean survival time and suicidal intention. The mortality rate was very high and was directly proportional to the dose of poison ingested.

Keywords

Suicide, Poisoning, Fumigant, Mortality rate.

Introduction

Fumigants are used in the control of insects, ro-dents and soil nematodes, being in the gaseous form at the time they exert their action and penetrate to areas otherwise inaccessible for pesticide application. Aluminium phosphide is an inorganic compound of aluminium and phosphide. Aluminium phosphide is a solid fumigant pesticide in use since 1940s[1]. It has been a boon for the agriculture industry in India and has emerged as an ideal fumigant[2] because of its low cost, easy transportation, high efficacy (phosphine gas is highly lethal for the target species, while sparing others), considerable safety for handling because of distinct odour. It is available over the counter and is marketed in India as Celphos, Alphos, Quickphos, Synfume, Phostoxin, Phosfume, Fumigran.

History

Earlier isolated cases of fatal exposure to phosphine gas have reported when aluminium phosphide was used as a grain fumigant. Acute accidental phosphine gas poisoning was reported in 1978 abroad a grain freighter at a Canadian port in which one child died and 29 crew members become acutely ill[1]. In the last two decades aluminium phosphide poisoning reported in different northern states of India. Incidence of aluminium phosphide poisoning has increased steeply during the last two decades. At present it has achieved alarming proportions and has gripped many states in northern India. In the word literature first suicidal case was reported by Zipf et al.[3] In India the incidence of aluminium phosphide poisoning was unknown before 1980.The first report of aluminium phosphide poisoning appeared in 1983. Near about 37 cases of aluminium phosphide poisoning were reported from Udaipur Medical College in one year to Press trust of India.[4]

Fatal Dose: Less than 500 mg of an unexposed pellet of aluminium phosphide is lethal for an adult (usu-al being 150 - 500 mg for a 70 kg individual). The inhalation of phosphine at a concentration of 290- 300 ppm is dangerous to life. At a level of 400-600 ppm it is lethal within half an hour, at a level of 1000 ppm it is rapidly fatal.[5]

Fatal Period: One hour to four days. Majority die within twenty-four hours.[6]

Signs & symptoms:

Mild inhalation exposure produces throat irritation, acute respiratory distress. Other symptoms include headache, tightness in the chest, fatigue, nausea, vomiting, diarrhoea, great thirst.

Moderate to severe toxicity produces ataxia, numbness, paraesthesia, tremors, diplopia, jaundice, muscular weakness, in coordination and paralysis.

Very severe toxicity produces acute respiratory distress syndrome, cardiac arrhythmias, convulsions and coma. Higher concentration of phosphine at 300 ppm can kill a person.

Ingestional toxicity could be mild, moderate or severe, depending on the dose of the fresh compound consumed.

Mild intoxication produces nausea, vomiting, headache and abdominal pain, and the patients usually recover.

The systemic symptoms and signs of moderate to severe ingestional toxicity are as follows:

G.I.T.: Nausea, vomiting, diarrhoea, retrosternal pain.

C.V.S.: Cardiogenic shock is the common cause of death. About 90% of patients died due to profound hypotension. ECG changes in aluminium phosphide poisoning are common and diverse. The ECG changes seen are sinus tachycardia, premature beats, junctional tachycardia, atrial fibrillation and intra ventricular conduction defects, early repolarisation syndrome, varying SA blocks, brady tachysyndrome and electrical alternans. ST segment changes are the commonest. ST depression being more common than elevation and few patients also having brady arrhythmias.[7] Complications include pericarditis, [8] myocarditis, acute congestive heart failure.

Respiratory System: Cough, dyspnoea, cyanosis, pulmonary edema, ronchi, bilateral basal crepitation, respiratory failure, ARDS.[9]

Hepatic: Jaundice, hepatitis, hepatomegaly.

Renal: Acute Tubular Necrosis, Renal failure.

C.N.S.: Headache, dizziness, restlessness, convulsions, acute hypoxic encephalopathy, coma.

Rare: Muscle wasting tenderness [10] and bleeding diathesis [11] due to wide capillary damage.

Organ damage is wide spread but appears to be hypoxic evident from ante mortem & postmortem finding.[5]

Aims and objectives

1) The exact sequence of signs and symptoms and its correlation with possible court questions (medico legal importance).
2) Minimum and maximum fatal period.
3) Various demographic features like age, sex, social background, marital status, intension of poisoning.
4) Relationship of the dose and freshness of aluminium phosphide tablets to mortality.

Material & Methods

50 cases of aluminium phosphide poisoning admitted to Medicine Department of Guru Nanak Dev hospital, attached to Government Medical College, Amritsar were studied during the period from 01-10-2004 to 15-04-2006. The diagnosis of aluminium phosphide poisoning was based on reliable history of ingestion, circumstantial evidences such as the production of the remaining tablets/empty con-tainer by the relatives, garlic/decaying fish like odour & evident clinical manifestations.

The confirmation was done by sil-ver nitrate paper test with gastric aspirate in every case. The data and information pertaining to the cases were collected by interviewing the patient, relatives or friends of the patient and recorded on the proforma to find out answers to possible

court questions. The sensitivity of 'silver nitrate paper test' is high even with low concentration of phosphine (even trace amount) darkens the silver nitrate pa-per. Its specificity is also high except sometimes when silver nitrate produces blackening due to reaction with H_2S in the air. For this reason an-other filter paper impregnated with silver nitrate has to be kept outside as control. The procedure adopted includes

With Gastric aspirate: Gastric contents were aspirated with the help of nasogastric tube. 5 ml of gastric aspirate with 15 ml of water were placed in a flask, a round strip of filter paper impreg-nated with $AgNO_3$ (0.1 N) were placed on mouth of flask and heated at 50^0C for 15-20 minutes followed by drying of filter paper. Blackening of filter paper is indicative of presence of phosphine (PH_3) gas. [12, 13]

Observations & discussion

The present clinico-medicolegal study of aluminium phosphide poisoning was done in the Department of Forensic Medicine & Toxicology and Medicine Department, Government Medical College, Amritsar with effect from 1-10-2004 to 15-4-2006.

There were 33(66%) males and 17(34%) females giving a distinct male preponderance as shown in table 1. Male: female ratio being 1.94:1. As far as the age group is concerned in both males and females a majority of the patients were i.e. 34% (24% males & 10% females) in the 21-30 years age group followed by the age group of 31-40(20% males &10% females). No case was reported in the age group of 0-10 years in both males and females. The maximum age was 63 years in males and 42 yrs in females and the minimum age was 16 years in both males & females. Majority of the patients in our study were young in the age group of 21-40 years. The study shows decline in incidence with increasing age. Frustration due to imbalance between ambitions and available avenues at an age when dependence on parents is usually weaned off seemed to be the root cause of poisoning in young age group persons. In the present study male: female ratio is 1.94:1. This ratio coincides with ratio observed by Katira et al 1990[14] (2.1:1), Gupta & Rao 1995[15] (1.8:1) in their studies. The high incidences in males may be because males are more exposed to stress & strain, economic instability and also to occupational hazards.

Table 1: Age and sex wise distribution of aluminium phosphide poisoning cases

Age in years	Male		Female		Total	
	No.	%age	No.	%age	No.	%age
0-10	-	-	-	-	-	-
11-20	4	8%	5	10%	9	18%
21-30	12	24%	5	10%	17	34%
31-40	10	20%	5	10%	15	30%
41-50	5	10%	2	4%	7	14%
Above 50	2	4%	-	-	2	4%
Total	33	66%	17	34%	50	100%

Regarding social background of the victims, it was found that incidences were more in urban areas (54%) as compared to rural areas (46%) as shown in table 2. There was a male preponderance in the both rural (28%) and urban (38%) population. The study coincides with the study of Singh et al 1995.[16] (44.50% rural & 55.50% urban). Our study is in contradiction to the study by Sepaha et al 1995[17] & Aggarwal et al 1995[18] who reported higher incidences in rural areas (60% & 56.82% respectively). Incidences are increasing in urban areas because of increasing stress & strain of life, failure on educational front, lack of employment opportunities and moreover our hospital is situated in urban area.

Table 2: Distribution of cases according to social background (rural/ urban)

SOCIAL	Male		Female		Total	
BACKGROUND	No.	%age	No.	%age	No	%age
RURAL	14	28%	9	18%	23	46
URBAN	19	38%	8	16%	27	54
TOTAL	33	66%	17	34%	50	100

Table 3: marital status of aluminium phosphide poisoning CASES

Marital status	male	Female	total			
	No.	%age	No.	%age	No	%age
MARRIED	23	46%	11	22%	34	68%
UNMARRIED	10	20%	6	12%	16	32%
TOTAL	33	66%	17	34%	50	100%

Table 3 showed out of 50 cases, 34 (68%) were married and 16 (32%) unmarried. 46%of males & 22% of females were married and 20% of males and 12% of females were unmarried. The incidence of aluminium phosphide poisoning was more in married patients (68%). The common causes of poisoning among married women could be the suspected husband's fidelity, family feuds and prevailing evil of dowry system in our society. In men frustration due to financial stress, domestic troubles, matrimonial disharmony and excess freedom were the possible reasons.

As per the history given by the relatives or by the patients, 86% (60% males and 26% females) patients had suicidal intent and 14% cases (6%males & 8% females) had accidentally ingested the poison. No case of homicidal poisoning was reported. Male (60%) highly outnumbered females (26%) in suicidal cases (Table 4). Abder-Rahman et al 2000[19] in a study encountered 10 fatalities due to aluminium phosphide poisoning with in three months period and observed that the circumstances of death were accidental in six cases, suicidal in two and homicidal in two. As to the underlying cause of poisoning, self poisoning (suicidal intent) was the motive in 86.67 % (Katira et al 1990 [14]), 88% (Chugh et al 1991 [20]), 20 % (Abder- Rahman et al 2000 [19]) of patients. It is obvious from various studies that incidences of accidental / homicidal poisoning are rare because highly pungent smell and distressing taste make this compound easily recognizable. Homicidal administration could only be explained on the basis that the unsuspecting victim might be a child or very old. Easy availability, cheapness, highly lethal nature had made it a handy tool for suicidal purposes.

Table 4: Distribution of patients in relation to intention of poisoning

Nature of poisoning	male		Female		total	
	No.	%age	No.	%age	No.	%age
SUICIDAL	30	60%	13	26%	43	86%
ACCIDENTAL	03	06%	4	08%	07	14%
HOMICIDAL	—	—	—	—	—	—
TOTAL	33	66%	17	34%	50	100%

Table 5 showed out of 50 patients studied, the main male victims

Table 5: Occupational status of individuals of aluminium phosphide poisoning

Occupation	Male	Female	Total			
	No.	%Age	no.	%Age	no.	%Age
Agriculture	06	12%	—	—	6	12%
Business	10	20%	—	—	10	20%
Service	11	22%	1	2%	12	24%
Students	02	04%	2	4%	04	08%
Housewives	—	—	14	28%	14	28%
Labourer	3	06%	—	—	3	06%
Unemployed	1	02%	—	—	01	02%
TotaL	33	66%	17	34%	50	100%

were serviceman (22%) followed by businessman (20%). The possible reasons were the stress and strain of life followed by economic fluctuations in their lives. The main female victims were housewives (28%) which were same as reported by Khosla et al 1988[21]. The possible reasons could be family conflicts/ stress, prevailing evil of dowry system. 12% males were engaged in agriculture, 8% (4% males & 4% females) were students and 2% males were unemployed. In highly competitive society, with higher expectations and unrealistic goal setting by the students could easily lead to their failures and leading to suicide with this deadly poison. Only 2% females were doing service. 6%cases were from labour class again points to socioeconomic deprived class succumbing to its use

Majority of the patients presented with nausea/vomiting (96%),

Table 6 : Clinical presentation of aluminium phosphide poisoning cases

Clinical features	No. of Cases	
	No.	%age
GIT		
Nausea/vomiting	48	96%
Diarrhoea	18	36%
Bleeding from GIT	03	.6%
Retrosternal / Epigastic pain/ discomfort	45	90%
Respiratory		
Breathlessness	40	80%
Cough	24	48%
CVS		
Palpitation	46	92%
Giddiness	47	94%
Restlessness	47	94%

giddiness (94%), restlessness (94%), palpitation (92%), retrosternal / epigastric pain /discomfort (90%), breathlessness (80%), Cough (48%) and diarrhoea (36%) were common (table 6). Bleeding from GIT was observed only in 6% cases. Even other workers observed that among gastrointestinal symptoms nausea, vomiting, severe epigastric pain/ retrosternal pain/ discomfort, were more common. These effects have been attributed to the gastrointestinal irritation produced by liberation of phosphine in the stomach. In our study 80% patients experienced breathlessness which is in accordance with the study by Singh, Rastogi & Singh 1989 [22] . Majority of the patients were restless in our study as well as in study by Singh, Rastogi & Singh 1989[22] as shown in table 7.

As shown in table 8 the major physical signs observed were shock (94%), Cyanosis (56%) anaemia (54%). Jaundice was present in 02% cases. There was tachypnoea in 58% cases. Pulse rate was below 60 / min in 2% cases and unrecordable in 50% cases. B.P was unrecordable in 50% cases and systolic blood pressure was below or equal to 90 mm of Hg in 44% cases. Acute cardiovascular collapse with low to unrecordable B.P, fast steady pulse is the commonest mode of presentation.[23] Shock was invariably present in majority of cases

Table 8: General physical examination of aluminium phosphide poisoning cases

Clinical feature	No. of Cases	
	No.	%age
Anaemia	27	54%
Jaundice	01	02%
Cyanosis	28	56%
Respiratory rate		
More than 20/min	29	58%
Less than 20/min	21	42%
Pulse rate		
More than 90/min	16	32%
60-90/min	08	16%
less than 60/min	01	02%
Unrecordable	25	50%
Blood Pressure		
Unrecordable	25	50%
SystolicB.Pd"90mmHg	22	44%
SystolicB.P>90mmHg	03	06%
Shock	47	94%

Table 9: Mortality incidence in aluminium phosphide poisoning cases

No. of patients	Expired		Survived	
	No.	%age	No.	%age
50	38	76%	12	24%

signifying that the cardiovascular system bears the burnt earliest and is most severely affected.

In our study mortality rate was 76%. Other workers have reported mortality ranging from 32% to 85%. This high mortality rate difference could be because of variation in the number of patients studied & facilities available to treat the patient. Mortality depends not only on the dose of the poison but also on the freshness of the compound, duration and severity of shock and presence or absence of complication and their management. The high mortality is due to rapid absorption of phosphine throughout the body producing organ damage and lack of any specific antidote available.

Out of 50 cases 12 survived and 38 died. Majority of cases (63.16%)

Table 10: Correlation of time elapsed between intake of poison & death of the patient

Time elapsed between intake of poison and death	No. of patients	
	No.	%age
0-1 hrs	-	-
+1-6 hrs	24	63.16%
+6-12 hrs	12	31.58%
+12-24 hrs	01	2.63%
More than 24 hrs	01	2.63%
Total	38	100%

expired within 1-6 hours while 31.58% cases expired within 6-12 hours & 2.63% cases expired within 12-24 hours. Only 2.63% cases

Table 7:

Study	No. of patients developing (%)							
	GIT			Respiratory		CVS		
	Nausea/ vomiting	Diarrhoea	Retrosternal/ Epigastric Pain /discomfort	Breath	Cough lessness	Palpitation	Giddi ness	Restless ness
Wilson et al 1980 [1]	72% / 45%	21%	-	59%	52%	-	62%	-
Singh, Rastogi &Singh 1989[22]	100%	-	-	78%	-	78%	78%	Majority
Gupta, Malik &Sharma 1995 [7]	100%	12%	100%	-	-	12%	-	-
Present study 2006	96%	36%	90%	80%	48%	92%	94%	94%

survived even for more than 24 hours. The minimum survival period between ingestion of poison and death was observed to be 3 hours 5 minutes and maximum survival period was 27 hours 50 minutes and the mean survival time was 4.56 hours. Our study is almost consistent with the findings of Singh et al 1985[24] (5.30 hours) and Siwach et al 1998[25] (within 6 hours). While Singh et al 1995[16] reported 11.30 hours time interval between ingestion of aluminium phosphide and death of victim. This can be due to the fact that their study is retrospective study and better means of life savings were available in their institute. The rapidity with which aluminium phosphide causes death is a measure of its high lethality compounded by absence of specific antidote as evidenced from our study in which 24 out of 38 cases expired with in first 6 hours.

The mortality in aluminium phosphide poisoning is directly related to the dose of poison consumed as shown in table 11. Out of 48% patients who consumed one tablet 24% survived resulting into 50% mortality. The patients who had 2 tablets or more did not survive. Katira et al 1990[14] reported 63.3% overall mortality in ninety patients. The number of aluminium phosphide tablets ingested ranged from ¼ to 6.Siwach et al 1998 [25] studied that there was direct positive correlation between dose of aluminium phosphide consumed and mortality rate. Those who consumed 3 or more tablets hardly survived.

Table 11: Relationship of dose of aluminium phosphide to mortality rate

No. of tablets	No. of patients died		No. of patients survived	
	No.	%age	No.	%age
1	12	24%	12	24%
2	24	48%	—	—
3	—		—	—
4 and above	02	04%	—	—
Total	38	76%	12	24%

Conclusions

If the credits of fumigants include enhanced economic potential in terms of increased production of food then their debits have resulted in serious health implications to man and his environment. The present study seeks to highlight the upsurge in the frequency of aluminium phosphide poisoning in this part of India with male preponderance, involving the younger generation as main victim and more common in married, urbanites & servicemen. Aluminium phosphide is a systemic lethal protoplasmic poison. Heart is the first organ to be affected. Toxic manifestations involving GIT, lungs, liver are common. Mortality rate is more or less directly proportional to dose of the poison. Lack of antidote is the biggest lacuna in its management. Easy availability, cheapness, its lethal nature are contributing factors for making it an agent of choice for suicides. Depressed persons may soon be tempted to try this agent to end their lives. Carelessness in storage and therapeutic error could lead to accidental exposure. The fundamental dictum is 'Prevention is better than cure'. A degree of safety can be achieved if they are used in prescribed manner and in ways recommended for safe storage. Introduction of hard, perforated, seal proof containers can help in averting this problem. Public awareness of risks could also reduce the occurrence of aluminium phosphide poisoning to a greater extent. Immediate first aid can be life saving and should be carried out at PHC level without any delay.

References

1. Wilson R, Lovejoy FH, Jaeger RJ, Landrigan PL. Acute phosphine poisoning abroad a grain freighter. The Journal of American Medical Association 1980; 244 (2): 148-150.
2. Gargi J, Rai H, Gurmanjit R. Aluminium phosphide: is ban a solution? Romanian Journal of Legal Medicine 1997; 5(2): 195-197.
3. Zipf K, Arndt T, Heintz R. Clinical observation of a case of phostoxin poisoning (German). Arch Toxicol. 1967; 22(4): 209.
4 Kabra S G, Narayanan R. Aluminium Phosphide Worse than Bhopal. Lancet 1988; 11: 1333.
5. Chugh SN. Aluminium phosphide poisoning. Journal of Association of Physician of India 1992; 40 (6): 401-405.
6. Reddy KSN. Toxicology. The Essentials of Forensic Medicine and Toxicology. 25th ed. K. Suguna Devi, Malakpet; Hyderabad 2005: 418-422, 445-447.
7. Gupta MS, Malik A, Sharma VK. Cardiovascular manifestations in aluminium phosphide poisoning with special reference to echocardiographic changes. Journal of Association of Physician of India 1995; 43 (11): 773-780.
8. Wander GS, Arora S, Khurana SB. Acute pericarditis in aluminium phosphide poisoning. Journal of Association of Physician of India 1990; 38 (9): 675.
9. Chugh SN, SantRam, Mehta LK, Arora BB, Malhotra KC. Adult respiratory distress syndrome following aluminium phosphide ingestion report of 4 cases. Journal of Association of Physician of India 1989; 37(4): 271-272.
10. Singh SB, Singh VP, Gupta S, Gupta RM, Sunder S. Tropical myositis- A clinical immunological and histopathological study. Journal of Association of Physician of India 1989; 37(9): 561-563.
11. Gupta MS, Mehta L, Chugh SN, Malhotra KC. Aluminium phosphide poisoning- Two cases with rare presentation. Journal of Association of Physician of India 1990; 38(7): 509-510.
12. Chugh SN, Santram, Chugh K, Malhotra KC. Spot diagnosis of aluminium phosphide ingestion: An application of a simple test. Journal of Association of Physician of India 1989; 37 (3): 219 – 220.
13. Mital HS, Mehrotra TN, Dwivedi KK, Gera M. A study of aluminium phosphide poisoning with special reference to its spot diagnosis by silver nitrate test. Journal of Association of Physician of India 1992; 40(7): 473-474.
14. Katira R, Elhence GP, Mehrotra ML, Srivastava SSL, Mitra A, Agarwala R, Ram A. A study of aluminium phosphide poisoning with special reference to electrocardiographic changes . Journal of Association of Physician of India 1990; 38(7): 471-473.
15. Gupta RS, Rao HK. Clinical profile of aluminium phosphide poisoning as seen at M.C Patiala. Journal of Association of Physician of India 1995; 43 (12): 907.
16. Singh D, Dewan I, Vasishat RK, Tyagi S. Aluminium phosphide poisoning- autopsy and histopathological findings. Journal of Forensic Medicine and Toxicology 1995; xii (1&2): 16-20.
17. Sepaha GC, Bharani AK, Jain SM, Raman PG. Acute aluminium phosphide poisoning. Journal Indian Medical Association 1985; 83(11): 378-379.
18. Agarwal R, Barthwal SP, Nigam DK, Saxena S, Shukla SK, Shukla N, Shukla RD- Allahabad. Changing patterns of acute poisoning in eastern U.P- hospital based study. Journal of Association of Physician of India 1995; 43(12): 907.
19. Abder Rahman HA, Battah AH, Ibraheem YM, Shomaf MS, El-Batainch N. Aluminium phosphide fatalities, new local experience. Med. Sci. Law 2000 Apr; 40(2): 164-168.
20. Chugh SN, Chugh K, Santram, Malhotra KC Electrocardiographic abnormalities in aluminium phosphide poisoning with special reference to its incidence, pathogenesis, mortality and histopathology. Journal Indian Medical Association 1991; 88(2): 32-35.
21. Khosla SN, Nityanand, Kumar P, Trehan V. Muscle involvement in aluminium phosphide poisoning. Journal of Association of Physician of India 1988; 36(4): 289-290.
22. Singh RB, Rastogi SS, Singh DS. Cardiovascular manifestations of aluminium phosphide poisoning intoxication. Journal of Association of Physician of India 1989; 37(9):590-591.

23. Sainani GS, Anand MP, Chugh MP et al. Toxicology. API Textbook of medicine 5th ed. Association of Physician of India; Bombay1992: 1394-1395.
24. Singh S, Dilawari JB, Vashisht R, Malhotra HS, Sharma BK. Aluminium phosphide ingestion. British Medical Journal1985; 290: 1110-1111.
25. Siwach SB, Singh H, Jagdish, Katyal VK, Bhardwaj G. Cardiac arrhythmias in aluminium phosphide poisoning studied by on continuous holter and cardioscopic monitoring. Journal of Association of Physician of India 1998; 46(7): 598-601.

Myocardial infarction resulting in head injuries- A medico legal point of view

Putul Mahanta

*Assistant Professor of Forensic Medicine and Toxicology, Gavhati Medical College, Assam.

Abstract

An alleged road traffic accident (RTA) victim of 57-year-old male was brought for a medico legal autopsy. A contusion on scalp with right temporoparietal subdural haemorrhage and acute myocardial infarction (AMI) with cardiac tamponade was found. Earlier he had a 18 hours hospital stay and received conservative treatment. His CT scan report also revealed right temporoparietal subdural haemorrhage with midline shift. ECG showed ST elevation. This case demonstrates that with each type of injury, the focus should always to be given to the entire vital organs like heart to rule out the natural causes or diseases of death for the sake of justice.

Keywords

Acute myocardial infarction, cardiac tamponade, head injury, subdural haemorrhage.

Introduction

Myocardial infarction is due to extended ischemia and if left untreated, leads to "ultimate cell necrosis"[1]. Acute myocardial infarction (AMI) may culminate in sudden death by ventricular fibrillation, cardiogenic shock, and cardiac rupture with lots of other complications[2]. AMI is one of the most common diagnoses in hospitalized patients in industrialized countries[3]. In up to one-half of cases of ST- segment elevation myocardial infarction (STEMI), a precipitating factor appears to be present beforehand, such as vigorous physical exercise, emotional stress, or a medical or surgical illness[3]. Here is a case of acute STEMI, discovered upon investigation of a patient who presented with a history of head injury following a stressful long driving. This presentation is unique as it describes a dead person with AMI with cardiac tamponade presenting as head injury with tremendous medico legal importance.

Case report

A 57-year-old male with known cardiac risk factors of hypertension, diabetes mellitus and being alcoholic was driving for a long distance of his company's car. Suddenly he felt retrosternal pain and tried to stop of his car but lost his control and his car just struck hard on a tree and sustained head injury. He had a brief loss of consciousness. Earlier, on the same day, he had suffered from retrosternal chest pain. In an unconscious state he was brought by an ambulance to the emergency department of Gauhati Medical College and Hospital. His CT scan report revealed right temporoparietal subdural haemorrhage with midline shift. ECG showed ST elevation. He was treated conservatively, but succumbed to death within 24 hours. As the case was presented with head injuries, thought to be the RTA one, autopsy was requested.

Autopsy findings

External findings:

- The face of the case was congested.
- One contusion of size [6.5x 4.5] cm over right fronto-temporoparietal region of the scalp found with intact skull (Figure-1).
- No external injuries over the chest wall.

Internal findings:

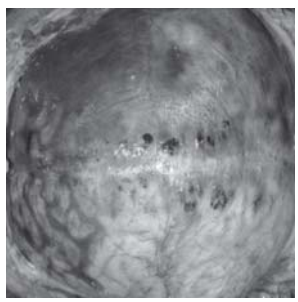
Figure 1: Contusion of scalp of the case over right fronto-temporoparietal region.

- General findings
- Normal chest wall with congested lungs.
- All other viscera were congested.
- Visceras preserved for chemical analysis gave negative reports for drug or alcohol intoxication.

A. Finding of brain:

- Congested brain with membrane.
- Sub-dural haemorrhage over right temporoparietal region of size [5X 4] cm (Figure-2).
- *On cut section*: Brain tissues found edematous with signs of raised intracranial pressure with midline shifting.

Figure 2: sub-dural haemorrhage over right temparoparital region.

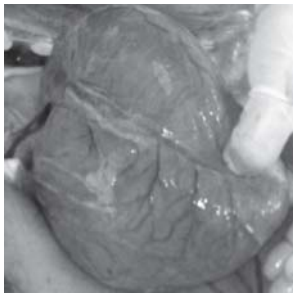


B. Cardiac findings:

- Pericardial cavity contains about 500ml of slightly hemorrhagic fluid with congestion and creamy white thickening of pericardium suggestive of pericarditis (Figure-3).
- Weight of the heart was 1200 gm.
- Cut section showed thicken left ventricular wall with focal transmural congested anterior myocardial wall.
- Coronaries were thicken and of cord like consistency.
- Multiple pale white areas of focal infarction were seen over left ventricular wall and at places over myocardium (Figure-4).
- Multiple petechial hemorrhages over myocardium at places.

Figure-4: Signs of atherosclerosis and AMI *(pale infarct area).*

Figure 3: Pericardial cavity showing *Cardiac Tamponade.*



Discussion

Cardiac injuries are the most commonly overlooked injuries in patients who die from trauma[4]. The presentation of AMI in the setting of injury is atypical on three counts. First, the main site of the trauma was the head with scalp contusion with subdural haemorrhage and not the chest. This case was most specific in the sense that the blows were to his head alone although there can be no doubt that a significant degree of force must have been transmitted up across the diaphragm as well as in an antero-posterior direction across the abdomen as he was the driver. Secondly there was an unusually large amount of hemorrhagic fluid (500 ml) present in the pericardium without evidence of cardiac rupture. Thirdly the case has evidences of AMI. Coronary heart disease (CHD) is one of the leading causes of death worldwide; 3.8 million men and 3.4 million women die each year due to CHD[5]. Methods for thorough history-gatherering to identify preexisting conditions, for early hemodynamic monitoring and intervention for AMI in the setting of trauma should be defined[6]. The relationship between AMI and trauma has been investigated by many researchers. Trauma has been suggested, in case series, as one of the non-atherosclerotic mechanisms leading to AMI, the leading cause of death in the USA. AMI following non-penetrating injury has been shown to carry significant morbidity and mortality. Direct trauma to the heart following blunt chest injury, was observed to carry the greatest risk for AMI. Abdominal or pelvic trauma also increased the risk for AMI[7]. Zajarias et al. reported a case of AMI following a blunt chest trauma from automobile airbag deployment[8]. Rapid accumulation of as little as 250-350 cc of blood can cause cardiac tamponade, i.e., progressive limitation of the pumping action of the heart and eventual cardiac arrest. Rupture of heart is more common in young individuals. In the old, blunt injuries can precipitate myocardial infarction. A diseased heart may rupture even with trivial trauma[9]. Tamponade can occur as a result of any type of pericarditis[10]. In this reported case, there were no signs of chest injury, though there may be possibilities of having blunt chest trauma. Therefore, most likely the patient presented here had developed AMI with complication of cardiac tamponade (though there is no obvious myocardial rupture) which may resulted as dizziness leading to the accident causing head injury ultimately succumbed to death.

Legal implications

At common law, a myocardial infarction is generally a disease, but may sometimes be an injury. This has implications for no-fault insurance schemes such as worker's compensation. A heart attack is generally not covered; however, it may be a work-related injury if results, for example, from unusual emotional stress or unusual exertion. Additionally, in some jurisdictions, heart attacks suffered by persons in particular occupations such as police officers may be classified as line-of-duty injuries by statute or policy. In some countries or states, a person who has suffered from a myocardial infarction may be prevented from participating in activity that puts other people live at risk, for example driving a car, taxi or airplane[11].

Conclusion

In conclusion, all cases of trauma should be assessed thoroughly to identify pre-existing conditions, such as AMI as in this case, for early monitoring and intervention. This case also indicates the needs of further study for better understanding the relationship between trauma and AMI. Legal provision should also be there to include sudden death for insurance coverage etc as it happens unexpectedly like that of accident.

Consent

Informed written consent was obtained from legal guardian for uses of the case materials for research purposes and publication of findings.

Competing interest

Have no competing interests.

Acknowledgements

Thanks to my wife MANMI, my kids Jacinth and Adriana amongst many others who have inspired and helped me a lot in different way.

References

1. Opie LH. In: The Heart: Physiology from Cell to Circulation. 3rd ed. Philadelphia, Pa: Lippincott; 1998.
2. Hasan Ekim, Mustafa Tuncer and Halil Basel: In: Repair of ventricle free wall rupture acute myocardial rupture after acute myocardial infarction: a case report. Cases Journal 2009, 2:9099dol:10.1186/1757-1626-2-9099.
3. Antman EM Braunwald E. ST-segment elevation myocardial infarction. In: Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL (Eds). Harrison's Principles of Internal Medicine. 16th ed. McGraw Hill. New York St Louis San Francisco, 2005; Volume II: 1448-1459.
4. Liedtke AJ, Demuth WEJ. In: Nonpenetrating cardiac injuries: A collective review. Am Heart J 1973; 86: 687–97
5. JLY Liu, N Maniadakis, AGray, and Mrayner. In: The economic burden of coronary heart disease in the UK Heart, Dec 2002; 88: 597-603.
6 Moosikasuwan JB. Thomas JM. Buchman TG. In: Myocardial infarction as a complication of injury. J Am Coll Surg. 2000; 190 (6):665-70.
7. Ismailov RM, Ness RB, Weiss HB, Lawrence BA, Miller TR. In: Trauma associated with acute myocardial infarction in a multi-state hospitalized population. Int J Cardiol. 2005; 105(2):141-6.
8. Zajarias A, Thanigaraj S, Taniunchi M. In: Acute coronary occlusion and myocardial infarction secondary to blunt chest trauma from an automobile airbag deployment. J Invasive Cardiol 2006; 18(1):E71-3.
9. Pillay VV. In: Textbook of Forensic Medicine and Toxicology. 14th ed. 2007; 186
10. Parvez N, Carpenter JL. In: Cardiac tamponade in Still disease: a review of the literature. South Med J. Aug 2009; 102(8):832-7.
11. Internet source: http://schools- wikipedia.org/wp/m/ myocardial_infarction.htm.

Fatal cardiogenic shock after electroconvulsive therapy: A case report

Manish Shrigiriwar*, Rajesh Bardale**

*Associate Professor, Dept. of Forensic Medicine, Indira Gandhi Govt. Medical College, Nagpur, **Lecturer, Dept. of Forensic Medicine, Govt. Medical College, Nagpur

Abstract

Modified electroconvulsive therapy is relatively safe procedure and on most of the instances, morbidity and mortality is related to cardiovascular complications. We report a case of fatal cardiogenic shock after electroconvulsive therapy.

Keywords

cardiogenic shock, electroconvulsive therapy, death

Introduction

Modified electroconvulsive therapy (ECT) is accepted as relatively safe procedure (1). On most of the instances, morbidity and mortality is related to cardiovascular complications and includes dysarrythmias, myocardial infarction, cardiac arrest, hypertensive response, transient myocardial dysfunction and circulatory collapse (2,3). Similarly pulmonary edema and cardiogenic shock is also recognized as an occasional complication of ECT but fatality has not been reported (4,5). Herein we are reporting a case of fatal cardiogenic shock after ECT.

Case report

A 53-year-old female patient was admitted to Regional Mental Hospital, Nagpur as a case of paranoid schizophrenia. No past history of ischemic heart disease, hypertension or diabetes mellitus was noted. Her medication history included administration of resperidone, olanzapine, haloperidol, lorazepam and carbamazepine. She was advised biweekly modified ECT and had received 10 modified ECT uneventfully. For 11th ECT, patient was taken on ECT table with stable vitals. Anesthesia was induced with125 mg sodium pentathol and ECT was instituted. After ECT, patient was sifted to recovery room. In recovery room, within minutes, patient had experienced difficulty in breathing. Anesthetist on duty attended the patient who was gasping and started resuscitative measures but could not review patient.

A forensic autopsy, conducted at Indira Gandhi Govt. Medical College & Hospital Nagpur, revealed an obese lady, 166 cm in height and 100 kg in weight. External examination revealed no injuries except for those from clinical procedures and resuscitative measures. There was a blackish area present on right side of head corresponding for electrode. Microscopic examination of skin showed congestion, edema and mild chronic inflammatory reaction in dermis. Internal examination revealed congested and edematous brain. The right and left lungs weighed 900 gm and 850 gm respectively and exhibited severe edema and congestion. Heart weighed 300 gm with eccentric coronary artery atherosclerosis with patent lumen. There was no evidence of an acute or remote myocardial infarction on gross and microscopic examination. Liver was enlarged. Uterus showed two subserosal fibroid. Microscopic examination of brain and lungs showed congestion and edema; liver showed congestion, edema and fatty changes. Spleen and pancreas were congested. Kidney showed congestion, edema and cloudy changes. Adrenal showed congestion and edema. Toxicological screening was negative.

Address for correspondence:
Dr Rajesh Bardale
Lecturer Dept. of Forensic Medicine Govt. Medical College & Hospital Nagpur – 440 003
Email: bardalerv@yahoo.co.in

Discussion

Cardiogenic shock is a state of inadequate tissue perfusion due to cardiac dysfunction (6). Development of cardiogenic shock after ECT has been reported and is attributed to myocardial stunning combined with neurogenic causes (4). Myocardial stunning is a prolonged but reversible myocardial contractile dysfunction that develops after ECT (3).

The seizures induced by ECT leads to transient, yet intense, stimulation of autonomic nervous system. Initially there is marked parasympathetic discharge with resultant bradycardia followed by sympathetic surge associated with marked increase in arterial blood pressure and heart rate as well as plasma catecholamine, vasopressin and adrenocorticotrophic hormone levels. Norepinephrine and epinephrine levels increase three and five-fold, respectively, over pre-ECT levels. These hemodynamic and humoral events produce a marked increase in myocardial oxygen demand that may result in myocardial stunning and may precipitate myocardial ischemia or even infarction in susceptible patients with coronary artery disease (1,3). The hemodynamic response after ECT is often accompanied by new left ventricular regional wall motion abnormalities and impaired systolic performance of left ventricle (7,8). In the earlier reports of neurogenic pulmonary edema and cardiogenic shock after ECT, a normal to minor non-specific and T-wave changes were observed in electrocardiogram with no evidence of myocardial infarction. Serial measurements of cardiac enzymes were normal. However, 2-D echocardiogram revealed mild dilatation of left ventricle with severe global hypokinesia and ejection fraction less than 20% thus exhibiting deranged myocardial function with myocardial stunning. Angiography revealed normal coronary arteries (4).

The present case has some similarities to the previously published case report of cardiogenic shock developing immediately after ECT (4). In the present case, there is no evidence of fresh or old myocardial ischemic lesion. Autopsy findings suggest cardiogenic shock as a cause of death. The cardiogenic shock is thought to be due to myocardial stunning that develops after ECT. Therefore, the clinicians should be aware that patients undergoing ECT might be at risk for hemodynamic instability with stunned myocardium-induced cardiogenic shock and at times, such shock may prove fatal.

References

1. O'Connor CJ, Rothenberg DM, Soble JS, Macioch JE, McCarthy R, Neumann A, Tuman KJ. The effect of esmolol pretreatment on the incidence of regional wall motion abnormalities during electroconvulsive therapy. Anesth Analg 1996;82:143-7.
2. Eitzman DT, Bach DS, Rubenfire M. Management of myocardial stunning associated with electroconvulsive therapy guided by hyperventilation echocardiography. Am Heart J 1994;127:928-9.
3. Zhu WX, Olson DE, Karon BL, Tajik AJ. Myocardial stunning after electroconvulsive therapy. Ann Intern Med 1992;117:914-5.
4. Ring BS, Parnass SM, Shulman RB, Phelan J, Khan SA. Cardiogenic shock after electroconvulsive therapy. Anesthesiology

1996;84:1511-3.
5. Buisseret P. Acute pulmonary oedema following grand mal epilepsy and as a complication of electric shock therapy. Br J Dis Chest 1982;76:194-5.
6. Hollenberg SM, Kavinsky CJ, Parrillo JE. Cardiogenic shock. Ann Intern Med 1999;131:47-59.
7. Messina AG, Paranicas M, Katz B, Markowitz J, Yao FS, Devereux RB. Effect of electroconvulsive therapy on the electrocardiogram and echocardiogram. Anesth Analg 1992;75:511-4.
8. Kadoi Y, Saito S, Seki S, Ide M, Morita T, Goto F. Electroconvulsive therapy impairs systolic performance of the left heart. Can J Anaesth 2001;48:405-8.

A case report of autohysterectomy

Renju Raveendran*, Anand T.P**

*Assistant Professor, Department of Forensic Medicine, T.D.Medical College, Alappuzha 688005, Kerala, **Department of Forensic Medicine, T.D. Medical College, Alappuzha 688005, Kerala

Abstract

Self inflicted incised wounds are common on various parts of the body especially with suicidal intention on the front of wrist, neck, chest etc. But incised wounds made on a prolapsed uterus as a desperate attempt to perhaps undertake a self executed surgical excision' does speak volumes on the state of mind of the victim. More so from the medico legal point of view since it brings to light the innumerable possibilities of deliberate self harm sometimes bordering on the bizarre, of methods that elude conventional forensic thinking.

Keywords

Uterovaginal prolapse, Incised wound, enterocoele, vascular anastomosis.

Introduction

This case report illustrates a unique situation where an elderly women inflicted multiple incised wounds on several sites of a utero-vaginal prolapse. Self inflicted superficial incised wounds are superficial, multiple, grouped together parallel to each other and placed over approachable parts of the body, more commonly on the anterior aspect of forearms, inner aspect of thigh, front of lower abdomen and anterior and lateral aspect of upper arm[1]. Suicidal wounds are always present over the front or accessible part of the body e.g. front of the neck, flexor surface of the wrist or elbow joint, groin and occasionally on the chest or back of legs, over radial border of forearm, abdomen or vagina especially when done by a determined suicide or lunatic[2]. The uniqueness of the case is evident from the foregoing explanation in that the site chosen is unusual and it belies a suicidal attempt. The case is being reported here for its extraordinary nature. The uterus and vagina have a vascular ramification received from a host of prominent blood vessels forming a highly vascular structure , all the more vulnerable to bleed profusely when cut. Death in this particular case was obvious the result of such exsanguination.

Case report

' A dead body of a 91year old elderly female was brought for postmortem examination to the mortuary of T.D. Medical College Hospital Alappuzha. As per the police inquest and requisition form for postmortem examination, the women had died due to bleeding resulting from cuts sustained 'somehow' on the protruding part of uterus. She was taken to the hospital where she succumbed to the injuries sustained.

Postmortem findings

External

Poorly nourished ,elderly female of height 136cm and weight 38kgs. Cataract present in both eyes. Whole of the uterus and cervix with prolapsed vaginal wall seen protruding through a dilated vaginal orifice. The prolapsed vaginal wall was pale, thickened and oedematous . Lips and nails were pale.

The antemortem injuries present on the body are as follows

1) Four sutured incised wounds of sizes varying from 3cm long and 0.5cm deep to 5cm long and 1cm deep seen arranged horizontally as well as obliquely as a cluster on the front aspect of the prolapse just below the vaginal outlet.
2) Another incised wound 6X4cm placed on the back aspect of the prolapsed vaginal wall which was seen communicating with the peritoneal cavity (enterocoel) through which the greater omentum ,fallopian tube and right and left ovary were seen protruding out. Multiple superficial linear incised wounds(3cm and 5cm in length) were seen arranged in a cluster parallel and horizontal to one another adjacent to the main wound
3) Sutured incised wound 4cm long and 1cm deep vertically placed on the right side of the prolapsed vaginal wall.
4) Uterine and vaginal blood vessels underlying these wounds were cleanly cut.

All internal organs were pale. The uterus and cervix were atrophic.

Fig. 1:

Fig. 2:

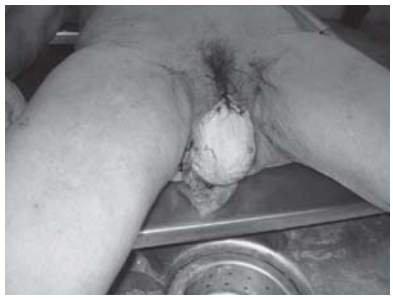


Fig. 3:

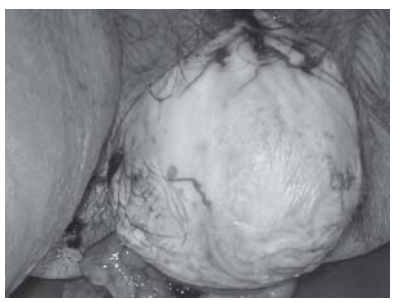
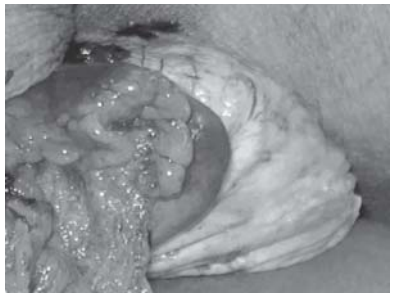


Fig. 1:



Stomach contents did not reveal any unusual smell or colour

Opinion as to cause of death was furnished as due to incised wounds involving uterine and vaginal blood vessels of the prolapsed uterus.

Discussion

Most important aetiologal factor in prolapse is atonicity and asthenia that follows menopause. It is mostly seen in post menopausal and multiparous women due to oestrogen deficiency. Another important aetiological factor is birth injury where the over stretching of pelvic floor muscles cause atonicity . Pudendal nerve injury during child birth resulting in prolapse is reversible in 60% of cases.[3]

A reason for high incidence of prolapse in India is that circumstances force poor women to resume their heavy work soon after delivery without any rest or pelvic floor exercise.[3]

Clinically genital prolapse can be broadly classified into vaginal prolapse and uterine prolapse, while vaginal prolapse can occur independently without uterine descent, uterine prolapse is usually associated with variable degree of vaginal descent [4]. Depending on the site, prolapse of vaginal wall is classified into:

A) Anterior vaginal wall prolapse, cystocoele and urethrocoele when it involves upper two thirds and lower one third of vaginal wall respectively.

B) Posterior Vaginal Wall prolapse- enterocoele (pouch of Douglas hernia) and rectocoele, when it involves upper one thirds and lower two thirds respectively.[3,4]

Depending on the degree of descent it can be classified as

First degree –Descent of cervix into vagina

Second degree- Descent of cervix upto introitus

Third degree – Degree of cervix outside introitus

Procidentia- All of the uterus is outside the introitus (as in the present case)

Arterial supply of vagina[5]

Branches of

(1) Uterine artery

(2) Vaginal artery- a branch of internal iliac artery

(3) Internal pudendal artery

(4) Middle rectal artery

The uterine and vaginal arteries form a longitudinal anastomosis along the anterior and posterior vaginal wall[5].

Arterial supply of uterus

The main arterial supply to the whole of uterus is by way of two uterine branches of internal iliac artery. These vessels anastomose superficially with the ovarian and inferiorly with the vaginal arteries. The two uterine arteries anastomose extensively with each other across the midline by small branches. Each uterine artery produces large number of branches which pass at once into the uterine wall dividing there into groups of anterior and posterior arcuate arteries[6].

Nerve supply

Lower third of vagina is pain sensitive and supplied by pudendal nerve through inferior rectal and posterior labial branches of perineal nerve. Upper two third is pain insensitive and are supplied by sympathetic L1,L2 and parasympathetic segments S2,S3 nerves derived from inferior hypogastric and uterovaginal plexus[7].

Conclusion

Ninety one years of age is too advanced a period in life whereupon any average human being can be expected to possess a normal state of mind fully balanced, sound in memory and rational in thought. And to be burdened with a huge mass of tissue constantly encumbering on one's freedom and well being, only goes to aggravate the shortfalls of dotage.

The nature and multiplicity of the wounds reveal a frantic attempt by the victim to get rid of a protruding body part that has become an annoyance, but at the same time obviously unaware of its consequence in that it could cut blood vessels though painless and bleed dangerously leading on to death. So putting everything together it can be concluded that death in this case was accidental in nature and the actions involved undoubtedly strange and inconceivable to an ordinary mind.

References

1. Principles of Forensic medicine including toxicology-Aburba Nandy third edition page363.
2. Forensic Medicine and Toxicology-J.B.Mukherjee page 360, 3rd edition.
3. Text book of Gynecology- Shaw, 13th edition.
4. Text book of Gynecology D.C.Dutta 3rd edition- page 193-194
5. Essentials of Human Anatomy-A.K Datta 6. Grays Anatomy page 1359 -60, 35th edition.
6. Human Anatomy- B.D.Chaurasia 4th edition page 365.

Embalming of cadavers by gravitational method

Rohit C. Zariwala*, Dimple S. Patel**

*Head & Associate Professor, Dept. of Forensic Medicine, **Associate Professor, Dept. of Anatomy, AMC MET Medical College, Maninagar, Ahmadabad.

Human Anatomy is the branch of science which studies the structure of human body by dissecting so as to understand the structural parts. Cadaver dissection forms the basic Of understanding human body in the field of medicine, dentistry and all paramedical sciences.

The artificial method of mummifying or embalming dead bodies was known to the ancient Egyptians, and the specimens of their mummies are to be found in the British Museum of London in a very well preserved condition, after thousands of years. (01). Cadaver dissection for anatomical study is mentioned in the Sushruta Samhita. (02)

Cadaver preservation by arterial injection was introduced by Wepner (1620-1695) and in England by William hunter 916818-1783).With the discovery of formaldehyde in 1863, the process of the preservation of the human cadaver has undergone great advancement . The procedure of dissecting a human body is tedious and time consuming. However it can be done only on a well preserved body by proper embalming so that the body is well preserved without dehydration and decomposition or the dissected parts may be displayed in the museum for teaching and learning.

Embalming a word for old English phrase to apply balm is derived from the Latin with em-encapsulate and balming or balsam – any aromatic resins produced by certain trees of the mint family in most modern cultures is an art and science of temporarily preserving human remains to forestall decomposition and make it suitable for display. The three goals for embalming are thus; preservation, sanitization and presentation of a dead body. Embalming has a very long and cross cultural history with many cultures giving it a greater religious meaning. Now the position of being embalmed is being crowded by numerous world famous personalities. An embalmer is someone who has been specifically trained in art and science of embalming. Embalming chemicals are variety of preservatives, sanitizing and disinfecting agent and additives used to temporarily prevent decomposition and restore natural appearance of the body. A mixture of these chemicals is known as embalming fluids.

Arterial fluid

The embalming fluids injected into the arteries for preservation of cadavers is called arterial fluid consisting of preservatives, antimicrobial agents, buffers, wetting agents moisture retaining agents, perfumes and diluents. The pH of the living body is maintained at 7.4. Formaldehyde combines with the normal and abnormal products of the dead tissues of the body. The amount of gas required to saturate the tissues is called the formaldehyde demand of the body. A diluted embalming solution of larger volume is required so that uniform distribution of formaldehyde throughout the body. If a small volume is injected the embalming solution spread superficially because of low resistance leaving the deeper tissues unaffected. After a day or two the deeper tissues soften and delay causing embarrassment to the embalmer. Arterial injection is forcing of fluid into an artery to reach the tissues through the arterioles and capillaries. In embalming the diffusion occurs at the capillary level so that tissues and the cells are well preserved. It is therefore the capillaries which form the main basis circulation. The total surface capillary exchange in man weighing 70 kgs is approximately 600 sq.mm. With about 40-50 million capillary of size 8x700 microns. There are several other factors besides the capillary resistance and chemical composition of the solution which determine the flow and diffusion of fluid into the tissues. These are the injection pressure osmosis, dialysis (diffusion) and gravity.

Gravity

In post embalming phase the fluid filled in vessels will gradually subside and disappear into the tissue by the combined action of osmosis, diffusion (dialysis) and by gravity. The dependants' part gets sodden and swollen or there may be even oozing of the fluids through the skin if the position of the subject is not altered for some time.

Advantages

-It is simplest, slowest of the injection methods.

-There is constant flow of embalming fluid as long as there is fluid in the percolator.

-Economical

-No energy required. Operation is quite and does not require any supervision.

Disadvantages

-Pressure range is limited.

-Refilling is tedious

-Prolongs the time of embalming.

-Distribution is uneven.

Aims of embalming are to achieve the following in a cost effective and safe manner

01) Prevention of growth of fungus.
02) Minimizing risk or fear of infection on contact with dead body.
03) Preserving a life like appearance with natural color.
04) Preventing putrefaction/desiccation.
05) Preventing contamination from insects and maggots.
06) Long term preservation of organs with minimal shrinkage/ distortion.
07) Preventing over hardening, maintain flexibility of internal organs.
08) Reduction of environmental chemical hazards(Formaldehyde)

The ideal time for embalming is as soon as possible or within a period of one week after death with arterial embalming by gravitation method. This is simplest and safest with gravity bottle placed at 3-4 feet above the height of embalming table providing a pressure of 0.6 kg/sq.cm. The maximum local temperature ranged between 19 and 36 degrees centigrade and the humidity between 30 and 100%. The body is placed on the embalming table with clothing removed. It is washed, positioned in the desired state with the upper limbs by side, the head slightly elevated by 10 cms and placed on a head rest in a slightly extended position. Nasal and oral cavities are are disinfected by soaking cotton with arterial solution and plugging the orifices. A skin incision of 5 cms length is made at the level of the upper border of thyroid cartilage along the anterior border of sterno-cleido-mastoid muscle. The muscle is pushed laterally to expose the carotid sheath. The common carotid artery is dissected and elevated to the surface. A vertical incision is given on the anterior wall of the artery and cannula is inserted toward the head region and tied with thread. The screw cap is opened and one liter of mixture is allowed to enter the head and face region. The cannula is withdrawn slowly and ligature is tied tightly. The cannula is put again facing downward and the remaining solution is allowed to

pass through it. The cannula is removed, the artery ligated and the incision sutured with muscle cutting needle and thread. These embalmed cadavers are then stored in tanks contain 10% formalin diluted in water. It is ideal that these cadavers are removed and washed thoroughly before the dissection room should be well ventilated with exhaust fan to eliminate the formalin vapor.

References

01) Modi's Medical Jurisprudences and Toxicology, 22nd Edition.
02) Embalming, Principles and legal aspects, 1st Edition.

Profile of medicolegal cases in northan tribal region of Andhra Pradesh

Ajay Khade*, Rajinsh Borkar**, Mohammed Shakeel Mohammed Bashir

*M.D Pharmacology, Associate Professor & Head in Pharmacology Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad, A.P., India, **M.D SPM, Assistant Professor in Community Medicine Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad, A.P., India, ***M.D Pharmacology Assistant Professor in Pharmacology Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad, A.P., India

Abstract

Objectives

A retrospective study of pattern of medico-legal cases (MLCs) in Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad, a medical college located in northern tribal area of Andhrapradesh was carried out to provide feedback to clinician, forensic experts, social workers and legal experts of Adilabad.

Materials and methods

Data was collected from records of patients admitted in RIMS Adilabad, from the period of 1st October 2008 to 30th September 2009. All the MLCs of one year period were studied.

Results

In all admitted cases 10.39% were MLCs. Physical injury observed in 78.25% while there was no injury in remaining 21.75% cases. Major reason behind MLCs were – RTAs (road traffic accidents) 30.18%, fall due to various reasons (17.34%), suicide/attempted suicide cases (13.82%), assault (11.22%) and animal attack (9.87%).

Conclusion

Our study concludes that unusual high number of suicidal tendencies and RTAs are indicators of tremendous impact of developmental activities in the region without substantial improvement at the socioeconomic front.

Keywords

MLC, Road traffic accidents, Fall, Suicide, Assault, Animal attack

Introduction

India is 2nd most populous country in the world with population of 1028 million as per 2001 census[1]. Growing population is responsible for enormous stress on existing environment, infrastructure and health care facility leading to change in lifestyle. This changed in lifestyle is not only affected urban Indian population but also the rural and tribal population of India leading to various environmental, socio-political and health related problems[1]. Change in socioeconomic and cultural habits created different kind of problems in rural and tribal regions[2] which are responsible for many health related crimes or variety of medico-legal cases (MLCs).

Adilabad a northern tribal district of Andhrapradesh is a typical rural cum tribal region with 73.52% tribal and rural population[3]. There is dearth of knowledge about health related crimes of this tribal region. Hence the present study was planned in Rajiv Gandhi Institute of Medical Sciences (RIMS) Adilabad to know the status of various kind of health related problems associated with legal procedures by evaluating the medico-legal cases. Further the study was carried out to evaluate the probable reasons behind these medico-legal cases.

Address for correspondence:
Dr. Mohammed Shakeel Mohammed Bashir
Tajmansion, Teachers colony, Thakur plot, Badatajbag, Nagpur, Maharashtra, India.
Email: drmsmbashir76@rediffmail.com

Materials and methods

The present study was a retrospective study approved by the institutional authorities of RIMS Adilabad. Data was collected from the medical record section of RIMS Adilabad. Records of only those patients who were admitted in RIMS Adilabad were analysed. Total 33370 cases were admitted during the period of October 2008 to September 2009. From the all admitted cases, record of MLCs was scrutinized and all the total 3466 medico-legal cases were included in the study. Parameters included were road traffic accidents (RTAs), fall, poisoning, assault, animal attack and MLCs of other categories. Data was analysed by using Microsoft excel 2007 programme.

Results

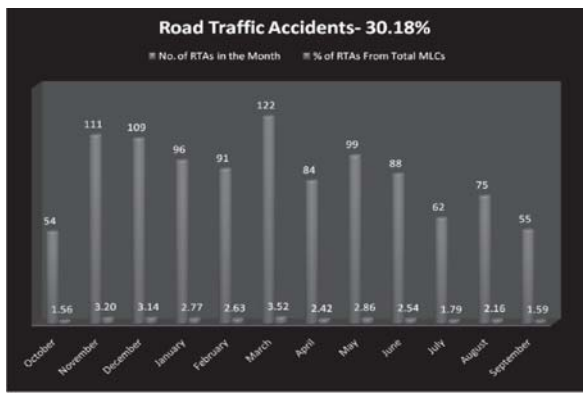
Table.1 Indicates pattern of MLCs. Physical injury was observed in 2712 MLCs while there was no visible physical injury in remaining 754 MLCs. Road traffic accidents (RTAs) were the leading cause of MLCs with 1046 cases while fall due to various reasons was the 2nd most important reason with 601 cases. Other significant MLCs were poisoning-541, assault-389 and animal attack-342 cases.

Table 1: MLCs Admitted during October 2008 to September 2009

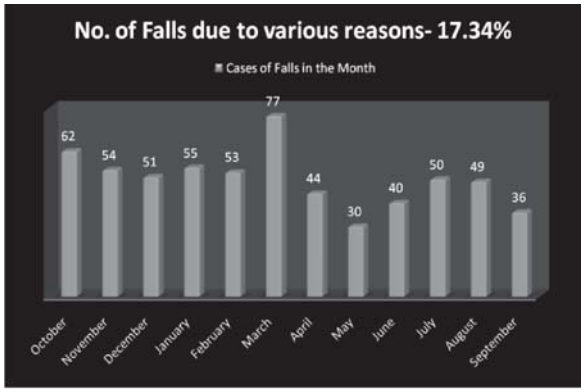
MLCs with visible physical injury		MLCs without visible physical injury	
RTAs	30.18%	Poisoning	15.61%
Fall	17.34%	Prisoners	5.80%
Assault	11.22%	Rape	0.35%
Animal attack	9.87%		
History not available	5.28%		
Burn	3.89%		
Other injuries	0.46%		
Total MLCs	78.25%	Total MLCs	21.75%

RTAs were less common during July to October season (Bar diagram1) while there was no seasonal variation in pattern of fall but peak was observed during the month of March (Bar diagram 2).

Bar diagram1:



Bar diagram 2 :



In poisoning category of MLCs, leading cause was insecticide intake followed by alcoholic poisoning and known/unknown tablet intake (Table 2). Consumption of insecticide (Figure 1) was started rising from the month of July and peak was in the month of September. It was also high during previous year's month of October and the rate of intake was almost stable during the season of November to June.

Trend of assault cases in the region was not uniform and distribution was vague not limited for any season (Table 3) although peak was observed in the month of March. While in the category of animal attack related MLCs (Table 4) majority of cases were due to wild animal attack (314 out of total 342 animal attack cases) and snake bite was the predominant cause with 228 cases. Snake bite cases were 6.58% of all MLCs. Incidence of bite was most common during the season of July to October (Figure 2) with peak cases observed during the month of July.

In other categories of MLCs, prisoners constituted predominated group with 201 cases while history was not available in 183 cases of injuries. Burn constituted another important group with 135 cases while least MLCs were of miscellaneous group with 16 cases and rape with 12 cases.

Discussion

Medico legal case (MLC) is a case of injury or ailment, etc., in which investigations by the law-enforcing agencies are essential to fix the responsibility regarding the causation of the said injury or ailment[4]. Generally trend of MLCs are different in urban and rural regions of India. Since Adilabad is a predominant tribal cum rural area [3] so it is quite expected that some unusual MLCs will be there that's why we have included typical tribal type of parameters like animal attacks etc.

RTAs was the leading cause of MLCs. National highway No.7 (NH-7) which connects south India to north India passes from the district. Previously it was of two lanes but now it is under the construction phase of 4 lanes. It is the probable reason behind the high level of RTAs and related MLCs in the district.

Fall means collapse to the ground. Person can fall due to various medical reasons [5] and fall can also occurs due to non medical reasons like accidental, homicidal, and suicidal or assaults [6]. Suspected falls when leads to injury, process of MLC registration occurs. Falls and RTAs together constitute almost 48% of MLCs indicating again high percentage of morbidity and mortality which can severely affect social and economic profile of the society. The region is already tribal in nature so impacts are severest on the society.

Hospitalization due to poison intake was the 3rd leading cause of MLCs. Common poisons used in this category was intake of insecticide and tablets. The pattern indicates that the tribal area have high rate of suicidal tendencies. In Adilabad majority of population 68.97% is exercising agriculture practice which depends mainly on monsoon[3]. Failure or late arrival of monsoon is responsible for damage to crops leading to heavy financial losses. Trend of high intake of insecticides during July to October with peak in September indicate tremendous impact of crop loss on regional social life. Similar observations were also made by others in nearby districts[7] and other part of rural India[8].

High rate of MLCs related with alcoholic poisoning indicates disastrous impact of alcohol in this rural/tribal region. Alcohol intake is common in persons involved in agriculture practice[9]. Higher incidence of alcoholic poisoning is because of use of Desi (country) liquor which is prepared by local available materials. Quality of Desi liquor is always substandard with lot of hazardous substances present in it[10].

Assault cases were another important medico-legal problem of the region as they constitute 4th leading cause of MLCs. Alcoholic poisoning is common in the region which indicate practice of alcohol intake is more common. It is one of reason behind more assault cases in the region because there is a casual relationship with alcohol intake and assault[11]. Literacy rate in this tribal cum rural district is 27.80% which is below the state average[3] so we can say that illiteracy is probably another reason of more assault related MLCs.

Forest and agriculture lands constitute 78% geographical area of the district with 43% thick forest which covers entire district and have variety of wild animals[3]. It is the probable reason behind high incidence of wild animal attack. Since wide human developmental activities are going on like national highway construction work which is affecting forest land. Our observations are similar with other study which indicates that man –animal conflict occurs if human activities are near forest land[12]. In animal related MLCs snake bite was predominant which was more common during the season of monsoon. Usually in India farmers walk barefoot in their fields, this makes them prone to snake-bites. Their houses are built of mud which provides easy access and shelter to snakes[13]. Our finding is similar to other study which was conducted in

Table 2:

Poisoning cases				
Poison	% From total MLCs		% From total poisoning	
Insecticide	12.15	Suicidal/attempted poisoning 13.82	77.82	Suicidal/attempted poisoning 88.54
Tablet	1.59		10.17	
Kerosene	0.06		0.37	
Unknown liquid	0.03		0.18	
Alcohol	1.62	Non suicidal 1.79	10.35	Non suicidal 11.46
Gas leak	0.14		0.92	
Food poisoning	0.03		0.18%	
Total Cases	**15.61**		**100**	

Table 3:

Assault cases												
Month	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep
No of Cases	30	35	34	29	26	40	37	32	48	28	33	17
% of MLCs	0.87	1.01	0.98	0.84	0.75	1.15	1.07	0.92	1.38	0.81	0.95	0.49
% of assault	7.71	9.00	8.74	7.46	6.68	10.28	9.51	8.23	12.34	7.20	8.48	4.37

Table 4:

Animal attack related MLCs			
Wild Animal Attack		Domestic Animal Attack	
9.06%		0.81%	
Snake bite	6.58	Rat bite	0.03
Scorpion bite	0.75	Dog bite	0.38
Insect bite	0.43	Pig bite	0.03
Unknown bite	0.75	Buffalo bite	0.03
Honey bee bite	0.14	Hit by buffalos	0.03
Bear attack	0.26	Hit by cow	0.03
Wild pig attack	0.12	HIT by bull	0.29
Unknown Wild animal attack	0.03		

Figure 1:

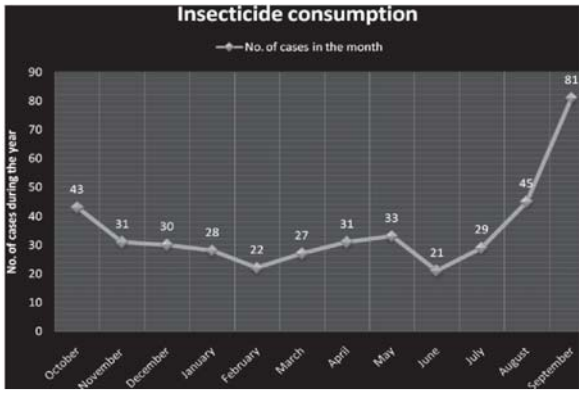
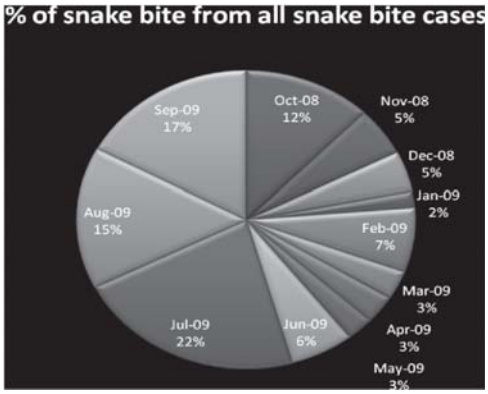


Figure 2:



nearby rural region of Maharashtra[14].

In other category of MLCs, most common MLCs were of prisoners and least was of rape. Unusual high number of prisoners related MLCs are due to inclusion of all prisoner cases as medico-legal case. As usual trend in India, rape cases are less in comparison to other crimes so MLCs are also least.

Conclusion

Developmental activities in the region are severely affecting rural and tribal lifestyle with high incidences of RTAs. Pace of development is not matching with our responsibilities towards environment, wild life, tribal culture etc. There is urgent need to concentrate on farmers' plight and socioeconomic development of tribal population of the region.

Acknowledgment

We are thankful to Mr. M. Salimuddin record section incharge, Mr. B.Vikram Naik record assistant and Mr. Pendhari Shankar technician of RIMS Adilabad for their invaluable help during the collection of data.

Referencess

1. Census of India , office of the registrar general & census commissioner, India, {accessed on December 3, 2009} Available from: http://www.censusindia.gov.in/
2. Subramanian SV, Davey Smith G, Subramanyam M. Indigenous health and socioeconomic status in India. PLoS Med. 2006 Oct;3(10):e421.
3. National informatics center Adilabad, Official website of Adilabad collectorate.mht, {accessed on December 28, 2009} Available from: http://www.adilabad.nic.in/
4. Jayapalan VK. Practical Medico-Legal Manual.1st Ed. Indian Academy of Forensic Medicine; 1988: 26.
5. Tideiksaar R, Kay AD. What causes falls? A logical diagnostic procedure. Geriatrics. 1986 Dec; 41(12):32-50.
6. Türk EE, Tsokos M. Pathologic features of fatal falls from height. Am J Forensic Med Pathol. 2004 Sep;25(3):194-9.
7. Gautami S, Sudershan RV, Bhat RV, Suhasini G, Bharati M, Gandhi KP. Chemical poisoning in three Telengana districts of Andhra Pradesh. Forensic Sci Int. 2001 Nov 1;122(2-3):167-71.
8. Batra AK, Keoliya AN, Jadhav GU. Poisoning: an unnatural cause of morbidity and mortality in rural India. J Assoc Physicians India. 2003 Oct;51:955-9.
9. Sundaram KR, Mohan D, Advani GB, Sharma HK, Bajaj JS. Alcohol abuse in a rural community in India. Part I: Epidemiological study. Drug Alcohol Depend. 1984 Sep;14(1):27-36.
10. Mohan D, Sundaram KR, Advani GB, Sharma HK, Bajaj JS. Alcohol abuse in a rural community in India. Part II: characteristics of alcohol users. Drug Alcohol Depend. 1984 Oct;14(2):121-8.
11. Brismar B, Bergman B. The significance of alcohol for violence and accidents. Alcohol Clin Exp Res. 1998 Oct;22(7 Suppl):299S-306S.
12. Vijayan S, Patil B P. Impact of Changing Cropping Patterns on Man-Animal Conflicts Around Gir Protected Area with Specific Reference to Talala Sub-District, Gujarat, India. Population & Environment. 2002;23(6):541-559
13. Bawaskar H S, Bawaskar P H. Profile of snakebite envenoming in western Maharashtra, India. *Trans R Soc Trop Med Hyg* 2002;**96:**79–84.
14. Punde D P. Management of snake-bite in rural Maharashtra: a 10-year experience. Natl Med J India. 2005 Mar-Apr;18(2):71-5.

Efficacy of preoperative ultrasonography in the evaluation of tumor thickness of tongue

Vijayalaxmi*, Ashok L**, Sujatha G.P.***

*Senior Lecturer, Department of Oral Medicine & Radiology Terna Dental College & Hospital Navi Mumbai, **Professor and Head Department of Oral Medicine & Radiology Bapuji Dental College & Hospital Davangere, ***Professor Department of Oral Medicine & Radiology Bapuji Dental College & Hospital Davangere

Abstract

Objectives

Comparison between the clinically measured vertical thickness of tumor with Ultrasonography keeping the Postoperative Histological measurement as a gold standard in patients with tongue carcinoma (uptoT4 clinical staging) with limited pharyngeal extension.

Materials and methods

Twenty five clinically and histologically proved cases of carcinoma of the tongue patients (uptoT4 clinical staging) were subjected to Ultrasonography to measure tongue tumor thickness using 5-8.2 MHz probe, before they underwent surgical resection. The Post surgical specimens were studied histopathologically for tumor thickness, which was considered as a gold standard. The values of Ultrasonographically measured and histologically measured vertical tumor thickness of tongue were tabulated for statistical analysis for comparison.

Results

Overall accuracy of ultrasonography as compared with histopathology was 64%, whereas 100% accuracy in T1 staged tumors and 12.5% in T2 staged tumors. The $p < 0.001$(HS)

Sonographic accuracy was 75%, 30%, & 11.11% in T1, T2 and T3 stages respectively. Ultrasound overestimation was found in 25% of T1 staged tumors and underestimation in 70% and 88.89% in T2 and T3 staged tumors respectively, with the $p<0.001$ (HS) & the Karl Pearson Product Correlation coefficient, $r = 0.6739$ being significant.

Conclusion

Preoperative ultrasonography can be used as an adjunctive diagnostic tool for precise surgical treatment plan in tongue cancer up to T3 clinical staging.

Keywords

Tongue cancer, Ultrasonography, Tumor thickness, Histopathology

Introduction

The incidence of Carcinoma of tongue has recently increased due to enhanced exposure to carcinogens & is in the range of 0.5 – 1 percent of all malignancies.[1] The most common presenting sign of carcinoma of tongue is a painless mass or ulcer, typical lesion develops on the lateral or ventral border of tongue and may proceed either to develop a fungating exophytic or infiltrate into deep layers of tongue producing fixation and induration.[2]

Vertical thickness of the tumor is the direct micrometer measurement by the pathologist, and in contrast to tumor depth, tumor thickness is independent of the local anatomy.[3] Predictive information obtained preoperatively, about tumor thickness would be of significant use in the treatment decision making.[4] This is made possible by some advanced imaging modalities like CT, MRI, Ultrasonography, and Radionuclide imaging. Ultrasonographic examination has the advantages of being noninvasive, rapid, reproducible and of comparable accuracy and it also provides a 3 dimensional view of the tumour. Various authors found that Ultrasonography (US) was better comparable to computed tomography and physical examination for confirmation of the extent of the disease.[1,2,3,4]

With this background, present study was undertaken to know the efficacy of preoperative ultrasonography in evaluation of tumor thickness in tongue cancer, where in 25 subjects of biopsy proven carcinoma of tongue were subjected to Ultrasonography prior to surgery. The vertical thickness of the post surgical specimen was measured histopathologically (HP) and was used as a gold standard to compare with ultrasonographic thickness.

Methodology

The study group was comprised of 25 clinically and histologically proved cases of carcinoma of the tongue patients. With the informed prior written consent, the patients were examined to asses the TNM staging of the malignancy and maximum vertical thicknesses of the tumors were noted. In 13 cases, 5 MHz probe was used and in 11 cases, 7.5 MHz probe was used and in 1 case, 8.2 MHz probe was used.All tumors were seen as hypo echoic, ill defined, and homogenous lesions destroying the normal architecture of the tongue. Special attention was given to measure the tumor thickness.

Later the patients were subjected to surgery. The area with maximum vertical thickness was carefully sectioned out, and specimen was then mounted in paraffin blocks, with the surface of the specimen representing the maximal depth of infiltration exposed. These blocks were then mounted on a microtome; serial sections were taken and mounted on glass slides. These specimens were then stained with H&E stains and viewed under microscope equipped with scale on stage. Vertical distance from the surface to the deepest part of the tumor was measured.30% tissue shrinkage compensation was given for tissue shrinkage induced by dehydration and hot-wax infiltration during paraffin processing [5] All the values were tabulated and subjected to statistical analysis for comparison.

The study group was comprised of 25 subjects, with the age ranging from 28 to 75 years of 19 males and 6 females(Table I). All patients except 1 had various habits of chewing pan and tobacco, cigarette, beedi smoking, and alcoholics. 24 patients were ulcerative and 1 patient had exophytic growth.

Out of 4 patients staged T1, there were 2 males & females each, out of 10 staged T2, there were 8 males and 2 females , out of 9 patients staged T3, there were 7 males and 2 females and there were 2 females in T4 stage.

Clinically measured vertical thickness of tumor

4 male patients had clinically palpable vertical thickness less than 2.0 cms, 8 male and 2 female had of 2.1 to 4.0 cms, 7 male and 2 female between 4.1 to 6.0 cms and 2 female had lesions more than 6 cms (Table II).

Vertical tumor thickness of tongue as measured by US

The minimum tumor thickness measured by US was 4.4mm and maximum was 44mm. 11 males and 4 females in tumor range of 0.0 – 20.0mm, 7 males and 1 female in tumor range of 20.1-40.0mm, and 1 male and 1 female in tumor range of 40.1-60.0mm

Histologically measured vertical thickness of tongue tumor

The minimum tumor thickness measured histological with 30% tissue shrinkage compensation was 2.86mm and maximum was 22.10mm. They were divided into 3 groups. 13 males and 1 female in range of 0.0 – 10.0mm,7 males and 3 females in range10.1-20.0mm, and 1 female in range of 20.1-30.0 mm

Comparison of tongue tumor thickness clinically and ultrasonographically

Out of 4 tumors staged T1 clinically were correctly estimated in 3 cases (75%), and 1 case (25%) was overestimated by US. Out of 10 tumors staged T2 correctly estimated in 3 cases (30%) and 7 cases (70%) were underestimated .Out of 9 cases tumors staged T3 clinically were correctly estimated in 1 case (11.11%) and underestimated in 8 cases (88.89 %). All 2 tumors staged T4 clinically were underestimated by US(Table III)

Out of total 25 patients, ultrasound correctly estimated in 7 patients (28%), underestimated in 17 (68%) and overestimated in 1 (4%). Overall accuracy of US when compared with clinical thickness was 28%. The p-value is <0.001,(HS) & Karl Pearson's product Correlation coefficient between Clinically measured thickness and US was significant, $r = 0.4200$

Comparison of tongue tumor thickness clinically and histopathologically

Tumors staged T1 clinically were correctly estimated where as those tumors staged T2, T3 and T4 clinically, were all underestimated histologically. Correct estimation was done in 4 (16%), underestimation in 21(84%) patients. Overall accuracy of clinical examination when compared with histopathological thickness was 16%(Table IV). p-value is 0.001. Karl Pearson's product Correlation coefficient was significant, $r = 0.4661$(Table V)

Comparison of tongue tumor thickness by Ultrasound & Histopathology

15 cases of tumors measuring less than 2 cms by US were correctly estimated histologically.8 cases measuring between 2-4 cms by US were correctly estimated in 1 (12.5%) and underestimated in 7 (87.5%) cases & also 2 cases measuring between 4-6 cms. Correct estimation was done in 16 (64%), underestimation in 9 cases (36%) Overall accuracy of US when compared with HP thickness was 64%(Table VI). The p-value is < 0.001 & Karl Pearson's product Correlation coefficient was significant, $r = 0.6739$

Comparison of clinical, sonographic and histological measurement

Out of 4 tumors, 3 tumors staged T1 (<2 cms) clinically were estimated correctly by US & HP and 1 tumor was overestimated (2-4 cms) by US but correctly estimated (<2 cms) by HP.

Out of 10 Tumors staged T2 (2-4 cms) clinically, were underestimated to be <2 cms in 7 cases and correctly estimated in 3 cases by US. All tumors were underestimated (<2 cms) histologically.

Out of 9 tumors staged T3 clinically (4-6 cms), were underestimated in 8 cases by US, i.e., 4 cases measured <2 cms and 4 cases measured between 2-4 cms. And 1 case was correctly estimated (4-6cms) by US. All cases were underestimated histologically i.e., 8 cases measured <2 cms and 1 case measured between 2-4 cms.

All tumors staged T4 clinically (>6 cms), were underestimated by US i.e., 1 case measured between <2 cms and 1 case measured between 4-6 cms. All tumors were underestimated by HP, 1 case measured <2 cms and 1 case measured between 2-4 cms.

The MD of clinically measured and US as studied by applying students paired t – test is 8.3996 and SD difference is 8.8423. The p-value is < 0.001(HS) (Table VII).

.Karl Pearson's product Correlation coefficient between two was significant, $r = 0.6739$.

Discussion

US reported to be either underestimated or overestimated by various authors in the literature. The reasons being, in the superficial lesions, the echo beam often passes obliquely from the submental approach and the lack of an ideal cross-section makes it difficult to evaluate the correct size and the degree of superficial extension. Overestimation by US could be because of failure to distinguish between inflammation, fibrous tissue and tumor [9,10,11,12].

Shintani et al[9] who could identify tumors less than 5 mm in thickness by ultrasound and not by CT and MRI, however our study did not include other imaging modalities.In our study US findings were not in accordance with palpatory findings in 18 cases, whereas Narayana et al[1], found this difference in 9 cases out of 13, overestimation in 3 and underestimation in 6 cases.

In our study 7 cases (28%) were correctly estimated by clinical examination and US whereas J. Ikezoe et al [13], found tumors to be of same size in 30 cases (53%) , by ultrasound and physical examination,17 (30%) were overestimated and underestimated in 7 (12%) cases out of 57.

Our study is comparable to that of studies of Shintani[4,9] et al in 1997 and 2001 who found a value of r was 0.985 & 0.988respectively.However, Pavelka R et al[10], found that there was correlation of sonography with surgical specimen in 14 tumors out of 20 (70%)and Fruhwald F et al[11,12] found US correctly detected the size in 93% of the cases. Narayana H M et al[1], found overall, US accuracy was 65% and accuracy by palpation was 45% amongst operated cases and US was more sensitive in detecting spread across the midline.

In our study 75% accuracy was found in estimating T1 tumors whereas Narayana et al[2], found 33% accuracy in diagnosing T1 tumor

Table I: Age and sex wise distribution of 25 patients

Age Range(Yrs)	Male	Female	Total
25-35	3	0	3
36-46	7	2	9
47-57	5	1	6
58-68	4	1	5
69-79	0	2	2

Table II: Comparison of clinically measured thickness with ultrasound measurement

Clinically measured thickness	N	Ultrasound staging		
		Correct estimation	Under estimation	Over estimation
T1 (0-2 cms)	4	3 (75%)	Nil	1 (25%)
T2 (2-4 cms)	10	3 (30%)	7 (70%)	Nil
T3 (4-6cms)	9	1 (11.11%)	8 (88.89%)	Nil
T4 (> 6cms)	2	Nil	2 (100%)	Nil
Total	25	7 (28%)	17 (68%)	1 (4%)

N = Number of patients

Table III: Comparison of Clinical measured thickness (cms) and Ultrasound Measurement (mm) by students paired t-test

Methods	Mean	SD	Mean diff.	SD diff.	Paired t value	p-value	Significance
Clinical measured thickness (cms)	3.6040	1.5046	- 14.8680	10.4427	- 7.1188	<0.001	HS
Ultrasound measurement (mm)	18.4720	10.9850					

by US. Freuhwald et al[12], found sonographic evaluation was in accordance with pathologic as well as clinical stage in 49 of 50 patients.

Conclusion

In Our study it was observed that Preoperative Ultrasonography is found to be more

accurate in measuring tumor thickness in T1 stage and hence it can be used as a

complemenatary imaging of tongue neoplasms.

Table IV: Comparison of clinically measured thickness with histologic measurement

Clinically measured thickness	N	Histological measurement		
		Correct estimation	Under estimation	Over estimation
T1	4	4 (100%)	Nil	Nil
T2	10	Nil	10 (100%)	Nil
T3	9	Nil	9 (100%)	Nil
T4	2	Nil	2 (100%)	Nil
Total	25	4 (16%)	21 (84%)	0 (0%)

N = Number of patients

Table V: Karl Pearson's product Correlation coefficient between Clinical measured thickness (cms), Ultrasound Measurement (mm) and Histo-pathological Measurement (mm)

Methods	Clinical measured thickness (cms)	Ultrasound measurement (mm)
Ultrasound measurement (mm)	0.4200*	—
Histopathological measurement (mm)	0.4661*	0.6739*

* Significant at 5% level of significance ($p<0.05$)

Table VI: Comparison of ultrasound and histological measurements

Ultrasound measurement	N	Histological measurement		
		Correct estimation	Under estimation	Over estimation
Less than 2cms	15	15 (100%)	Nil	Nil
2-4cms	8	1 (12.5%)	7 (87.5%)	Nil
4-6cms	2	Nil	2 (100%)	Nil
Total	25	16 (64%)	9 (36%)	0 (0%)

Table VII: Comparison of Clinical measured thickness (cms) and Histo-pathological Measurement (mm) by students paired t-test

Methods	Mean	SD	Mean diff.	SD diff.	Paired t value	p-value	Significance
Clinical measured thickness (cms)	3.6040	1.5046	-6.4684	3.4589	-9.3503	<0.001	HS
Ultrasound measurement (mm)	10.0724	3.8937					

Photographs

Photo 1: Ultrasound scanning of patient

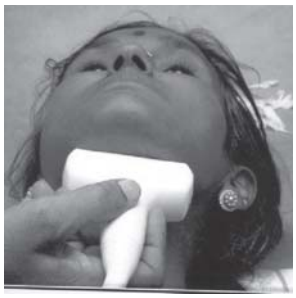
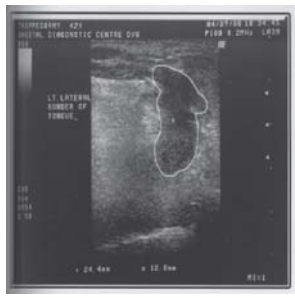


Photo 3: Surgical specimen of the tongue with tumor mass

Photo 2: ultrasonography of tumor of the tongue



References

1. Narayana HM, Panda NK, Mann SBS, Katariya S, Vasishta RK. Ultrasound versus physical examination in staging carcinoma of the mobile tongue. The Journal Laryngol and Otol 1996; 110: 43-47.
2. Shafer WA, Hine MK, Levy BM. A textbook of Oral Pathology. 4th edn. Saunders Elsevier Science: 1993.
3. Kuriakose MA, Loree TR, Hicks WL, Welch JJ, Wang H, DeLacures MD. Tumor volume estimated by computed tomography as a predictive factor in carcinoma of the tongue. Br J Oral Maxillofac Surg 2000; 38: 460-465
4. Shintani S, Nakayama B, Matsuura H, Hasegawa Y. Intraoral ultrasonography is useful to evaluate tumor thickness in tongue carcinoma. The Am J Surgery 1997; 173: 345-347
5. Culling CSA, Allison RT, Barr WT. Cellular Pathology Techniques. 4th edn.. London Butterworth; 1985.
6. Prince S, Bailey BMW. Squamous carcinoma of the tongue: review. Br J Oral Maxillofac Surg 1999; 37: 164-174.
7. Lidstad ST, Bigelow ME, Remensnyder JP. Squamous cell carcinoma of the tongue : A comparison of the anterior two thirds of the tongue with its base. Am J Surg 1983; 146: 456-461.
8. Mettler FA, Schultz K, Kelsey CA, Khan K, Sala J, Kligerman M. Gray-scale ultrasonography in the evaluation of neoplastic invasion of the base of the tongue. Radiology 1979; 133: 781-784
9. Shintani S, Yoshihama Y, Ueyama Y, Terakado N, Kamei S, Fijimoto Y, et al. The usefulness of intraoral ultrasonography in the evaluation of oral cancer. Int J Oral Maxillofac Surg 2001; 30: 139-143.
10. Pavelka R, Streinzer W, Zrunek M, Fruhwald F, Neuhold A, Seidl G.

Evaluation of real-time sonography in the pretherapeutic staging of malignant tumors of the tongue and mouth floor. Laryngol Rhinol Otol (Stuttg) 1986; 65(11): 632-9. (Pubmed.com). Accessed on 29/8/06

11. Fruhwald F, Neuhold A, Seidl G, Pavelka R, Mailath R, Zrunek M. Realtime sonography in the diagnosis and follow-up of malignant tongue tumors. II. Detection and staging. Rofo 1986; 144(2): 174-8. (Pubmed.com). Accessed on 29/8/06.
12. Fruehwald F, Salomonowitz E, Neuhold A, Pavelka R, Mailath G. Tongue cancer : Sonographic assessment of tumor stage. J Ultrasound Med 1987; 6: 121-137.
13. Ikezoe J, Nakanishi K, Morimoto S, Takashima S, Inoue T, Makasi N and Kozuka T. Sonographic staging of cancer of the mobile tongue. Acta Radiologica 1991; 32 Fasc 1 ; 6-8

Comparative studies of some toxic ions like Pb^{2+} & Cd^{2+} on the reproductive functions in female rats: A case study

Vaneet Dhir

Assistant Professor, Sikh National College (NAAC, B^{+}), Qadian 143516, Punjab, India

Abstract

In my previous work[1-2] I (Dhir) worked on the physicochemical interactions in between the biomolecules (eccrine) with series of cations and also studied the importance of hydrophobic character of big biomolecules. Therefore; in this pilot project I (Dhir) studied the effect of toxic cations like Pb^{2+} & Cd^{2+} on the various biological aspects. Heavy metal toxicity is a serious worldwide problem which adversely affects the growth, health, reproductive performance and life span of all living organisms. Leads (lead acetate) and cadmium(cadmium sulphate) being a toxic cumulative poison and an environmental pollutant, experiments were conducted at an oral chronic dose of (60 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 was studied. It was observed that the chronic dose of lead caused an elevation in the level of proteins, acid phosphatase, alkaline phosphatase, alanine aminotransferase and aspartate aminotransferase in all the soft tissues studied indicating tissue damage, whereas it was observed be me (Dhir) the effects were more severe in case of cadmium. However no literature was available so far as to compare the toxicity level of cadmium with lead (because the cadmium ions or compounds are available in trace amount but I (Dhir) cannot rule out the possibility of cadmium pollution). Therefore it is necessary to compare lead with cadmium. Cadmium also inhibited the level of acetylcholinesterase in all the tissues. Fertility tests by pairing treated females with males showed that lead and cadmium treated female showed irregular oestrous cycle and the fertility rate dropped to 40 %(in case of lead) and 50 % (in case of cadmium) as female pups of lead treated mothers showed loss in weight, high mortality rate, poor growth rate and late vaginal opening. Histological studies of ovary showed atresia (figures-5-8) in all the stages of folliculogenesis sustaining the poor fertility observations. It was also observed by me (Dhir) that there must be some relation in terms of anionic part of salt is being considered because I (Dhir) have taken lead in the form of acetate(because acetate are available in solution easily to feed the rats) while cadmium in the form of sulphate.

Introduction

Lead has no known biological function and any lead 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 [3]. 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. The toxic effects are many, ranging from morphological tissues damage at higher concentration to lesser biochemical effects at lower concentrations [4].

Lead is known to be toxic when present in traces and enters human body as a result of environmental pollution[3]. 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[4-5]. Children exposed to lead are reported to have adverse effects on central nervous system and kidneys[4]. Maternal blood lead level as an environmental factor is an apparent predictor of low birth weight and child body mass ratio[5] and low to moderate environmental exposure increases the risk for spontaneous abortion [6]Anaemia which is frequently observed in lead poisoning was a result of decrease lifetime of erythrocytes and synthesis of heme [7-8]. In Ludhiana (Punjab, India), the analysis of water samples of Budha Nallah after the input of effluents by dying industries and pesticide manufacturing units indicate that the concentration of lead has increased manifold[8] and the mean daily intake of lead was 162.32 ± 19.1 mg/day [9].

Mating involving one lead toxic parent have recorded significant decrease in litter size, birth weight and survival rate [10-12]. A variation in the time of vaginal opening and a significant disturbed oestrous cycle was also observed in lead toxicity [13]. Since the absorption of lead indicated toxicity in humans is great due to the intake through food, air, and water, it became imperative to carry out a systematic study on the effect of chronic oral dose of lead on female reproductive functions and also to record the various enzymatic changes in rats. These findings would be useful in understanding the various effects on sensitive species and also extrapolating, with care the results for humans.

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 lead and cadmium 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). Statical 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 [14] daily between 9:30 a.m. to 10:30 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.

The vaginal smears were examined immediately under the microscope while still wet and the cellular components were judged to determine the various stages of oestrous cycle with the help of following criteria: Diestrus : leucocytes only; Proestrus: epithelial cells with nuclei; Oestrus: vaginal cornification with total absence of leucocytes; and Metoestrus: leucocytes with few cornified epithelial cells.

For histopathological study, a piece of ovary was fixed for twenty four hours in alcoholic bouins fluid. The animals were sacrificed at 30, 60, 90 days after dose administration and ovaries were removed, cleaned of adjoining tissues and fixed in alcoholic bouins solution. The tissue was then processed for histological studies. Further serial paraffin sections were cut at 7mm. These sections were stained with haematoxylin eosin and stained serial sections of ovaries were examined under light microscope and morphological characteristics of normal and arteric follicles observed.

Fertility tests were conducted by treating female rats continuously for three months with lead(@ 60mg/kg/day) and cadmium (@ 40mg/kg/day) and housed with mature normal untreated males. The males were separated from females after formation of vaginal plug. The female were observed for entire gestation period of 28 days and the

parameters of birth rate, litter size, morphological alterations, survival rate of pups, body weight from birth to 60 days, and vaginal opening in female pups for the litter were recorded. The surviving pups were then administrated lead @ 60 mg/kg body and cadmium @ 50 mg/kg body weight, respectively after weighing up to 60 days of age.

Results and discussions

Biochemical parameters

Daily oral administration of lead and cadmium (60 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[14]. Increase in level of acid phosphatase in liver and kidney might be suggestive of increase physiological phagocytosis[15] and the moderate amount of acid phosphatase activity in regressing luteal cells of the ovary indicated lysosomal activity in luteolysis[16]. 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. Ryan (1981) had also associated a relationship of acid phosphatase being a lysosomal enzyme playing a phagocytic role in follicle cells during atresia. 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[17].

Both lead and cadmium caused a significant increase in alkaline phosphatase level (Table 1) in plasma, liver, kidney and ovary. While the effects are more in case of cadmium as compared to lead. It has been suggested that an increase in alkaline phosphatase level occur due to the 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[18]. 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[17].

Lead and cadmium (the dose rate of 60 mg/kg/day and 40 mg/kg/day) for 90 days produced an overall increase in the levels of alanine aminotransferase in plasma, liver and ovary and a non significant rise in its level kidney (Table 2). Alanine aminotransferase is present in liver, kidney, heart, skeletal muscles, intestines and RBC (Doxy 1971) and its increased values are specific indicator of hepatocellular (liver) damage (Kaneko 1989). Lead and cadmium also produced significant increase in aspartate aminotransferase in liver, plasma and ovary while the effect of lead on aspartate aminotransferase in liver, plasma and ovary is more as compared to cadmium (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 and cadmium on muscles increasing the permeability of cell membrane cannot be excluded (Thomson 1971).

Daily oral administration of lead and cadmium produced significant decrease in AChE level in liver, kidney and ovary and non significant decrease in plasma (Table 3). It was considered that decrease in AchE activity was responsible for behavioural and locomotor changes recorded in lead intoxicated calves (Golter and Michelson, 1975). Acetylcholinesterase in the luteal cells of ovary hydrolyses acetylcholine in the production of acetic acid which is used subsequently in the pathway for production for steroidogenes for hormone production in the goat ovary (Bhattacharya, 1978). Thus decrease in AChE activity in the rat ovary might be an indicator of the lack of steroidogenesis resulting in poor fertility.

Significant increase in the levels of proteins in liver, kidney, ovary and non-significant increase of proteins in plasma were observed

Table 1: Effect of lead and cadmium 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)	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 (cadmium)	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)	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 (cadmium)	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)	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 (cadmium)	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)	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 (cadmium)	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)	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 (cadmium)	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)	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 (cadmium)	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)	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]	2846.250±19.330[ab]
Kidney (cadmium)	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)	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 (cadmium)	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]

a-Statistically significant different ($P<0.05$) when compared to values of control animals.
b-Statistically significantly different ($P<0.01$) when compared to control animals.
All values given are the mean of 3 animals except control
Control values given are the mean of 4 animals.

Table 2: Effect of lead and cadmium on tissue aminotransferases.

Organ	Control	Treatment					
		15 Days	30 Days	45 Days	60 Days	75 Days	90 Days
Alanine aminotransferase (n mol pyruvate formed / min/ml) (Mean ± S.D.)							
Plasma (lead)	12.22±0.22	3.9300 ± 0.187[ab]	12.880 ± 0.160[ab]	12.966 ± 0.050[ab]	13.430 ± 0.720[ab]	15.420 ± 0.260[ab]	19.315 ± 0.410[ab]
Plasma (cadmium)	12.22±0.22	3.999 ± 0.245[ab]	13.256 ± 0.356[ab]	13.996 ± 0.247[ab]	14.564 ± 0.897[ab]	17.898 ± 0.478[ab]	20.345 ± 0.478[ab]
Liver (lead)	428.310 ± 19.64	570.146 ± 16.07[ab]	658.940 ± 5.380[ab]	477.900 ± 21.17[a]	491.070 ± 2.570[ab]	541.090 ± 40.17[ab]	566.090 ± 11.56[ab]
Liver (cadmium)	428.310 ± 19.64	587.455 ± 19.45[ab]	688.360 ± 7.238[ab]	523.204 ± 29.78[a]	545.184 ± 3.65[ab]	558.125 ± 46.34[ab]	577.87 ± 12.33[ab]
Kidney (lead)	52.400 ± 10.73	55.240 ± 0.650	59.930 ± 4.480	60.500 ± 13.09	58.600 ± 8.900	65.620 ± 6.250	68.996 ± 9.610
Kidney (cadmium)	52.400 ± 10.73	57.342 ± 0.732	60.143 ± 5.790	63.235 ± 15.16	65.125 ± 9.909	68.657 ± 7.235	72.569 ± 10.450
Ovary (lead)	6.560 ± 0.180	9.490 ± 0.310[ab]	15.87 ± 8.650[ab]	17.47 ± 10.30[ab]	24.78 ± 0.760[ab]	20.83 ± 5.480[ab]	31.83 ± 0.630[ab]
Ovary (cadmium)	6.560 ± 0.180	9.989 ± 0.245[ab]	16.89 ± 9.455[ab]	18.898 ± 13.24[ab]	26.899± 0.899[ab]	24.353 ± 6.453[ab]	33.24 ± 0.783[ab]
Aspartate Aminotransferase (n mol pyruvate formed / min/ml) (Mean ± S.D.)							
Plasma (lead)	2.15 ± 0.04	5.09 ± 0.26[ab]	4.30 ± 0.79[ab]	5.93 ± 0.53[ab]	5.92 ± 0.77[ab]	5.78 ± 0.05[ab]	6.99 ± 1.02[ab]
Plasma (cadmium)	2.15 ± 0.04	6.88 ± 0.56[ab]	6.78 ± 0.89[ab]	7.94 ± 0.74[ab]	7.98 ± 0.89[ab]	9.88 ± 0.15[ab]	10.59 ± 2.04[ab]
Liver (lead)	263.10 ± 21.57	275.91 ± 12.57	294.82 ± 9.210	299.92 ± 12.32[a]	298.03 ± 11.24	304.44 ± 33.62	313.81 ± 26.74[ab]
Liver (cadmium)	263.10 ± 21.57	279.91 ± 14.47	299.88 ± 10.22	301.78 ± 14.82[a]	305.23 ± 14.26	308.74 ± 35.67	318.97 ± 232.88[ab]
Kidney (lead)	209.60 ± 10.11	231.94 ± 13.85	263.41 ± 2.050[ab]	292.75 ± 11.56[ab]	294.07 ± 13.82[ab]	319.76 ± 12.27[ab]	344.35 ± 14.41[ab]
Kidney (cadmium)	209.60 ± 10.11	239.25 ± 16.23	256.24 ± 4.45[ab]	289.34 ± 13.56[ab]	298.23 ± 16.77[ab]	319.28 ± 13.88[ab]	355.25 ± 16.56[ab]
Ovary (lead)	4.03 ± 0.74	4.140± 0.62	7.120 ± 0.49[ab]	7.520 ± 0.13[ab]	12.93 ± 0.58[ab]	15.15 ± 2.05[ab]	22.74 ± 1.74[ab]
Ovary (cadmium)	4.03 ± 0.74	5.245± 0.82	7.450 ± 0.89[ab]	7.620 ± 0.43[ab]	12.83 ± 0.88[ab]	16.45 ± 3.35[ab]	24.64 ± 3.84[ab]

a-Statistically significant different (P<0.05) when compared to values of control animals.
b-Statistically significantly different (P<0.01) when compared to control animals.
All values given are the mean of 3 animals except control
Control values given are the mean of 4 animals.

Table 3: Effect of lead and cadmium on tissue acetylcholinesterase and proteins.

Organ	Control	Treatment					
		15 Days	30 Days	45 Days	60 Days	75 Days	90 Days
Acetylcholinesterase (n mol Acetylcholine hydrolysed / min/ml) (Mean ± S.D.)							
Plasma (lead)	0.1075 ± 0.09	0.045 ± 0.029	0.060 ± 0.030	0.070 ± 0.024	0.090 ± 0.080	0.076 ± 0.067	0.056 ± 0.070
Plasma (cadmium)	0.1075 ± 0.09	0.097 ± 0.049	0.099 ± 0.020	0.120 ± 0.034	0.129 ± 0.094	0.099 ± 0.087	0.035 ± 0.060
Liver (lead)	3.70 ± 0.56	0.500 ± 0.050[ab]	0.820 ± 0.088[ab]	0.647 ± 0.068[ab]	0.552 ± 0.070[ab]	0.909 ± 0.066[ab]	0.713 ± 0.050[ab]
Liver (cadmium)	3.70 ± 0.56	0.4903 ± 0.040[ab]	0.856 ± 0.093[ab]	0.765± 0.093[ab]	0.539 ± 0.090[ab]	0.129 ± 0.096[ab]	0.836 ± 0.088[ab]
Kidney (lead)	0.560 ± 0.05	0.417 ± 0.064[a]	0.263 ± 0.010[ab]	0.464 ± 0.073[ab]	0.246 ± 0.095[ab]	0.310 ± 0.048[ab]	0.156 ± 0.034[ab]
Kidney (cadmium)	0.560 ± 0.05	0.465 ± 0.044[a]	0.265 ± 0.040[ab]	0.577 ± 0.0782[ab]	0.438 ± 0.098[ab]	0.295 ± 0.088[ab]	0.196 ± 0.054[ab]
Ovary (lead)	0.459 ± 0.07	0.257 ± 0.057[ab]	0.157 ± 0.040[ab]	0.367 ± 0.047	0.388 ± 0.047	0.275± 0.017[ab]	0.149± 0.090[ab]
Ovary (cadmium)	0.459 ± 0.07	0.336 ± 0.037[ab]	0.298 ± 0.032[ab]	0.415 ± 0.069	0.4598 ± 0.087	0.499± 0.067[ab]	0.505± 0.099[ab]
Proteins (g/100ml) (Mean ± S.D.)							
Plasma (lead)	0.77±0.096	0.076±0.005	0.079±0.004	0.082±0.008	0.086±0.002	0.081±0.077	0.092±0.033
Plasma (cadmium)	0.77±0.096	0.056±0.003	0.088±0.023	0.094±0.007	0.092±0.005	0.093±0.087	0.121±0.065
Liver (lead)	0.385±0.0176	0.381±0.010	0.388±0.023	0.450±0.020[ab]	0.476±0.052[ab]	0.437±0.012[ab]	0.441±0.002[ab]
Liver (lead) (lead)	0.385±0.0176	0.399±0.024	0.423±0.043	0.475±0.056[ab]	0.499±0.043[ab]	0.501±0.013[ab]	0.567±0.003[ab]
Kidney (lead)	0.298±0.016	0.284±0.004	0.286±0.032	0.403± 0.057[a]	0.464±0.016[ab]	0.470±0.025[ab]	0.477±0.011[ab]
Kidney (cadmium)	0.298±0.016	0.293±0.034	0.299±0.064	0.427± 0.093[a]	0.594±0.045[ab]	0.689±0.045[ab]	0.704±0.031[ab]
Ovary (lead)	0.0715±0.038	0.0710±0.0049	0.094±0.0125	0.098±0.004	0.154±0.036[ab]	0.161±0.004[ab]	0.170±0.023[ab]
Ovary (cadmium)	0.0715±0.038	0.0878±0.0077	0.077±0.0226	0.099±0.013	0.199±0.089[ab]	0.151±0.024[ab]	0.146±0.026[ab]

a-Statistically significant different (P<0.05) when compared to values of control animals.
b-Statistically significantly different (P<0.01) when compared to control animals.
All values given are the mean of 3 animals except control
Control values given are the mean of 4 animals.

Table 4: Fertility assessment of lead (60 mg/kg) and cadmium (60 mg/kg) on treated female rats.

Number of days of treatment	Treated (female)	Untreated (male)	parturition	Size of litter (number)	Gestation period (days)	Died at birth time (number)
90	2	1	½ (lead)Nil (cadmium)	11	20-22	2 (lead)1(cadmium)
90	2	1	0/2	*	*	*
90	2	1	½	8	21-24	3(lead)
90	2	1	½	9	21-23	4(lead/cadmium)
90	2	1	½	7	22-24	2(lead)
0	None (control)	10 females, 3-4 males	10/10	6-10	25-27	0(lead/ cadmium)

*Female rats did not show any signs of pregnancy till the end of the experiment.

Table 5: Effect of lead and cadmium 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 Treated	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] (lead) 10.388±2.45[ab](cadmium)	21.290±2.46 (lead) 19.38±3.58 (lead)	35.21±0.21[ab](lead) 30.99±0.234[ab] (cadmium)	-
B	5.306±0.370[ab]	9.8260±2.27[ab](lead) 9.768±3.14[ab](cadmium)	16.4404.29(lead) 15.230±3.03(lead)	33.80±0.39[ab](lead) 31.67±0.58[ab](cadmium)	-
C	5.570±0.233[ab]	10.912±1.03[ab] (lead) 09.876±1.05[ab] (lead)	20.990±1.56(lead) 18.678±1.89(cadmium)	31.82±0.00[ab](lead) 30.65±0.03[ab](cadmium)	30.21 ±0.00[ab](lead, died on day 63), 26.61 ±0.05[ab](cadmium, died on day 55)
D	5.490±0.150[ab]	10.560±1.97[ab](lead) 09.384±1.82[ab](cadmium)	20.765±1.14(lead) 19.325±2.28(cadmium)	31.48±0.00[ab](lead) 18.98±0.04[ab](cadmium)	-

Table 6: 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 (lead/cadmium)	6-10	6-10	6-10	6-10
60	11	9	8(lead)6(cadmium)	6	6	3	Died
60	Nil	Nil	Nil	Nil	Nil	Nil	Nil
60	8	5	5 (lead)7 (cadmium)	4 (lead)3 (cadmium)	4(lead)6 (cadmium)	2(lead)4 (cadmium)	Died (lead / cadmium)
60	9	5	5(lead)7 (cadmium)	3(lead)5 (cadmium)	3 (lead)6 (cadmium)	1(lead)2 (cadmium)	1(lead)3 (cadmium)
60	7	5	3(lead)2 (cadmium)	2(lead)1 (cadmium)	2(lead/ (cadmium)	1(lead)Nil- (cadmium)	Died(lead/ (cadmium)

Table 7: Effect of lead and cadmium on body weight of treated female.

Treatment dose (mg/kg)	0 day	15 days	30 days	45 days	60 days	75 days	90 days
0	122 4.52	132±3.33 (lead) 120±4.24 (cadmium)	145±2.26(lead) 135±2.04 (cadmium)	151±2.61(lead) 149±2.20 (cadmium)	165±4.21(lead) 155±2.93 (cadmium)	170±2.17(lead) 156±3.16 (cadmium)	173±2.36(lead) 150±1.26 (cadmium)
60	1202.95	122±4.32[ab](lead) 119±2.12[ab] (cadmium)	125±2.26[ab] (lead) 118±1.86[ab] (cadmium)	130±1.56[ab](lead) 120±1.33[ab] (cadmium)	127±4.56[ab](lead) 120±3.44[ab] (cadmium)	125±4.03[ab](lead) 120±2.66[ab] (cadmium)	126±3.92[ab](lead) 117±2.88[ab] (cadmium)

Fig. 1: Shows the conc. Of acid & alkaline phosphatise (tissue) in plasma; ¨,lead; ,cadmium; D, • lead; ·, cadmium.

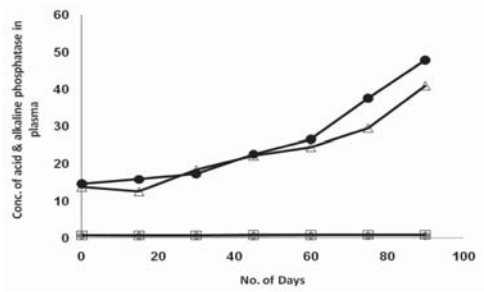


Fig. 2: Shows the conc. of acid & alkaline phosphatise (tissue) in Liver; D, lead; ´, cadmium; •, lead; ·, cadmium.

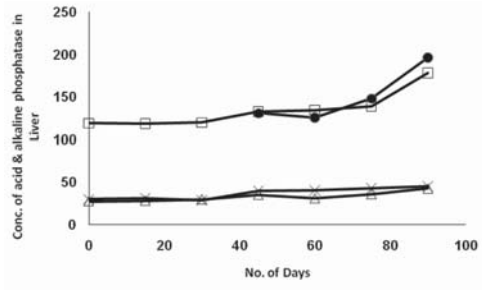


Fig. 3: Shows the conc. of acid & alkaline phosphatise (tissue) in kidney; , lead; •, cadmium; D, lead; , cadmium.

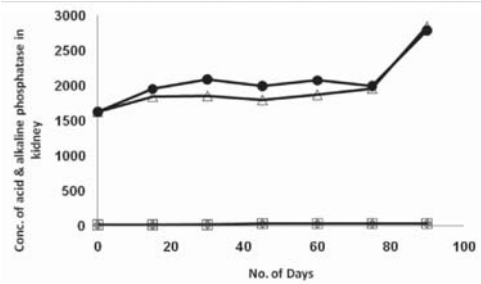


Fig. 4: Shows the conc. of acid & alkaline phosphatise (tissue) in ovary; ¨, lead; •, cadmium; , lead; ·, cadmium.

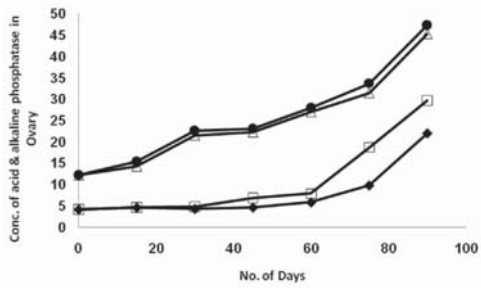


Figure 5: Various stages of follicles undergoing atrsia (HE stain) 100X(Lead).

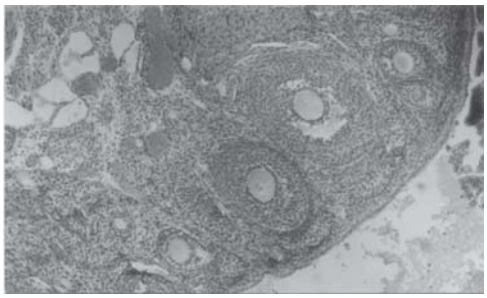


Figure 6: Incipient antral stage follicle undergoing atresia (HE stain) 100X(Cadmium).

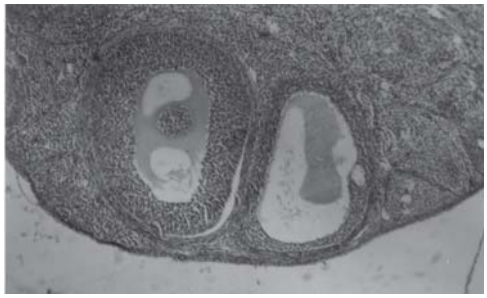


Figure 7: Antrum formed Graafian follicle undergoing atresia with complete detachment of granulose from theca shows advanced stage in atresia (HE stain) 100X (Lead).

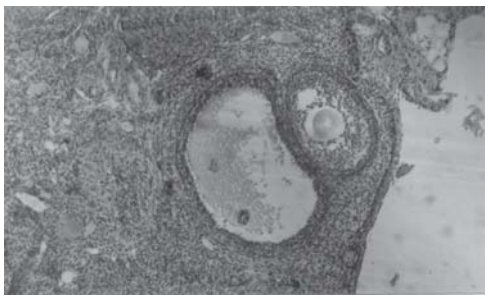
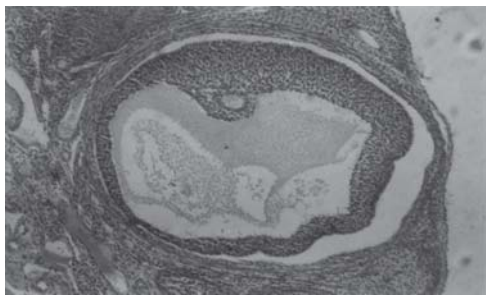


Figure 8: Antrum formed Graafian follicle undergoing atresia with complete detachment (arrowheads) of granulose from theca shows advanced stage in atresia (HE stain) 100X (cadmium).



following daily oral dosing of lead (Table 3). The elevation of proteins is reported to occur in conditions when the cells are subjected to wide variety of environmental assaults including toxins, poisons and pollutants and is mainly due to stimulation of the synthesis of acute phase protein and corresponding m-RNA (Puga and Rodrigues, 1974) which buffer them from harm (Welch, 1993). 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 lead on fertility of rats indicated that lead at a dose of 60 mg/kg caused 40% reduction in the fertility rate while cadmium at a dose of 45 mg/kg caused 50% reduction in the fertility rate (Table 4) 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 5-8). Most reproductive functions are controlled by sex steroids, the possibility that changes in the synthesis / breakdown of these hormones may alter reproductive capacity in man and other animals exposed to lead cannot be excluded. Chronic dosage of lead and cadmium probably imbalances this delicate interplay of hormones and disallows implantation in rat[15].

In addition to the observations made above, the treated females showed irregularity in estrous cycle. Der et al. (1975) and Ronis et al. (1998a) have also reported irregularity in estrous cycle of female albino rats. Female pups of treated mother also showed late vaginal opening, poor fur growth, significantly lower body weight (Table 5) and decrease foetal survival ratio (Table 6). Maternal-foetal transfer of nutrients is an established phenomenon and death of young ones of lead poisoned mothers could represent the placental transfer of lead[18]. Rat fed lead and cadmium showed significant decrease in body weight (Table 7) while the decrease in weight is more in case of cadmium as compared to lead. Furthermore environmental toxicants, teratogenic compounds can have drastic affects on the survival rate of embryos when ingested at crucial early stages of gestation (Christianson, 1992). Parshant et al.[21](2009) in medico-legal update (jounal) 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 and cadmium 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. Lead and cadmium caused high mortality rate in pups and also slows down their growth rate.

Valuable observations

Fig 1. Indicate the effect of lead and cadmium on the concentration of acid and alkaline phosphatise in the blood plasma. It is clear from the trend appeared that the effect of lead and cadmium on the acid phosphatise is almost similar with the 0-90 days while the effect of lead and cadmium on the alkaline (basic) phosphatise is very severe as compared to the acid phosphatise. It is almost clear from the fig1. That trend increase sharply with the number of days. If we compare the trend within no. of days then there is sharp increase in the 60-90 days as compared to 1-59 days. Thus it is concluded that effect of lead and cadmium toxic ions are more severe on the alkaline phosphatise as compared to acid phosphatise.

Fig 2. Indicate the effect of lead and cadmium on the concentration of acid and alkaline phosphatise in liver. It is clear from the trend appeared that the effect of lead and cadmium on the acid phosphatise in liver is almost similar with the 1-90 days while the effect of lead and cadmium on the alkaline (basic) phosphatise has sharp increase in 70-90 days as compared to the acid phosphatise(1-45days). It is almost clear from the fig2. That trend increase with the number of days. If we compare the trend within no. of days then there is sharp increase in the 60-90 days(effect of cadmium on alkaline phosphatise) as compared to 1-44 days. Thus it is concluded that effect of cadmium toxic ions on the alkaline phosphatise are more severe as compared to effect of lead ions on the acid phosphatise.

Fig 3. Indicate the effect of lead and cadmium on the concentration of acid and alkaline phosphatise in kidney. It is clear from the trend appeared that the effect of lead and cadmium on the acid phosphatise in kidney is almost similar with the 1-90 days while the effect of lead and cadmium on the alkaline (basic) phosphatise has sharp increase from the beginning 1-90 days as compared to the acid phosphatise(1-90days). It is almost clear from the fig3. that trend increase with the number of days (in case of basic phosphatise). If we compare the trend within no. of days then there is sharp increase in the 77-90 days (effect of cadmium on alkaline phosphatise) as compared to 1-90 days(acid phosphatise). Thus it is concluded that effect of cadmium toxic ions on the alkaline phosphatise in kidney are more severe as compared to effect of lead ions on the acid phosphatise.

Fig 4. Indicate the effect of lead and cadmium on the concentration of acid and alkaline phosphatise in ovary. It is clear from the trend appeared that the effect of lead and cadmium on the acid phosphatise in ovary is almost similar with the 1-90 days while the effect of lead on acid phosphatise is very steep in 60-90 days which is similar with the effect of cadmium. While cadmium on the alkaline (basic) phosphatise has sharp increase from the beginning 60-90 days as compared to the acid phosphatise(1-59days). It is almost clear from the fig4. that trend increase with the number of days(in case of basic phosphatise). If we compare the trend within no. of days then there is sharp increase in the 61-90 days(effect of cadmium on alkaline phosphatise) as compared to 1-30 days(acid phosphatise) while there is small decrease in 25-45 days . Thus it is concluded that effect of lead and cadmium toxic ions on the acid & alkaline phosphatise in ovary are more severe as compared to effect of lead and cadmium ions in the plasma, liver & kidney. While I (Dhir) unable to provide explanation for the decrease in the concentration of acid & alkaline phosphatise in ovary in the days 25-45 days.

Acknowledgement

I (dhir) specially acknowledge Mrs. Pooja Dhand {M.sc (Zoology, P.A.U Ludhiana), B.Ed}for guiding me to complete this pilot project and also to provide valuable data for the necessary comparison of lead with cadmium in terms of toxicological effect.

References

1 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.
2 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 copared to old classical theory. Medico-Legal Update., 9(2):48-52.
3 Aurrichio F, Migliacci A and Castoria Y (1981) Dephosphorylation of oestradiol receptor in vivo. Biochem. J., 198: 699.
4 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.
5 Christianson WT (1992) Stillbirths mummies , abortions and early embryonic death. Vet. Clin. North Amer., 8: 623-639.
6 Der R, Fahim Z, Hilderbrand D, Fahim M (1985) Combined effect of lead and less protein on growth, sexual development and metabolism in female rats. Res. Commun. Chem. Pathol. Pharmacol., 9: 723-738.
7 Doxey D L (1971) Veterinary Clinical Pathology, pp. 58, Bailliere Tindall and Cassell Ltd., London.
8 Drotman RB and Lawehorn GT (1978) Serum enzymes as indicators of chemically induced liver damage. Drug. Chem. Toxicol., 1: 163-171.
9 Gill BS, Roy RS and Saigal RP (1984) Histoenzymological study of the liver in induced chronic aflatoxicosis in the rabbit. Mykosen, 27: 259-264.
10 Foulkes EC (1990) Biological effects of heavy metals. Vol. II Metal Carcinogenesis U.R.C. Press, Boston.
11 Golter M and Michaelson I A (1975) Growth, behaviour and catecholamines in lead exposed neonatal rats. Reapprasil Science, 187: 359-365.
12 Goody WW, Schrader WT and Malley BWO (1982) Activation, transformation and subunit structure of steroid hormone receptor. Endocr. Rev., 3: 141.
13 Gouda IM, Aziz SAA, Ahmed AA, Lotgi MM and Soliman MM (1985) Changes in some liver functions in experimentally lead poisoned goats. Archiv. Fur. Exp. Vet., 40: 242-249.
14 Hedlund K and Nilsson OA (1971) Hormonal reuirements for the uterine attachment reaction and blastocyst implantation in the mouse, hamster and guinea pig. J. Report Fert., 26 : 267-269.
15 Aurrichio F, Migliacci A and Castoria Y (1981) Dephosphorylation of oestradiol receptor in vivo. Biochem. J., 198: 699.
16 Sandhu H S and Brar RS (2000) Textbook of Veterinary Toxicology, pp. 95-99, Kalayani Publishers, Ludhiana, Punjab, India.
17 Voss G and Sachsse K (1970) Red cell and plasma cholinesterase activities in microsample of human and blood determined simultaneously by a modified acetycholine / DTNB procedure. Toxicol. App. Pharmacol., 16: 764-772.
18 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.
19 Ronis MJJ, Badger TM, Shema SJ, Roberson PK and Shaik F (1998a) Effects on pubertal growth and reproduction and reproduction in rats exposed to lead perinatally or continuously throughout development. J. Toxicol. Environ. Health., 13 (4): 327-341.
20 Ryan RJ (1981) Follicular atresia: some apeculations of biochemical markers and mechanism. In : Dynamics of ovarian Function. Schwartz N B and Hunzickerdunn M (eds.), Raven Press, New York.
21 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.

Incidence of metopism in skulls of adult people from Belgaum, Karnataka

Vijay Kumar A.G*, Ravidra S. Honnungar**, Ajay Kumar T.S***, Vinay R. Hallikeri****

*Post Graduate, Dept. of Forensic Medicine & Toxicology, KLE University's, J.N.Medical College, Belgaum, Karnataka, India, **Assistant Professor, Dept. of Forensic Medicine & Toxicology, KLE University's, J.N.Medical College, Belgaum, Karnataka, India, ***Post Graduate, Dept. of Forensic Medicine & Toxicology, KLE University's, J.N.Medical College, Belgaum, Karnataka, India, ****Post Graduate, Dept. of Forensic Medicine & Toxicology, KLE University's, J.N.Medical College, Belgaum, Karnataka, India

Abstract

The frontal suture is a dense connective tissue structure that divides the two halves of the frontal bone of the skull in infants and children. It usually disappears by the age of six, with the two halves of the frontal bone being fused together. If it does not disappear it may be called a "metopic suture" or "sutura frontalis persistens." The incidence of metopism was observed in 7 skulls (7%), out of 100 skulls studied, being 70 cases (70%) male and 30% (30 cases) female. The Incomplete metopic suture was found in 39 skulls (39%), being more frequent in males (26 cases; 66.7%) than in females (13 cases; 33.3%). The most common shape was linear (16 cases; 41.1%) followed by V shape (09 cases; 23.1%). In remaining 52 of the skulls studied signs of metopic suture were not found. Metopic sutures are of no clinical significance, although they can be mistaken for cranial fractures. The knowledge of morphological pattern of the metopic suture is important for the radiological and surgical point of view as well as for the forensic experts.

Key words

Metopic suture, Metopism.

Introduction

The frontal suture is a dense connective tissue structure that divides the two halves of the frontal bone of the skull in infants and children. It usually disappears by the age of six, with the two halves of the frontal bone being fused together. If it does not disappear it may be called a "metopic suture" or "sutura frontalis persistens." If the suture is not present at birth (craniosynostosis) it will cause a keel-shaped deformity of the skull called trigonocephaly. In some individuals the suture can persist (totally or partly) into adulthood, it is referred to as a metopic suture (or a persistent metopic suture). The suture can either bisect the frontal bone and run from nasion to bregma or persist as a partial metopic suture (where part of the suture survives and is connected to either bregma or nasion) or as an isolated metopic fissure. When complete metopic sutures are present from nasion to bregma, then it is called Metopism. This metopism is more common in higher races & in brachycephalics. Different countries showed different incidences of metopic sutures. They are 7% -8% in European populations, 1% in African population & 4 -5% in Mongolians. It also differs in different states of India namely 4% in Dravidians, 5% in Punjabis, 2.35% in UP subjects.

Material and methods

In this study, all the cases brought for post-mortem examination at mortuary of KLEs PRABHAKAR KORE HOSPITAL AND MRC, BELGAUM between 1ST January 2009 to 31st December 2009 have been studied. Totally 100 skulls were studied for the presence of metopic sutures. The analysis and description of the sutures were made by macroscopic observation of the skulls. Then, these skulls were divided into two initial groups for each sex: those having a complete metopic suture and those with incomplete suture. The metopic sutures were considered complete when they extended from the nasion to the bregma uninterruptedly and incomplete when extending from the nasion to varied points of the frontal bone anterior to the bregma. The incomplete sutures of both sexes were classified according to their morphology as: linear, those single and shallower sutures; V-shaped, those bifurcated and double, those being independent from their origin.

Corresponding author:
Dr. Vijay Kumar A.G
Post Graduate, Dept. of Forensic Medicine & Toxicology KLE University's J.N. Medical College, Belgaum-590010, Karnataka State, INDIA
Telephone: 919916735739
E mail: vijay.fmt@rediffmail.com

Results

The incidence of metopism was observed in 7 skulls (7%), out of 100 skulls studied, being 70 cases (70%) male and 30% (30 cases) female [Table 1]. The Incomplete metopic suture was found in 39 skulls (39%), being more frequent in males (26 cases; 66.7%) than in females (13 cases; 33.3%). The most common shape was linear (16 cases; 41.1%) followed by V shape (09 cases; 23.1%)[Table 2]. In remaining 52 of the skulls studied signs of metopic suture were not found.

Discussion

The cranial sutures influence the growth of the whole skull and despite having been studied for decades there is no consensus about the correlation between cranial development and suture closure.

Table 1:

Skull	Male		Female		Total
	No.	%	No.	%	
Metopism	5	71.4	2	28.6	7

As for the complete suture this study detected seven (7%) instances of complete metopism, male constitutes 71.4% and female 28.6%. In a study carried out by del Sol *et al*. in 400 skulls of Brazilians, the incidence of metopism found was of 2.75% (female 2.96%, 4/135 and male 2.64%, 7/265).[1]

In the study done by Breathnach *apud* Baaten *et al.* the incidence of metopism was seen in 7% European skulls and those described by Czarnetzki *apud* del Sol *et al.,* who found metopism in 11.2% of German female skulls.[2]

On the other hand, the smaller incidences were among blacks, Nigerians and fell to 1.0% in Africans.[3,4]

Data greater than of this work were obtained by other investigators, such as Aschley-Montagu *apud* Del Sol *et al.* in Bolivians (20.24%); Arensburg *et al.* in Bedouins (23%); Comas *et al. apud* del Sol *et al.* in Alpine skulls (63.2%) and in Genoese skulls (41.6%).[5]

In Incomplete sutures males (26 cases; 66.7%) are outnumbered compare to female (13 cases; 33.3%). The most common shape was linear (16 cases; 41.1%) followed by V shape (09 cases; 23.1%). In the study done by del Sol *et al.* in Brazilian skulls, linear shape was found in 64.35% (74/400) and Ajmani *et al*. studied skulls from Nigerians and observed linear sutures in 24.27% (50/206) and the Hindu skulls studied by Agarwal *et al.* the incidence of this type of suture was 23.12% (295/1276).[6]

Conclusion

Metopic sutures are of no clinical significance, although they can

Table 2:

Skull	Male		Female		Total	
	No.	%	No.	%	No.	%
Linear	11	42.3	5	38.5	16	41.1
"V"	6	23.1	3	23.1	9	23.1
Double	4	15.4	1	07.7	5	12.8
Single And Shallower	2	07.7	1	07.7	3	07.7
Bifurcated And Double	2	07.7	2	15.3	4	10.2
Independent From Their Origin	1	03.8	1	07.7	2	05.1
Total	26	100	13	100	39	100

be mistaken for cranial fractures. The knowledge of morphological pattern of the metopic suture is important for the radiological and surgical point of view as well as for the forensic experts. According to the international standards reported in the scientific literature it is concluded that the index of metopism found is within the global patterns. Obtaining more conclusive results demands the amplification of the number of skulls evaluated, as well as carrying out comparative studies between the different regions of country.

References

1. Del Sol M, Binvignat O, Bolini PDA and Prates JC. Metopismono individuobrasileiro. Rev. Paul. Med., 107(2):105-7, 1989.
2. Baaten, PJJ, Haddad M, Abi-nader K, Abi-ghosn A, Al-kutoubi A and Jurjus AR. Incidence of metopism in the Lebanese population. Clin. Anat., 16:148-51, 2003.
3. Bryce TH. Observations on metopism. J. Anat., 51:153-66, 1917.
4. Ajmani ML, Mittal RK and ain SP. Incidence of the metopic suture in adult Nigerian Skulls. J. Anat., 137(1):177-83, 1983.
5. Arensburg B, Goldstein MS and Nathan H. Metopism in Bedouin crania from the Negev of Israel. Z. Morphol. Anthropol. 68:293-7, 1977.
6. Agarwal SK, Melhotra VK and Tewari SP. Incidence of the metopic suture in adult Indian crania. Acta Anat., 105:469-74, 1979.

A study to establish a relationship between serum cholesterol level & unnatural fatalities among the population of the Chandigarh Zone of North West India

Y S Bansal*, Dalbir Singh**

*Associate Professor, **Addl Prof & Head, Department of Forensic Medicine, PGIMER, Chandigarh

Abstract

Even with the advancement of science and the highly improved medical and therapeutic facilities available in the modern world, the incidence of unnatural deaths has only increased spirally. Various factors have been analysed to unveil the reasons behind. Serum cholesterol levels have been found to be associated with an increase in the incidence of violent deaths, particularly suicides. This study was undertaken with an aim to establish a correlation between serum cholesterol levels and unnatural deaths in the Chandigarh Zone of North West India.

A total of 358 cases and 45 healthy volunteers were studied. Seventy eight percent of the cases under study were of deaths due to accident, 18% were suicides and 4% were homicides. The mean serum cholesterol levels were 101.2 mg% in accidental deaths, 98.5 mg% in suicides and 88 mg% in homicides; however the levels in the controls were 176.6 mg%. Cases of homicidal deaths in the age group 40 - 49 yrs exhibited the least mean serum cholesterol levels of 69.9 mg%.

Keywords

Serum cholesterol, Unnatural deaths, Suicides, Accidents, Homicides

Introduction

The extent of unnatural deaths is a gross indicator of the socio-economic condition and mental health of the society.

Previous prevention trials have established that lowering serum cholesterol concentration reduces incidence of coronary heart disease [1,2]; however reduction of the total mortality as a result of the cholesterol regimes has not been demonstrated[3] . This has led to several hypothesis linking the lowered Cholesterol level with mortality due to violent deaths i.e. suicidal, homicidal & accidents [4]. Low Cholesterol concentration was associated not only with a fall in incidence of death from coronary heart disease but also with an increase in death due to suicide [5]. It has also been documented that low serum cholesterol concentration may lead to a behaviour that increases the risk of violent deaths[3].

Analysis of the various factors influencing the trends of unnatural deaths in a society, besides reducing the workload on the health care institutions, law enforcing agencies and judiciary; will also help the policy makers in planning better living conditions for the future generations, thereby contributing towards a better tomorrow.

Material & methodology

This was a prospective study conducted with an aim to establish a correlation between serum cholesterol levels and unnatural deaths in the Chandigarh Zone of North West India so that the results would enable us to provide the serum cholesterol levels as a prognostic factor in the follow up of cases of attempted suicide and as an advancement of the existing knowledge on the role of serum cholesterol in unnatural deaths. A total of 358 cases of unnatural deaths, which were brought to the mortuary of the department of Forensic Medicine, PGIMER, Chandigarh, for medico-legal autopsy were studied. Forty five healthy volunteers from the hospital staff, after due informed consent, were taken as controls for the study.

Following cases were included in the study:

1. Cases of unnatural deaths brought for medico-legal autopsy
2. Healthy volunteers

Following cases were excluded from the study:

1. Any pre-existing medical ailment like diabetes mellitus, coronary artery disease, hypertension, obesity or any other metabolic disorder.

Ten cc of blood was collected from the right femoral vein from the dead body during autopsy and from the right cubital vein of the control groups. Serum was separated from blood by centrifugation at 1500 rpm for 10 minutes and cholesterol levels were then estimated by enzymatic method using the kit "Infinite Cholesterol Liquid supplied by Accurex biomedical Pvt. Ltd., Thane, India.

Results

A total of 358 cases were included in the study. Of these, 279 (78%) were cases of deaths due to accidents, 65 (18%) were suicidal and 14 (4%) were homicidal deaths. Of the 279 accidental deaths, majority were due to vehicular accidents 187 (67%), followed by trauma 47 (17%) and burns 43 (15%). Majority of the suicides were due to poisoning 58 (89%), followed by burns 6(9%). Similarly, majority of the homicides were due to trauma 11 (79%) followed by burns 3 (21%). Gender wise, males accounted for 266 (74%) cases; the male : female ratio being 2.8:1. Table-1.

Taking the serum cholesterol levels into consideration, it was found that the mean cholesterol level in accidental deaths was 101.16mg%, with males showing a mean cholesterol level of 102.75mg%, and females, 95.37mg%. The mean serum cholesterol level in suicidal deaths was 98.49mg%, being 102.67mg% in males and 92.97mg% in females. Homicidal deaths exhibited a mean cholesterol level of 88.09mg% with males showing 79.64mg% and females 109.2mg%. However the controls showed a mean cholesterol level of 176.55mg%, being 173.31mg% in males & 182.44mg% in females. Table - 2., Fig-1

As regards the different age groups, the <18yrs group showed mean cholesterol level of 83.7 mg% in suicidal deaths, followed by 94.5 mg% in accidents and 98.7 mg% in homicides; whereas the control group in this age groups showed a mean cholesterol level of 162.5 mg%.

In the 18-29, 40-49 and >50 yrs age groups, cases of homicidal deaths exhibited the least mean cholesterol level i.e.76.8 mg%, 69.9 mg%, 90.6 mg%, respectively, compared to the controls which exhibited the mean cholesterol levels of 146.8 mg%, 180.5 mg% and 189.6 mg%, respectively.

In the 30-39yrs age groups, the cases of accidental deaths showed the least mean serum cholesterol level of 99.3 mg% as compared to 181.1 mg% in the control.

Taking all the categories into consideration, cases of homicidal deaths in the age groups 40-49 yrs exhibited the least mean serum cholesterol level of 69.9 mg%.

Discussion

Lowering of cholesterol is popular due to its direct role in the causation of ischaemic heart diseases. Restricting saturated fat and cholesterol is a safe way to reduce the risk of coronary heart disease and other atherosclerotic disorders[6]. However, evidence suggests that

Corresponding Author:
DR Y S Bansal
Assoc. Prof, Department of Forensic Medicine, PGIMER, Chandigarh
E-mail: yogender_bansal@rediffmail.com

naturally low or clinically reduced cholesterol is associated with increased non-illness mortality. Investigations in monkeys reveal that reductions in plasma cholesterol increase the tendency to engage in impulsive or violent behavior through a mechanism involving central serotonergic activity[7]. Similarly, many studies have shown an unusual correlation between lowering of cholesterol levels with that of unnatural fatalities, notably suicides[3,8]. Low cholesterol levels may predispose an individual to aggression, impulsivity, and violence[9] Many studies have found that patients with mood disorders have lower cholesterol levels[10]. The role for serotonin metabolism was suggested by Engelberg[11] and subsequently elaborated by Salter[12] to explain a possible association between low cholesterol levels and violent death.. Low serotonin concentrations have been observed in depression and suicide.

In a study of patients with antisocial personality disorder[13] and in another study of adolescent boys with conduct disorder [14], there was an association between low cholesterol and violent behavior. Of the two studies conducted at the Whiting Forensic Institute, one involved 50 patients admitted to the forensic hospital for violent crimes [15]. The 21 subjects found to be "more violent" had lower cholesterol levels than the 29 "less violent" subjects. The second study involved 106 patients admitted for crimes of violence [16] . The group with lower cholesterol had a higher mean frequency of aggressive incidents compared with the high-cholesterol group. These studies suggest that lower cholesterol levels are related both to the frequency of violence and to its intensity.

In this study , we observed a highly significant and strong association between lower cholesterol levels and deaths due to unnatural fatalities($P<0.001$) when compared with controls. Although serum total cholesterol levels were correlated with age, the primary finding was independent of age or sex . The finding confirms the utility of examining the association between cholesterol level and unnatural fatalities in this zone. The results extend the discussion of low cholesterol beyond unnatural deaths to violent behavior. Currently, one popular hypothesis for the association is the role of cholesterol in serotonin reuptake[17]. However, this study was not designed to explain this hypothesis. The author is also working on a project to assess the role of serotonin in

Table 1: Sex wise distribution of the cases with respect to the manner of death

		Male	Female	Total			
	Mannerof death	No	%age	No	%age	No	%age
Accidental	Burn	19	88.2	24	11.8	43	15.41
	Trauma	33	70.2	14	29.8	47	16.8
	Poison	2	100.0	-	-	2	.7
	R.T.A.	165	88.2	22	11.8	187	67
	Total	219	78.5	60	21.5	279	77.9
Homicidal	Trauma	10	10.9	1	9.1	11	78.5
	Burn			3	100	3	21.4
	Total	10	71.4	4	28.6	14	3.9
Suicidal	Poison	37	63.8	21	36.2	58	89.2
	Burn			6	100	6	9.2
	Hanging			1	100	1	1.6
	Total	37	56.9	28	43.1	65	18.2
Grand total		266	74	92	26	358	100
Control		29	64.4	16	35.6	45	12.4

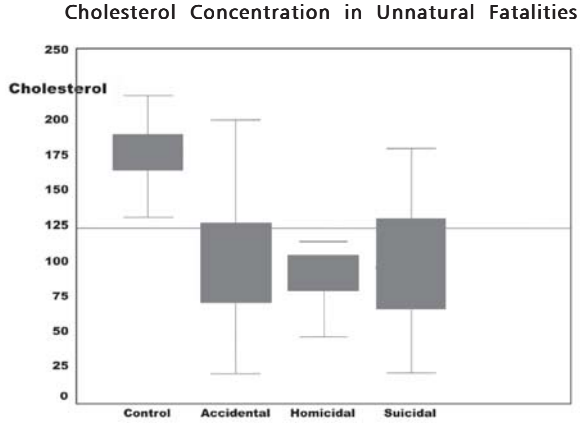
Table 2: Cholesterol concentration in the various categories of case under study

		Male			Female			Total		
	Manner	No. of case	Mean Cholesterol (mg%)	p-value (with control)	No. of case	Mean Cholesterol (mg%)	p-value (with control)	No. of case	Mean Cholesterol (mg%)	p-value (with control)
Accidental	Burn	19	94.83 ±35.9651	8.033***	24	88.7442 ±36.1369	10.150***	43	91.4344 ±35.7608	12.67***
	Trauma	33	105.5706 ±55.9106	6.130***	14	102.4014 ±46.2079	5.911***	47	104.6266 ±52.7273	8.33***
	Poison	2	120.3500 ±81.5294	0.915ns				2	120.3500 ±81.5294	0.973ns
	R.T.A.	165	102.8842 ±46.1047	11.100***	22	98.1250 ±38.2538	8.547***	187	102.3243 ±45.1864	14.47***
	Total	219	102.7500 ±46.9552		60	95.3705 ±39.1511		279	101.1630 ±45.4254	
Homicidal	Trauma	10	79.6400 ±25.2832	9.803***	1	93.4000	excluded	11	80.8909 ±24.3419	11.49***
	Burn				3 ±35.3023	114.4667	3.218ns	3 ±35.3023	114.4667	2.991ns
	Total	10	79.6400 ±25.2832		4	109.2000 ±30.6885		14	88.0857 ±29.1878	
Suicidal	Poison	37	102.6700 ±42.2255	8.127***	21	96.5705 ±40.1219	8.283***	58	100.4616 ±41.2271	11.382***
	Burn				6 ±31.2846	91.1750	6.554***	6 ±31.2846	91.1750	6.390***
	Hanging				1	28.0000	excluded	1	28.0000	excluded
	Total	37	102.6700 ±42.2255		28	92.9654 ±39.2530		65	98.4895 ±40.9440	
Control		29	173.3117 ±28.1745		16	182.4375 ±22.2001		45	176.5564 ±26.3188	

***p<0.001, **p<0.01, *p<0.05, ns- non significant

Table 3: Serum cholesterol concentration with control in the various categories of cases under study, age wise.

Age(	Accidental		Homicidal		Suicidal		Total		Control
in years)	Male	Female	Male	Female	Male	Female	Male	Female	
<18	92.2035	98.0987	83.3000	114.0000	93.2000	77.9620	91.98	94.06	162.5000
	±46.9762	±40.8815	.	.	±9.7734	±45.2991	±43.33	±40.95	±3.5355
	23	15	1	1	3	5	27	21	2
18-29	109.3300	99.7065	70.3200	86.4000	102.0963	96.2270	106.87	96.76	146.8438
	±66.9840	±40.2830	±30.5900	±9.8995	±46.7398	±38.1524	±61.67	±39.13	±30.0186
	56	23	3	2	19	23	78	48	8
30-39	101.7226	87.4250		150.0000	101.1860		102.68	98.43	181.0921
	±43.8404	±35.9008		.	±40.7879		±43.05	±38.80	±20.0452
	39	8		1	10		49	9	19
40-49	106.5602	93.5800	69.8500		108.6333		106.53	98.21	180.5440
	±37.8534	±31.9634	±32.7390		±48.2763		±38.76	±31.65	±15.7316
	45	6	2		3		50	6	5
50-59	97.7146	76.5250	102.8400		120.8000		97.51	76.53	189.5683
	±26.7010	±50.0214	±5.8272		±63.0739		±25.21	±50.02	±23.3311
	26	4	3		2		31	4	11
>60	98.5370	97.6300	53.9200				99.19	99.30	
	±29.0918	±48.9601	.				±31.12	±42.57	
	30	4	1				31	4	



suicides. As such, cholesterol levels were determined from the blood samples collected at the time of post-mortem examination and not when the incident happened. However, it is unlikely that cholesterol levels fluctuate quickly. Similarly no correlation was found with time since death also.

Conclusion

Serum cholesterol concentration was significantly lower in the accidental, suicidal & homicidal group. Despite the robustness of these findings, the possible implications of low cholesterol levels having any behavioral changes, has gained relatively little attention among medical practitioners.

Our findings are limited due to the small sample size. However, our finding of an independent association between unnatural fatalities and low cholesterol levels is highly statistically significant . It appears that low total serum cholesterol levels might be potentially useful in predicting risk for aggression or suicides. Further studies with larger sample size and different zones are needed to replicate the findings and enhance its generalization with respect to other groups.

References

1. Frick MH, Elo O Hapa K, et al. Helsinki Heart study. Primary prevention trial with gemfibrozil in middle-aged men with dyslipidemia. N.Engl J Med 1987;317:1237-1245.
2. Lipid Research Clinics Program: The lipid research clinics coronary primary prevention trials results: I. reduction in the incidence of coronary heart disease. JAMA 1984;252:351-364.
3. Muldon MF, Manuck SB, Matthew KM. Lowering cholesterol concentrations and mortality: a quantitative review of primary prevention trials. BMJ 1990; 301:309-314.
4. Santiago JM, Dalen JE: Cholesterol and violent behavior. Archives of Internal Medicine 1994;154:1317-1321.
5. Frantz I D, Dawson EA, Ashman PL. Test of effect of lipid lowering by diet on cardiovascular risk. Atherosclerosis 1989;9:129-135.
6. Dayton S,Pearcee M L,Hashmoto S, Dixon W J,Tomivasu U. A controlled clinical trial of a diet high in unsaturated fat in preventing complication of atherosclerosis. Circulation 1969;39-40 (Suppl II);1-63.
7. Kaplan J R, Muldoon M F, Manuck SB.Assessing the Observed Relationship between Low Cholesterol and Violence-related Mortality.New York Academy of Sciences. 1997;836:57-80.
8. Lindberg G, Rastam L, Guliberg B, Eklund GA. Low serum cholesterol concentration and short term mortality from injuries in men and women.BMJ 1992;305:277-9.
9. Golomb BA. Cholesterol and violence: is there a connection? Ann Intern Med. 1998;128(6):478–487.
10. Pae CU, Kim JJ, Lee SJ, et al. Aberration of cholesterol level in first-onset bipolar I patients. J Affect Disord. 2004;83(1):79–82.
11. Engelberg H. Low serum cholesterol and suicide. Lancet 1992;339:727-9.
12. Salter M. Low serum cholesterol and suicide. Lancet 1992;339:1 169.
13. Virkkunen M: Serum cholesterol in antisocial personality. Neuropsychobiology1979; 5:27- 30.
14. Virkkunen M, Pentinnen H: Cholesterol in aggressive conduct disorder: a preliminary study. Biological Psychiatry 1984;19:435-

Is informed consent sole responsibility of the doctor?

Abhay Shete

Visiting Consulting Gynecologist Obstetrician & Infertility Specialist & Lecturer Bhagubai Changu Thakur College of Law, PITRUASHISH"Plot.6, Road 2, Sector 19, Near Fire Station, Panvel, Raigad 410206, Maharashtra, India

Abstract

The law as it stands today has placed responsibility of "informed consent" squarely & firmly on the doctors, lack of taking it amounting to negligence which is separate from duty of care. The doctors on the other hand find it difficult to stand up to the legal expectations as can be seen from recent judgments. Lack of established norms, illiteracy, different social, cultural & traditional practices makes the task of the doctors more difficult. The doctors have a long way to go starting with adopting the patient autonomy approach & doing away with the paternalistic pattern of managing patients. They will have to take help of family elders, community leaders & social workers in explaining the current trends in medical field before implementing the surgical methods & procedures failing which the issues may snowball in to major issues detrimental to the doctor-patient relationship. The State can help the doctors by doing its duty of educating its subjects as mentioned in the Directive Principles of the Constitution of India. A well informed patient is the ultimate solution to the vexatious litigations faced by the doctors. The role of the State cannot be undermined considering the political, infrastructural & financial resources at its disposal. Finally the doctors should not compromise the treatment based on patient autonomy approach & take a firm stand to deter the options that are detrimental to the health & wellbeing of the person & the society. The legislators, administrators & the judiciary should come together & take steps to set limits & standards of disclosure before the doctors' start resorting to defensive practice which is not in good interest of the society at large.

Introduction

The origin of the concept of consent dates back to the days of Greek and Roman laws when life was deemed to belong to man as his self-owned property with unquestionable rights. Man then had complete dominance on life and personal liberty of himself, his wife, children and slaves, giving him exclusive powers to consent to any injury being caused or bartering them away for pecuniary benefits. Thus suicide was considered as inherent right of every free man and 'might is right' was the predominant unwritten code of conduct. Later on as state took the responsibility to protect every individual, the entire notion of ownership underwent a sea change. Consent was defined to mean "to agree to do something; to give ones permission to do something" or to mean concurrence of the will coupled with conciseness or comprehension of the act which is consented to. It also meant "voluntary agreement, compliance or permission". However, mere surrender to the will of another or submission, void of the element of voluntariness would not amount to consent; that is, the every consent involves submission but mere submission by no means amounts to consent.[1] Under section 13 Indian contract act, consent is defined as two or more persons agreeing upon "the same thing in the same sense". Section 87 to 92 of Indian Penal Code relate to the consent in criminal law. It revolves on the premise that a person can consent to any harm, short of death or grievous hurt. Therefore, when a surgeon operates on a patient likely to cause his/her death but not intending to cause it, and acting in good faith for the benefit of the patient, commits no offence provided a valid consent was obtained.

Evolution of informed consent

Informed consent law developed from the intentional tort of battery. This law protects the individual from unwanted physical touching of the body by one having neither express nor implied consent of the person touched nor a privilege to do so. In a medical setting battery is said to occur when the physician performs a treatment without the consent of the patient or carries out a substantially different procedure than the one for which consent was given or exceeds the scope of the consent or a different physician than the one to whom the consent was given carries out the procedure. As informed consent was increasingly recognized basis for physician liability, the courts reasoned that any consent based on inadequate information was vitiated by the failure of the physician to disclose and therefore that an unconsented touching occurred characteristic of a battery. However characterizing the tort in battery placed too great a burden on the medical profession and didn't emphasize enough on regulatory approach of law. The Phrase 'informed consent' itself is from the case Salgo v. Leland Stanford Jr. University Board of Trustees[2] which is regarded to have given birth to the doctrine of informed consent. In the instant case the doctor failed to warn the patient of the risk of paralysis inherent in the performance of the operation as a result of which the patient suffered severe paralysis of the lower limbs. The Court observed:

"A physician violates his duty to his patient and subjects himself to the liability if he withholds any facts which are necessary to form the basis of an intelligent consent by the patient to the proposed treatment."

This was one of the first cases to acknowledge that a patient needs adequate information about the nature of proposed treatment, the risks & the alternatives to make an intelligent decision about whether or not to undergo the treatment.[3] According to Robertson[4] the doctrine of informed consent means that a doctor is required to give his patient sufficient information about proposed treatment so as to provide him with the opportunity of making an 'informed' or 'rational' choice as to whether to undergo treatment.

What is informed consent?

Informed consent is the legal embodiment of the concept that each individual has the right to make decisions affecting his or her well-being. It is generally accepted that individuals should consider the risks and potential benefits flowing from their decisions. To do so, decision makers should have knowledge of those risks and potential benefits. The law protects the individual's right to give informed consent by requiring the disclosure of information by the party to whom consent is given. In case of the doctor patient relationship, the onus of disclosure of information lies with the doctor and the right to decide the manner in which his/her body will be treated lies with the patient. Hence, it becomes the duty of the doctor to disclose information on the risk emanating from the treatment to the patient. Risk may be defined as 'exposure to a chance of injury or loss'.[5]

In Samira Kohli v.Dr.Prabha Manchanda and Anr.,[6] the two issues raised before the bench were:

1. Whether informed consent of a patient is necessary for surgical procedure involving the removal of reproductive organs?
2. When a patient consults a medical practitioner, whether consent given for diagnostic surgery can be construed as consent for performing additional or further surgical procedure, without the specific consent for such additional or further surgery?

The Supreme Court in its decision held that, consent in the context of a doctor-patient relationship, means grant of permission by the

patient for an act to be carried out by the doctor, such as a diagnostic, surgical or therapeutic procedure. Consent can be implied in some circumstances from the action of the patient, consent form for Hospital admission and medical treatment, to which Appellant's signature was obtained by the Respondent can safely be presumed to constitute the contract between the parties. In Medical Law, where a surgeon is consulted by a patient, and consent of the patient is taken for diagnostic procedure/surgery, such consent cannot be considered as authorization or permission to perform therapeutic surgery either conservative or radical (except in life threatening or emergent situations). Similarly where consent by the patient is for a particular operative surgery, it cannot be treated as consent for an unauthorized additional procedure involving removal of an organ, only on the ground that such removal is beneficial to the patient or is likely to prevent some danger developing in future, where there is no imminent danger to the life or health of the patient. Doctor is required to communicate all inherent and potential hazards of the proposed treatment, the alternatives to that treatment, if any, and the likely effect if the patient remained untreated. Standard of disclosure is however subject to only two exceptions viz. where there was a genuine emergency and where the information would be harmful to the patient. Consent given only for a diagnostic procedure, cannot be considered as consent for therapeutic treatment. The Court established the Principle of Necessity - Restriction which is restricted to cases where the patient is temporarily incompetent (being unconscious), to permit the procedure delaying of which would be unreasonable because of the imminent danger to the life or health of the patient.

The consent should be 'express consent' & its nature differs from the 'real consent' in UK, to the 'informed consent' in America. In UK, the elements of consent are defined with reference to the patient & consent is considered to be valid & real when:

i. The patient gives it voluntarily without any coercion,
ii. The patient has the capacity & competence to give consent &
iii. The patient has the minimum information about the nature of the procedure to which he is consenting to.

On the other hand, 'informed consent' developed by the American courts, while retaining the basic requirements of consent, shifts the emphasis on the doctor's duty to disclose the necessary information to the patient to secure his consent. "Informed Consent" is the consent that is given by a person after receipt of the following information:

i. The nature & purpose of the proposed procedure or treatment.
ii. The expected outcome & the likelihood of success.
iii. The risks, the alternatives to the procedure & supporting information regarding those alternatives
iv. The effect of no treatment or procedure, including the effect on the prognosis & material risks associated with no treatment.
v. Instructions regarding what should be done in case of failure of procedure or becomes harmful.

In Nizam Institute of Medical Sciences v, Prasanth S Dhanuka & Ors.[7] A young 20 year old student developed paraplegia following the surgery done for removal of neurofibroma by a cardiothoracic surgeon as the tumor was in the thoracic cavity. The surgery was preceded by failed attempts to biopsy the tumor under ultrasound & CT scan control. The cardiothoracic surgeon, who operated on the patient had taken consent for excision biopsy but extended the scope of surgery by removing complete tumor along with 4th rib thereby destroying the intercostal vessels leading to paraplegia. The National Commission held that consent given by the complainant for excision biopsy cannot be taken as an implied consent for surgery save in exceptional cases. The neurosurgeon was not referred the case preoperatively & it was contended that the spread of the tumor into the spinal area made it mandatory for the neurosurgeon to be involved right from the beginning & not at a belated stage. The National Commission observed that the case should have been referred to both the cardiothoracic and the neurosurgeon preoperatively, a view which was upheld by the Supreme Court. The Court laid emphasis on the inviolable nature of the patient's right in regards to his body & his right to decide whether he should undergo the particular treatment or surgery or not. The Supreme Court remarked that a doctor cannot extend the scope of surgery without the consent of the patient at the risk of repetition except in order to save the life or preserve the health of the patient. The Supreme Court held the cardiothoracic surgeon negligent for not consulting the neurosurgeon preoperatively & for attempting to remove it without the assistance of a neurosurgeon.

Objections to doctor being solely responsible for informed consent

1. The concepts & technological advances specializations & super-specializations have rapidly changed the treatment modalities & options. There are wide ranges of therapeutic options available each with marginal benefit over the other. Given the fact that the doctor explains all the options, the patient who fails to respond to the chosen line of therapy will attribute the lack of success to the ill-chosen therapy & blame the doctor for not providing correct information to make a competent choice.
2. The law is clear that no physician can absolutely avoid liability under the informed consent laws unless he/she discloses every known risk & alternative to every patient. However the Courts in UK, US & India have failed to enumerate clear limits as to the level of disclosure of information that would constitute informed consent.
3. As the physician has pressures to be productive & lacks the time required for in-depth counseling, it is easier not to counsel. Most medical encounters involve decisions that are less clear-cut & physicians have difficulty in fully informing patients about treatment options. Some patients tend to rely too much on physician opinion & several studies have identified that as many as 2/3rd of patients prefer that physician make the decision for them because they lack the competence.
4. Taking of informed consent is not necessarily in the interest of the patient. In T.T.Thomas v. Elisa Thomas[8] the judge observed: "Why should a doctor insist on informed consent is not for the safety of the patient, but for the protection of the physician or the surgeon as the case may be. Every surgery, whether minor or major, is fraught with some degree of hazard or risk which varies in accordance with the seriousness of the disease. If a patient collapses during the course of a surgery or during the course of a treatment, law gives protection to the medical man, provided he establishes that the risky step was adopted with the express or implied consent from the patient. The consent factor may be important very often in cases of selective operations which may not be eminently necessary to save the patient's life."
5. In K Alemelu v Govt. of Tamil Nadu[9] the High Court remarked "Most of the patients in India cannot understand medical terms, concepts & treatment procedures. They accept whatever is done in their interest based on rough & ready diagnosis of doctor's intuition or experience. The stark reality is that for a vast majority in the country, the concept of informed consent & choice in treatment has no meaning or relevance". It is fact that what the patient really understands is questionable given the poor tolerance to failure of desired consequences of treatment especially among the illiterate Indians. After explaining all the risk to the patient in a language he/she understands, the willingness of the relatives to accept the unfortunate outcome is not forthcoming. This is reflected in violent attacks seen on doctors & establishments causing law & order problems which unfortunately are not adequately handled by the law enforcing agencies.
6. The doctors are at the end of the corrective system in the chain of events from causation of the disease to its manifestation & culmination in to recovery, disability or death of the patient. Why the doctors should be held responsible for not giving favorable result to the manifested disease when those who caused the disease are left to go scot free? Take for example carcinoma of the bronchus caused by smoking tobacco or by inhalations of the pollutants by a patient. Assuming the patient dies during the

surgery or due to anaphylaxis caused by the administered drugs, why should the tobacco company or the pollution causing industry not be held responsible for causing the cancer in first place? Just because the doctor happens to face the patient's relatives doesn't mean that he or she has to bear the brunt of the entire chain of events that started with harvesting of tobacco, manufacturing of cigarettes, smoking habit of the masses, failure of the government to adequately curb the menace ending with patient developing cancer & having to face the risk of surgery.

7. The doctor treating the patient is party to the contract. The law has put the responsibility of explaining the risk of treatment squarely on the treating doctors. It is assumed that the patient has the capacity to understand & choose the treatment based on intelligent choice made after deliberate discussions with other members of the family & the treating doctor. The doctor also is expected to voice his opinion & concerns on the choice of treatment to be exercised by the patient. How can the doctor ensure that the patient has understood the treatment modalities or the options clearly & is making an intelligent choice? The choice made may not be based on the facts & the opinion of the doctor but on many other factors.
8. What is the doctor supposed to do if he is not in agreement with the option chosen or there are reservations on the choice of treatment options by the patient? In the event of unexpected results the patient would seek cover of inadequate information given by the treating doctor. Can one of the parties to the contract be given the entire responsibility of ensuring the terms of contract while the other is given the privilege of seeking cover under the guise of having not been given proper & specific information? It would not be a fair contract & the ideal situation would be both the parties agree to the terms in same light & in same the same sense. For this to happen, the patient should seek information of the disease, treatment options and outcomes not from the treating doctor but from independent source before coming to the table to sign the informed consent contract.
9. The State is supposed to perform its duty of educating its subjects according to the Directive Principles enshrined in the Constitution of India. Extending the role of the State in educating its subjects, if the patient fails to understand the nuances of treatment & the possible outcomes can it be a mute spectator to the unfortunate litigations? Is it not the duty of the State to accept the responsibility of educating its subjects on various issues related to health and to protect the rights of both the parties to the contract? The current situation is lop-sided with the State & the judiciary assuming that the patient being in a fiduciary relationship with the doctor may be exploited for commercial gains. Such assumption is against natural justice as the doctors are presumed to be guilty of hiding information from the patient, which in the first place should have been available to the patient from independent sources.

Seeking solution to the complicated issue of informed consent

The current practice of holding the doctors solely responsible for taking informed consent is neither good in law nor is in the interest of the society. The doctors feel that they are penalized no matter how they counsel the patients. As most of the patients are interested only in the result & hear only the positive aspect of the counseling before opting to get treated, he/she feels that the doctor failed in proper counseling & cheated them. One of the solutions is that the State can take on the responsibility of imparting the information required by a patient to make decisions. The State has the required infrastructure in the form of post offices, revenue offices, schools having primary & secondary school teachers. The senior officers in these places can be taught to have access to the information on the net or in an officially published manual that explains the disease process, procedure to be adopted & all the favorable & unfortunate outcomes. The person who needs information should approach these people, who will grant them the access to the web site or manual & help them understand the core issues involved. As these officers & teachers belong to the same cross section of society as the patient, they will be in a better position to bring out communication gaps in the doctor, patient & his/her relatives. In case of complications or unfavorable outcomes of the treatment, these officers can mediate between the aggrieved patient/relatives to settle the dispute thereby reducing the litigations. More importantly these officers may serve to endorse the disputed aspect of informed consent. The patient cannot later on claim that he/she was not adequately informed or was misinformed. Importantly Right to Education now being made a fundamental right the State will fulfill its obligation towards the same. Education does not mean only formal education & health education is a part of the whole aspect of patient education. These attempts of imparting education will be helpful in the long run as they can be used successfully for imparting information on preventive aspects of disease control. The State having the necessary funds & the infrastructure, can certainly implement the above option thereby promoting a healthy doctor-patient relationship. By doing so, the State will be performing its duty of imparting health education to its subjects, which is their fundamental right. Medical councils of respective branches of medicine can be roped in to set up information details to be put up on the web or printed as manuals.

Conclusion

Informed consent cannot be taken as the sole responsibility of the treating doctor. The State should take upon itself to educate its subjects especially when Right to Education is fundamental Right. Faulting the doctor for not taking adequate informed consent when in fact the information given may not have been properly understood has negative impact on the providers of health services. This may prove detrimental in the long run and is not in the interest of the society. Some steps need to be taken actively to reduce the burden of providing information to the patients by the treating doctors.

Estimation of stature from the length of ulna in living adults

Abhilasha Wahane, M.P Fulpatil, R.A Kamble

Department of Anatomy, Indira Gandhi Government Medical College , Nagpur, Maharashtra

Abstract

An attempt was made to find out correlation and to derive regression formula between length of right and left Ulna and height of an individual in Vidarbha region of Maharashtra. The material consisted of 600 individuals, 300 Males and 300 Females between the age group of 18 to 23 years. Measurements were taken at a fixed time to avoid diurnal variation. The height was recorded with the help of an Anthropometer and length of both sided Ulna was recorded with spreading caliper. The observed data was subjected to statistical analysis. The result showed that there is a definite correlation between length of right and left Ulna and height of an individual. Simple linear regression equation can be used for estimation of height. However, if multiple regressions equations are used, the height can be better estimated.

Keywords

Estimation of stature, Length of Ulna.

Introduction

The study of skeletons is an exact science permitting the identification in terms of individual's age, sex, race and height (stature). The anthropometric study of bones convey information regarding race, sex, age and height of a person which is of an interest to the Anatomist in academic field as well as in Medico-legal work. To assess the height of an individual from measurement of different parts of the body has been of immense interest to anthropologists. For a long time height has been one of the factors in description of the impressiveness of an individual. Climate, Heredity and nutritional status of population are reported to have an effect on stature and length of long bones. The climate and dietary habits of the people of different regions of India vary. Racial and ethnic variations also exist in the population therefore opinions based on the study of residents of one state are not necessarily applicable to another state. Estimation of stature of an individual in Maharashtra (India), by using the formulae given by western workers involves an error of 5-8%. (Athawale, 1963)[2]. Many of the previous authors have worked on the cadavers received in the Anatomy departments. Opinions differ as to whether the cadaveric length is the same, or more, than the height in the living. According to Trotter and Gleser (1952)[11] the increase in height after death is 2.5 cm when the measurement is taken in recumbent posture.

Telekka(1950) [10]worked on bones of Finns expressed the opinion that each racial group needs a separate formula for estimation of stature. John K Lundy(1983,1985)[6,7] discussed the regression equation and mathematical and anatomical method of estimating stature from long limb bones. Various workers have shown significant correlation between height and different parts of the body. The forearm long bone Ulna is mostly subcutaneous throughout its length and easily approachable to measure, hence it is selected for the present study. The ossification of the upper limb long bone is completed by the age of 18 to 23 years.

Material and method

In the present study 600 students were taken. Males and Females constituted the equal number. The age group taken was from 18 to23 years. The subjects taken for the study were Medical students, Nursing students of Indira Gandhi Government Medical College Nagpur and students of other faculties. The subjects with any obvious congenital or acquired deformity of spine or extremities were not included.

Measurement technique

For measuring the height of the subject an Anthropometer fixed against wall was used. The subject was asked to stand against the anthropometer with feet together and bare, trunk braced, eyes forward and lateral Palpebral Commissure and the tip of the auricle in the same horizontal plane. A scale was placed on the head of the subject to note the exact reading on the anthropometer.The reading thus obtained was taken as standing height of an individual. Length of Ulna was measured with the help of spreading caliper from tip of olecranon process to tip of styloid process with elbow flexed and palm spread over opposite shoulder. All the above measurements were taken at a fixed time to eliminate discrepancies due to diurnal variation. These measurements were taken three times and their Mean value was taken as final measurement. The data so collected was then subjected to statistical analysis.

Observations

The statistical data obtained from the calculation and analysis were tabulated in table 1 to 4. The correlation coefficient of height and length of right Ulna is 0.87368 (r_1), height and length of left Ulna is 0.86044 (r_2) for males. The correlation coefficient of height and length of right Ulna is 0.85 (r_1), height and length of left Ulna is 0.83 (r_2) for females. The value of (r_1 and r_2) implies that there is positive correlation.

Table 1: (Observations and Calculations for Males)

s.no	Variable	Average	Std.dev.	Summation	Summation of square	Summation of product	Coeff. of variation
1	Height(y)	169.81	7.2525	50944.1	8666732		0.0427
2	Length of right ulna($x_{1)}$	27.005	1.1364	8101.5	219167	1377899	0.04208
3	Length of left ulna($x_{2)}$	26.9	1.1302	8068	217357	1372166	0.04203

Table 2: (Observations and Calculations for Females)

s.no	Variable	Average	Std.dev.	Summation of square	Summation of product	Summation of variation	Coeff.
1	Height(y)	155.27	6.1501	46582.7	7244469		0.0427
2	Length of right ulna($x_{1)}$	25.54	0.8770	7572.5	191372.5	1177201.6	0.04208
3	Length of left ulna($x_{2)}$	25.13	0.8850	7538.1	189644.1	1171835	0.04203

Figure 1:

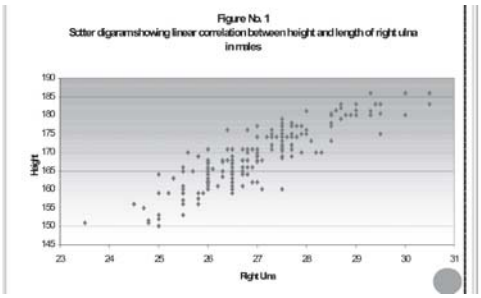


Figure 2:

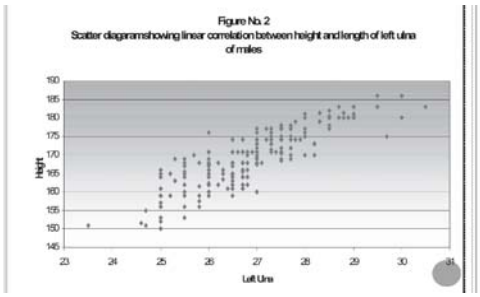


Figure 3:

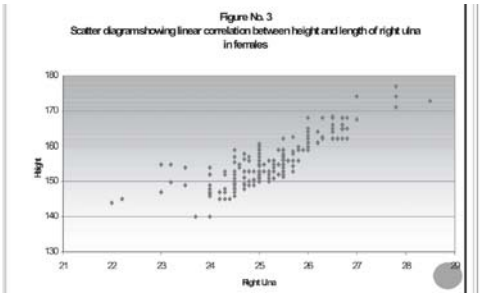
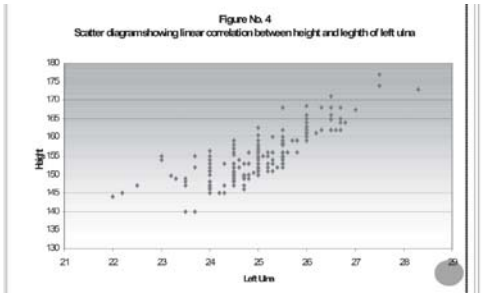


Figure 4:



't' test for correlation coefficient is applied to test the statistical significance. In males for Right Ulna (t=31), for Left Ulna (t=29.15). In Females for Right Ulna (t=28.29), for Left Ulna (t=25.72). The value of 't' is found to be statistically significant. The calculated value of t is significant at 5% level of significance than the tabulated value. Simple linear regression formulae were derived.

Estimation of height from right Ulna and Left Ulna respectively for males

Est $y_1 = 19.41 + 5.58\ X_1 \pm 6.93$

Est $Y_2 = 21.15 + 5.52\ X_2 \pm 7.25$

Estimation of height from right Ulna and Left Ulna respectively for Females

Est $Y_1 = 4.18 + 5.98\ X_2 \pm 6.29$

Est $Y_2 = 10.31 + 5.76\ X_3 \pm 6.72$

Alongside figure is the graphical representation where length of ulna is plotted on the X-axis and height on Y-axis. The scatter diagram clearly shows that linear relationship exists between X and Y. Thus there is positive relationship between x and Y.

Discussion

Estimation of stature from Ulna of an individual in Maharashtra by using the formulae given by western workers involves an error of 5-8% (Athawale, 1963)[2].Values for male sample nearly coincide with findings of the previous workers like M.C Athawale (1963)[2], N.B Joshi et al (1965)[3].there are no comparable female samples in these previous studies. In the present study scatter diagrams show that there exist positive linear relationship between height and length of Ulna. This holds true for male and female sample. These findings coincide with the study of P.M kolte and P.C Bansal (1974)[4] who worked on cadavers.

Mall G.et al (200) [8] studied sex determination and estimation of stature from the long bones of the arm and found maximum ulnar length (mean 26.5 cm in males, 23.8 cm in females).These findings nearly coincide with the present study.

Simple linear regression equations derived for estimation of height from length of Ulna from Vidarbha region (Maharashtra).

1) Estimation of height from right Ulna and Left Ulna respectively (for Males)

Est $y_1 = 19.41 + 5.58\ X_1 \pm 6.93$

Est $Y_2 = 21.15 + 5.52\ X_2 \pm 7.25$

2) Estimation of height from right Ulna and Left Ulna respectively (for Females)

Est $y_1 = 4.18 + 5.98\ X_1 \pm 6.29$

Est $Y_2 = 10.31 + 5.76\ X_2 \pm 6.72$

Conclusion

In the present study the value of 't' was statistically significant which indicates that height of an individual is related to length of Ulna. This holds true for sample size taken. Further statistical analysis signifies that height of an individual may be dependent on length of Right and Left Ulna at the same time. Simple linear regression equation derived can be used for Estimation of height. However if Multiple regression equations are used the height can be better estimated.

Bibliography

1) Allbrook ,D: The estimation of Stature in British and East African males based on the tibial and Ulnar bone lengths. Jour For.Med 8:1587,1961.
2) Athawale,M.C: Estimation of height from lengths of forearm bones. A study of one hundred Maharashtrian male adults of age between twenty five and thirty years. A.J.P.A 21: 105-112,1963.
3) Joshi N.B.:M.P.Patel,M.G.Amin and A.V.Dongre: Use of Tibia and Ulna in estimation of total body height.ind jour.Med.Res.53:831-834,1965.
4 Kolte P.M. and Bansal P.C: Determination of regression formulae for reconstruction of stature from the long bones in Maharashtrian of Marathwada region. Jour.Anat .soc.India, 23:6-11,1974.
5) Lal C.S and Lala J.k: estimation of height from tibial and Ulnar lengths in north Bihar. Jour .Ind. Med. Essn. 58(4),1972.
6) Lundy J.K.: Regression equations for estimating living stature from long limb bones in South African Negro, S. African journal science 79.337-338, 1983
7) Lundy J.K.: The mathematical versus Anatomical Methods of stature estimate from long bones. American Journal of Forensic Medicine and Pathology Vol.6 no.1 pp.73-76 March 1985.

8) Mall G.et al: Sex determination and estimation of stature from long bones of the arm. Department of Legal Medicine, University of Munich, Germany (2001)
9) Siddiqui M.A and Shah M.A: Estimation of stature from Long bones of Punjabis. Indian J.Med.Res.32:105-108, 1944.
10) Telekka A: On prediction of human stature from the long bones.Acta Anat.9: 103-111, 1950
11) Trotter M, and Glesser G.C: Estimation of stature from long bones of American Whites and Negroes.A.J.P.A.10:463-514, 1952.
12) Trotter M and Glesser G.C: A re-evaluation of estimation of stature based on the measurement of stature taken during life and of long bones after death. A.J.P.A 16:79-124, 1958.
13) Wells L.R estimation of stature from long bones. A reassessment Jour. For.Med:171-177, 1959.

Tissue microarray – A plethora of multiple data

Akhilesh Chandra*, Anil Singh**, Manjunath Badni***, Rohit Jaiswal****, Sarita Chaudhary****

*P.G. Student, **Professor and H.O.D, ***Reader, ****Lecturer, Department of Oral Pathology & Microbiology, Sardar Patel Post Graduate Institute of Dental & Medical Sciences, Lucknow, India

Abstract

Tissue microarray (TMA) technology enables high-throughput tissue analyses to keep pace with the rapid process of advance discovery. With this technique, up to 1000 minute tissue samples are brought into an array format and analyzed simultaneously. The TMA technology is a fast, cost-effective, and statistically powerful method that will substantially facilitate translational research. The ability to study archival tissue specimens is an important advantage as such specimens are usually not applicable in other high-throughput genomic and proteomic surveys.

Keywords

Tissue microarray, Immunohistochemistry, Squamous Cell Carcinoma

Introduction

Biomedical research is being revolutionized by the ability to carry out the investigations on a genome-wide scale.[1] Completion of the human genome sequence has provided the basic structural information on all human genes.[2] In the post-genomic era with the availability of vast information on the simultaneous expression levels of thousands of genes by high-density cDNA microarrays, a platform for high-throughput analysis of tissue specimens was inevitable. Tissue microarray (TMA) technology though initially utilized in target validation of cDNA and other high-throughput approaches, is now becoming an invaluable tool for clinical research.[3]

The conventional investigation of fresh frozen or paraffin wax embedded tissues is too expensive and time consuming to be applied to the characterization of hundreds or thousands of genes or gene clusters associated with distinct tumor entities or other diseases. Thus, technique that can facilitate research on a large series of tissues in parallel in a single experiment was required.[4]

In microarray technique, first described by Kononen *et al.* in 1998, a high precision punching instrument was introduced, which enabled the exact and reproducible placement and relocalisation of distinct tissue samples.[4] TMA sections provide targets for parallel in situ detection of DNA, RNA, and protein targets, and consecutive sections allow the rapid analysis of hundreds of molecular markers in the same set of specimens.[5]

The horizons for the use of TMA technique are now widening, and further applications have been proposed for routine pathology purposes.[4]

Tma construction

For TMA construction, core tissue biopsies (diameter 0.6 mm; height 3–4 mm) are taken from hundreds of different "donor" paraffin-embedded tumor blocks and precisely arrayed into a new "recipient" paraffin block (45 × 20mm) using a custom-made precision instrument. These small tissue core biopsies are punched from selected regions of the donor blocks using a thin-wall, stainless steel tube sharpened like a cork-borer. Punched tissue blocks remain fully interpretable for all morphological and molecular analyses that may subsequently become necessary.[6]

Hematoxylin and Eosin stained sections overlaid on the surface of the donor blocks guide sampling from morphologically representative sites in the tumors.[2] The selection of cylinders with 0.6 mm diameters allows preservation of histologic information while as many as 1000 specimens can be arrayed in each recipient block, with minimal damage to the original blocks. Specimens are arranged in the recipient block in subdivisions to assist interpretation of the immunohistochemical analyses.[5]

An adhesive coated tape sectioning system assists in cutting the tissue microarray block. 5 μm sections of the resulting tumor TMA block are transferred to glass slides using the paraffin sectioning aid system supporting the cohesion of 0.6- mm array elements. At least 200 consecutive sections of 4 to 8μm thickness can be cut from each tumor array block.[5]

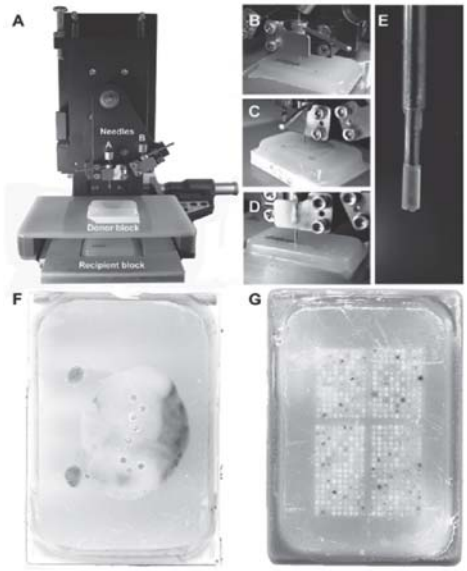


Fig. 1 Tissue microarray (TMA) construction.

(A) Arraying device with A and B two hollow needles. (B) A 0.6-mm hole is punched in the recipient block using needle B. (C) A tissue cylinder is punched from the donor block using needle A. (D) The tissue cylinder is released into the hole of the recipient block. (E) Tissue cylinder sitting on the tip of needle A. (F) Donor block showing only little damage even after multiple punches. (G) Completed TMA

Most of the work required for TMA relates to logistics. (*i*) Histologic samples meeting the requirements for the intended array must be identified from databases. (*ii*) All slides from all these cases must be collected from the slide archive and carefully reviewed by a pathologist. (*iii*) Representative areas must be marked on the slide to guide the person who will do the punching work. (*iv*) The corresponding paraffin blocks must be collected. Depending on the size of the planned TMA, thousands of glass slides and paraffin blocks may have to be moved. (*v*) A database must be generated that includes the specimen identifier and all histologic and clinical information. Comprehensive software solutions facilitate the high-throughput analysis and storage of TMA immunostainings.[6]

Dan et al. (2004) proposed a new methodology for TMA preparation and produced highly-qualified tissue chips of colorectal tumors. It was

claimed that using the refitted common microscope to produce tissue microarray is a simple, reliable, cost-effective and well-applicable technique.[7]

Types of tma

Virtually all types of analysis that can be applied to tissue sections, including immunohistochemistry (IHC), fluorescence in situ hybridization (FISH), RNA in situ hybridization (RNA-ISH) can be used on TMA sections. In principle, three different kinds of arrays have been utilized for this purpose; namely, prevalence TMAs, progression TMAs, and prognostic TMAs.

Prevalence TMAs contain tumor samples from one or several tumor entities without further clinicopathological information. They are used to evaluate the prevalence of a particular marker in a tumor population of interest. It could be used to comprehensively study the epidemiology of molecular features in all types of human neoplasia. In an early study, the known frequencies of amplification for cyclin-D1, c-myc, and *HER2* were confirmed in a multitumor TMA containing 17 different cancer types.

Prevalence TMAs can also be made from nondiseased normal tissues. Normal tissue TMAs are particularly instrumental for addressing questions related to pharmacology research. For example, if a new drug is developed, then it is crucial to explore the normal tissue distribution of the target protein to estimate potential side effects of the therapy. In diagnostic IHC it can also provide negative and positive control tissues.

Progression TMAs contain samples of different stages of one particular tumor type. Progression TMAs have been successfully utilized to detect associations between tumor phenotype and genotype in a wide variety of cancer types. Progression TMAs are also suited to study potential heterogeneity between primary tumors and their metastases.

Prognosis TMAs contain samples from tumors with available clinical follow-up data. They are optimally suited to evaluate suspected associations between genetic alterations and clinical outcome. TMAs have also been constructed from a variety of experimental tissues including cell lines or xenografts. For example, cell line TMAs can be easily made from formalin-fixed paraffin-embedded cell chunks and are especially suited to quickly identify cell lines expressing a particular gene for subsequent functional analyses.[6]

Applications

Various applications for TMAs can be summarized, including:

- Testing and optimization of probes and antibodies, improved utilization of pathology archives and tissue banks.
- International and other large-scale collaborations, e.g., studies of rare tumors or molecular profiling of tissues from multicenter clinical trial materials.
- Teaching and quality-control tool to improve standardized interpretation of morphologic, immunohistochemical, and molecular analyses in pathology.
- Standardized molecular or immunohistochemical detection of targets.
- Rapid translation of results from cell lines, xenografts, and animal models to human cancer.[5]

Quality control

Quality control in IHC is one of the major problems in daily practice. Even though IHC as been in use now for decades, there is still a high variability of intralaboratory and interlaboratory results, mainly because of differences in antigen retrieval, staining protocols, antibodies used, and in the interpretation of staining results. Therefore, approaches to guarantee a high level of quality are highly variable. The use of TMAs offers an alternative method of quality control for research and non-research purposes.[4]

In essence, two approaches to the use TMAs for these purposes have been described. A certain antigen profile with defined end points for a positive or negative staining result can be defined. The requirement for such quality improvements has been demonstrated repeatedly by TMA based quality assessment studies.

Another simple, but effective and reliable method has been described. With the use of internal control tissues in "mini-TMA format" some major problems in IHC could be reduced or even circumvented. This approach also offers an alternative for the usually time consuming work to evaluate new antibodies and reagents.[4]

Advantages

The TMA technique has a number of distinct advantages over traditional pathology methods using large sections. Improved standardization, capacity and speed of analysis, as well as the potential of automatization of array construction and analysis, are strong advantages of this technology.[5]

The TMA technology provides the platform to keep pace with high-throughput lead identification methods such as cDNA or protein arrays.[6]

Studies showing that clinicopathologic associations with molecular markers can reliably be identified on TMAs will be important to validate and increase the acceptance of TMA in the near future.[5]

Limitations

The most obvious drawback of this technology was initially believed to be the possibility that small tissue cylinders might not adequately represent the whole sections particularly in case of tumors because of intratumor heterogeneity of protein expression. Camp *et al*. reported that two cores of 0.6 mm from the same block of breast cancer provided equivalent information on estrogen receptor indices as the whole sections. Thus increasing the number of cores collected from the sample and increasing the core diameter to 1-2 mm can resolve this limitation.

Other practical limitations of TMA are related to loss of tissue cores during processing and the unreliability of IHC staining. The former can be prevented by using the adhesive tape transfer method and the later by frequent interlaboratory quality control checks.[3]

Tissue microarray analysis in head and neck squamous cell carcinoma

The squamous cell carcinomas of head and neck (HNSCC) are heterogeneous in terms of its distribution with each of these subentities having varied biologic and clinical characteristics. TMA by its high-throughput technology has demonstrated its power in revealing cellular alterations in large series of specimens in HNSCC, thereby determining its clinical outcome and prognostic potential.[8]

Freier *et al.* (2003) analysed the expression of cyclin D1, c-myc, erbb1 and erbb2 by IHC on a TMA containing 547 primary HNSCC. Cyclin D1 and c-myc were overexpressed at higher frequencies in primary pharyngeal and laryngeal carcinomas compared with primary oral carcinomas, while erbb1 and erbb2 overexpression was associated with oral site. Furthermore, cyclin D1 overexpression correlated with stage IV primary carcinoma suggesting that HNSCC as a heterogeneous group of tumors, depending on anatomic sites and clinical stage, having variable expressions of the oncoproteins.[9]

Karsai et al. (2007) performed an immunohistochemical study on TMA in one of the largest cohorts of 664 tumors to study the loss of p16INK4A protein expression and p53 alteration. Both loss of p16(INK4a) expression and p53 alterations differed significantly across both tumor sites and stages being more prevalent in the hypopharynx than in the other tumor sites and in advanced tumor stages. An increased DNA methyltransferase 1 protein levels occurred preferentially in tumors with aberrant p53 and negative p16INK4A expression. These changes were related and perceived to be important particularly in early carcinogenesis of HNSCC.[10]

Future development

The TMA technology has successfully passed the evaluation phase and is an established standard tool.[6] Major changes that can be expected in the field of TMA technology include automation of TMA construction and analysis. One of the advantages of TMAs for automated image analysis is the exact X-Y-positioning of each specimen. Automation could help to archive the raw image data and to support the decisions by pathologists.

A multitude of different possibilities seems realistic, and some are already in use. For example, the use of "paraffin wax tissue banks" in pathology departments for the retrospective evaluation of new tumor markers for individual patients.[4] This valuable research material can be used for organization of long-term tissue banking, for education purposes and facilitating multicentric studies.[3]

Tissue microarray technology has the potential to significantly accelerate molecular studies and has become one of the most promising tools in head and neck cancer research.[8]

Pathologists have to integrate this new technology to successfully establish their discipline as an integral component of the contemporary multidisciplinary approach to medical and surgical treatment of diseases.[5]

References

1. Nazmul-Hossain ANM, Patel KJ, Rhodus NL, Moser KL. Microarrays: Application in dental research. *Oral diseases 2008; 14: 25-29*
2. Kallioniemi OP, Wagner U, Kononen J Sauter G. Tissue Microarray technology for high-throughput molecular profiling of cancer. *Human Molecular Genetics 2001; 10(7): 657-662*
3. Avninder S, Ylaya K, Hewitt SM. Tissue Microarray: A simple technology that has revolutionized research in pathology. *J Postgrad Med 2008; 54: 158-162*
4. Packeisen J, Korsching E, Herbst H, Boecker W, Buerger H. Demystified . . Tissue microarray technology. *J Clin Pathol: Mol Pathol 2003; 56: 198-204*
5. Moch H, Kononen J, Kallioniemi OP, Sauter G. Tissue Microarrays: What Will They Bring to Molecular and Anatomic Pathology? *Advances in Anatomic Pathology 2001; 8(1): 14-20*
6. Simon R, Mirlacher M, Sauter G. Tissue microarrays. *BioTechniques 2004; 36: 98-105*
7. Dan HL, Zhang YL, Zhang Y, Wang YD, Lai ZS, Yang YJ, Cui HH, Jian YT, Geng J, Ding YQ, Guo CH, Zhou DY. A novel method for preparation of tissue microarray. *World J Gastroenterol 2004; 10(4): 579-582*
8. Radhakrishnan, Solomon M, Satyamoorthy K, Martin LE, Lingen MW. Tissue microarray – a high-throughput molecular analysis in head and neck cancer. *J Oral Pathol Med 2008; 37: 166-176*
9. Freier K, Bosch FX, Flechtenmacher C, et al. Distinct sitespecific oncoprotein overexpression in head and neck squamous cell carcinoma: a tissue microarray analysis. *Anticancer Res 2003; 23: 3971-3977*
10. Karsai S, Abel U, Roesch-Ely M, et al. Comparison of p16(INK4a) expression with p53 alterations in head and neck cancer by tissue microarray analysis. *J Pathol 2007; 21: 314-322*

Polymorphous low-grade adenocarcinoma: A case report

Arun Singh*, Bastian T.S.**, Ceena Denny E***.

*P.G. Student, **Professor and H.O.D. Department of Oral & Maxillofacial Pathology, Sardar Patel Post Graduate Institute of Dental & Medical Sciences, Lucknow, ***Reader, MCODS, Mangalore

Abstract

Polymorphous Low-Grade Adenocarcinoma is a rare, malignant salivary gland tumor, which is found almost exclusively in minor salivary glands. It is a relatively uncommon carcinoma which arises almost exclusively in minor glands, particularly of the palate. It is characterised by its morphological diversity and cytological uniformity. The striking histological feature is architectural diversity combined with benign cytologic feature.

Keywords

Polymorphous Low-Grade Adenocarcinoma (PLGA), Pleomorphic Adenoma (PA), Adenoid Cystic Carcinoma (ACC).

Introduction

Polymorphous low-grade adenocarcinoma (PLGA) is a malignant salivary gland tumour with a predilection for the minor glands and is composed of a wide variety of lobular and cribrifrom patterns, and has a low potential for metastasis.

PLGA was described by Freedman (1983) under the name of lobular carcinoma, and by Batsakis and others (1983) as terminal duct carcinoma. Evans and Batsakis (1984) eventually coined the term PLGA. [4]

This tumour occurs almost exclusively in the region of the minor salivary glands, and usually presents as a slowly growing, indolent mass. Soft tissue of the palate followed by the lips, buccal mucosa, tongue, floor of the mouth and pharynx are the most affected areas. An intraosseous lesion has been described once, and that lesion presented as a maxillary odontogenic cyst. The main histological aspects of PLGA are cytological uniformity and a broad spectrum of growth patterns within the same lesion. These growth patterns are represented by lobular, cribriform, tubular, trabecular, papillary and cystic structures. The use of immunohistochemistry should be considered when the presence of overlapping architectural, background and cellular features in a neoplasm of minor salivary gland presents the pathologist with a diagnostic dilemma.[6] PLGA is characterized by infiltrative growth, morphologic diversity and cytologic uniformity. It usually shows several overlapping histological patterns with pleomorphic adenoma (PA) and adenoid cystic carcinoma (ACC).[4]

PLGA affects people in the age range from 30 to 70, with a female predilection in a 2:1 ratio. It is rare in extraoral locations, including major salivary glands. Sixty percent of the cases occur on the hard or soft palate, followed by 13% of the cases occuring in the buccal mucosa, 10% in the upper lip, 6% in the retromolar area, and 9% in the rest of the oral cavity. The lesion is normally described as a painless, slow growing mass, covered by non-ulcerated mucosa. In some cases it may be adhered to deep planes and it can reach sizes between 1 and 4 cm.[5]

Case report

A 69 years old male patient reported to Department of Oral Medicine & Radiology with chief complaint of difficulty in swallowing and speech and palatal swelling since 6 months. It started as a small swelling and gradually increased in size, but patient neglected it as it was a painless swelling. His medical history was non-contributory. He had the habit of tobacco chewing for the past 30 years. He gave the history that the swelling grew quite rapidly in the past 2 months. Upon intraoral examination, (Fig 1) a firm painless swelling was observed about 3 cm in diameter mostly in the soft palate and partially boundering the hard palate which appeared well demarcated. The overlying oral mucosa was intact. Swelling which was mostly on the left side, had pinkish colour. The lesion was rubbery in consistency and no tenderness on palpation was observed. No regional lymphadenopathy was detected. The well circumscribed soft tissue mass was on soft palate mostly and It had no secondary changes. Occlusal radiograph revealed no bony destruction.

Histopathological examination the sections revealed tumour cells proliferating in islands cords and sheets (Fig.2). Many areas revealed tubular pattern and cribriform pattern of proliferation (Fig.3). The tumour consisted of cuboidal to columnar isomorphic cells in which nucleoli appeared inconspicuous. The stroma appeared hyalinized in

Fig. 1: Intra oral photograph showing palatal swelling crossing the midline.

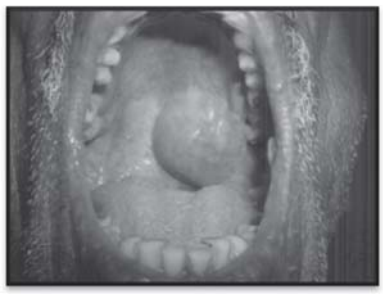


Fig. 2: The tumour cells prolifereating in tubular & cord pattern. X 20

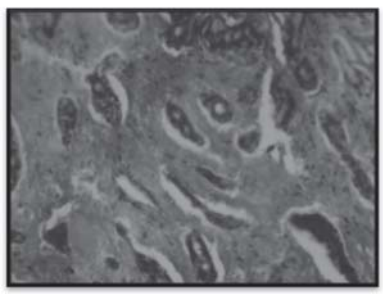
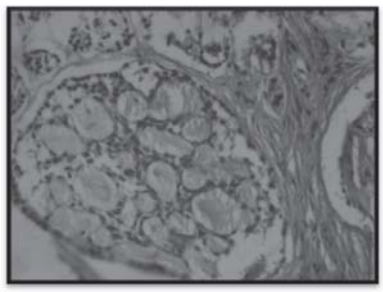


Fig. 3: The characteristic cribrifrom pattern of arrangement of cells X 40



many areas, however necrosis was not evident in any of the section.

Polymorphous low grade adenocarcinoma is a rare malignant epithelial tumour mostly related to minor salivary gland. In 1983, two separate groups of investigators reported on low-grade adenocarcinomas of minor salivary gland origin and termed as terminal duct carcinoma and lobular carcinoma. Terminal duct carcinoma was used to emphasize the proposed histogenesis of the tumor, which was thought to be the progenitor cell of the distal or terminal duct portions of the salivary gland unit, that is, the intercalated duct reserve cell. Lobular carcinoma was used because these salivary gland adenocarcinomas demonstrated areas of an infiltrative single - file growth pattern that is similar to that of the lobular carcinoma of the breast. In reality, these reports, although using different names, were describing the same neoplasm. In 1984, Evans and Batsakis described a group of oral minor salivary gland neoplasms and used the term polymorphous low grade adenocarcinoma, Which emphasized the salient features of these tumors, namely, the varied histomorphology and the malignant, albeit indolent, behavior. The authors stated that this group of neoplasms included those tumors previously termed terminal duct carcinoma, lobular carcinoma, papillary carcinoma and trabecular carcinoma.[2] In the second edition of WHO classification of salivary gland tumors published in 1991, it was classified under a separate category.[1]

At present, polymorphous low grade adenocarcinoma is the accepted term for this neoplasm. Diagnosis is usually late and can take weeks or even years because of its slow growth. This lesion can erode or infiltrate bone tissue. Histology shows a non-encapsulated lesion with infiltrative margins. It is named as polymorphous due to its different growth patterns: tubular, solid, papillary, microcystic, cribiform, fascicular, and cords. The tumour can infiltrate bone tissue and even present perivascular and perineural invasion.[5]

Microscopically, PLGA shows histopathologic features characteristic of many benign and malignant salivary glands neoplasms, particularly several overlapping histological patterns with pleomorphic adenoma (PA) and adenoid cystic carcinoma (ACC).[5] These diagnostic difficulties often occur during frozen section examination or when the biopsy is small. The cytomorphologic features can be quite similar, thus rendering a definitive diagnosis of PLGA of a small biopsy at the time of frozen section virtually impossible. Furthermore, because mixed tumors of minor salivary glands most often are unencapsulated, differentiation from PLGA based on that feature is not reliable. The distinction between PLGA and pleomorphic adenoma usually can be made by identifying the presence of infiltrative growth, especially when combined with the presence of neurotropism. Adenoid cystic carcinoma (ACC) can mimic the growth patterns identified in PLGA, especially the proclivity for perineural invasion. However, in contrast to PLGA, the cells in ACC tend to be smaller, with hyperchromatic nuclei, less cytoplasm, a higher nuclear-to-cytoplasmic ratio, and coarser nuclear chromatin. The differences in nuclear morphology are particularly striking and are nearly pathognomonic.[3] It is difficult to confirm PLGA only by histopathology, so it is necessary to perform immunohistochemical analysis. The immunohistochemical reactivity in PLGA includes consistent demonstration of cytokeratin (high and low molecular weight), epithelial membrane antigen (EMA), and S-100 protein. Both the luminal and nonluminal cells are reactive for these antigens. Carcinoembryonic antigen (CEA) and muscle-specific actin are variably immunoreactive. Glial fibrillary acidic protein (GFAP) immunoreactivity has not been consistently evaluated in PLGA. One report did not identify any immunoreactivity for GFAP in seven cases of PLGA, where as other studies identified focal positive immunoreactivity in limited cases.[2]

Electron microscopy has been used to identify the cell-types and their organisation of the salivary gland tumours. As for PLGA, variable results have been found. Dardick et al studied a PLGA from nose and demonstrated a gradual transition from myoepithelial cells which were associated with redundant basal lamina, excessive amounts of glycosaminoglycans and positive staining of S100 to luminal epithelial cells with the development of a true ductal type of lumen.[7]

The best treatment is surgical excision including the subjacent bone, if necessary. This surgery is frequently followed by radiotherapy. The prognosis is good and recurrence rate ranges between 17% and 24%. Metastasis is unusual (9%) but in case it occurs, it mainly affects regional lymph nodes.[5]

In our case, complete surgical excision was chosen as the treatment, since studies sustain that it provides an excellent long-term prognosis. Local recurrences may manifest after a long period, and this data suggests that close patient assessment for recurrent tumour is necessary. However in the present case no recurrences have been observed even after 2 years, following surgical treatment.

Bibliography

1. Arathi N, Atul M. Bage. Polymorphous low grade adenocarcinoma of parotid gland: a rare occurrence. Indian Journal of Pathology and Microbiology. 2009; 52(1): 103-105.
2. Gary L. Ellis; Paul L. Auclair; Douglas R. Gnepp; Surgical Pathology of the Salivary Glands – Volume 25 in the Series Major Problems in Pathology.
3. James T. Castle, Lester D. R. Thompson, R. Allen Frommelt, Bruce M. Wenig, Harvey P. Kessler. Polymorphous Low Grade Adenocarcinoma- A Clinicopathologic Study of 164 Cases. Cancer 1999; 86: 207–19.
4. Kazuko Takubo, Rieko Doi, Kazunori Kidani, Motoki Nakabayashi, Masayuki Sonoda, Fumihiro Ohtake, Isamu Kodani, Yasushi Horie and Kazuo Ryoke. Polymorphous Low-Grade Adenocarcinoma Arising at the Retromolar Region: A Rare Case of High-Grade Malignancy. Yonago Acta medica. 2007;50:17–22.
5. Maria Fernanda Pintor, Liberto Figueroa, Benjamin Martinez. Polymorphous low grade adenocarcinoma: Review and case report. Med Oral Patol Oral Cir Bucal. 2007 Dec 1;12(8):E549-51.
6. MHCG de Magalhaes, RP de Magalhaes, VC de Araujo1 and SOM de Sousa. Case Report Polymorphous low grade adenocarcinoma presenting an uncommon radiographic aspect. Dentomaxillofacial Radiology (2006) 35, 209–212.
7. Y-C Wei, C-C Huang, C-Y Chien, J-C Hwang, W-J Chen. Polymorphous low-grade adenocarcinoma of the nasopharynx: a case report and brief review. J Clin Pathol 2008; 61:1124-1126.

Cheiloscopy- a growing concept in forensic odontology

Kunal Jha*, Sabyasachi Saha**, G.V.Jagannath***, Sahana S****

*Post Graduate Student, I year Department of Preventive and Community Dentistry, Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow, **MDS Professor & HOD Department of Preventive and Community Dentistry Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow, ***MDS Reader Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow, ****MDS Senior Lecturer, Sardar Patel Post Graduate Institute of Dental and Medical Sciences, Lucknow

Introduction

The identification of the criminal or culprit and the victim in crime, accidents, mass disasters is of paramount importance from a social, emotional and legal view point. Lip prints have gained importance since there is a growing demand placed upon law enforcement to provide sufficient physical evidence to link a suspect to a crime or a contact between a victim and a suspect. Till date finger prints are considered to be the most important form of evidence but lip prints are gradually gaining importance in the forensic science arena.[1]

Any process that possesses the possibility of assisting the forensic field in identifying a suspect should be pursued and, if discovered pertinent, utilized in the act of criminal investigations and legal proceedings. The use of lip prints falls into this category and because they have been proved reliable and trustworthy to link a suspect to a crime, more emphasis should be given to this field.[2] Cheiloscopy is a forensic investigation technique that deals with identification of humans based on lips traces.[7] Lip print analysis or Cheiloscopy is a process that provides both qualitative and quantitative results thus its application in the forensic field should be widely accepted by both law enforcement and the legal professionals.

There is conclusive evidence that lip prints are suitable for the successful comparison, analysis and identification of a person to a crime. In fact there have been convictions of perpetrators who were positively identified via the analysis of their known lip prints to those found at the crime scene. Studies also indicate lip prints are classified as individual characteristics and similar to fingerprints, no two people possess the same prints.[3] Given this information, it is interesting to wonder whether lip prints are hereditary. The physical attributes regarding the shapes of family members' lips can clearly be identified as being hereditary. [4]

Milestone studies

Lip prints have been with us since the beginning of man. Similar to the prints on a person's finger, lips also possess furrows that can be classified into various types for identification purposes. Unlike fingerprints however, lip prints have not been as popular a study due to limited research in this field.

During the early 1900's, anthropologists merely mentioned the existence of lines on lips without providing any type of evidence or studies regarding their use in the forensic science field pertaining to identification. LeMoyne Snyder was the first one to introduce the possibility of utilizing lip prints to identify individuals. Hence he is considered the Father of cheiloscopy. In his studies, Snyder described an interesting case where in a woman was struck by a vehicle. During the investigation, a lip print was discovered on the left front fender of the vehicle suspected to have hit her. After comparing the lifted lip print to the female's lips, investigators discovered a match thus placing the vehicle at the scene of the crime.[5]

Perhaps the greatest research of Cheiloscopy completed has been from Japanese doctors Suzuki and Tsuchihashi in 1970 and 1974 wherein lip prints were obtained from 280 and 1,364 Japanese citizens (respectively). [1]

The research concluded "no lip print showed the same pattern in the investigation of 1,364 Japanese subjects (757 males and 607 females)." The study further stated: "With regard to the dissimilarity of the lip print, as far as the 1,364 subjects used in this study are concerned, there were no two identical lip prints.[2] This means that each human lip print has its own individual characteristics, and although the numbers so far studied are relatively small, it is noteworthy that the data indicate a strong possibility of the absolute dissimilarity of lip prints. Therefore, it may be concluded that the lip print can be used as one of the techniques for identification in the field of forensic odontology.

Dr. Suzuki examined 18 pairs of uni-ovular twins discovering numerous similarities between the lip prints but no exact match. He reported "It was assumed that personal lip prints may show dissimilarity amongst individuals, and that this lip groove pattern could be influenced by hereditary factors, some of which were formed by the study of twins. This finding was important information due to the fact both uni-ovular twins contain the same DNA but not the same fingerprints. The discovery of two different forms of physical identification for such twins was exciting and pertinent for the forensic science field.[1]

Other than the aforementioned collected lip prints from the twins, the only other analysis of lip prints connected with families was reported by Hirth, Gottsche and Goedde (1975). In this study, lip prints were obtained from 76 families for the purpose of determining whether there was a genetic basis of ridge-pattern in the lips. A branched pattern was prominent on the upper lips while simple patterns (long and short vertical grooves) were more frequent on the lower lips. The results of the study proved a genetic basis of lip prints.[6]

Lincoln C. Petersen conducted a study in 2006 to determine whether lip prints had hereditary characteristics or not. There were 81 participants comprising of 20 different biological families. From the research gathered in this study it was indicative that lip print characteristics are indeed hereditary, either directly from the parents or from the grandparents. It is also important to note that while the children possessed the same characteristics as their parents and/or grandparents, it should be stressed that the characteristics located on the lips were not in the exact location as their parents, suggesting each person possessed his/her own individually unique lip prints.[6]

Although limited research has been completed on the subject of Cheiloscopy and lip prints, a common response within all the literature reflects a positive indication for the utilization of lip prints for personal identification.

Indian perspective

In India, research in the field of Cheiloscopy has been increasing. Not only have the laboratories developed a new technique in identifying suspects or criminals from the description of their lips, the Forensic Sciences Laboratory in Bangalore has established a comprehensive classification system for the micro-structural (grooves and wrinkles found on lips) and macro-structural (shape and size of lips) patterns of lips.

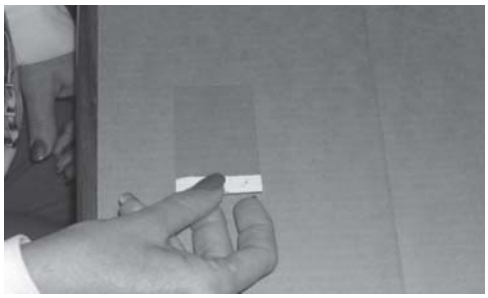
Preeti Sharma, Susmita Saxena and Vanita Rathod of the Department of Oral Pathology and Microbiology, Subharti Dental College, Meerut, UP conducted a study of lip prints in gender identification. They found that lip prints are useful in identification of gender as the lip pattern is different in males and females.[7]

Murkey P.N., Sutay Seema, Khandekar I.L. have studied lip print pattern, the relationship of gender, and genetic composition with lip print in 204 individuals of Sewagram of Maharashtra including 10 groups of twins. They found that persons with similar genetic composition had similar lip prints but there was no exact match. [7]

Dr. Anil Aggarwal, Professor of Forensic Medicine has studied in detail the importance and uniqueness of lip prints. The author suggests that lip prints are a unique feature and can be used as an adjunct to finger prints.[7]

Techniques

There are two methods of recording Lip prints. In the first method, the person simply presses his or her lips onto a piece of plastic that is subsequently processed with black magnetic detection powder. While the latent print is successful in being developed, the characteristics are somewhat difficult to analyze utilizing a common, household magnifying glass. It would stand to reason that these prints would be greatly enhanced using more advanced technological instruments found in a forensic laboratory. The first method of obtaining lip prints is by utilizing a cut piece of clear plastic on which the lip print would be placed. Using a black magnetic detection powder, the latent lip prints are then developed. This method serves as a twofold experiment. The first being a positive way to develop a latent lip prints such as the way one would be discovered at an actual crime scene. The second positive result is the the fact that the latent lip prints can be successfully developed after five months. As with any experiment, this procedure is accomplished by adhering to extremely specific directions.[8]



The second method in obtaining lip prints is the use of lipstick and blank index cards. Two lip prints are obtained on one index card for two reasons; the first is the possibility that one print may contain clearer lines over the other. Secondly, it goes to reason that the applied lipstick may be extremely heavy (depending on how forceful the participant is). By rolling the lips on the card the second time using the same applied lipstick, some may be clearer as the first sample removed the thicker layer of lipstick. The same thought process applies when lifting a latent print at a crime scene that was developed with powder. The first lift is often dirty in appearance due to excessive powder while the second lift obtains a cleaner looking print. The process is extremely helpful in analyzing, as two prints are available on one card.[8]

Types of lip prints

Type I: represents a lip possessing full vertical grooves.

Type I! (pronounced "one-dash") has partial grooves running vertically on the lip.

Type II represents branched grooves.

Type III represents intersected (diamond) grooves that look similar to crosses.

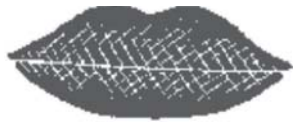
Type IV represents the reticular (rectangular) pattern similar to wire mesh or boxes .

Type I: (Full Vertical Grooves)

Type II: (Short Vertical Grooves)

Type II: (Branched Grooves)

Type III: (Diamond Grooves)



Type IV: (Rectangular Grooves)

Because most lips contain more than one type of pattern, the lips are divided into four quadrants. Each quadrant is studied and the various types of lip prints are recorded. Each quadrant is read from the center of the lip outward toward the corner of the lip. The upper and lower lips are divided through the center by an imaginary vertical line, thus producing left and right upper and lower quadrants. A branched pattern is prominent on the upper lips while simple patterns (long and short vertical grooves) are more frequent on the lower lips.[7]

Conclusion

Lip prints are hereditary yet considered to be individualistic, each possessing their own unique characteristics. For this reason it is safe to suggest lip prints can and should be included in the forensic sciences

arena as a legitimate means of identifying persons of interest connected with criminal activity.

Of course lip prints may never be on the same level as fingerprints when it comes to identification; however, it is interesting to know that certain countries around the world are creating databases and programs centered around the characteristics and appearances of lips specifically for the purpose of solving crimes.

References

1. Suzuki, K., Tsuchihashi, Y. Personal Identification by Means of Lip Prints. Journal of Forensic Medicine, 1970; 5: 52 – 57.
2. Tsuchihashi, Y. Studies on Personal Identification by Means of Lip Prints.Forensic Science Journal, 1974 ;15: 233 – 248.
3. Castello, A., Alvarez, M. & Verdu, F. Just Lip Prints? The Faseb Journal, 2004; (7): 615 – 616.
4. Ehara, Y. & Marumo, Y.. Identification of Lipstick Smears by Fluorescence Gas Chromatography. Forensic Science International, 2004;(10) : 1 – 10.
5. Hansen, M. The Fine Print. ABA Journal, 2000 ;(18): 134-138.
6. Hirth, L., Gottsche, H. & Goedde, H.W. Lip Prints—Variability and Genetics,National Library of Medicine, 1975; (15): 47 – 62.
7. Kasprzak, J. Cheiloscopy. Forensic Science International, 2000; (5) : 358 – 362.
8. Kasprzak,J.Possibility of Cheiloscopy. Forensic Science International, 2002; (7) : 145 – 151.

Lead toxicity in children: A review

Pradeep Kumar K. N*, Amitha M. Hegde**

*Post Graduate Student, **Senior Professor and HOD, Department of Pedodontics and Preventive Children Dentistry, A. B. Shetty Memorial Institute of Dental Sciences, Deralakatte, Mangalore 575018, Karnataka, India

Abstract

Lead is a widely used metal, but it is simultaneously a versa-tile, subtle, and persistent poison. Lead is toxic to humans, with the most deleterious effects on the hemopoietic, nervous, renal and reproductive systems. The main sources of lead exposure are paints, water, food, dust, soil, kitchen utensils, and leaded gasoline. The majority of cases of childhood lead poisoning are due to oral ingestion and absorption through the gut. Hyperactivity, anorexia, decreased play activity, low intelligence quotient, and poor school performance have been observed in children with high lead levels. Lead crosses the placenta during pregnancy and has been associated with intrauterine death, prematurity and low birth weight. Lead toxicity is a significant but preventable health problem. Medical practitioners and par-ents need to be made more aware and educated regarding the problem of lead toxicity.

Introduction

Lead is a widely used metal, but it is simultaneously a versa-tile, subtle, and persistent poison. Significant exposure to lead is an environmental threat to optimal health and to physical development in young children that affects all so-cioeconomic groups[1]. The most susceptible populations are children particularly toddlers, infants and the fetus[2]. In 1991, the Centers for Disease Control (CDC) and Prevention statement concerning lead poisoning in young children re-defined elevated blood lead levels as those $>10\,\mu g/dl$[1].

The effects of lead toxicity on young children were first described in 1892 in Brisbane, Australia[3]. Lead poisoning in chil-dren is a characteristic disease usually occurring between the second and third year of life[4, 5]. The characteris-tic features of lead toxicity, including anemia, colic, neurop-athy, nephropathy, and coma. However, the deleterious effects of lead may be efficiently prevented by applying specific regu-lations to its use[6]. The removal of lead from gasoline in 1990, regarded by many as one of the major public health triumphs of the 20th century, was a major victory for the environment and had an immediate impact[7].

Sources of lead

Repeti-tive hand-to-mouth activity is now recognized as a ma-jor contributor to the total body burden of lead in children. Sources include, in-utero transmission of lead from the mother to child through the placenta[4, 5, 8, 9], human milk and infant formula[10]. Environmental sources are leaded paint, water pipes with lead joints; lead glazed ceramics, lead painted toys, leaded gasoline, certain canned foodstuff, air, dust, soil, and eye cosmetics and fishing weights[2]. Children with calcium deficiency and excessive exposure to lead can lead to the accumulation of lead in children[11].

Absorption of lead

Lead may enter the body by ingestion through the intestine, through the lungs by inhalation or through the skin. Respiratory lead absorption is prima-rily dependent on particle size. The percentage of inhaled lead reaching the bloodstream is estimated to be 30-40%. Rate of absorption through the gastrointestinal tract depend on the nutritional status and the age of the indi-vidual exposed. Infants and young children absorb an average of 50% of the ingested quantity of lead. Absorption through the gut is the predominant route for children and increases when dietary intakes of iron, calcium, phosphorus, zinc, manganese, copper, chromium and magnesium are low. Such deficiencies are common due to refined food diets and poor eating habits[4, 11, 12]. Epidemiologic evidence suggests that diets higher in fat and total calories are associated with higher blood lead concentrations at 1 year of age[13].

Distribution of lead

Once absorbed, lead accumulates in three compartments: blood, soft tissues, and bone. In blood, approximately 99% of the lead is found in the erythrocytes, leaving about 1% in the plasma and serum[14]. The kinetics of lead transfer from blood to soft tissues is low and takes approxi-mately 4 to 6 weeks. Half life of lead in blood is 35 days; soft tissue pool is 40 to 50 days whereas it is 10 to 20 years in skeletal pool. Blood lead concentrations reflect the intake of only the previous 3 to 5 weeks and thus cannot be used as indices of chronic exposure.

In children, total body burden of lead found in the skeleton is about 73%. Lead crosses the placental barrier, beginning at 12 weeks of gestation. Concentrations of lead in umbilical cord blood were found to be 80-100% of the maternal blood lead level[12, 15]. If intake of lead exceeds $3.5\,\mu g$ / kg /day, accumulation occurs in the body[16].

Excretion of lead

Inorganic form of lead is not metabolized; however, alkyl lead com-pounds are oxidized by the hepatic P 450 system. Excretion of lead is low, 90% through the kidneys and 10% through bile. Although minute amounts of lead are excreted through the sweat and the nails, these routes do not have any practical significance. In general, lead is excreted extremely slowly from the body, thus facilitating accumulation in the body[12]. Even vitamin C may increase renal excre-tion of lead[13].

Epidemiology

Blood lead level for children less than 12 years was greater than 10 μg /dl for 51.4 % of children in India. Possible causes are increased urban population growth and vehicles using leaded petrol, using lead-tin alloy to coat the inside of copper eating utensils, practice of wrapping foodstuffs in newspaper printed with ink containing lead; incorporation of lead in grinded spices and grains due to the wear of machinery bushings, brass fittings and tinned metal surfaces[17].

Effects of lead in children[18]

Nervous system

- Encephalopathy
- Acute encephalopathy
- Alters function of developing brain
- Convulsions
- Head ache
- Cerebral Palsy
- Neurotransmitter release disrupted

Peripheral nervous system

- Reduced touch sensitivity
- Slow nerve conduction velocity

- Foot/ hand drop
- Balance altered
- Dizziness

Growth & development

- Delayed neurodevelopment [e.g. in sitting up, walking, talking]
- Stature and growth rate reduction
- Impaired pituitary-thyroid endocrine system
- Osteoporosis
- Weight loss

Cognitive development

- I.Q. levels decrease
- Cognitive function deficits
- Verbal function / linguistic deficits
- Learning difficulties
- Decreased educational performance
- Short term memory
- Autism

Behaviour

- Aggression, violence, hostility, anti-social or delinquent behaviour
- Attention problems; distractibility, restlessness
- Hyperactive behaviours
- Inappropriate / uncontrolled behaviours
- Irritability
- Lethargy
- Increased school absenteeism

Hearing

- Hearing impairment
- Auditory evoked response patterns altered
- Auditory processing altered

Sight

- Retinal degeneration
- Depressed sensitivity of rod photoreceptors
- Perceptual function deficits
- Visuo-spatial skills deficit

Movement and muscular

- Visual-motor skills deficits [hand-eye coordination]
- Fine motor dysfunction
- Motor function deficits
- Impaired muscular strength and endurance
- Paralysis
- Somatic complaints [aches and pains]

Digestive system

- Impaired Vitamin D metabolism
- Colic
- Loss of appetite
- Vomiting
- Constipation, diarrhoea, anorexia
- Abdominal cramps

Renal (kidneys), blood and circulation

- Acute and chronic nephropathy
- Queensland nephritis
- Microcytic hypochromic anemia

Oral cavity

- Defective enamel and dental caries
- Reduced saliva formation

Death

Acute and chronic toxicity:

Overt signs of acute toxicity include dullness, restlessness, ir-ritability, poor attention span, headaches, muscle tremor, ab-dominal cramps, kidney damage, hallucinations, and loss of memory with encephalopathy, all occurring at blood lead lev-els of 80-100 μg/dl in children. There is no safe level of blood lead below which children are not affected. Chronic toxicity is not common in children[19].

Investigations for lead determination

Diagnostic Testing

Some experienced clinicians measure the blood lead concentration in children with growth retarda-tion, speech or language dysfunction, anemia, and attentional or behavioral disorders. A low blood lead concentration in a school-aged child does not rule out earlier lead poisoning be-cause hand-to-mouth activity decreases and the child's body mass increases [20].

Laboratory testing

Includes increased blood lead levels; raised delta-amino levulinic acid in blood and urine, increased free red cell protoporphyrin or Zn protoporphyrin, and urinary corproporphyrinogen[16].

The free erythrocyte protoporphyrin levels reflect impaired heme synthesis. As this test is not sensitive enough to identify blood levels between 10 and 25 ug/dl, it is no long-er recommended as a screening test[21].

If either blood lead or free erythrocyte protoporphyrin is elevated, then other tests for lead effects on the kidneys (urea nitro-gen, creatinine, and urinalysis) and blood (complete blood count with smear) should be performed. It is important to note that urea nitrogen and creatinine are not sensitive in-dicators of renal damage, since it is known that they do not rise until a large part of renal function is lost[22].

Radiological examination may reveal radiopaque densities or radiographic evidence of paint chip ingestion; "lead lines", across the metaphyses of long bones and along the margins of flat bones, such as the iliac crest. Ra-diological examination is not a sensitive method for diag-nosing acute lead poisoning[23].

The various methods of blood lead estimation include Anodic stripping voltammetry, Atomic absorption spectrophotometry and inductively coupled plasma mass spectrometry[2].

Treatment

CDC guidelines for management of children with blood lead levels are as below[24]:

Chelating agents

Chelation therapy with any agent should not be undertaken unless exposure has defini-tively been curtailed, since its use in the presence of contin-uing exposure may result in enhanced absorption of lead and a worsening, rather than amelioration, of toxicity[25].

Dimercaprol (BAL)

Dimercaprol, also known as British Anti-Lewisite (BAL), acts by increasing the urinary excretion of lead. It was the first chelating agent found to be used. Due to sulfide odor, patients often complain of the taste and bad feeling when the drug is administered. The recommended dose by the manufacturer is 3-5 mg/kg IM every 4th hourly. Despite the high incidence of side effects (fever, allergy), BAL has remained in use for more serious lead poisoning because of concerns that Calcium DiSodium EDTA therapy may translocate lead into the central nervous system and increase the potential for encephalopathy. Traditionally, pre-treatment with BAL has been recommended to avoid precipitation

Class	Blood lead levels	Management
I	d"9 ìg/dL	No action. Class I child is not considered to be lead poisoned.Nutritional and educational intervention and remove from source.
IIA	10-14 ìg/dL	Community wide screening and prevention programme.Repeat blood test after one month. Repeat lead test after 3 months.
IIB	15-19 ìg/dL	Remove from source. Repeat blood test after one month.
III	20-44 ìg/dL	Remove from source. Give Dimercapto succinic acid (DMSA) only if blood lead remains high.
IV	45-69 ìg/dL	Remove from source. Oral Chelation therapy (DMSA) recommended.
V	> 70 ìg/dL	Remove from source, Medical emergency. British Anti-Lewisite (BAL) and Ethylene Di amine Tetra Acetic acid (EDTA) Chelation therapy.

of encephalopathy[26].

Calcium disodium edta (cana2edta)

CaNa2EDTA, a chelating agent was first used in the year 1950. It increases the urinary excretion of lead. Because the use of CaNa2EDTA may cause increased lead concentration in the central nervous system, it should be administered after BAL is given. Very low bio-availability from oral intake necessitates parenteral administration. Treatment with CaNa2EDTA should usually be performed in a hospital setting on patients with normal renal function and with careful monitoring of renal parameters[26].

Succimer (2, 3-meso-dimercaptosuccinic acid or dmsa)

This is an oral chelation agent, chemically similar to BAL, has a high therapeutic index, and is absorbed through the gastrointestinal tract. The recommended dose by the manufacturer is 10 mg/kg three times a day for five days, followed by 10 mg/kg twice a day for two weeks[26]. The most common adverse effects of succimer are abdominal distress, transient rash, ele-vated hepatocellular enzyme concentrations, and neutropenia. The drug is unpleasant to administer because of a strong "rotten-egg" odor[27].

Ascorbic acid

Serum ascorbic acid level was inversely related to the blood lead level in children. Vitamin C supplements in modest doses (100 to 1000 mg per day) may be an attractive adjunct to the management of patients with mild lead toxicity[28].

Prevention of lead toxicity

Primary prevention

Educate the parents to take adequate measures to minimize their child's exposure to common sources of lead such as paint and dust; and to less common sources, such as water or contaminated soil. Also, discussions about nu-trition and the importance of dietary iron may help to prevent elevated blood lead levels. Educational brochures are available to assist in preventive edu-cation. Public health efforts to prevent lead exposure through the removal of environmental lead hazards continue to be a most effective measure. The child's residence and site of routine care are most important, because high lead exposures occur most frequently where children spend the majority of their time[24].

Secondary prevention

Lead poisoning and its sequelae can be prevented by blood lead screening followed, when appropriate, by education and case management. Other candidates to be considered for targeted screening include children 1 to 2 years of age living in housing built before 1950, children of ethnic or racial minority groups who may be ex-posed to lead-containing folk remedies, children who have emigrated (or been adopted) from coun-tries where lead poisoning is prevalent, children with iron deficiency, children exposed to contam-inated dust or soil, children with developmental delay whose oral behaviors place them at signifi-cant risk for lead exposure, victims of abuse or neglect, children whose parents are exposed to lead (vocationally, avocationally, or during home renovation), and children of low-income families.

Screening for lead exposure should be considered in the differential diagnosis of children with unexplained illness such as severe ane-mia, seizures, lethargy, and abdominal pain[24].

Risk Factors for Lead Exposure and Prevention Strategies[24]

Risk Factor	Prevention Strategy
Environmental	
Paint	Identify and abate
Dust	Wet mop, frequent handwashing
Soil	Restrict play in area, ground cover, frequent handwashing
Drinking water	2-minute flush of morning water; use of cold water for cooking, drinking
Old ceramic cookware,	Avoid use
Some imported cosmetics, toys, Crayons	Avoid use
Home renovation	Proper containment, ventilation
Buying or renting a new home	Inquire about lead hazards
Host	
Hand-to-mouth activity (or pica)	Frequent hand washing
Inadequate nutrition	High iron and calcium, low-fat diet; frequent small meals
Developmental disabilities	Frequent screening

Summary

Lead toxicity is a significant but preventable health problem. Blood lead levels confirm poisoning, and their determination helps with differential diagnosis. Identification of the various lead sources that sur-round us can help towards prevention of lead toxicity. Medical practitioners and par-ents need to be made more aware of the problem. They must fully understand that "the less lead in the developing brain, the better". Continued public health initiatives to remove lead from the environment, in concert with routine lead screening of young children; will be the key for meeting the goal of the Centers for Disease Control and Prevention to eliminate childhood lead poisoning by the year 2011.

References

1. Centers for Disease Control and Prevention. Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control, October 1991. Atlanta, GA: US Dept of Health and Human Services, 1991.
2. ATSDR-Case studies in environmental medicine. Lead Toxicity, September 1995.
3. Fison DC. The Royal Children's Hospital, Bisbane, 1878 to 1978. Med J Aust, 1978; 2:137-38.
4. Markowitz M. Lead Poisoning. Pediatr Rev, 2000; 21:327–35.
5. Shannon MW, Graef JW. Lead intoxication in infancy. Pediatrics,

1992; 89:87-90.
6. Landrigan PJ, Silbergeld EK, Froines JR, Pfeffer RM. Lead in the modern workplace. Am J Public Health, 1990; 80:907–8.
7. Rosner D, Markowitz G: A "gift of God"?. The public health controversy over leaded gasoline during the 1920s. Am J Public Health 1985; 75:344–52.
8. Gulson BL, Jameson CW, Mahaffey KR et al. Pregnancy increases mobilization of lead from maternal skeleton. J Lab Clin Med 1997; 130:51-62.
9. Gulson BL, Mizon KJ, Korsch MJ, Palmer JM, Donnelly JB. Mobilization of lead from human bone tissue during pregnancy and lactation—a summary of long-term research. Sci Total Environ. 2003; 303:79 -104.
10. Gulson BL, Jameson CW, Mahaffey KR et al. Relationships of lead in breast milk to lead in blood, urine, and diet of the infant and mother. Environ Health Perspect. 1998; 106:667-674.
11. K.R. Mahaffey. Nutritional Factors in Lead Poisoning. Nutrition Reviews 39:353-362, 1981.
12. Philip AT, Gerson B. Lead poisoning – Part I. Clin Lab Med, 1994; 14: 423–44.
13. Centers for Disease Control and Prevention. Managing Elevated Blood Lead Levels Among Young Children. Recommendations From the Advisory Committee on Childhood Lead Poisoning Prevention. Atlanta, GA: Centers for Disease Control and Prevention; 2002.
14. Rabinowitz MB. Toxicokinetics of bone lead. Environ Health Perspect, 1991; 91: 33–37.
15. Papanikolaou N C et al. Lead toxicity update. A brief review. Med Sci Monit, 2005; 11(10):329-336.
16. Klaasen CD, Watkin JB. Casarett and Daulls. Toxicology- The basic science of poisons. 6 Ed. New York:Mc Graw-Hill, 2003, p 35312.
17. Mike van Alphen. Lead Poisoning in India. Lead action news 1999 Vol 7 No 1.
18. Vance Vella et al. Health impacts of lead poisoning. Lead action news Vol 6 No 2.
19. Guidelines for drinking water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization, 1996; 254–75.
20. Esteban E, Rubin CH, Jones RL, Noonan G. Hair and blood as substrates for screening children for lead poisoning. Arch Environ Health. 1999; 54:436-440.
21. Bergdahl I, Schutz A, Gerhardsson L et al. Lead concentrations in human plasma, urine and whole blood. Scand J Work Environ Health, 1997; 23: 359–63.
22. Philip AT, Gerson B. Lead poisoning-Part II. Effects and assay. Clin Lab Med, 1994; 14: 651–70.
23. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Lead. Prepared by Clement International Corporation. Contract No. 205-88-0608. Atlanta, GA, US Department of Health and Human Services, April 1993.
24. Screening for blood lead levels. American academy of paediatrics, committee on environmental health. Pediatrics 1998; 101(6): 1072-78.
25. Porru S, Alessio L. The use of chelating agents in occupational lead poisoning. Occup Med (Lond), 1996; 46: 41–48.
26. Treatment guidelines for lead exposure in children. American Academy of Pediatrics, Committee on Drugs. Pediatrics, 1995; 96: 155–60.
27. Treatment of Lead-Exposed Children (TLC) Trial Group. Safety and efficacy of succimer in toddlers with blood lead levels of 20-44 μg/dl. Pediatr Res. 2000; 48:593-599.
28. Simon JA, Hudes ES. Relationship of ascorbic acid to blood lead levels. JAMA, 1999; 281: 2289–93.19. Chisolm JJ Jr, Kaplan E. Lead poisoning in childhood - comprehensive management and prevention. J Pediatr. 1968; 73:942-950.

Common peroneal component of sciatic nerve piercing piriformis muscle: Piriformis syndrome versus sciatica

Rakhi Rastogi[1], Virendra Budhiraja[2], Ajay Kumar Asthana[3]

[1]Department of Anatomy, Subharti Medical College Delhi-Haridwar By Pass Road, Meerut 250002, U.P., India, [2]Department of Anatomy, Subharti Medical College Delhi, Haridwar By Pass Road, Meerut 250002, U.P., India, [3]Department of Anatomy, Subharti Medical College Delhi, Haridwar By Pass Road, Meerut 250002, U.P., India

Abstract

Piriformis syndrome and sciatica can seem quite similar, particularly in terms of symptoms and this similarity in how they feel has caused considerable confusion for doctors and patients alike. We studied emergence of sciatic nerve in the gluteal region with relation to piriformis muscle in 40 cadavers on 80 sides. In 78 cases both tibial and common peroneal component of sciatic nerve were found emerging below the piriformis muscle to enter into the gluteal region, while in 2 cases common peroneal component pierced piriformis muscle and divided the muscle into upper and lower slips. We discussed the finding in terms of possible sciatic nerve entrapment and correlated it with piriformis syndrome and sciatica.

Keywords

Common peroneal nerve, sciatic nerve, piriformis syndrome, sciatica.

Introduction

Sciatic nerve is the thickest nerve in human body. Normally sciatic nerve reaches the gluteal region from the pelvic fossa by passing below the piriformis muscle. The nerve divides into tibial and common peroneal nerve in the lower part of posterior compartment of thigh [1]. The two terminal branches of sciatic nerve may arise directly from the sacral plexus [2]. When the nerve divides in the pelvis, the common peroneal usually pierces the piriformis muscle [3].

Material and methods

During routine dissection for medical undergraduate conducted on formalin preserved 40 cadavers of both sexes aged 50-70 years at Subharti Medical College, Meerut. The gluteal region was opened on 80 sides and sciatic nerve was observed in relation with piriformis muscle.

Observation

Out of 40 cadavers i.e. 80 sides, in 78 cases both component of sciatic nerve (tibial and common peroneal) were found emerging below piriformis muscle to reach gluteal region from pelvic cavity (figure 1)

In 2 cases common peroneal nerve was observed passing through the piriformis muscle to enter into gluteal region and thus dividing piriformis muscle into an upper and lower slip (figure 2).

Discussion

Piriformis syndrome is caused by entrapment of the sciatic nerve as it exits the greater sciatic notch in the gluteal region [4]. There are two normal variations for the exit of the sciatic nerve in this region. In first case sciatic nerve lies inferior to the piriformis muscle and superior to the gemellus superior muscle [5]. Entrapment in this area is likely due to a myospasm or contracture of either of these two muscles [6]. The second common site of entrapment is when the sciatic nerve or one of the braches of sciatic nerve actually pierces the piriformis muscle, which can occur in 1% - 5% of all humans[7,8,9].

In our study we found two such cases in which common peroneal component of sciatic nerve pierced the piriformis muscle and divided it into upper and lower slip. Here the myospasm and or contraction of piriformis muscle itself can lead to pain along back of thigh to the knee, loss of sensation, numbness and tingling in the sole of the foot, resulting in piriformis syndrome [8, 10].

The piriformis syndrome (pseudo sciatica) often mimic its more notorious counterpart known as sciatica, and that being the case; it is often misdiagnosed as sciatica [11]. But since the most effective treatment for the two conditions varies significantly, it is important to determine the correct diagnosis. In most cases, sciatica can be differentiated from piriformis syndrome with a couple of simple test maneuvers.

To begin, from a seated position, one straightens the knee on the side of sciatic pain, holding the leg straight and parallel to the floor, and if this position causes increase in symptoms, it is a good indicator of true sciatica [12]. The second maneuver is done in two parts. First

Figure 1: Sciatic nerve emerging below piriformis muscle PM – Piriformis muscle, SN- Sciatic nerve

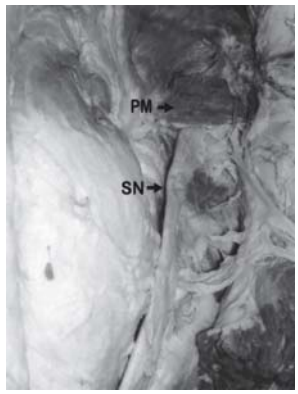
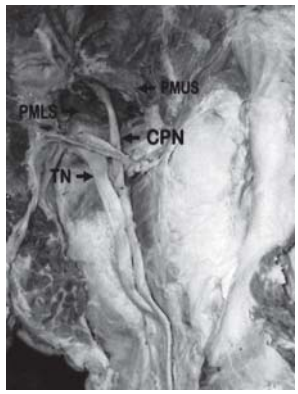


Figure 2: Common peroneal nerve piercing piriformis muscle CPN – Common peroneal nerve, TN – Tibial nerve, PMLS – Piriformis muscle lower slip, PMUS – Piriformis muscle upper slip



Corresponding Author:
Dr. Rakhi Rastogi
Associate Professor Department of Anatomy Subharti Medical College Delhi-Haridwar By Pass Road, Meerut – 250002 (U.P.) – INDIA
Ph: +91 9897898515, Fax: 91121 – 2439127
E-mail: rakhirastogi1207@gmail.com

from the sitting position one bends the leg and pulls the knee on the painful side towards the same-side shoulder, there is usually no major increase in pain in this position. The second part of maneuver is to pull the knee towards the apposite side shoulder. An increase in sciatica like symptoms is a strong indicator of piriformis syndrome [12].

The treatment plan of sciatica is mainly physiotherapy including, various exercise methods of Mckenzie [13]. The management of piriformis syndrome includes injections of local anesthetic and steroid in piriformis muscle. Older techniques of injection were done blindly, newer techniques involve the use of muscle electromyography or computed tomography (CT) to identify the piriformis muscle, and the use of nerve stimulator to identify the sciatic nerve [5, 6]. Surgery may be considered when there is involvement of piriformis in sciatic nerve entrapment, in which the muscle may be thinned, divided or excised. The obturator internus, gemelli and quadratus femoris muscles can compensate for the loss of piriformis function, because these muscle shares common insertions with the piriformis muscle [5, 6].

References

1. Williams PL, Bannister LH, Berry MM, Collins P, Dyson M, Dussek JE, Ferguson MWJ. Gray's Anatomy 38th Edition, Edinburgh, Churchill Livingstone. 1995; P 1284.
2. Bergman RA, Afifi AK, Miyauchi R. Compendium of human anatomical variations. Baltimore, urban and schwarzenberg. 1988; 148.
3. Urgrenovic S, Jovanovic I, Krstic V, Stojanovic V, Vasovic L, Antic S, Pavlovic S. The level of sciatic nerve division and its relations to the piriformis muscle. Vojnosaint Pregl 2005; 82: 45-49.
4. Porta M. A comparative trial of boutlinum toxin type A and methyl prednisolone for the treatment of myofascial pain syndrome and pain from chronic muscle spasm. Pain 2000; 85: 101 – 105.
5. Parziale JR, Hudgins TH, Fishman LM. The piriformis syndrome. Am. J. orthop. 1998; 25: 819 – 823.
6. Barton PM. Piriformis syndrome: a rational approach to management. Pain 1991; 47: 345-352.
7. Jaijesh Paval, Satheesha Nayak. A case of bilateral high division of sciatic nerve with a variant inferior gluteal nerve. Neuroanatomy. 2006; 5: 33-34.
8. Rich B, Mckeag D. When sciatica is not a disease: detecting piriformis syndrome in actives patients. Phys. Sports Med. 1992; 20: 104 – 105.
9. Robinson D. Piriformis syndrome in relation to sciatic pain. Am. J. surg. 1947; 73: 355-358.
10. Lam AW, Thompson JF, Mc.Carthy WH. Unilateral piriformis syndrome in a patient with previous melanoma. Aust. NZJ surgery 1993; 63 (2): 152-153.
11. Sridhara CR, Izzo KL. Terminal sensory branches of the superficial peroneal nerve: an entrapment syndrome. Arch. Phys. Med. Rehabil. 1985; 66: 789 – 791.
12. George Bestsecks. Sciatica versus piriformis syndrome. Article Alley 2008.
13. George F. Best, D.C. Sciatica self care by best health and wellness 2008; P. 8 – 16.

Laws related to women in India

Kadu Sandeep S.*, Burungale Sham**, Mattu Neha V***, Khare Suraj J****

*Asso. Prof., Dept. of Forensic Medicine and Toxicology , ** Asst. Prof., Dept. of Community Medicine, ***Asst. Lecturer, Dept. of Forensic Medicine and Toxicology, ****Intern, Padmashree Dr. Vitthalrao Vikhe Patil, Foundation's Medical College, Ahmednagar, M.S.

Abstract

The word 'WOMAN' differs from the word 'MAN' by first two alphabets. But actually word 'WOMAN' has vast meaning. It is a symbol of tolerance, love and forgiveness. It is a fairer sex. A woman plays several roles in her life as a mother, daughter, sister, wife. Firstly she is under protection of her father and then is handed over to her husband for the rest of her life. Then when will she be independent ? When will she be free to lead a life for herself ? When will she get freedom ? Are we really protecting her ? Is she really safe in society ?

All though we say, we have modernized our ways of life and the system has changed, our society is still a male dominated society. Women are not given a position, status, and dignity which they deserve. This is evident from the practices like child marriage, female infanticide, abortions that are still prevalent in some part of India.Women are an integral part of our life. They are working efficiently and have achieved success in almost every field. They are at par with men.We have taken a review of different legal rights and statutory provisions given to women in India. As a forensic expert we are dealing with different medico legal cases related to women. The knowledge of different laws related to women will help in dealing with rape cases, sexual harassment at work places etc.

Keywords

Woman, Laws, Constitution, Indian Penal code etc.

Introduction

In India the first movement for women's right centered around the three major problems of infant marriages, enforce widowhood and property rights for women. Several woman activits focused attention on gender-based oppression of women.

Gandhiji often said that, "Women has been suppressed under custom and law for which man was responsible. He said, "To me the female sex is not the weaker sex; it is the nobler of two, for it is even today the embodiment of sacrifice, silent suffering, humility, faith, knowledge".1926 he spoke against the double standards for men and women related to morality and dovorce. [1]

1. Constitutional rights to women

The constitution of India incorporated a number of provisions for the protection and development of women and placed them at par with men. Article 14 of the constitution states that, "State shall not deny any person to equality before law or the equal protection of the laws within the territory of India". Article 14, 15 specially 15 (3), 16 and 39 deserve special mention in this regard. Rights guaranteed under article 14, 15, 16 are fundamental rights and if they are violated by the state, a citizen can move to high court or even the Supreme Court. [2]

2. Right to equal share in the property of father

The Hindu Succession Act. 1956, provides equal shares to women in the properties of their father. Section 14 of the act gives women the right to acquire absolute ownership of property. Under this section, woman have not been given absolute power to dispose of her property as she thinks fit. [3]

3. Womens right related to police

She cannot be called to police station or anywhere for interrogation. She can be questioned at her residence in presence of her family member. She can consult her lawyer. She can be told reason of arrest and right of bail. A female constable must be present at the time of arrest and she can take one relative or friend along with her. A medical examination should be done every 48 hours while she is in custody. She has to be produced before a magistrate before 24 hours of arrest. If poor can demand competent counsel at State expenses.

4. Domestic violence (criminal remed)

The law dealing with dowry is 'Dowry Prohibition Act of 1961'. This Act defines 'Dowry' as any property or valuable security given or agreed to be given either directly or indirectly (1) by one party in a marriage to the other party of marriage or (2) by the parents of either party in a marriage or by any other person to either party of marriage of the said parties. The act was amended in 1984 and 1986 to make the provision of law more stringent and to plug the loopholes.

Existing criminal law provisions can be use to initiate proceedings against the husband or his relatives in the court of Magistrate where the incidence took place. A complaint can be filed under section 498-A for any physical or mental harassment. If the abuse / harassment / torture / ill- treatment is not linked with dowry demands. However, if any monetary / financial demands have been made and she has been subjected to cruelty, then she can file the complaint under both the provision i.e., for harassment and dowry. Offences under section 498-A are cognizable and non-bail able i.e., Police Officer can arrest without a warrant and can not grant bail themselves. The accused party has to obtain a Bail Order from the concerned court. [4]

5. Civil remedy (domestic violence)

Sometimes women due to personal reasons may not like to go for criminal remedy i.e., registration of FIR or filing the Criminal Complaint. In the said circumstances, there are certain Civil Remedies are available for the Case Of Domestic Violence.

She can file suit under Order VII Rule I Of Code Of Civil Procedure in the Family Court or where there is no Family Court in A Civil Court against the husband or relatives who are harassing her to seek any of the following orders.

That the spouse or/ and his relatives are restrained from ousting her and her children from matrimonial house. That exclusive possession of the matrimonial house should be granted to her and to her children. That the spouse or/ and his relatives are restrained from entering the premises or any part of it, which is in her occupation.

6. Rape (375, 376 ipc)

Rape is an offence not against any individual but like all the crimes in the Indian Penal Code, it is the crime against the state. Once incidence occurs it has to be reported immediately without any delay to the police station of Jurisdiction. As far as the complaint must be given in writing containing all relevant fact in order to avoid manipulation. Then police will investigate the matter. The information, i.e., the person filing the complaint is entitled to a copy of the first. The victim must go under medical examination conducted by a female medical officer. The trial is conducted in a court of Sessions. Like all criminal matters the

victim cannot have independent lawyer, unless she makes an application for appointing a Special Prosecutor. [5]

Different punishments of rape

1. Sexual intercourse by a man with a woman against her will or without her consent obtained fraudulently Minimum Imprisonment for 7 yrs or upto life imprisonment fine.
2. Intercourse by a Public Servant with woman working in office Imprisonment for 5 yrs and Fine
3. Intercourse by a Superintendent of Jail,/ Remand with any woman in the Jail/ Remand Imprisonment for 5 yrs and Fine.
4. Intercourse by a member of Management and Staff of Hospital with any woman in the Hospital Imprisonment for 5 yrs and Fine.
5. Unnatural Offence Voluntary Carnal Intercourse against the order of nature with any woman i.e., oral sex, man or animal Imprisonment for 10 yrs and Fine.

7. Sexual harassment at work place

No specific law regarding sexual harassment . Supreme Court has laid down guidelines in Vishaka's case (AIR 1997 Supreme Court 3011).Any unwelcome sexually determined behaviour - direct or implicit viz., physical contacts and advances. Demand or request for sexual favours. Any other unwelcomed physical , verbal , non-verbal conduct of sexual nature. Such acts commited amounts to sexual harassment. Where the victim has reasonable apprehension.

(a) That such conduct is humiliating, (b) Constitutes health and safety problem

When the victim has reasonable ground to believe that her objection to such act would disadvantage, would prejudice her in connection with work, recruitment, promotion. When non-consent or/ objections to such act entails adverse consequences.

8. The indescent representation of woman (prohibition) act., 1986

The act prohibits depiction in any manner a figure of woman , body or any part thereof , in a such a way that has the effect of being indecent of or derogatory to or denigrating woman. In intends to prevent woman being depicted as sex object in the media for commercial gain or prevent the co-modification of woman. This act indents to prohibit indecent representation of women in writing, paintings and figures or in any manner i.e., likely to corrupt or injure the public morality or morals.

9. Immoral traffic (prevention) act , 1956

In India prostitution is tolerated and regulated. Prostitution is per say not a crime. Prostitution is not abolished / prohibited / ban. The act intends to criminalize and penalize the Institution / Industry (brothel, brothel keepers, procurers, pimps, touts, middle man, land lords etc.).But actually ends up criminalizing and penalizing. Trafficking of persons- male / female is prohibited. Prevention of sexual exploitation of person for commercial purposes. Soliciting and / or seduction in public place is crime. Offences involving children / minor – stringent punishment.

10. The medical termination of pregnancy act,1971

Abortion in India has been legalized since the year 1971.

A pregnancy may be terminated by Registered Medical Practitioner on following grounds :-

1. Where the length of pregnancy does not exceeds 12 weeks, one Registered Medical Practitioner is required.
2. Where the length of pregnancy exceeds 12 weeks but does not exceeds 20 weeks , two Registered Medical Practitioner are required.
3. If the doctor/s is / are of the opinion that continuance of pregnancy would involve a risk to the life of pregnant woman or would cause grave injury to her physical or mental health.
4. The continuation of pregnancy would cause substantial risk and if the child were born it would suffer such physical or mental abnormalities as to be seriously handicapped.
5. If the pregnancy is caused by rape and the anguish cause by such pregnancy is presumed to constitute a grave injury to the mental health of the pregnant woman.
6. If the pregnancy occurred as a result of failure of family planning device, the anguish caused by such unwanted pregnancy may be presumed to constitute a grave injury to the mental health of a pregnant woman
7. No pregnancy shall be terminated without consent of a pregnant woman.
8. Termination of Pregnancy shall be made only in the hospital established or maintained by Government or such other place approved by government. [7]

11. The pre-natal diagnostic techniques (regulation and prevention of misuse) act, 1994 as amended :- 2003

The Pre-Natal Diagnostic Techniques (Regulation And Prevention Of Misuse) Act' was passed in 1994 and rules were framed in 1996.The Main Objective Of The Act is to provide for the Regulation of the use Pre-Natal Diagnostic Techniques for the purpose of detecting Genetic and Metabolic Disorders. The act is prohibitory as well as regulatory both in nature. There is prohibition of misuse of Pre-Natal Diagnostic Techniques of the sex of the foetus. All offences under act are cognizable , non-bailable and non-compoundable. The violation are punishable with imprisonment , which may extend to 5 years and fine. In case of registered medical practitioner , his name shall be reported by the appropriate authority to State Medical Council concerned for taking necessary action like suspension of the registration if charges are framed by the court. For the removal of his name from register of the council on conviction for the period of Permanently for the subsequent offence. [8]

12. Ragging

The supreme court has led down certain guidelines in Vishwa Jagriti Mission Vs. Central Government (2001, Supreme Court Cases , page 577).Where in Educational Institution in which the student is studying is empowered to be the disciplinary authority and that the police station should not be resorted unless it is unavoidable. Any disorderly conduct by words spoken or written or by an act that has the effect of treating or handling with rudeness any other student including in ruddy or undisciplines activities. Which cause or is likely to cause annoyance, hardship, or psychological harm or to raise fear or apprehension in a fresher or Junior Student; Asking the students to do any act or perform something which such student will not do it.

13. The commision of sati prevention act , 1987

This act intends to prevent commission of Sati by any one. Any person who commit Sati or does any act towards such commission is punishable with imprisonment that may extend to one yr or fine or with both. Person abets the commission of such Sati , either directly or indirectly , shall be punishable with life imprisonment and fine. Encouraging a woman to commit Sati. Making the woman believe that the commission of Sati would result in some spiritual benefit to her or to the deceased husband's family. Encouraging a woman to remain fix in her resolve to commit Sati and thus instigating her. Glorification of Sati is punishable with imprisonment for not less than 1 yr which may not extend upto 7 yrs and fine of Rs.5,000/- to Rs.30,000/-.

14. Law on molastation (section 354 ipc)

An assault or use of criminal force with intent to outrage the modesty of any woman shall be punished with imprisonment upto 2 years of fine or with both.

15. Law on eve teasing (section 509 & 214 ipc)

On intention to insult the modesty of a woman either by uttering any word or making any sound or gesturing or exhibiting any object , is punishing with imprisonment upto 1 year or with anyone who annoys other by doing obscene act in public places, or sing songs or recites or utters and obscene songs, ballad or words in public place shall be punished with imprisonment upto 3 month or with both.

16. Child marriage restrain act 1929 (act no. 19 of 1929)

This Act is expedient to restrain the solemnization of child marriages. It extends to whole India except Jammu & Kashmir. According to this act child means person who, if male has not completed 21 yrs of age and if a female has not completed 18 yrs of age. Child marriage means marriage to which either of contracting party is child either a male or female. Child marriage is punishable with simple imprisonment may extend to three months and shall also be liable to fine.

Minor means a person of either sex who is under 18 yrs of age where a minor contracts a child marriage any person has charge of marriage whether parent or guardian or any other capacity lawful or unlawful who does any act to promote the marriage or permits it to be solemnized or negligency falls to prevent shall be punishable with simple imprisonment which may extend to three months and shall also be liable to fine. It is cognizable offence. [9]

17. Protective legal measures for female workers in india [10,11]

The justification for protective legislation for women workers has been recognized by all member countries of the ILO (Bhagoliwal 1995 . Following are the legislations (Kartikeyan & Gaurav 2002) related to protects of rights of female worker in India :

Mica Mines Labor Welfare Fund Act 1946 : It is compulsory to appoint a female member in the Advisory Committee.

The Factories Act, 1948 : Prohibition of employment of women in hazardous occupations. Maternity have for 12 wks. Separate toilets and washing facilities for male and female workers. Provision of creches where fifty or more women are employed.

ESI (General) Regulation, 1950 : Claims for maternity benefits becomes due on the date of issue of the medical certificate of miscarriage, confinement premature delivery or pregnancy related illnesses.

The Plantation Labor Act, 1951: Working mothers are given time off, during duty hours, for breastfeeding children. Provision of crèches where fifty or more women are employed.

The Mines Act, 1952 : Prohibition of employment of a women in underground mines which is more risk to health.

Maternity Benefit Act, 1952: Prohibition of employment of women in underground mines which is more risk to health.

Maternity Benefit Act. 1961: Eligible for maternity benefits after completion of 80 days of service. Maternity benefit to be allowed on submission of medical certificate. Cash benefit to be paid by employer in organizations where perinatal and postnatal care is not provided free of charge. the pregnant employee should not be given any work that could adversely affect her health or that of the fetus.

According to Amendment in 2008 the duration of maternity benefit is increased upto 6 months.

The Bidi and Cigar Workers (Conditions of Employment) Act, 1966 : Provision of crèches where fifty or more women are employed.

The contract Labor (Regulation and Abortion) Act 1970 : Provision of crèches where 20 more women are employed. Female employee (except midwives and nurse) should not be required to work beyond nine hours between 7 PM and 6 Am. However this has been recently amended by Supreme Court in 2005 and woman can also work in night shift if security is ensured. Separate toilets and washing facilities for male and female workers are provided.

Limestone and Dolomite Mines Labor Welfare Fund Act. 1972: It is compulsory to appoint a female member in the Advisory Committee.

Equal Renumeration Act, 1976: Payment of equal renumeration to male and female workers for the same or similar nature work.

Bidi workers Welfare Fund Act, 1976: It is compulsory to appoint a female member in the Advisory Committee.

The Inter-State Migrant Workman (Regulation of Employment and Conditions of Services) Act 1979 : Separate toilets and washing facilities for male and female workers are provided. Payment of equal renumeration to male and female workers for the same or similar nature work.

18. Relaxation in criminal proceedings

There are certain safe guards to protect women under Criminal Procedure Code Of 1973. Section 51 (2) provides that whenever it is necessary to cause a female to be searched, the search shall be made by another female with strict regard to decency. Under Section 437 Clause (1) a woman is entitled to be released on bail even if she is guilty of an offence punishable with death or life imprisonment.

Whenever a woman is required to be examined as a witness in connection with a case, she should not be summoned to the Police Station. If the officer want to investigate the woman, he must go to her residence. If this provision is violated and if the woman, is called to the Police Station and kept under restraint, the officer is punishable under section 341 and 342 I.P.C.

If a woman is sentenced to death, is found to be pregnant, the High court shall order the execution of the sentenced to be postponed and may if thinks fit, commutes the sentence to life imprisonment under section 416 I.P.C. [12]

Conclusion

Though we have discussed above laws, the fact is that many laws are on paper only. We do not have effective administrative system for the implementation of above Laws. Political commitment and lots of motivation is required for social workers and women for effective implementation of above laws. Legal literacy can play a key role in awareness of different laws.

Let us make an effort to change the scenario and make this word better and safe place for the women.

References

1. Statutary Protection to Women In India : Mrs. Ch. Ramkumari Sahu, Mr. Ch. Suvarna Raju.Central India Law Quarterly, Vol. VII : I.
2. Article 32, The Constitution Of India.
3. AIR 1994 Pat. 3,4 & 5.
4. Deccan Herald, Banglore 5th Nov, 1983.
5. Modi's Medical Jurisprudence and Toxicology (23rd edition) K. Mathiharan and Amrit K Patnaik, Butterworths, 2005, 895 – 899.
6. AIR 1997, Supreme Court, 3011.
7. Principles Of Forensic Medicine And Toxicology, Apurba Nandy,

New Central Book Agency,2005, 401 – 408.
8. Textbook Of Forensic Medicine And Toxicology, V. V. Pillay, 14th Edition, Paras Publication, 2004, 580 – 582.
9. Govt. of India, The Child Marriage Restrain Act 1986 (19 of 1929, 1st October 1929).
10. Women's Bureau, Twenty facts on Women Workers, Fact Sheet No. 88 – 2. United States Of Labour Washington DC 1988 : 1– 2.
11. National Health Programs of India, J. Kishore, 5th Edition 2005, P. 478.
12. Criminal Procedure Code of 1973, Section 51 (2).

MEDICO-LEGAL UPDATE

Aster-06/603, Supertech Emerald Court, Sector – 93 A
Expressway, NOIDA 201 304, UTTAR PRADESH
Mobile: +91-9891098542
Email: medicolegalupdate@hotmail.com
Website: www.medicolegalupdate.org

CALL FOR SUBSCRIPTIONS

About The Journal

Print-ISSN:0971-720X, Electronic - ISSN:0974-1283, Frequency: Six monthly(2 issues per volume).

Medico Legal Update is a journal which brings latest knowledge regarding changing medico legal scenario to its readers. The journal caters to specialties of Forensic Medicine, Forensic Science, D. N. A. fingerprinting, Toxicology, Environmental hazards, Sexual Medicine etc. The journal has been assigned international standard serial number (ISSN) 0971-720X. The journal is registered with Registrar of Newspapers for India vide registration numbers 63757/96 under Press and Registration of Books act, 1867. The journal is also covered by EMBASE (Excerpta Medica Database) from 1997 and by INDEX COPERNICUS, POLAND.

Medico-Legal Update is a quarterly peer reviewed journal. The journal has also been assigned E- ISSN 0974-1283 (Electronic version). The first issue of the journal was published in 1996.

Journal Title	Pricing of Journals		
Medico-Legal Update	Print Only	Print+Online	Online Only
Indian	INR 6000	INR 8000	INR 4500
Foreign	USD 400	USD 500	USD 300

Note for Subscribers

Advance payment required by Cheque/demand draft in the name of "Medico-Legal Update" payable at New Delhi.
Cancellation not allowed except for duplicate payment.
Claim must be made within six months from issue date.
A free copy can be forwarded on request.

SEND REMITTANCE TO :

Prof. RK Sharma, Editor

MEDICO-LEGAL-UPDATE

Aster-06/603, Supertech Emerald Court, Sector – 93 A
Expressway, NOIDA 201 304, UTTAR PRADESH
Mobile: +91-9891098542
Email: medicolegalupdate@hotmail.com
Website: www.medicolegalupdate.org

Published, Printed and Owned : Dr. R.K. Sharma
Designed and Printed : Process & Spot
Published at : Aster-06/603, Supertech Emerald Court, Sector – 93 A
Expressway, NOIDA 201 304, Uttar Pradesh
Editor : Dr. R.K. Sharma