



Indian Journal of Forensic Medicine & Toxicology

An International Journal

Indian Journal of Forensic Medicine & Toxicology

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Print-ISSN: 0973-9122 Electronic - ISSN: 0973-9130 Frequency: Six Monthly

"Indian Journal of Forensic Medicine & Toxicology" is a peer reviewed six monthly Journal. It deals with Forensic Medicine, Forensic Science, Toxicology, DNA fingerprinting, Anatomy, Forensic Odontology, Forensic Nursing, Sexual Medicine and Environmental Medicine. It has been assigned International standard serial No. p-0973-9122 and e-0973-9130. The Journal has been assigned RNI No. DELENG/2007/21789.

The Journal is indexed with **Index Copernicus** (Poland) and is covered by **EMBASE** (Excerpta Medica Database). The journal is also abstracted in **Chemical Abstracts** (CAS) database (USA).

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

Published, Printed and Owned 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



Indian Journal of Forensic Medicine Toxicology

www.ijfmt.com

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Role of dentist in identification in mass disaster

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Abstract

Personal identification is always a major task in any mass disaster event including natural, accidental or criminal. Identification can be done by visual, fingerprint, dental and DNA evidence. A disaster identification team should be set up to handle the problems and plan for the future. Advances in photographic, radiographic, and computer technology have provided the forensic dental team with additional resources to enable recovery, documentation, storage, and comparison of forensic dental evidence.

Key Words

forensic, dental, disasters

Introduction

The identification of large number of casualties in mass disasters are complex and fraught with hazards, both physically and emotionally¹. The term multiple (mass) fatality incident (MFI) evokes images of a chaotic event, initiated by a destructive force, which results in numerous deaths necessitating identification². The ultimate aim of all disaster victim identification is to establish every victim by comparing and matching accurate ante-mortem and postmortem data³.

In mass disasters, physical features are often destroyed. Because teeth are heavily calcified, they can resist fire as well as a great majority of traumas. Generally, teeth and restorations are resistant to heat, unless they are exposed directly to flame. Preservation is possible in most cases⁴. Essentially, the same methods that are used for single body identification may be applied in multiple identification in mass disasters. However complications may occur because of the magnitude of the task⁵.

Types of multiple fatality incident (MFI)

Mass disaster events can be classified in one of three ways: Natural, Accidental and Criminal. Each type of MFI event results in the death of numerous victims. However, the problems faced by forensic dental team responsible for identifying the decedents may vary, depending on the type of mass disaster².

Natural mass disasters include earthquakes, tornadoes, hurricanes, volcanic eruptions, fire storms, tsunamis, and floods. Victims may be scattered throughout broad areas, extending for miles. In addition, many victims in natural MFI situations may be unknowns who cannot be presumptively identified. Several countries or states can be affected, as in the 2004 Indian Ocean tsunami disaster.

Accidental MFI events are most often associated with transportation accidents, fires, industrial and mining accidents, and military accidents. These situations usually occur over short time periods and are associated with closed populations e.g. airplane, bus, or train passengers; mine or factory workers.

Unlike natural and accidental MFIs, criminal mass disasters involving death may occur over extremely long time

periods and wide ranges of territory e.g., different cities or states. It includes serial homicide, mass suicide and acts of terrorism. Dental structures in these situations may not always be available for postmortem review. Also perpetrators often employ physical, chemical or thermal means to accomplish the destruction of fingerprints, teeth and other evidences⁶.

The acts of bioterrorism may include exposure to biologic agents, chemical toxins and the discharge of nuclear devices. Thus the dentist involved in MFI recovery and identification after an act of terrorism may additionally be required to assist medical workers in providing care for the injured².

Methods of identification

The common and legally admissible methodologies used to identify the human remains in mass disaster are: Visual identification, Finger prints identification, Dental identification and DNA evidence.

Visual identification

Visual methodology is least reliable due to subjective factors and stressful situation in which the relative or friend is during identification of the victims.

Finger prints or foot print identification

Finger print mode is long respected, but is subjected to availability of antemortem prints on file or retrievable latent prints from personal effects. Trauma and fire associated with mass disaster sometimes destroy postmortem finger print and foot print evidence.

Dental identification

Dental structures are highly resistant to destruction, but dental identification is also subject to available antemortem dental records on radiographs.

An individual's dentition can provide for a unique combination of decayed, missing, and filled teeth. Furthermore, the mineral composition of teeth is such that a tooth is resistant to fire, extreme temperatures, and decomposition. Dental structures represent the hardest and most resilient tissues of the body. Bernstein noted that nearly 70% of identifications in airline mass disasters are based upon dental comparisons. Paramount in the identification process is the ability to secure antemortem records and the accuracy of such documentation⁷.

Dental identification uses the teeth, jaws and orofacial characteristics in general as well as the specific features of dental work with metallic or composite fillings, crowns, bridges and removable prostheses as well as distinctive configuration of bony structures of jaw (mandible and maxilla), the presence and shape of teeth including the roots, configuration of maxillary sinuses and long standing pathology such as prior fractures and orthopedic procedures⁸.

DNA evidence

DNA comparison is legally admissible in jurisdictions. If the immediate relatives of the victim are available, the genetic make up may be established without antemortem DNA record of the victim. DNA typing, using teeth as a source for recovering DNA was first used by Ballantyne in 1997⁵. DNA typing is being applied in the identification of victims of mass disaster such as an aircraft

accident. The bodies of the victims are usually severely traumatized and fragmented and may also be burned. Initially, traditional methods of identification are used, while DNA typing was reserved only for use when all other methods had failed.

The victim identification strategy involved a comparison between the DNA profiles obtained from the victims' frozen tissue, preferably muscle with blood samples obtained from close biological relatives⁹.

The polymerase chain reaction (PCR) technology is remarkably useful in amplifying very small amounts of either the so-called genomic DNA found in the nucleus or the mitochondrial DNA (Mt DNA) found in the mitochondria that are inherited exclusively from our mothers.

The human genome contains at least several predominant satellite DNA sequences, a different mixture of which is found at each centromere. The number of copies of a satellite DNA vary over a wide range so that remarkable comparisons can be identified that discriminate between two people.

MtDNA is very useful in forensic identification, as it is inherited only from the maternal line and is the best way to test relatedness if there are several generations between ancestor and living descendant. The sex of skeletal bones or teeth can be rapidly determined with enormous accuracy in a segment of the human X or Y chromosome encoded AMEL gene⁹.

Disaster identification team

Problems of body fragmentation, mutilation, commingling and incineration, idiosyncratic dental records from numerous regions, poor working conditions and psychological stresses all confound the identification process¹. Therefore properly trained individuals work as a team to deal with such disasters. Generally, the team includes a coordinator or head of the team, forensic pathologists, forensic photographers, police identification experts and various specialists with experience related to the particular type of disaster, in addition to the forensic odontologist⁴. This organized team is trained to face emergency situations. In its training pro-grams mock mass disaster exercises are included.

Training sessions can be used to counsel the dental team and to inform members of the posttraumatic stress often associated with this type of forensic work. This stress is a result of the sensory and psychological insults encountered by the dentist who is dealing with human death on a large scale⁵.

The dental experts may have an integral role in the following areas –

- Recovery of significant material at the site, as they are part of field recovery team
- Preparation, reconstruction, examination and documentation of post-mortem material at the mortuary.
- Collecting and transcribing of ante-mortem dental records at the identification centre and direct communication with dentists supplying the records.
- Sorting and comparison of the antemortem and postmortem data.

Dental identification team organization

The dental team which is involved in the process of identification can be divided into home team and away team.

Home team (antemortem subsection):

Home team is responsible for the collection of antemortem data and also transmits the information to the away team. Before starting the actual work the team leader should brief police officers about the procedures to be followed. All antemortem dental records including charts, radiographs, models and photographs of missing victims should be collected from dentists, hospitals and other sources. The radiograph

should be of good quality, properly mounted, labeled and original. In addition, copies of victim's dental treatment progress notes should be collected as well¹⁰. Once the records are collected they have to be entered in a standardized antemortem data form (eg. antemortem record chart, Interpol design). Entry should be made by at least two members of the team.

The dental data needs to be collected in the country of origin, by a forensically trained dentist or dentists. Family practitioners are either too busy to prepare adequate records, or not aware of the unique requirements of disaster victim identification (DVI) charting and radiographs¹¹.

Away team (Postmortem dental examination subsection):

This team performs examination of the victim, prepares the postmortem dental data and compares with the antemortem data provided by the home team. Examination of the remains is carried out by two or three members and cross checked. Extra and intra oral radiographs and photographs, panoramic radiograph (OPG) should be taken. Resection of the jaws, when permitted, significantly simplifies the postmortem radiographic technique¹². While taking radiographs, necessary precautions should be taken to avoid exposing the people working in the area. Postmortem computed tomography (CT) is a fast and because of the non-invasiveness, a great help in the no-touch documentation of fragile and brittle teeth in carbonized bodies⁸.

Dental charts must be filled accurately. The postmortem forms are divided into various subsections according to sex, age and types of dental treatments. When only portions of body are recovered, the forms can be kept under "unsexed" subsection. A master copy is prepared with all postmortem records in numerical order.

Comparison methods

Comparison of postmortem and antemortem dental records, radiographic films or digital images is a common procedure for establishing identity of human remains. Comparison may be performed by manual method or by using computers. Classical methods for forensic dental identification are clinically used radiological documentation techniques such as dental periapical radiographs, bitewing films and panoramic radiographs (OPGs). Use of computers reduces tedious and time consuming manual sorting of records⁵.

Technologic aids in mass disaster identification analysis

Advances in photographic, radiographic, and computer technology have provided the forensic dental team with additional resources to enable recovery, documentation, storage, and comparison of forensic dental evidence in MFIs, as well as in other situations requiring forensic dental expertise (e.g., bite mark analysis, documentation of human abuse). Among these advances are developments in the following²:

Digital photography

The basic digital camera used for forensic evidence documentation should include a through-the-lens (TIL), light-metering, SLR, 35-mm digital camera body with interchangeable lenses or an adjustable lens capable of normal range (30 to 50 mm) to macro range (90 to 100 mm) focal length. A removable flash memory card with adequate storage capacity is also required.

Digital radiography (DR) equipment

Electronically generated and stored radiographic imaging can be accomplished by the following:

- Scanning normally processed radiographic film into a computer.
- Using a phosphor substrate shaped and used like radiographic film to expose and scan radiographic information into the

- computer by a special propri-etary device.
- Using a sensor sized and shaped like a radio-graphic film that is made of a scintillation screen and a charge-coupled device (CCD) or complementary metal oxide semiconductor (CMOS).

Direct digital radiography (DDR)

When ener-gized by radiation, this device creates a direct image on the pixels of its CCD or CMOS. This radiographic image is then sent to a computer through wire or wireless technology. Thus because of its ability to save time, DDR technology is recommended for clinical and forensic casework.

Cone-beamcomputed tomography (CBCT)

CBCT provides a 3D imaging modality to collect a complete maxillo-mandibular- facial anatomic volume of data. Application of CBCT in forensic dental situations can overcome intraoral access prob-blems with some specimens (e.g., fourth-degree burn cases).

Portable hand-held x-ray generation devices (e.g., Nomad manufactured by Aribex, and MinXray HF70DUL Type A

The forensic dentist is able to expose film or digital radiographs quickly and effortlessly with a battery-powered unit that can be carried to the body on the gurney in the morgue. Additional applications for the use of these devices in the dental office include exposure of radiographs on pediatric or sedated patients or those having end-odontic therapy.

X-ray fluorescence (XRF) methodology

Analysis of dental materials in cre-mation and other difficult forensic identification cases may be facilitated by analysis of specimens with this technology.

Computer software technology

The advent of computer software has assisted MFI dental identi-fication teams in filing, storing, sorting, and matching bits of antemortem and postmortem information. Computer assistance has proved beneficial in disas-ters involving hundreds of victims. Commonly used programs include the following:

FBI-NCIC program, based on the California Dental Identification System, developed by Dr. Norman Sperber and Dr. Robert Siegel.

CAPMI-4 (Computer-Assisted Postmortem identi-fication-version 4.0), developed by Dr. Lewis Lorton of the U.S. Army Institute of Dental Re-search and maintained by the Armed Forces Insti-tute of Pathology (It was first used in 1985 in support of the Arrow Air-U.S. military charter avia-tion runway accident in Gander, Newfoundland.)

WinID3 dental comparison software, developed by Dr. James McGivney (St. Louis, Mo.) (Bridged with the Dexis DR program, WinID3 facilitated comparison of antemortem and postmortem dental records in Hurricane Katrina recovery efforts and various transportation and industrial MFI events.)

Each of these computer software systems is user friendly, can be run on readily available and accessible hardware, is automated and capable of networking and relies on objective data entry².

Problems during mass disaster identification

It has been found that the major problems which are faced in a forensic identification at the time of mass disaster include large number of human remains that can be scattered throughout broad areas. The bodies may be fragmented, commingled and severely burned or there may left only skeletal remains.

Difficulty in acquisition of meaningful medical/dental records and radiographs as well as internal and external documentation and communication problems can be present.

Proper facilities may not be available to carry out

examination, because usually temporary mortuaries are set up in the scene of disaster.

Legal, jurisdictional, organizational and political issues may further complicate the identification problem.

Conclusion

Disaster Victim Identification, is a difficult task and has to involve the active participation of many other organizations and agencies. This can only be brought to a successful conclusion if properly planned and well deployed. Well trained officers or volunteers in collecting antemortem data from the bereaved families are needed. Manpower strength available for instant activation should be established. These should include local personnel, law enforcement agencies, transportation companies as well as specialists such as forensic odontologists, forensic pathologists, forensic anthropologists, fingerprint specialists etc., with good teamwork. Equipment and supplies should be maintained in a ready condition at all times. The material should be periodically checked and outdated materials should be replaced as necessary. Preparation of morgue facilities should be done by considering the following factors - space, security, communication facilities, electrical provisions, technological support, accessibilities to disaster sites, assembly point, viewing space.

It is very important to establish a communication centre, the people in charge of the centre should responsible for the release of the information to the media. The education and training of the disaster team and related group should be done periodically to ensure readiness of the team, to update and refine the protocol for better performance.

References

1. Pretty IA, Sweet D. A look at forensic dentistry—Part 1: the role of teeth in the determination of human identity. *Br Dent J* 2001;190:359–66.
2. Neville BW, Damm DD, Allen CM, Bouquet JE. *Oral and Maxillofacial Pathology 3rd Edition*. Forensic Dentistry. 2009; 887-916
3. Sirisup N, Kanluen S. Role of Forensic Doctors in Thailand's Tsunami: Experiences from Chulalongkorn Medical School. *J Med Assoc Thai* 2005; 88(4):335-338
4. Avon SL. Forensic Odontology: The Roles and Responsibilities of the Dentist. *J Can Dent Assoc* 2004; 70(7):453–8
5. Dayal PK, Srinivasan SV, Paravathy RP. *Text Book of Forensic Odontology*. 1st Edition 1998, Paras Medical Publishers, Hyderabad
6. Toms C, Rogers CB, Sathyavagiswaran L. Investigation of Homicides Interred in concrete-The Los Angeles Experience. *J Forensic Sci* 2008; 53(1): 203-207
7. Flint DJ, Dove SB, Brumit PC, White M, Senn DR. Computer-aided Dental identification: An Objective Method for Assessment of Radiographic Image Similarity. *J Forensic Sci* 2009; 54(1):177-184
8. Thali MJ, Markwalder T, Jackowski C, Sonnenschein M, Dirnhofer R. Dental CT Imaging as a Screening Tool for Dental Profiling : Advantages and Limitations. *J Forensic Sci* 2006;51(1):113-119
9. Slavkin HC. Sex, enamel and forensic dentistry: a search for identity. *J Am Dent Assoc* 1997;128:1021-1025
10. Cardoza AR. Dental Forensic Identification in the 2003 Cedar Fire. *J Calif Dent Assoc* 2004;32(8):689-693
11. Kieser JA, Laing W, Herbison P. Lessons Learned from Large-scale Comparative Dental Analysis Following the South Asian Tsunami of 2004. *J Forensic Sci* 2006;51(1):109-112
12. Mincer HH, Chaudhry J, Blankenship JA, Turner EW. Postmortem Dental Radiography *J Forensic Sci* 2008;53(2):405-407

Acute copper sulphate poisoning: A case report

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Abstract

Copper sulphate toxicity is a rare event in the US but it is commonly used as a form of suicide in India. It is commercially available and found in products of fungicides, insecticides, and is used in whitewashing, leather manufacture and to bind colors to fabrics. However, the pathophysiology of acute copper intoxication is not well understood and its management has not been established. Here a case of suicidal ingestion of copper sulphate in a young male is presented along with a brief description about the management and literature review regarding copper sulphate poisoning.

Key Words

Copper sulphate; acute poisoning; fatal.

Introduction

Copper is an essential trace material in Humans. It is vital for the functioning of certain enzymes such as Cytochrome C Oxidase¹. Copper sulphate is odorless, transparent blue triclinic crystals or crystalline granules or powder, having a pH of 4.0, specific gravity 2.28 at 15.6 C and a solubility of 31.6 per 100 cc of water. The compound is Stable under ordinary conditions of use and storage. When heated to decomposition, Hazardous decomposition products like cupric oxide and sulfur oxide may form. It is used in dyeing cotton and silk, manufacturing green and blue pigments, for electroplating with copper soap, ink for marking tin; hair dye; insecticide mixtures² (Bordeaux mixture, etc.) for treating the "white disease" of vines caused by Oidium, preserving bides, wood, and railway ties, tanning leather³, electric batteries, process engraving, destroying algae, etc., in pools and as primary standard in analytical chemistry. Due to its easy availability it is commonly used as a form of suicide in India⁴.

Case report

A 28 yrs old male, rickshaw puller by profession, was brought to the casualty wing with the history of ingesting some poisonous substance at home. He was allegedly suffering from depression for past few days regarding some financial problem. He was declared brought dead by the attending doctor and the body was sent to the mortuary for PM examination.

Fig. 1: Showing blue stains over external wall of stomach and its adjoining region.



During autopsy it was a dead body of young male of average built. Face was congested and no injuries were present over the external surface of the body.

During internal examination, a bluish colored material mixed with mucous was found to be present inside the esophagus. On opening the abdominal cavity, same material was found to stain the external walls of the stomach and the adjoining omentum and intestines (fig 1).

On opening the stomach, about 150 ml of bluish material was present inside it and its walls were congested showing patchy hemorrhages at places (fig 2).

The viscera were sent for chemical analysis whose report shows presence of copper sulphate in the stomach and intestinal contents. The cause of death given was acute copper sulphate poisoning.

Discussion

The lethal dose of cupric sulphate has been described to be as low as 1 gm¹. Ingested copper induces mucosal irritation, nausea, vomiting and diarrhea. Ionized copper is readily absorbed from stomach and intestine, and the serum copper level increases rapidly. The element is bound to albumin and ceruloplasmin, and is taken up by liver, kidneys, lungs and red blood cells. Hemolytic anemia and renal tubular necrosis may follow 36-48 hrs after exposure. The primary route of excretion is through bile and feces².

The toxicity of copper at cellular level is probably related to sulfhydryl groups. Copper inhibits sulfhydryl moieties of Glucose-6-phosphate Dehydrogenase and Glutathione, thereby reducing their free radical scavenging activities. Copper induces hemolysis through oxidation of hemoglobin sulfhydryl groups. Copper also inhibits Na⁺/K⁺-ATPase and increases the permeability of cell membrane. Since copper is known to damage human skeletal muscle cells³, copper intoxication could cause rhabdomyolysis. Although a case of copper-induced acute rhabdomyolysis in Wilson's disease was reported⁴, rhabdomyolysis in acute copper intoxication has been rarely reported. This might be because myoglobinuria might be overlooked by the coexistence of hemoglobinuria secondary to hemolytic anemia. The treatment for ingested copper overdose includes dermal decontamination, cautious gastric lavage and supportive therapy⁵. Dimercaprol, penicillamine and edetate calcium disodium might be considered for massive copper ingestion, and persistent symptomatology or

Fig. 2: Showing Stomach content.



persistently elevated serum copper concentrations. For serious poisoning, it is considered best to administer dimercaprol intramuscularly 4 mg/kg/dose every 4 hours for 5-7 days. Penicillamine is usually administered orally in doses of 250-500 mg/dose every 8-12 hours. Edetate calcium disodium is also a drug of choice, but the agent has not been approved in Japan for copper intoxication and so was not used in this case. Dialysis or hemoperfusion has not been demonstrated to increase the elimination of copper, since copper binds to serum and tissue proteins. However, chelated copper would be removed from serum by diuresis and dialysis.

References

1. Haddad LM, Whinchester JF. Clinical management of poisoning and drug overdose. 2nd Edn. WB Saunders. Philadelphia. 1990; 1030-1031.
2. Walsh FM, Crosson FJ, Bayley M et al. Acute copper intoxication: pathophysiology and therapy with a case report. *Am J Dis Child.* 1977;131:149-151.
3. Chuttani, Gupta, Gulati, Gupta. Acute copper sulfate poisoning. *Am J Med.* 1965; 39: 849-854.
4. Klein WJ Jr, Metz EN, Price AR. Acute copper intoxication: a hazard of hemodialysis. *Arch Intern Med.* 1972; 129: 578-582.
5. Stein RS, Jenkins D, Korn ME. Death after use of cupric sulfate as emetic. *JAMA* 1976;235: 801.
6. Jantsch W, Kulig K, Rumack BH. Massive copper sulfate ingestion resulting in hepatotoxicity. *Clin Toxicol.* 1985; 22: 585-588.
7. Benders AA, Li J, Lock RA, Bindels RJ, Bonga SE, Veerkamp JH. Copper toxicity in cultured human skeletal muscle cells: the involvement of Na⁺/K⁺-ATPase and the Na⁺/Ca²⁺-exchanger. *Pflugers Arch.* 1994; 428: 461-467.
8. Propst A, Propst T, Feichtinger H, Judmaier G, Willeit J, Vogel W. Copper induced acute rhabdomyolysis in Wilson's disease. *Gastroenterology.* 1995; 108: 885-887.
9. Leikin JB, Paloucek. *Poisoning & Toxicology Handbook.* 2nd ed. 1996-97. Lexi-Comp Inc., Ohio, 1995: 896-898.

Forensic implications of parasomnias

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Introduction

Parasomnias are a group of undesirable clinical events which appear during sleep or are exacerbated by sleep¹.

These are divided into two categories:

1. Arising from NREM sleep and
2. Arising from REM sleep¹.

Out of seventeen parasomnias, five parasomnias like confusional arousal, sleep walking, sleep terror, rhythmic movement disorder and REM behaviour disorders interestingly make up 80% of parasomnias seen in the general population. These five conditions are gaining importance in forensic medicine because of the occurrence of violence.

Recent research activities have shown about 2% of the adult population suffering from the above sleep disorders are involved with violent behaviours^{1,2}. Sleep walking, sleep terrors and confusional arousal, the three main pillars of violence, are common sleep disorders to occur mostly in children and 20% of adults³ making it 1 – 2% of the adult population.

Violence during sleep needs to be distinguished from the conditions where the patient may appear to sleep but is actually awake.

Violence during sleep thus has characteristic features which involve:

1. No signs of premeditation of attack,
2. No existence of previous animosity,
3. Lack of motive for attack – attack is senseless, impulsive and abrupt,
4. No personal gain from violence,
5. Complete amnesia of event though in a few cases the aggressor may regain awareness towards the end of the incident,
6. No desire to conceal the evidence about the incident,
7. Incident is out of character for the aggressor,
8. Occurrence of guilt and surprise after the incident,
9. Period of action is usually brief,
10. Should have history or have been investigated by sleep laboratory for presence of sleep disorder,
11. Lack of support that the episode was due to direct drug abuse, medication or a general medical condition,
12. A blank staring face, relative unresponsiveness and the inability to be awakened except with great difficulties,
13. Clinically significant distress or impairment in social, occupational or other important functions related to sleep walking,
14. Last but not least, the sleeper is unresponsive to the situation and intense stimulation occurring during the incident.

Sleepwalking

In sleepwalking the patient characteristically gets out of bed, stands, walks and carries out complex tasks. Sleepwalking may take the form of slow wandering and may be agitated, running or other associated motor behaviours. Sleepwalking may also follow sleep terrors or confusional arousal. However, in sleepwalking, violence occurs only after sleepwalking has been triggered and is common in the adult male.

The newest helpful investigatory information for sleepwalking includes:

1. arousal from slow wave sleep (SWS)
2. Hypersynchronous delta wave (HSDW)
3. Increase in cyclic alternating pattern
4. Result of frequency analysis of delta wave activity
5. Provoking the sleepwalking incident in a sleep laboratory, sleep depriving the person or using alcohol prior to bedtime to trigger the episode are two examples.

Confusional arousals

Confusional arousals are sudden, partial arousals from SWS (slow wave sleep) resulting in complex behaviours but most importantly the person involved does not leave the bed. These are associated with severe cognitive impairment and followed by amnesia. The victim in this condition touches the sleeping individual or attempts to arouse the individual sleeper. In this condition, 100% of the cases conform to the generally held belief of provocation and or proximity triggering sleep related violence. This diagnosis also has shared pathophysiology with sleepwalking.

Sleep terrors

Sleep terrors are often started by a frightening image or images with marked sympathetic nervous stimulation. The manner of presentation may be agitated, panicky or anxious. In most cases visual imagery of sleep terror is less complicated and complex than a typical nightmare. The images usually are of an attack or assault. Thus in response to violent imagery violent behaviour is produced in the sleeper.

Many sleepers have sleep terrors followed by sleepwalking with agitated complex behaviour leading to violence. Sleep terrors thus can easily be differentiated from sleepwalking and confusional arousal by history in most cases.

Rhythmic Movement Disorder (RMD)

Rhythmic movement disorder also called "jactatio capitis nocturna" is diagnosed by repetitive banging or rocking motions just before and during light sleep. The presentation includes head banging, head rolling, body rocking type and body rolling type. RMD is alarming and many cases cause self violence.

REM sleep behaviour disorder

REM Sleep Behaviour Disorder is identified by a feature when during REM sleep, active paralysis of all somatic musculature except the diaphragm and extraocular muscles occurs. There is absence of expected REM sleep atonia which thus allows "acting out" of dreams with dramatic and significant violent or injurious consequences.

Interestingly there is no evidence of aggression in the wakeful state of the patient. Many neurological conditions including Parkinson's disease, multiple system atrophy and dementia cause this condition. As males predominate this condition, the related sexual hormone to aggression and violence has been suggested to be one of the causative factors. Polysomnographic

investigations confirm an increase in tonic and/or phasic electromyographic activity more often accompanied by muscle twitching, vocalization and flailing during REM sleep. If these progress then talking, laughing, shouting and direct injurious action towards the bed partner occurs. There are often vivid violent dreams which may be repetitively causing an increased number of bouts of violent activity.

Etiological considerations

The occurrence of the above disorders require predisposing, priming and precipitating factors.

Predisposing factors have a genetic and familial basis. In first degree relatives the development of sleep walking is ten times more than normal⁴ and recent publication of HLA gene D₈B₁ is present in 35% compared to 13.3% of normal population. Priming factors include conditions and substances that increase slow wave of sleep or make arousal from sleep more difficult. These factors are sleep deprivation, alcohol, medications, fever and situational stress.

Priming factors with genetic predisposition are present in deeper sleep, fragmented sleep and/or difficult arousal from sleep. Elevated quantity or percentage of SWS has been seen in sleep walking^{5,6}. Besides, decreased power in delta range in the first NREM period and a different trend of delta power during night are seen when SWS is analyzed by frequency analysis.

Increased number of arousals in sleep walkers during SWS is one of the most common reported sleep laboratory studies and in the opinion of many sleep scientists could be used as a potential diagnostic marker^{7,8,9,10,11,12-17} in both a clinical or forensic setting. This observation has lead to acceptance that sleepwalking and related disorders belong to NREM instability.

Sleep deprivation remains the most common finding in sleep walkers. All forms of sleep deprivation are known to increase arousal threshold¹⁸ and also cause the occurrence of complex behaviour from SWS.

Medications

Medications are reported to be associated with sleepwalking and almost all psychotropic drugs as well as many other type 7 drugs. However, no controlled study of the role of medication has been done.

Alcohol

Multiple studies^{5,6,19} have confirmed the association of alcohol abuse with the occurrence of sleepwalking. An automatism defense of alcohol abuse is very common in sleepwalking.

Fever

Fever has been responsible in many cases of sleepwalking in children and teenagers but not found to be a cause in adults.

Stress

Stress is frequently noted as a factor in sleepwalking^{5,6,19} but should be carefully differentiated from psychiatric disorders.

Precipitating factors/triggers

In most cases specific triggers are observed and these include a) sleep disordered breathing, b) periodic leg movement (PLMs) c) noises d) touch.

Forensic implications

Forensic scientists have to look for all the evidence

supplied by 1. history 2. predisposing factors, 3. priming factors and 4. precipitating factors or triggers.

Drug or alcohol related situations and behaviours must be separated from sleepwalking. Violent behaviour caused by malingering, a fugue, temporal lobe epilepsy, seizures and encephalopathy must be ruled out. Guidelines for assessment of possible sleep related violence should be followed as described earlier (page 2-3).

Forensic implications of investigation described above should be kept in mind.

Common finding of occurrence of arousal during SWS by sleep laboratory is not conclusive in establishing or refuting a tendency towards sleepwalking¹⁴.

Sleep deprivation studies performed after a criminal act describes defendants' current behaviour but may only be indicative of past of the behaviour.

A sleep specialist should remember that medications can cause episodes when the person involved has no other factors related to sleepwalking. Thus utilizing a sleep laboratory to prove that medication caused the sleepwalking still lacks the basis of accepted forensic science.

Forensic implications of alcohol induced automatism has been the defense plea in many cases but in most jurisdictions in the world, voluntary alcoholic intoxication is not accepted as a complete defense of a criminal act. During automatism the question of what happened during the incident should be analyzed. The claim of alcohol induced sleepwalking should be first evaluated for the possibility that criminal behaviour has occurred by alcohol intoxication alone before considering the much less likely defense of sleepwalking²⁰.

Experimental attempts to stimulate sleepwalking episodes have been tried but the real goal of establishing the fact that the crime was committed on the day of the episode is not completely met by this. Secondly, the provocation study may not involve the priming or precipitating factors of a crime episode. Lastly, the sleep laboratory has a very artificial environment and thus cannot be the absolute defense for a criminal act.

It is worthwhile and important to be familiar with the concept of Junk Science and the goal should be to have an impartial expert or "amicus curiae". Most of the sleep societies have guidelines for expert witnesses which should be followed.

Conclusion

Sleepwalking and related disorders are the end result of complex set of predisposing, priming and precipitating factors. However, having few of these factors might not be enough to produce sleepwalking and sleep related crime. On the other hand, sleep laboratory based investigation in its own right cannot retroactively establish the diagnosis.

Still nothing as yet in sleep medicine produces definite proof for the crime committed during sleepwalking. However, inclusion of integrated evidence including history, clinical facts (including predisposing, priming and precipitating factors), differential analysis and investigations including that of a sleep laboratory, all based on current sleep science, will help to form most often the correct diagnosis.

The future requires more research in the basic and clinical science of sleep medicine including: genetic, neurologic, neuroplastic and socioenvironmental components for clarifying the violence related to sleep medicine in general and to sleepwalking and sleepwalking related disorders, in particular.

References

1. Singh AN. Parasomnias and Management. International Medical Journal Sep 1997;4(3):3-7.

2. Ohayon MM, Caulet M, Priest RG. Violent behaviour during sleep. *J Clin Psychiatry* 1997;58:369-76.
3. Mahowald MW, Schenck CH. Finally – Sleep science for the courtroom. *Sleep Medicine Reviews*. Elsevier 2007 11, 1-3.
4. Shneerson JM, Sleep violence. *British Journal of Hospital Medicine* June 2009: Vol 70 No 6, 332-335.
5. Kales A, Soldatos CR, Bixler EO, Ladda RL, Charney DS, Weber G. Hereditary factors in sleepwalking and night terrors. *Br J Psychiat* 1980;137:111-8.
6. Mahowald M, Cramer Bornemann MA. NREM sleep-arousal parasomnias. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th ed. Philadelphia PA: Elsevier Saunders; 2005. p. 917-25.
7. Broughton R. NREM arousal parasomnias. In: kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 3rd ed. Philadelphia, PA: W.B. Saunders; 2004. p. 693-706.
8. Joncas S, Zadra A, Paquet J, Montplaisir J. The value of sleep deprivation as a diagnostic tool in adult sleepwalkers. *Neurology* 2002;26-58(6):936-40.
9. Pressman MR. Hypersynchronous delta sleep EEG activity and sudden arousals from slow wave sleep in adults without a history of parasomnias: clinical and forensic implications. *Sleep* 2004; 24(4):706-10.
10. R. v Catling, 2005.
11. R. v Lowe, 2004.
12. Espa F, Dauvilliers Y, Ondze B, Billiard M, Bisset A. Arousal reactions in sleep walking and night terror adults: the role of respiratory events. *Sleep* 2002;25(8):871-5.
13. Espa F, Ondze B, Billiard M, Bisset A. Sleep architecture, slow wave activity, and sleep spindles in adult patients with sleep walking and sleep terrors. *Clin Neurophys* 2000; 111: 929-39.
14. Brozen B, Foldvary NR, Dinner DS, Loddenkemper T, Lim L, Gollish JA. The value of the unexplained polysomnographic arousal from slow-wave sleep in predicting sleepwalking and sleep terrors in a sleep laboratory patient population. *Sleep* 2003;26:A325.
15. Gadreau H, Joncad S, Zadra A, Montplaisir J. Dynamics of slow-wave activity during the NREM sleep of sleepwalkers and control subject. *Sleep* 2000;23:755-60.
16. Broughton R. Phasic and dynamic aspects of sleep: a symposium review and synthesis. In: Terzano MG, Halasz PL, Declerck AC, editors. *Phasic events and dynamic organization of sleep*. New York: Raven Press 1991. p. 185-205.
17. Schenk CH, Pareja JA, Patterson AL, Mahowald MW. Analysis of polysomnographic events surrounding 252 slow-wave arousals in thirty-eight adults with injurious sleepwalking and sleep terrors. *J Clin Neurophysiol* 1998; 15(2): 159-66.
18. Blatt I, Peled R, Gadoth N, Lavie P. The value of sleep recording in evaluating somnambulism in young adults. *EEG Clin Neurophysiol* 1991;78:407-12.
19. Bonner MC. Acute sleep deprivation. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th ed. Philadelphia, PA: Elsevier Saunders; 2005. p. 51-66.
20. Mahowald MW, Schenck CH. Parasomnias: sleepwalking and the law. *Sleep Med Res* 2004(4):321-39.
21. Pressman MR. Factors that predispose, prime and precipitate NREM parasomnias in adults: Clinical and forensic implications. Elsevier: *Sleep medicine reviews* (2007) 11, 5-30.

An unusual case of death due to buffalo gore - A case report

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Abstract

The cows and buffalos are most commonly domesticated animals in the rural India. Deaths resulting from these animals are quite naturally common in the rural population. People who are passers by the road side, near a herd of buffalos are also prone of injuries caused by goring of these animals. A case report is presented here which signifies that death can occur in a patient gored by a Buffalo, where even though there was no penetration of the horns, but still the patient died as a result of injuries from the blunt impact from the horns and also the impact injuries sustained to the head from being thrown off the ground.

Key words

Bull gore, Buffalo gore, domesticated animal, non-penetrating injuries.

Introduction

In spite of urbanization, millions of injuries and hundreds of deaths occur as a result of animal attacks all over the world.¹ Bull gore injuries are frequently observed in villages and are different from other casualties like stab injuries and road side accidents². The cattle and bull are easily domesticated animals which cater to the needs of the population at large. The bulls are usually domesticated to plough the agricultural fields, for transportation of agricultural products and even people in our rural India. Besides these, the cows and buffaloes provide us with milk, which is the prime ingredient for various dairy products. These domesticated animals sometimes get angry for no reason and will gore the people nearby causing various types of injuries depending upon the anatomical region involved which may sometimes be fatal.

The injuries caused by the horns of the bulls, cows and buffaloes are of various shapes, size and direction and are goring in nature and violent. The wounds produced are contusions, laceration, criss-cross wounds, penetration of body cavities and rarely fractures. The subcutaneous tissues and muscle are frequently affected². Men who come in contact with these large animals in their day to day routine work are in fact at risk of injuries caused by these animals. To quote with while grazing, while feeding, while milking the animal's human beings are at greater risk of being gored by these animals². The problem of stray cattle, which has led to many road accidents, including fatalities is quite rampant. Amongst these, deaths caused by bulls and other cattle's are quite common in rural India³.

The term "Bull horn" injury is not strictly correct because the injury is more often due to the horn of a cow or buffalo

rather than a bull⁴. The horns of the bull and cow being projected more forward and being gored will usually result in a penetrating injury to the abdomen and perineal region depending on the position of the victim. The horns of the buffalo not being so vertical (as shown in photograph 1) will result in a non-penetrating injury which is also fatal. In this case report, we present an unusual case of a old man who died as a result of buffalo gore, since he was thrown to a distance due to goring, resulting in secondary injuries to his head from impact to a Tractor trailer parked on the road side (shown in photograph 2).

Case report

An old man aged about 70 years was gored by a buffalo while he was walking beside a herd of buffaloes on the evening of 13/01/10 at his village about 40 kilometers from the city of Davangere. This man being short stature i.e the animal gored and threw him for a distance due to which his head hit the Tractor trailer parked beside the road. He was admitted to S.S.I.M.S. Hospital & R.C on the same evening and the patient died on 21/01/10. Autopsy was conducted at Mortuary, Department of Forensic Medicine, J.J.M. Medical College and the following external injuries were noted.

- A contusion of size 6cms x 3cms was present over the left side of the chest 22 cms below in the mid-clavicular line, situated 10 cms to the left of midline of the sternum, which was due to the horns of the buffalo.
- Abrasion of size 13cms x 4cms present over the upper part of the left side of the back situated 3 cms below the left shoulder joint (shown in photograph 3).
- Bone deep laceration of size 12cms x 4cms present over the left temporo-parietal region situated 10 cms above the left mastoid process (shown in photograph 3). This was due to the secondary injury sustained from the head hitting the Tractor Trailer as the victim was thrown off the ground. Internal examination-
- Sub-Dural haematoma of size 50gm was present over the right temporo-parietal region (shown in photograph 4 &5)
- Sub- Arachnoid hemorrhage was present over the left temporo-parietal region.
- Multiple contusions of size 2cm X 1cm, 1.5cm x 1cm, 1cm x 1cm were present through out the thoracic part of the aorta (shown in photograph 6).
- Laceration of the spleen of size 2cm x 1cm was present on the upper surface of the spleen (shown in photograph 7).
- Liver, Kidney, Lungs and brain were pale
- About 1000ml of blood was present in the peritoneal cavity.

The cause of death was due to hemorrhagic shock as a result of sub-dural haematoma and laceration of spleen sustained from Buffalo gore injury.

Discussion

The factors determining the severity of injury are

1. **The weight of the individual:** The lighter individual will be carried off the ground with greater ease as compared to a stout person, there by resulting in lesser damage by

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Photograph - 1: Buffalo with inturned horns.



Photograph -2: Tractor trolley parked along the roadside caused secondary injuries.



Photograph -3: Secondary injuries caused by the tractor trolley.



Photograph - 4: Subdural hematoma involving the right temporo-parietal region.



the horn, but more harm may follow due to the fall resulting in bony injuries⁴. In our case as the deceased was an old man with 156 cms stature and weighed, 52 kgs, so he was literally thrown off the ground by the Buffalo due to which his head hit the parked Tractor trailer resulting in secondary injuries and death rather than the horns entering the body cavities.

- 2. **Force of impact:** The force of impact is naturally more when a bull is charging, than when it is stationary⁴. In our case since the old man was walking next to a herd of Buffaloes, the force of impact was less, an was thrown off the ground.
- 3. **Shape and length of the horns:** A severe injury is to be expected with a shorter but sharper horn as compared to long, stub, in turned or flayed out horns⁴. In our case

Photograph - 5: Subdural hematoma involving the right temporo-parietal region.



Photograph - 6: Contusion in the posterior part of the thoracic aorta.



Photograph - 7: Rupture of the spleen on its superior surface



since the injury was caused by a Buffalo, which usually has a smooth in turn horns, only a mild injury was observed in the form of contusion at the site of impact.

The blunt injuries to the chest constituted to 8.5% with 42.8% of these injuries having rib fracture with surgical emphysema and pneumothorax in the study conducted by Rau and Reddy⁵. In our case injury in the form of contusion of the left side of the chest was present but with no rib fractures. Laceration of the spleen was also present in our case.

The horns were classified as safe and unsafe horns. In the former, the tip of the horn is blunt and has wide smooth curvature form its base. These horns are commonly seen in Buffaloes and produce contusions and blunt injuries. The unsafe horns are further classified as potentially dangerous and dangerous. The potentially dangerous horns are long, curved, directed forwards and with smooth tapering ends. These types of horns produce lacerations and rarely penetrate the body cavities. The dangerous type have short sharply curved with slightly outward deviation and pointed ends. The cows and some bulls possess these horns and cause violent and severe injuries which also penetrate the body². In our case we could appreciate only blunt injury on the left side of the chest, emphasizing the fact that the horns were blunt and had wide smooth curvature from its base which is typically seen in a Buffalo gore.

This case is unusual because the deceased person was thrown off the ground, which usually occurs in case of young children and old people with short stature and less weight, resulting in impact injuries to the near by objects. Though there were no penetrating horn injuries, but still the patient died as a result of internal injuries sustained due to the blunt impact to

the nearby tractor. This signifies that a person may die not only because of the penetrating injuries but also because of the impact to nearby objects without a penetrating injury as in our case.

Conclusion

The injuries caused by the bull and buffaloes are varied depending upon the site of impact. To prevent injuries to human beings dehorning (amputation) is the only solution. Besides fixation of brass cups to the horns will prevent the injuries to some extent. Some unruly animals are tied with a wooden log to their necks which will retard the animal's speed of walking and by seeing them the pedestrians are also warned. To prevent any casualties it is advised to keep a safe distance from these animals when we are passing by a herd of cows and buffaloes.

References

1. Dogan KH, Demirci S, Erkol Z, Sunam GS, Kucukkartallar T. Injuries and Death Occuring as a Result of Bull Attack. *Journal of Agro medicine*, 2008; 13(3): 191-196.
2. Rau JBV. Bull gore injuries in Rural areas. *Indian Journal of Surgery* Oct -Nov 1982; 664-671.
3. Rani M, Rohit, Sharma A. Dikshit PC. Injuries by Bull Horns: Patterns and Prevention Protocols. *Anil Aggrawal's Internet Journal of Forensic Medicine and Toxicology*, 2010; 11(1)
4. Sekhon MS, Khatri HL, Grewal SS, Marya SKS. Bull horn injury. *Indian Journal of Surgery*, Aug- 1983; 486-488.
5. Rau JBV, Reddy RSN. Avulsion of jejunal segment-blunt abdominal trauma. *The Clinician*, 1975; 39: 320- 322.

A study of estimation of stature from length of fingers in Mysore

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Abstract

This study looks into the possibility of estimation of stature from the length of the fingers. 50 male and 50 female subjects were taken as subjects. All were aged more than 21 years and less than 30 years. Their family origins were from Mysore and neighbouring areas. The subjects' height and finger lengths were measured and independent linear regression equations were obtained. The best finger to predict the height in case of males was found to be the left thumb and in case of females it was the right thumb.

Key words

Finger length; Height; Stature

Introduction

The identification of mutilated or fragmented remains is a challenge for the medico-legal expert. The stature or the height is an important part of the identity of a person and although various methods and formulae are available for the estimation of stature, most of them utilize bones such as femur, tibia, humerus and radius. Very few workers have correlated height and finger length.

Macdonnel studied 3000 English criminals and compared the stature with the length of the middle finger¹. Tyagi et al studied from Delhi. They found accurate or near accurate correlation between stature and finger lengths and have suggested that the index finger is best for the prediction of stature in both males and females²¹.

The height of a person increases from intra-uterine life up to 20 to 21 years³. Trotter and Glesser found that there is a loss of height for each two decades of age over the age of 30 years⁴. Therefore in the present study the age of the subjects was taken as more than 21 years and less than 30 years.

Materials and methods

The subjects were 50 male and 50 female staff and students of various institutions of JSS Mahavidyapeetha, Mysore. All were aged more than 21 years and less than 30 years and their family origin was in Mysore, Mandya, Hassan or Chamrajnagar. Subjects with congenital or acquired skeletal abnormalities were excluded from the study.

Informed written consent was obtained prior to taking measurements. To measure the height an anthropometer was used.

The subject was asked to stand erect and barefoot against a wall. The feet were kept parallel to each other. The heels, buttocks and back touched the wall and the head was kept in the eye-ear plane, eyes facing forwards. The anthropometer was placed in front of the subject, perpendicular to the floor. The lower horizontal bar was brought in contact with the head at the vertex, in the mid-sagittal plane. The distance between the floor and the horizontal bar gave the height of the person.

To measure the length of the fingers a sliding caliper was used. The subject was asked to place the hands on a flat table,

palms facing downwards. The proximal point, that is the phallangion was noted by palpating the joint space. The distal point, that is the dactylion was the distal-most point of the finger. The ends of the caliper were placed on these anatomical landmarks and the distance between them gave the length of the finger.

Independent linear regression equations to calculate the height were obtained for each finger, separately for males and females.

Results

Conclusion

In case of male subjects, all the values showed a high degree of correlation and all values were statistically significant (p value < 0.05). Therefore any of the ten fingers can be used to calculate height. The best finger was found to be the left thumb (with the highest r² value).

Among the female subjects, only some of the values were statistically significant. These fingers were the left middle, left thumb, right index, middle, ring fingers and thumb. Therefore

Table 1: Results obtained for male subjects.

Finger Length(cm)	Regression Equation (Height =)	Percentage Explained(r ²)	P Value
Li	112.311 + 6.441 (Li)	0.23	0.000483
Lm	117.107 + 5.269 (Lm)	0.21	0.000949
Lr	127.121 + 4.4779(Lr)	0.15	0.005501
Ll	129.261 + 5.331(Ll)	0.18	0.002421
Lt	111.796 + 9.225 (Lt)	0.33	0.000015
Ri	110.536 + 6.587 (Ri)	0.26	0.000150
Rm	120.203 + 4.945 (Rm)	0.21	0.000846
Rr	118.968 + 5.290 (Rr)	0.23	0.000369
Rl	124.648 + 5.895 (Rl)	0.20	0.001101
Rt	115.785 + 8.598 (Rt)	0.26	0.000167

Table 2: Results obtained for female subjects

Finger Length(cm)	Regression Equation (Height =)	Percentage Explained(r ²)	P Value
Li	135.399 + 2.666 (Li)	0.06	0.081620
Lm	115.765 + 4.431 (Lm)	0.15	0.006285
Lr	124.891 + 3.707 (Lr)	0.10	0.023805
Ll	141.244 + 2.358 (Ll)	0.04	0.143001
Lt	121.162 + 6.198 (Lt)	0.16	0.004138
Ri	130.119 + 3.255 (Ri)	0.80	0.047776
Rm	117.546 + 4.263 (Rm)	0.14	0.006966
RR	123.531 + 3.824 (RR)	0.11	0.017878
RL	137.938 + 2.759 (RL)	0.07	0.060755
RT	116.077 + 7.153 (RT)	0.18	0.002503

Abbreviations:

LI- left index finger length RI- right index finger length
LM - left middle finger length RM- right middle finger length
LR- left ring finger length RR- right ring finger length
LL- left little finger length RL- right little finger length
LT- left thumb length RT- right thumb length

we recommend that only these fingers be used to estimate height in case of females. The best finger to predict height among females was found to be the right thumb.

This study was done on living subjects, therefore when only bones are recovered (without soft tissues), 2.5 to 4 cm should be added to the height obtained. This study took subjects from Mysore and neighbouring areas. There is scope for further study among people from other parts of the country as well.

The procedure of measuring the finger lengths and using them in the appropriate regression equations to determine the stature is a simple one. In case of fragmented or mutilated remains, this method can prove to be significantly useful in estimating the stature and in the ultimate identification of the individual, that is, fingers point to identity.

References

1. Krogman WM. The human skeleton in forensic medicine. 2nd ed. Springfield: Charles C Thomas, 1986: 153-187.
2. Tyagi AK, Kohli A, Verma SK and Aggarwal BBL. Correlation between stature and fingers length. International journal of Medical toxicology and Legal Medicine 1999; 1(2): 20-22.
3. Nandy A. Principles of forensic medicine. 2nd ed. Calcutta: New Central Book Agency, 2001: 86-89.
4. Saukko P and Knight B. Knight's forensic pathology. 3rd ed. London: Arnold 2004: 114-116.

Is dermatoglyphics a reliable criteria for quantifying oral and systemic diseases- A review

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Abstract

The word Dermatoglyphics was coined by Dr. Harold Cummins in 1926, at the University of Oklahoma. Dermatoglyphics have been correlated with skin patterns and external body features that can be used to construct diagnostic models for the purpose of personality identification as well as in diagnosis of phenotypic appearances. Today dermatoglyphics have been correlated with a number of oral and systemic diseases, and has been used in such diverse fields as Dentistry, Medicine, Genetic Research, Psychiatry and Anthropology. Dermatoglyphic examination is a very cost effective method and a reliable criteria for quantifying disease process, since sufficient evidences are there to prove same. what is being awaited is a mass education for same, it is expected that this review will be a much awaited step to give momentum to this reliable genetic marker i.e. Dermatoglyphic examination related to health and disease.

Introduction

The word Dermatoglyphics comes from two Greek words (derma, skin and glyphe, carve) and refers to the friction ridge formations which appear on the palms of the hands and soles of the feet¹. The term was coined by Dr. Harold Cummins at the University of Oklahoma in 1926. Considered the 'Father of Dermatoglyphics,' his research in the 1920s and '30s was contrary to the prevailing medical wisdom of his day. Twenty years later, when his findings were verified by the scientific establishment, Dermatoglyphics moved from obscurity to acceptability as a diagnostic tool.

The basis of considering dermatoglyphics is as follows- In humans, the development of the primary palate and the lip is completed by the 7th week of intra uterine life and that of secondary palate by 12th week. Abnormalities in these areas are influenced by a combination of environmental and heredity factors, but only when the combined factors exceed a certain level, can these abnormalities be expected to appear, this threshold theory advanced by studies of Carter and Matsunga is now generally accepted. The dermal ridges develop in relation to the volar pads, which are formed by the 6th week of gestation and reach maximum size between 12th and 13th weeks. This means that the genetic message contained in the genome - normal or abnormal is deciphered during this period and is also reflected by dermatoglyphics². Several studies have also shown that dermatoglyphic pattern are genetically determined^{3,6}. Today dermatoglyphics have been correlated with a number of oral and systemic diseases, and has been used in such diverse fields as Pediatric Medicine, Genetic Research, Psychiatry and Anthropology. Though there have been numerable diseases correlated to dermatoglyphics, review of literature is scanty on the subject. Present review attempts to sequentially encompass pathogonomic significance of dermatoglyphics in Oral as well as Systemic diseases. This sequential analysis of literature on this topic may lead to better research opportunities and diagnostic criteria through the field of forensic medicine and toxicology.

Dermatoglyphics as a diagnostic aid

From the mid 1930's onwards, after the work of Harold Cummins the hand was coming to be recognised as an important diagnostic aid in the diagnosis of congenital syndromes such as mongolism. LS Penrose had studied the hands of people with Down's Syndrome and other conditions of congenital mental defect. In 1931, he penned an article for The Lancet correlating the absence of the medial digital crease on the little finger with congenital mental retardation, research that proved to be but the start of a long and detailed investigation into the relevance of the hand in the clinical diagnosis of congenital conditions.

Oral diseases

Oral cancer

Oral cancer is the most common cancer in India. Related to multifactorial etiology most common cause is tobacco smoking. Being comprising 40% of all cancers. It has been demonstrated that arches and loops are more frequent in patients of oral cancer and there is a reduced frequency of atd angles and patterns in IV inter-digital area⁷. Other studies have shown that the dermatoglyphic finding can serve to strengthen a diagnostic impression when combined with other clinical features of patients with oral tumors⁸.

Dental caries

Dental caries is defined as an infectious disease of microbial origin. Caries are classified according to the location, extent and severity. Dermatoglyphic interpretation of dental caries have been proved in certain studies^{9,10}. Dermatoglyphic examination has revealed that caries free subjects increased frequency of whorls and where as caries free students have an increased frequency of loops on all fingers. This can be a valuable parameter in longitudinal studies and epidemiologic surveys since it is not only cost effective but a reliable method of quantifying dental caries.

Bruxism

Bruxism refers to the habitual, involuntary grinding or clenching of the teeth, usually during sleep and sometimes associated with stress. Characteristic dermatoglyphic patterns have been reported in subjects of bruxism. Bruxism patients demonstrated an increase in frequency of whorls and a decrease in frequency of ulnar loops, a lower frequency of atd angle than controls. Augmentation of I loops and t triradii and diminution of IV, H and t'' triradii were observed in bruxism patients. When combined with other clinical features in bruxism, dermatoglyphics can serve to strengthen a diagnostic impression. This can be of great significance in pediatric dentistry since susceptibility to bruxism can be pre determined at an earlier stage¹¹.

Cleft lip and palate

Cleft Lip and palate is the most common congenital malformation affecting head and neck. Both may appear together or separately approx 45% cases are CL+CP with 30% being isolated CP and 25% being CL. Studies have shown that, differences in frequency of dermatoglyphic pattern types and pattern dissimilarity exist between individuals with orofacial clefts and their unaffected relatives and between both groups and controls, with the major effect seen in female subjects.^{12,13} An increase in the ulnar loop pattern on distal phalanges on the ten fingers and an increase in the atd angle in the oral cleft children has been reported which denotes degree of developmental instability of the individual

Periodontal diseases

Periodontitis refers to inflammation of the gingival tissues in association with some loss of both the attachment of the periodontal ligament and bony support. Periodontitis may be rapidly progressive, generalised or juvenile. Dermatoglyphic pattern have been observed in all these variants of periodontitis. It has been observed that patients have a decreased frequencies of twinned and transversal ulnar loops on all fingers of the patients with Juvenile periodontitis, a decreased frequency of double loops on all fingers and an increased frequency of radial loops on the right second digits of the patients with Rapidly progressive periodontitis, and the increased frequencies of concentric whorls and transversal ulnar loops on all fingers of the patients with Aggressive periodontitis. In the light of these findings dermatoglyphics could be used together with the other diagnostic methods such as clinical and radiologic investigations and in the identifying of the patients from distinct groups of Periodontal Diseases¹⁴.

Systemic diseases

Incontinentia pigmenti

Incontinentia Pigmenti is a relatively rare inherited disorder, primarily affecting skin, eyes and central nervous system. Oral manifestations include oligodontia, delayed eruption and hypoplasia of the teeth. Both primary and permanent dentitions are affected. Remarkable dermatoglyphic findings in this disorder are hypothenar loops associated with distally displaced axial triradii on both palms, reduced total finger and summed palmar a-b ridge-counts¹⁵.

Schizophrenia

Schizophrenia is a mental disorder characterized by abnormalities in the perception or expression of reality. Distortions in perception may affect all five senses, including sight, hearing, taste, smell and touch, but most commonly manifest as auditory hallucinations, paranoid or bizarre delusions, or disorganized speech and thinking with significant social or occupational dysfunction. Studies have shown that schizotypal personality disorder group show more minor physical anomalies and dermatoglyphic asymmetries than the normal comparison group and higher cortisol levels than both of the other groups¹⁶.

Respiratory diseases

Patients with respiratory diseases such as Bronchitis, Asthma and Hypoventilation syndrome are quite common in dental settings now a days owing to changing lifestyle and unstable variation in weather conditions. Oral halitosis is sometimes chief complaint in such patients. Dermatoglyphic pattern type frequencies have been shown to be altered in

congenital central hypoventilation syndrome. In particular, there is an increase of arches in females and ulnar loops in males, with the largest differences for the left hand and for individuals with congenital central hypoventilation syndrome¹⁷.

Congenital heart disease

Patients with congenital heart disease are quite commonly treated in dental settings. Management of such patients require certain special parameters. Typical dermatoglyphic pattern have been observed in such patients and analysis revealed that the atd angle was significantly increased in CHD. Except for endocardial fibroelastosis and atrioventricular canal, all subtypes of CHD had a wider atd angle than controls.

In ventricular septal defect, patent ductus arteriosus, tetralogy of Fallot, and multiple cardiac defects, the increase is shown to be statistically significant¹⁸.

Type I diabetes

Dermatoglyphics in insulin – dependent diabetes or diabetes mellitus type 1 (t1dm)

Diabetes Mellitus (DM) can be defined today, in the light of new progress in the fields of etiology, pathogenic, diagnosis and therapy as a heterogeneous etiological syndrome, characterized by a profound and complex turbulence of the energetic metabolism^{1,6}. Studies performed on twins, on families with 2-3 children suffering from diabetes as well as molecular genetics ones have shown that from the genetic point of view, T1DM is a multifactorial disease^{1,4,11,15}. The genes that control the susceptibility, genesis and release of the disease are situated on several chromosomes (the pairs^{2,3,6,7,11,15,16}).

The typical dermatoglyphic features include substantial reduction of the frequency - for loops (L), - with a sensible increase of the frequency for whorls (W) and arches (A). An important increase of frequency for the radial orientation of digital models (A,L,W). These findings of digital dermatoglyphic distortions could help in markers design for the detection of the diabetogen risk in population (screening method), at a very cheap cost. The appearance of these markers, before the clinical manifestation of the disease, makes possible their use in prevention programs of diabetes mellitus insulin dependent¹⁹.

Dermatoglyphic peculiarities in population studies

Blood groups

Dermatoglyphic have been seen to be correlating with blood group patterns. Dermatoglyphic studies have shown that The general distribution of pattern of finger print show high frequency of loops whereas whorls are moderate and arches are least in frequency. Almost same order is noticed in both Rh-positive and Rh negative individuals or A, B, AB and O blood groups. Blood group A show more loops (Rh +ve 54.26%, Rh-ve 60%) while, blood group AB show more whorls (Rh +ve 43.34%, Rh - ve 60%). This suggests an association between finger print pattern and blood group. The distribution of different pattern of fingerprints in individual fingers also show some peculiarities in relation to blood group. The total finger ridge count (TFRC) was observed to be significantly greater in blood group B²⁰.

Mentally retarded

Mentally retarded refers to those individuals who have

an I.Q. score below 69. Such patients do pose a challenge in clinical settings particularly in pediatric dentistry where patients are a difficult task to manage. Mental retardation can be mild (50-69), moderate (35-49) severe (20-34) or profound (below 20). Proper dermatoglyphic interpretation of such subjects at an earlier age can be a clinical parameter of susceptibility at a later stage. Studies have shown that finger print patterns such as whorls, Arch, Loop, Radial Loop, Ulnar Loop and their respective Total Ridge Counts of both hands show intensity and incidence of Patterns and TRC in the mentally retarded subjects compared to the controls. Such studies can be used as the basic data which is useful for future research, biometric analysis and multi disciplinary studies²¹.

Biological significance

Dermatoglyphics have notably variable characteristics that are not duplicated in other people, even in monozygotic twins or even in the same person, from location to location. Thus, dermatoglyphics may be in a position to become the primary means of assessing complex genetic traits, and also useful for the evaluation of children with suspected genetic disorders and diseases with long latency, slow progression, and late onset.

Dermatoglyphic examination should be adopted by more researchers for oral and systemic related studies, leading to more early breakthroughs and new discoveries. This should aid in early diagnosis, treatment and better prevention of many genetically related disorders²².

Conclusion

In the light of above review of literature it is no wonder that in coming times dermatoglyphic examination may be a routine investigation for certain diseases such as dental caries, neurofibromatosis etc. as it is in some parts till date (Stanford sleep center, USA). Dermatoglyphics do have a sure scientific basis for their role as a genetic marker in various diseases. Recording dermatoglyphics has undergone marked changes till date, methods such as Matrix-assisted laser desorption/ionisation²³ have been devised. Image analysis of dermatoglyphics have been used to quantify drugs²⁴. Dermatoglyphics have been correlated with skin patterns and external body features that can be used to construct diagnostic models for the purpose of personality identification as well as in diagnosis of phenotypic appearances^{25,26}. Subclassification of dermatoglyphics into whorls, loops and arches gives a more wider criteria to imply them into diagnostic field. Utilized together with newly developed methods and insights gained in recent studies of other aspects of dermatoglyphics, they should significantly advance the studies of the relationship between dermatoglyphic variation and medical disorders²⁷.

Fig. 1: atd angle, tab angle and various positions of axial triradii

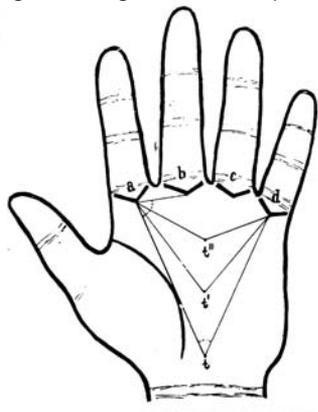


Fig. 2: Various finger tip pattern types



Dermatoglyphic examination is a very cost effective method and a reliable criteria for quantifying disease process, since sufficient evidences are there to prove the same. What is being awaited is a mass education in this sphere. It is expected that this review will be a much awaited step to give momentum to this reliable genetic marker i.e. Dermatoglyphics and its analysis in examination related to disease states.

References

1. Cummins H. Revised methods of interpretation and formulation of palmar dermatoglyphics. *Am J Phy Anthr* 1929; 12: 415-502.
2. L Mathew, AM Hegde, K Rai Dermatoglyphic peculiarities in children with oral clefts. *J Indian Soc Pedod Prev Dent* 2005;23:179-82 .
3. Holt S.B. The hypothenar radial arch-A genetically determined epidermal ridge configuration. *Am J Phy Anthro*. 1975;42:229-32.
4. Uchida J.A, Solton H.C. Evaluation of dermatoglyphics in Medical genetics. *Pediatr clin North Am*;10:409
5. Pons J. Genetics of a-b ridge count on human palm. *Ann Hum Genet*. 1964;37:273.
6. Glanville EV. Heredity and line of palmar dermatoglyphics. *Am J Hum Genet* 1965;17:420-4.
7. Elluru V, Anjana B, Vaishali K, Arvind S. Palmar dermatoglyphics in oral leukoplakia and oral squamous cell carcinoma patients. *JIAOMR*. 2008; 20(3): 94-99.
8. Polat MH, Gululmsir P, Banu K. Dermatoglyphic findings in patients with oral cancers. *Balkan Journal of Stomatology* 2004 2004, vol.; 8, br. 8(2)2, str.: 105-108. 105-108
9. Atasu M. Dermatoglyphic findings in dental caries: A preliminary report. *J Clin Pediatr Dent* winter 1998; 22:147-9.
10. Sharma A. Somani. R. Dermatoglyphic interpretation of dental caries and its correlation to salivary bacteria interactions – An in vivo study. *J Indian Soc Pedod Prev Dent* 2009; 27(1): 17-21.
11. Polat MH, Azak A, Evlioglu G, Malkondu OK, Atasu M. The relation of Bruxism and dermatoglyphics. *J Clin Pediatr Dent* 2000; 24(3): 191-4.
12. Balgir RS, Mitra S. Congenital cleft lip and cleft palate anomalies: A dermatoglyphic study. *J Postgrad Med*. 1986, 32: 18-23.
13. Scott NM, Weinberg SM, Neiswanger K, Brandon CA, Marazita ML Dermatoglyphic pattern types in nonsyndromic cleft lip with or without cleft palate (CL/P) and their relatives in the Philippines. *Cleft Palate-Craniofacial Journal* 2005 ;42:362-366.
14. Atasu M., Kuru B., E. Firatli and H. Meriç Dermatoglyphic findings in periodontal diseases. *Int J of Anthr* 2005; 20(1-2): 63-75.
15. Tanboga I, Kargul B, Ergeneli S, Aydin Ym, Atasu M. Clinical features of incontinentia pigmenti with emphasis on

- dermatoglyphic findings. *J Clin Pediatr Dent* . 2001; 26(2): 161-165.
16. Weinstein DD, Diforio D, Schiffman J, Walker E, Bonsall R, Minor Physical Anomalies, Dermatoglyphic Asymmetries, and Cortisol Levels in Adolescents With Schizotypal Personality Disorder. *Am J Psychiatry* April 1999; 156: 617-623.
 17. Todd, E. S., Scott, N. M., Weese-Mayer, D. E., Weinberg, S. M., Berry-Kravis, E. M., Silvestri, J. M., et al. Characterization of Dermatoglyphics in PHOX2B-Confirmed Congenital Central Hypoventilation Syndrome. *Pediatrics*. 2006; 118: e408-e414
 18. Alter M, Schulenberg R. Dermatoglyphics in congenital heart disease. *Circulation*. 1970 Jan; 41(1): 49-54.
 19. Tarca A, Tuluc E. Dermatoglyphics in insulin – dependent diabetes or diabetes mellitus type 1 (T1DM). *J of Prev Med*. 2005; 13 (1-2): 43-53.
 20. Bharadwaja, A. Saraswat, PK. Aggarwal SK, Banerji P, Bharadwaja S, Pattern of finger-prints in different ABO blood groups 2004. *JIAFM*; 26(1). 6-9
 21. Sajjad N Durrani A.K.S Ruhk L Ghafoor M. Dermatoglyphic analysis of finger prints (pattern and TRC)in Mentally Retarded population of Quetta.2008;23(24):1-8.
 22. Kamboj M Dermatoglyphics. 2008 *Br Dent J* Jan 26; 204(2): 51
 23. Wolstenholme R, Bradshaw R, Clench MR, Francese S. 2009 Study of latent fingermarks by matrix-assisted laser desorption/ionisation mass spectrometry imaging of endogenous lipids. 2009. *Rapid Commun Mass Spectrom*. Oct; 23(19):3031-9.
 24. Goucher E, Kicman A, Smith N, Jickells S. The detection and quantification of lorazepam and its 3-O-glucuronide in fingerprint deposits by LC-MS/MS.2009. *J Sep Sci*. 2009 Jul;32(13):2266-72
 25. Dermatoglyphics in the prognostication of constitutional and physical traits in man. *Sud Med Ekspert*. 2009. Jul-Aug; 52(4): 18-20.
 26. Coughlin MJ, Kaz A. Correlation of Harris mats, physical exam, pictures, and radiographic measurements in adult flatfoot deformity. *Birth Defects Orig Artic Ser*. 1991; 27(2): 193-228.
 27. Schaumann BA, Opitz JM. Clinical aspects of dermatoglyphics. 1991 *Birth Defects Orig Artic Ser*; 27(2): 193-228

Growth pattern of laryngeal cartilages in human fetuses- An analysis

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Abstract

With the advent of super-speciality of foetal therapy and emergence of various invasive and non invasive approaches aimed at correction of foetal defects, the developmental and topographical anatomy along with growth in dimension of the organ and attainment of characteristic morphology gains significance. The greater bulk of available information is based on animal experimentation. Since the data on human fetuses would be more reliable and befitting the clinical needs, this study is a sincere attempt to put forward the inference derived on growth rate and pattern of laryngeal cartilages during the intra uterine life. The study has been done on 31 fetuses their intrauterine life being 4th month onwards. The larynx of these fetuses were dissected out and relevant measurements made to derive the growth rate which has been shown to follow a steady course with a spurt in the 4th to 5th month period of IUL.

Introduction

Laryngeal development starts in the 4th week of embryonic development. Most of the anatomical characteristics of larynx develop by the 3rd month of foetal life. Structure of the larynx can be understood from the diagram given below.

The larynx consists of three paired and three unpaired cartilages. Unpaired cartilages include-thyroid cartilage, cricoids cartilage and epiglottis. Thyroid cartilage is hyaline cartilage and it is the largest of all cartilages. Cricoid is also hyaline cartilage also called signet ring cartilage due to its ring shape. Epiglottis is fibro elastic cartilage. Paired cartilages include one pair each of arytenoids, corniculate and cuneiform.

Level of larynx is at c3 at birth, by 5 years it reaches c6 and up to c7 by 15-20 years. Descent of larynx continues throughout life.

Furthermore the growth of laryngeal cartilages occurs primarily during puberty and in males it is more marked and slightly delayed. Almost all dimensions are greater in males and sexual dimorphism is least distinct before puberty (balboni 1953). According to zrunek et al (1988) dimensions of male larynx are in general 10-30% greater than the female larynx.

The derivation of different laryngeal cartilages has been variously assigned to different branchial arches. Thyroid is derived from 4th arch and also takes some contribution from 5th arch (arey, 1965), hypobranchial eminence (warwick and Williams, 1973) or even from 6th arch (sharp, 1963). No controversy exists about the origin of cricoids and arytenoids cartilages which develop from 6th arch.

In addition to the growth in the dimensions of an organ extensive changes in proportion, brought about by variations of growth rate, also occur to establish the characteristic organ-morphology. This prompted us to make an endeavour to find out the growth pattern of the laryngeal cartilages by taking the various measurements at different periods of intrauterine life.

To study the growth of the cartilages-31 fetuses were obtained individually after delivery, preserved and grouped into 5 groups on the basis of age determined with the help of table given by arey (1965). The age of foetus ascertained by this

standard table is most authentic and reliable one (Harrison 1981, warwick and Williams 1973a). There were 5-7 fetuses in each month of IUL starting from 4th month onwards whose larynx were dissected and measurements made.

Result

Thyroid cartilage - means of the angle between two laminae in group 1 to 5 are 111,111,113,115,113 respectively. The difference between any two groups is not significant statistically. This indicates that the pattern of angulations remains almost constant. Distance between posterior borders of the laminae increased steadily with a maximal increase in 4th to 5th month. After 6th month there is no further increase. Comparing the length of various borders of thyroid cartilage, the values are in decreasing sequence for superior, inferior, anterior and posterior borders respectively. Thickness is maximum at middle of the posterior border. Midline thickness shows further lower values.

Cricoid-in all the groups coronal diameter is greater than sagittal diameter. This is applicable to both internal and external diameter. These two diameters are seen to become nearly equal by 7th month of IUL. There is a significant increase in height of cricoids lamina but not so significant in cricoids arch. The ratio between vertical height of arch and ring is approximately 1:3 the ratio in adults being 1:4 (Warwick and Williams 1973b) the growth in thickness of cricoid lamina is not significant beyond 6th month of IUL. While the growth of the arch is quite irregular. Initially, during 4th month it is rapid which comes to a standstill only to increase again in 7th month.

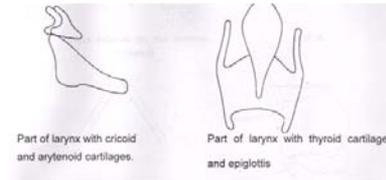
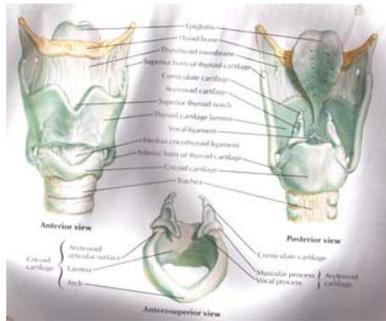
Epiglottis-the growth in height and width is steady and beyond 7 months of IUL it is about 11.05mm.the growth in thickness is maximal between 5th and 7th month.

Arytenoid-from 5th month onwards there is a regular growth in height of the arytenoids till its size becomes 5.92 mm in foetus of more than 7 months. Both anteropost and transverse diameter at base increases rapidly in 4th month which is in concurrence with the foetal growth pattern (Harrison 1981 warwick and Williams 1973a)

Discussion

Considering the growth of thyroid cartilage it is evident that the value of the angle between two laminae i.e.113 is in accordance with the value reported by balcony and pashley

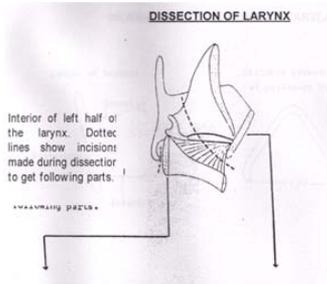




In the epiglottis the height of 11.05 is a significant value from clinical viewpoint as alteration in size of epiglottis is usually associated with a condition called laryngomalacia, a common cause of laryngeal stridor wherein the epiglottis becomes long and redundant.

The study thus shows that each parameter takes independently its own peculiar course of growth to contribute its share to characteristic laryngeal morphology. Majority of the measurements show a spurt in 4th to 5th month of IUL.

The significance of metrical analysis of laryngeal framework, which is responsible for keeping air passage patent, is further accentuated by increasing involvement of endoscopy in neonatology. In the view of the likely possibility of extension of endoscopic manipulation in foetal therapy, it is hoped that the information gained from the present study may establish their relevance in foetal therapy in foreseeable future.



(1986). And it remains fairly constant throughout. Since the distance between the posterior borders is increasing steadily without increase in the angle, the aforementioned increase is likely due to horizontal growth which is in conformity with the general growth pattern being especially rapid during 4th month (Harrison 1981, Warwick and Williams 1973) following which the growth is mostly vertical rather than horizontal. The ratio of growth of superior and inferior border is 1:1 and remains so postnatally as reported by Zank (1998). Strikingly, the growth of superior border is continuous and relatively rapid in comparison to other borders which grow at a slower rate with periods of arrest in between. This variance in growth rate is seemingly important in acquisition of the particular shape of the cartilage in the newborn. The superior cornu in all groups is longer and narrower as compared to the inferior one which is relatively shorter and broader. This proportion corresponds to that seen in adult larynx (Warwick and Williams 1973 b).

Cricoid being the narrowest dimension in air passage has invited lot of scientific attention, in view of ever growing importance of involvement of endoscopic evaluation in neonatology. The measurements done show that the cricoids is an elliptical cavity till 5th month of IUL which becomes circular by 7th month which is matching with one described by Spector (1985). In our study the minimum internal diameter of cricoids in fetuses beyond 7th month of IUL is 4.4mm which is in concordance with the lowest value in infants mentioned by Tucker (1932) and Maran (1988). The pattern of growth in height of lamina and arch is influential in establishing the shape of cricoids.

Acknowledgement

The help and facilities provided by my colleagues in Sangita Nursing Home and Apoorv Medical Centre are thankfully acknowledged.

References

1. BAKER DC Jr & Savetsky L (1996): congenital partial atresia of larynx, *laryngoscopy* 76:616
2. Balboni G (1953): first data on proportion of human larynx, *monit zool ital (Firenze)* 62
3. Bucher O (1942) *FUNCTIONAL MORPHOLOGY OF FRAMEWORK OF HUMAN LARYNX*
4. GRACIA M (1855) *OBSERVATIONS OF HUMAN VOICE, PHILOSOPHICAL MAGAZINE and journal of science*
5. Harrison HS, Fuqua WB and Giffin RB jr (1965): congenital laryngeal cleft.
6. Harrison RG (1981): introduction to human embryology
7. Lissner H (1911): studies on the development of human larynx, *am J Anat* 12:27
8. Simonetta B (1929) thyroid cartilage in man and sheep, *valsalva* 5:181
9. Smith H & Bain AD (1965) congenital atresia of larynx, *ann otol* 74: 338
10. Tucker L (1958) on the laryngeal mirror and its mode of employment, *zietschrip.d.ges.aerzte ZU Wien* 17:271
11. Wilmes H (1930) larynx in horse and cow, *anat anz* 69:122
12. WB Saunders Co, London p:2431

Successful treatment of body packers with many packages

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Abstract

During the last decade, increase rate of the drug traffic and those customs, have led the smugglers to attempt various methods. One of these methods of illicit drug smuggling is body packing. Body packers are people who illegally carry drugs, mostly cocaine as well as opium and/or heroin, concealed within their bodies. The packets are inserted in the mouth, rectum, or vagina in order to get across borders without being detected.

The smuggling of drugs by internal concealment of these illicit goods is a growing problem for custom services and law enforcement officials, not to mention risks for the carrier from a medical point of view. Individuals engaged in such activities called body-packer.

Here we present 11 cases of body-packers who were part of a smuggling gang, arrested by police and admitted to our hospital in a period of 24 hours. These 11 men were part of 90 body-packers who arrested in Tehran. Radiological examination including plain abdominal radiographs and CT scan images demonstrated the presence of multiple enteric packages.

Non-surgical conservative evacuation of bowel contents was induced by the administration of laxatives. Analysis of the packages, revealed crack and cocaine as concealed drugs.

Key words

Drug smuggling, body packing, body packer, crack, cocaine

Introduction

The smuggling of drugs by internal concealment is called body-packing^{1,2}. Body-packers may also be called "swallowers" "internal carriers" "courier" or mules³.

Another term, "body stuffer" occasionally and inappropriately used synonymously with body packing refers to the swallowing of relatively small amounts of loosely wrapped drug because of the fear of arrest⁴.

One of the major businesses with high benefit is concealment and transit of the narcotic agents (e.g. opium, heroin) in all over the world^{5,6}.

Body packing and body stuffing are the two major methods for smuggling of the narcotic⁷.

Body packing and Body stuffing are the terms used for the intra corporeal concealment of illicit drugs mainly opium, heroin, cocaine and marijuana^{7,8,9}.

To transport, these drugs are wrapped in the forms of capsules, condoms, balloons, plastic bags or finger of latex gloves located in various anatomical cavities or body orifices^{7,10}.

Identification of suspected persons is difficult for the customs agents at the national borders or airports^{10,11,12}.

The body packers are especially prone to rupture and then toxicity occurs. In addition the gastrointestinal obstruction may occur and also many reports in the literatures have shown the potentially fatal consequences of rupture or leaking of these packets among the gastrointestinal tract.

Suspicious person observation and signs such as person's

behavior, origin and destination of their traveling, doubtful declaration about their journey, halitosis and seating without mobility and the specific smell of their mouth due to acid effect of stomach on their packet's cover may notify police for body smuggling cases.

Thus when they refer to the hospital and undergo physical examination, plain abdomen films, CT scan, contrast study of the stomach and bowel and urinalysis (e.g. EMIT), the physician can be led to a comfortable diagnosis of the body smuggling of the suspicious person^{10,12}.

The patient who represents the toxicity or mechanical GI obstruction should undergo immediate surgical removal of the packets¹³. The asymptomatic body packer remains controversial^{13,14}.

U.S. Federal agencies report that body packing has increased recently, possibly because the increased border security since the events of September 11, 2001, has made conventional smuggling more difficult. Alternatively, more body packers may be detected simply as a result of increased airport security. New York's Kennedy International Airport reported 193 body-packing arrests from October 2001 to April 2002, as compared with 202 during the entire preceding year¹⁵. The majority of heroin reaching the United States is from Southeast Asia or South America.

Iran is considered by smuggling networks to provide the best transit for opium and/or heroin in its geographical region. The drug is trafficked to Iran mainly from Afghanistan, predominantly by land or air routes. The quantities of opium and/or heroin seized in Iran have steadily increased in recent years (UNDCP 2002), and the rates of opium and/or heroin poisoning are climbing comparing the past¹⁶. Although early body packers were predominantly young men¹⁷, the practice now crosses demographic groups.

The first reports of people smuggling drugs via body packing were published during the 1970s¹⁸. In 1973, two physicians from Toronto admitted a patient in whom small bowel obstruction developed 13 days after he had swallowed a condom filled with hashish¹⁹. The first nonfatal reported case of intoxication related to the ingestion of balloons filled with cocaine occurred in Florida in 1975²⁰. The first fatality from ingestion of packages of drugs to avoid detection from Customs officers was reported in 1977²¹.

Body packers usually carry about 1 kg of drug, divided into 50 to 100 packets of 8 to 10g each, although persons carrying more than 200 packets have been described^{22,23}. Each packet of cocaine, heroin, or amphetamine contains a life-threatening dose of drug²⁴. Many cases of accidental poisoning and also death in connection with body packing are described in detail in the toxicology and forensic literatures and were generally due to sudden rupture of an ingested drug package^{25,26,27}.

Our present recommendations for asymptomatic body packers include activated charcoal and whole bowel irrigation (WBI) with polyethylene glycol (PEG)²⁸. WBI is continued until rectal effluent is clear and no drug-filled bags are detected on a contrast study of the bowel.

We present 11 cases of recent smuggling of illicit drugs by packing the material into layers of plastic, forming capsules



which were then ingested. These 11 men were part of a group of 90 body-packers who were working together as a smuggling gang²⁹. Our research in literature did not reveal any previous report of body-packing on this scale to be arrested and admitted at the same time.

Case report

Tehran metropolitan area police arrested 90 suspected body-packers in several safe houses and airport in a cooperative movement between Iranian, Turkey, and Pakistani police department. These body-packers had African nationality and used Iran as a hub for smuggling drugs from Pakistan to EU countries, Far East and also Middle East countries²⁹.

Eleven of these men (age range, between 25-38 years) admitted to Loghman-Hakim Hospital after preliminary radiological examination by plain X-ray, for management of their condition. Nine men demonstrated multiple enteric packages in their X-ray. Confirmatory CT scan was performed for most of the patients.

These men remained under observation in the hospital. Following administration of sorbitol for 11 patient and polyethylene glycol for 4 patient, packages start to pass. Patients remained on solid diet restriction because of need for probable surgical intervention (admission range, between 1-10 days).

Examination of package substance revealed small amounts (12-20 g) of powder (crack and cocaine) enclosed within mechanically sealed rubber balloons, highly resistant to rupture and leakage.

In the period of hospitalization maximum amount of packages which recovered from a given patient was 157. Above images show passed packages in single defecation. No surgical intervention needed and all the cases managed conservatively.

Discussion

Body-packing is a method used to smuggle moderate amounts of high-profit illicit drugs such as cocaine and heroin. In recent years increased border security and use of new technologies has made conventional smuggling more difficult in many parts of the world. As a result methods like body-packing have been developed³.

A man who had developed small bowel obstruction following swallowing a condom filled with hashish was the first case of body-packing to be reported, dated to 1973³⁰.

Body-packers usually carry maximum 100 packages³, although persons carrying more than 200 packages have been described with less weight^{31,32}. Occasionally body-packers smuggle more than one type of drugs at the same time³³.

To postpone natural evacuation, especially for long distances, the body-packers take high dosages of anticholinergic drugs and even refuse to eat or drink during the journey³⁴.

Asymptomatic and even most of the symptomatic body-packers require radiological evaluation for diagnosis and sometimes confirmation of diagnosis. Plain radiography and

ultrasonography^{35,36}, have been used as rapid screening tools. CT scan^{37,38}, or barium enhanced radiography could provide a more definitive answer when a strong suspicion of body-packing exists³.

The most important medical complications of body-packing include partial or complete gastrointestinal tract obstruction and drug intoxication following leakage or rupture of the covering materials, however most cases do not experience complication and packages may pass spontaneously or pass with the use of laxatives but rarely surgical removal indicated. The main indications for surgery remain cocaine toxicity and bowel obstruction^{39,40}.

A comprehensive treatment algorithm has been created for management of body-packers by other researcher³.

Following reasons may account the large number of body smugglers in Iran/Tehran:

1. -Special geopolitical location of Iran.
2. -Increasing the production of the narcotic substance in neighbor countries of Iran according to the UNDCP reports.
3. -Severe control of fighting narcotic agent staff in Iran which causes the smuggler transports successfully the narcotic substance through internal transporting (Body smuggling).
4. -Less of knowledge and prevalence of the idleness in Third world countries including Iran.
5. -Afraid of referring to the hospital when symptoms of intoxication occur because the smugglers have a hard punishment.
6. -Method of packaging in comparison with the mechanical packaging of Mc carrion and Wood is very simple and not to be calculated.

There are no exact reports in actual numbers of body packers, because all of these persons haven't been taken captive and only in some of them symptoms of body packer syndrome is being revealed.

Autopsy of body packers may give the comprehensive data about packaging methods, exact number of packets, type of the transported illicit drug and location of the packets in the body.

Hospital physicians may neglect this type of gastrointestinal foreign body if they aren't aware of the body packer syndrome whereas immediate help to every poisoned patient is necessary at the first minutes of intoxication.

Conclusion

The detection of drugs concealed internally for smuggling is a problem that its diagnosis requires vigilance and a high level of suspicion, in the domain of management, in the majority of patients, conservative management will result in passing of ingested packages without further need for surgical intervention. However Medical therapy is effective and acceptable when patient's status is monitored carefully in the intensive care unit. However, the immediate surgical intervention should be kept in mind as an alternative. The physician should confirm the passage of all packets from the GI tract using imaging study before discharge of the patients

from hospital. Usually, passing of two or three packet-free stools during continuous WBI therapy for 12h, with a negative abdominal radiograph, is a reasonable end-point. Also, after surgical removal, the physician will need to confirm that the GI tract is free of drug-filled packets.

References

1. Roberts J.R, Price D, Goldfrank L and Hartnett L. The bodystuffer syndrome: a clandestine form of drug overdose. *Am J. Emerg. Med* 1986; 4: 24–27.
2. Wetli C.V, Mittleman R.E. The “Body Packer Syndrome”-toxicity following ingestion of illicit drugs packages for transport. *JFSCA* 1981; 26: 492–500.
3. Traub S.J, Hoffman R.S, Nelson L.S. Body packing—the internal concealment of illicit drugs. *NEJM* 2003; 349: 2519–2526.
4. June R, Aks SE, Keys N, Wahl M. Medical outcome of cocaine bodystuffers. *J Emerg Med* 2000; 18:221-224.
5. B.G. Brogdon, MD, *Forensic Radiology*. First Ed. CRC Press (Boca Raton Boston London New York Washington, D.C). 1998 PP: 251-255
6. Haugen - OA; Dalaker - M; Svind Land - A. Smuggling of narcotics in body Cavities. *Tidsskr- Nor- Laeg foren*. 1994 Sep 10; 114 (21): 2501-2
7. HADDAD AM, clinical management of poisoning and drug overdose, 3 rd ED. PHILADELPHIA, SAUNDERS 1998 PP: 506-516-528-538.
8. Ichikawa - K; Tajima - N; Tajima - H; Murakami - R; Okada - S; Hosaka - J; Ito - K et al Diagnostic imaging of body packers. *nippon - Tagaku - Hoshasen - Gakkai - Zasshi - 1997 Feb*; 57(3) : 89-93
9. Maibrain - ML; Neels - H; Vissers - K; Demedts - P; Verbracken - H; Dealemans - R; Wauters - A. A massive, near - Fatal cocaine intoxication in a body stuffer, A case report and review of literature. *Acta - Clin - Belg*. 1994; 49 (1): 12-8
10. Heineman - A; Miyashi - S; Iwersen - S; Schmoldt - A; Puschel - K. Body packing as a cause of unexpected sudden death. *Forensic science- International*. 1998 Mar. 92(1) 1-10
11. Stichenwirth - M; Stelway - Carion - C; Klupp - N; Honigschabls; Vycudilit - W; Bauer - G; Risser - D. Suicide of a body packer. *Forensic - Sci - int*. 2000 Jan 24; 108 (1): 61-6
12. Aldrighetti - L; Paganelli - m; Giacomelli - M; Villa - G; Ferla - G. Conservative management of cocaine - packet ingestion: experience in Milan. The main Italian smuggling center of south American cocaine. *Panminerva - Med*. 1996 Jun; 38 (2): 111 - 6
13. Miller - JS; Hendren - SK, Liscum - KR. Giant gastric ulcer in a body packer. *S. Trauma*. 1998 Sep; 45(3): 617-9
14. Aldrighetti - L; Graci - C; Paganelli - M; Vercesi - M; C atena - M; Ferla - G. Intestinal occlusion in cocaine packet ingestion *Minerva - Chir*. 1993 oct 31; 48 (20); 1233-7
15. Claffey, M. 2002. Stampede of drug mules at Kennedy: after 9/11 lulls, heroin & ecstasy busts soar. *New York Daily News* May 5:5.
16. Abdollahi, M., Jalali, N., Sabzevari, O., Hoseini, R., and Ghanea, T. 1997. A retrospective study of poisoning in Tehran. *J. Toxicol. Clin. Toxicol*. 35:387–393.
17. Gill, J. R., and Graham, S. M. 2002. Ten years of “body packers” in New York City: 50 deaths. *J. Forensic Sci*. 47: 843–846.
18. Pidoto, R. R., Agliata, A.M., Bertoline, R., Nainini, A., Rossi, G., and Giani, G. 2002. A new method of packaging cocaine for international traffic and implications for the management of cocaine body packers. *J. Emerg. Med*. 23: 149–153.
19. Deitel, M., and Syed, A. K. 1973. Intestinal obstruction by an unusual foreign body. *Can. Med. Assoc. J*. 109:211–212.
20. Mebane, C., and DeVito, J. J. P. 1975. Cocaine intoxication—a unique case. *J. Florida Med. Assoc*. 62:19–20.
21. Suarez, C. A., Arango, A., and Lester, L. 1977. Cocaine-condom ingestion: Surgical treatment. *JAMA*. 238:1391–1392.
22. Gherardi, R. K., Baud, F. J., Leporc, P., Marc, B., Dupeyron, J. P., and Diamant-Berger, O. 1988. Detection of drugs in the urine of body packers. *Lancet* 1:1076–1078.
23. Bulstrode, N., Banks, F., and Shrotria, S. 2002. The outcome of drug smuggling by ‘body-packers’ -the British experience. *Ann. R. Coll. Surg. Engl*. 84: 35–38.
24. Traub, S. J., Hoffman, R. S., and Nelson, L. S. 2003. Body packing—the internal concealment of illicit drugs. *N. Engl. J. Med*. 26: 2519–2526.
25. Patel, F. 1996. A high fatal postmortem blood concentration of cocaine in a drug courier. *Forensic Sci. Int*. 79: 167–174.
26. Wetli, C. V., Rao, A., and Rao, V. J. 1997. Fatal heroin body packing. *Am. J. Forensic Med. Pathol*. 18:312–318.
27. Gill, J. R., and Graham, S. M. 2002. Ten years of “body packers” in New York City: 50 deaths. *J. Forensic Sci*. 47:843–846.
28. Hoffman, R. S., Smilkstein, M. J., and Goldfrank, L. R. 1990. Whole bowel irrigation and the cocaine body packer: a new approach to a common problem. *Am. J. Emerg. Med*. 8: 523–527.
29. <http://www1.irna.ir/en/news/view/line16/0708180351162035.htm> accessed 2007-08-21
30. Hadjibabaie M, Rastkari N, Rezaie A et al. The Adverse Drug Reaction in the Gastrointestinal Tract: An Overview. *International Journal of Pharmacology* 2005; 1: 1-8.
31. Gherardi RK, Baud FJ, Leporc P et al. Detection of drugs in the urine of body-packers. *Lancet* 1988; 1: 1076-1078.
32. Shadnia S, Faiaz-Noori M.R, Pajoumand A, Talaie H, Khoshkar A, Vosough-Ghanbari S et al. A case report of opium body packer; review of the treatment protocols and mechanisms of poisoning. *Toxicology Mechanisms and Methods* 2007; 17: 205-214.
33. Gill JR, Graham SM. Ten years of “body-packers” in New York City: 50 deaths. *J Forensic Sci* 2002; 47:843-846.
34. McCarron MM, Wood JD. The cocaine ‘body-packer’ syndrome: diagnosis and treatment. *JAMA* 1983; 250: 1417-1420. 43. Hierholzer J, Cordes M, Tantow H, Keske U, Mäurer J, Felix R. Drug smuggling by ingested cocaine-filled packages: conventional x-ray and ultrasound. *Abdom Imaging* 1995; 20: 333-338.
35. Alzen G, Banning S, Günther R. Sonographic detectability of narcotic drug containers in the gastrointestinal tract. Experimental research in the dog. *Rofo* 1987;146:544-7 .
36. Taheri MS, Hassanian-Moghaddam H, Birang S, Hemadi H, Shahnazi M, Jalali AH et al. Swallowed opium packets: CT diagnosis. *Abdom Imaging* 2007; 4; [Epub ahead of print].
37. Hartoko TJ, Demey HE, De Schepper AM, Beaucourt LE, Bossaert LL. The body packer syndrome — cocaine smuggling in the gastro-intestinal tract. *Klin Wochenschr* 1988; 66: 1116-1120.
38. Kersschot EA, Beaucourt LE, Degryse HR, De Schepper AM. Roentgenographical detection of cocaine smuggling in the alimentary tract. *ROFO* 1985;142:295-298.
39. Lancashire MJ, Legg PK, Lowe M, Davidson SM, Ellis BW. Surgical aspects of international drug smuggling. *BMJ* 1988; 296:1035-7
40. Silverberg D, Menes T, Kim U. Surgery for “body packers”—a 15-year experience. *World J Surg* 2006 ;30:541-6.

Section 377 Indian penal code and its present scenario

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Abstract

"Gays present in the court room hailed the judgement and greeted one another with hugs"- Times of India.

Netherland was the first country to legalize homosexuality. Since then, many other countries legalized it. Today, homosexuality is recognized across the globe.

The following article aims to put forward Sec. 377 I.P.C, its meaning, pros and cons of decriminalizing Sec. 377 I.P.C and critical overview of the judgement of WR(C) No. 7455 of 2001.

Key Words

Sec.377 I.P.C, Decriminalization.

Pronouncing the order in Naz Foundation (India) Trust v. Government of NCT, Delhi and Others, Writ petition (Civil) No. 7455 of 2001, a division bench of Chief Justice A.P. Shah and Justice S. Muralidhar of Delhi High Court in its order on July 02, 2009, said that Section 377 of the IPC, insofar as it criminalizes consensual sexual acts of adults in private, is violative of Articles 21 [Right to Protection of Life and Personal Liberty], 14 [Right to Equality before Law] and 15 [Prohibition of Discrimination on Grounds of Religion, Race, Caste, Sex or Place of Birth] of the Constitution.

However, the court clarified that "the provisions of Section 377 will continue to govern non-consensual penile non-vaginal sex and penile non-vaginal sex involving minors." The judges also said that by adult they meant "everyone who is 18 years of age and above." According to them "A person below 18 would be presumed not to be able to consent to a sexual act," The Bench further said that "this clarification will hold till, of course, Parliament chooses to amend the law to effectuate the recommendation of the Law Commission of India in its 172nd Report which, which would remove a great deal of confusion." The judgment also made it clear that it would not result in re-opening of criminal cases involving Section 377 that had already attained finality¹.

Legislation Chapter XVI, Section 377 of the Indian Penal Code was introduced during British Rule of India. The Indian Penal Code (IPC), was drafted in 1860 by Lord Macaulay as a part of the colonial project of regulatory and controlling the British and Indian Origin subjects. It reads:

"Unnatural offences: Whoever voluntarily has carnal intercourse against the order of nature with any man, woman, or animal shall be punished with imprisonment for life, or with imprisonment of either description for a term which may extend 10 years, and also be liable to fine."

Explanation: Penetration is sufficient to constitute the carnal intercourse necessary to the offense described in this section.

"Whoever": The word "Whoever" used in the section signifies a singular in character. The person who commits the offence.

"Voluntarily": The word "Voluntarily" as used in the section is one of the essential ingredients signifying the mens-rea of Sec.377. The word "voluntarily" is defined under Sec. 39 of IPC which states that "a person is said to cause an effect

voluntarily when he causes it by means whereby he intended to cause it, or by means which, at the time of employing those means, he knew or had reason to believe to be likely to cause it." The voluntariness comprises of consciousness, belief, and desire.

"Carnal intercourse": According to Advanced Law Lexicon, 'Carnal' means pertaining to flesh; sensual. The word "carnal intercourse" is defined as indulging in sexual act against the order of nature with any man, woman, or animal.

Legal battle

In 2001, a petition filed by AIDS Bhed Bhao Virodhi Andolan (ABVA) was dismissed.

In 2003 Naz Foundation (an NGO related to HIV/Aids issues) filed a petition in the Delhi High Court asking for Section 377 to be read down by decriminalizing consensual sex among adults.

In September 2003, the Government insisted on retaining Section 377 on the grounds that Indian initiation society's disapproval of homosexuality was strong enough to justify it being treated as a criminal offence even where adults indulge in it in private.

In February 2006, the Supreme Court ordered the High Court to reconsider the constitutional validity of Section 377.

In November 7, 2008, when the verdict was reserved, the previous UPA government had opposed scrapping of section 377 of the Indian Penal Code. There were contradictions within the Government as the Home Ministry had opposed scrapping of section 377 while Ministry of Health came out openly in support of the gay rights activists. The Centre had said that homosexuals comprise only 0.3 per cent of the population and the right of rest 99.7 per cent of the population to lead a decent and moral life in society would be violated if such behaviour (gay sex) is legalized.

The question is whether to repeal the law or to scrap it?

It provides necessary alarm in the minds of individuals that intercourse through voluntary using their dominant relationship like teacher-student, senior – junior, master – servant, guru – disciple, etc. would be punished under the penal laws.

India being a secular state, arguments based on religion, which considers homosexual relations ship, as sacrilegious cannot be claimed for.

It was the lone voice of the then Health Minister who supported decriminalizing of homosexual offence, on the basis that it impedes awareness programme and effective treatment.

Medically speaking, according to American Psychiatric Association homosexuality is perfectly normal behavior i.e. due to abnormal androgen – estrogen ratio.

A group of 47 eunuchs from Lucknow were studied in details by K.B. Kunwar et.al. Most of them had a compulsive urge to homosexuality, but there were a few who were latent homosexuals and some indulged occasionally with an exploratory sense or when they were deprived of contact with members of the opposite sex³.

According to Kinsey, 4 percent of Americans are exclusively homosexuals while Desmond Curran and Denis Parr found the figure to be 5 percent in a series of private patients⁴.

In 1993, a group of medical researchers at the National Cancer Institute (NCI) led by Dr. Dean H. Hamer released a study of 40 pairs of brothers that linked homosexuality to the X chromosome. The research, published in *Science*, reported that 33 of the pairs of brothers had DNA markers in the chromosome region known as Xq28. The study won an enormous amount of media attention, and Hamer's own activities as a homosexual activist within NCI were ignored when Hamer offered interviews only when reporters agreed not to identify him as a homosexual⁵.

In a later interview, Hamer said, "Homosexuality is not purely genetic. Environmental factors play a role. There is not a single master gene that makes people gay. I don't think we will ever be able to predict who will be gay⁶."

Elliot Gershon, chief of the clinical neurogenetics branch of the National Institute of Mental Health, said, in relation to genetic "There's almost no finding that would be convincing by itself in this field. We really have to see an independent replication⁷."

A recent study of sexual practices in rural India by the United Nations Population Fund (UNFPA) found that 'male-to-male sex is not uncommon. In fact a higher percentage of men in the study reported having male-to-male sex than sex with sex workers. This was true of both married as well as unmarried men. Close to 10 per cent unmarried men and 3 per cent married men reported having had sexual intercourse with other men in the past 12 months.' The survey covered 50 villages in five districts of five states with feedback on sexual practices from close to 3,000 respondents and in-depth interviews on intimate habits from 250 people⁸.

According to S.13. of the Hindu marriage Act, 1955, a wife can apply for annulment of marriage if the husband has been guilty of rape, sodomy or bestiality.

The 172nd report of Law Commission of India recommends that rape laws be changed to [a] make it gender neutral; [b] make special provisions for child sexual abuse; and [c] repeal section 377 of the I.P.C.

In 1992, the World Health Organization removed homosexuality from its list of mental illnesses in the International Classification of Diseases (ICD 10). Guidelines of the ICD 10 reads: "disorders of sexual preference are clearly differentiated from disorders of gender identity and homosexuality in itself is no longer included as a category."

Critical review

❖ Age of consent:

- **According to Hindu Marriage Act, 1955, (Section 5 (iii))** States one of the conditions for a Hindu Marriage: -A Marriage may be solemnized between any two Hindus, if the following conditions are fulfilled, namely: -The bridegroom has completed the age of (twenty-one years) and the bride, the age of (Eighteen years) at the time of the marriage.
- **Indian Majority Act of 1875** declares that a person shall be deemed to be a major when he has completed the age of eighteen years, subject to the condition that the court has appointed no guardian. But, if the Court has appointed a guardian or a ward, the Act provides that the age of majority shall extend to twenty-one years.
- **Section 376 of the I.P.C. Staaes:** 1. Whoever, except in the cases provided for by Sub-Section (2) comments, rape shall be punished with imprisonment of either description for a term which shall not be less than seven years but which may be for life or for a term which may extend to ten

years and shall also be liable to fine unless the woman raped is his own wife and is not under twelve years of age, in which case, he shall be punished with imprisonment of either description for a term which may extend to two years or with fine or with both.

- **Section 361 Kidnapping from Lawful guardianship** Whoever takes or entices any minor under (sixteen) years of age if a male, or under (eighteen) years of age if a female or my person of unsound mind, without the consent of such guardian, is said to kidnap such minor or person from lawful guardianship.
- **The Prohibition of Child Marriage Act, 2006** states that "child" means a person who, if a male, has not completed twenty-one years of age, and if a female, has not completed eighteen years of age.

Section 377 IPC does not fix any age limit, the act is considered to be an offence irrespective of age and consent. Stating the age "everyone who is 18 years of age and above" also conflicts with the juvenile justice act.

❖ The court clarified that "the provisions of Section 377 will continue to govern non-consensual penile non-vaginal sex and penile non-vaginal sex involving minors."

Consensual (adj.): means existing or entered into by mutual consent without formalization by document or ceremony.

- "Whoever voluntarily" if we look, is the person who committed the offence and had foresight of the consequences. Voluntarily does not include consensus. If we put forward consensus in place of voluntarily then Sec. 312 I.P.C (defines miscarriage) and Sec. 322 I.P.C (voluntarily to cause grievous injury) will have a different perspective all together.

Conclusion

To conclude with the Delhi High court has given the rein to the Parliament, for the bill to be passed.

Only to state that decriminalization would provide equality, freedom from mental and physical pressures and pave way to legislation of marriage with same sex, adoption, etc.

On the other hand it violates equality, deteriorates the moral fabric of the society, would create vacuum in the laws involving juveniles.

What ever the outcome be it has put a challenge on existing societal norms and demands reforms in criminal and civil laws.

To put it all together, we personally feel that the section should be repealed and not to decriminalize as the Naz Foundation has put forth with.

References

1. Writ petition (Civil) No. 7455 of 2001; Naz Foundation (India) Trust v. Government of NCT, Delhi and Others.
2. B.V. Subrahmanyam, Modi's Medical Jurisprudence & Toxicology, 22nd Ed.
3. Kunwar K.B, Current Medical Practice, Jan 1966, 19- 27.
4. Kinsey, BMJ, 14 Sept 1957, 639.
5. Dean Hamer, The Science of Desire (New York, New York: Simon & Schuster, 1994), p. 82.
6. From speech in Salt Lake City in Lili Wright, "Science of Desire Is Topic for 'Gay Gene' Finder," Salt Lake Tribune, 28 April 1995.
7. Gershon Elliot, "Evidence for Homosexuality Gene," Science, Vol. 261, 16 July 1993: 291.
8. <http://www.indianexpress.com/oldStory/31566/>

Injuries in riders and pillion riders of fatal two wheeler road traffic accidents in city of Guwahati

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Abstract

Background

In modern times, increase in number of road traffic accidents has become an epidemiological concern. Accidents involving two wheelers are also becoming common.

Materials and methods

All two-wheeler accidents involving riders and pillion riders were taken up for the study period of one year from 1st September 2004 to 31st August 2005. A pretested proforma was used to determine the nature of the incident and type of vehicle involved. All 76 cases were autopsied and findings recorded and analyzed.

Results

Incidence of fatal two wheeler accidents was 16.59%. Most fatal riders were between 21– 30 years (35.52%) while pillion riders were between 31– 40 years. M:F ratio was 4. 43:1. Maximum cases (31.59%) were seen in spring season. Most accidents occurred between 6PM – 12 Mid Night. Accidents in the urban areas accounted for 42(55.26%) cases. Skull fracture was present in 22(64%) pillion riders and in only 16(38%) riders. Rib fractures were seen in 33.3% of riders and 17.64% of pillion riders. Laceration to the brain was seen in 16(38%) riders and 18(52.94%) pillion riders. Laceration to the liver and spleen constituted 19% each in riders while it was 14.7% and 17.6% respectively in pillion riders. Laceration of the heart was seen in 3 riders (7.14%) and 2 pillion riders (4.76%).

Conclusion

The study highlights the importance of enforcing speed limits on roads, enforcing laws requiring riders and pillion riders of two wheelers to wear helmets, upgrade road infrastructure and lastly to strengthen the health facilities for the victims.

Key words

Road traffic accidents; two wheeler; pillion rider; rider.

Introduction

With modern lives becoming increasingly busy, automobiles have become an invariable means of transport. At the same time, there is an increase in number of road traffic accidents and has become an epidemiological concern. The hike of fuel prices have forced people to increasingly use two-wheelers as a mode of travel and subsequently accidents involving two wheelers are also becoming common.

WHO (1) reported in 2001, that majority of fatal road traffic accident victims were pedestrians, motorized two-wheeler riders and bicyclists. On the view of the growing trend, the following study was conducted on fatal injury patterns of two-wheeler riders and pillion riders.

Materials and method

The study was conducted in the Department of Forensic Medicine, Gauhati Medical College and Hospital from 1st September 2004 to 31st August 2005. 76 cases of two-wheeler accidents involving riders and pillion riders were taken up for the study. Autopsies, where the nature of the incident could not be ascertained were not included. Pedestrians involved in two wheeler accidents were not included. All decomposed bodies were excluded from the study.

A detailed history was taken. A pretested proforma was used to extract information regarding the nature of the incident and type of vehicle involved in the accident. All 76 cases were autopsied and findings recorded and analyzed.

Result

Out of the total 1795 autopsies conducted during the study period, road traffic accidents constituted 458(25.52%). Of these, 76 cases (16.59%) involved two-wheeler riders and pillion riders. Fatal riders constituted 55.26% cases while rest 44.73% victims were pillion riders. Male victims (81.51%) outnumbered females (18.42%) in both groups. Majority of victims were from age group 21– 30 years (35.62%) as shown in Table No. I.

Incidence of two wheeler road traffic accidents were higher in spring (31.59%) followed by winter (21.05%). Majority of accidents (30.26%) occurred in between 6 PM – 12 midnight followed by (25%) cases during 6AM -12 midday. Accidents in the urban areas accounted for 42% (55.26%) cases while 18 (23.68%) cases occurred in rural areas.

Most common external injury was abrasion followed by contusion and laceration. In both riders and pillion riders, multiple abrasions were commonly seen; (45.23%) in the upper limbs and (42.85%) in lower limbs among riders while 58.82% in upper limbs and 44.11% in lower limbs among pillion riders. Twenty six cases (61.9%) showed contusion to the head among riders and 58.82% among pillion riders. Chest showed maximum contusions. Lacerations commonly involved the face and head in both the groups as shown in Table No. II.

Both riders and pillion riders showed maximum number of fractures on the head and chest. However, 22(64%) pillion riders suffered from fractures in the head compared to 16(38%) riders as shown in Table No. III. Rib fractures were seen in 33.3% of riders and 17.64% of pillion riders. Fracture of the long bones of the extremities was more commonly seen in riders; 4.76% in the upper limbs and 14.28% in lower limbs, compared to 4.76% in the upper extremities and 5.8% in lower extremities of pillion riders

Among internal injuries, laceration was more common than contusion in both riders and pillion riders. Brain was found lacerated in thirty four cases and pleura in fifteen cases in both groups combined. Contusion of the brain and the lungs was more common in the riders as shown in Table No. IV.

Discussion

Incidence of fatal two wheeler accidents among deaths resulting from vehicular accidents was 16.59% (76 cases). This

was similar to the study done by Ghose P.K.(2). Devi T.M. et al³ found an incidence of 22.76% which more or less can be comparable to present study.

Most fatal riders were of the age group 21–30 years (35.52%) similar to the findings of Devi T.M. et al³. and Ghose P.K.⁴. As this age group leads to an active lifestyle with more mobility and they frequently expose themselves to traffic hazards. Majority of pillion riders were in the age group 31–40 years followed closely by 21–30 years, which is in contrast to the study done by Devi et al³, who found 21-30 years age group preponderance. Frequently as age advances, people prefer to pillion ride than to drive themselves and so this may be the cause for majority of fatal pillion riders to belong to the 3 rd decade compared to riders.

We found preponderance of males over females seen with a ratio of M:F- 4.43:1. This was similar to studies done by Salgado M.S.L⁵ and Adeyemo A.O et al⁶. Males frequently engage themselves in outdoor activities and are thus more vulnerable to traffic hazards. However, Devi et al³. who found a M:F ratio of 9.2:1.

Highest numbers of fatalities were seen in spring accounting 31.59% cases. This was in contrast to study by Kumar A et al⁷ who found in rainy seasons. Dusty and windy weather leading to poor visibility, celebrations of a number of cultural festivals during this season leading to increased alcohol consumption, overcrowding on the streets and rash driving may be the cause of leading fatalities in spring.

Table I: showing the age distribution of riders and pillion riders involved:

Group	Age (Years)	Riders	Pillion Riders	Total	Percentage
I	0-10	0	1	1	1.31
ii	11-20	2	7	9	11.84
iii	21-30	19	8	27	35.52
iv	31-40	5	9	14	18.42
v	41-50	10	5	15	19.73
vi	51-60	5	3	8	10.52
vii	61-70	0	0	0	0
viii	71-80	0	1	1	1.31
ix	81-90	1	0	1	1.31
Total	42	34	76	100	
Percentage	55.26	44.73	100		

Table III: showings fractures among riders and pillion riders

Body Parts	Riders		Pillion Riders	
		Percentage		Percentage
Skull	16	38	22	64
Face	7	16.66	5	14.7
Neck	1	2.3	0	0
Thorax	14	33.3	6	17.64
Pelvis	3	7.14	1	2.9
Upper Limb	2	4.76	1	2.9
Lower Limb	6	14.28	2	5.8

Table II: Number of cases showing external injuries among riders and pillion riders:

Parts Involved	Abrasion		Contusion		Laceration	
	Riders	Pillion Riders	Riders	Pillion Riders	Riders	Pillion Riders
Head	5 (11.9)	3(8.82)	26(61.9)	20(58.82)	9(21.4)	7(20.58)
Face	11(26.19)	10(29.4)	5(11.5)	4(11.76)	15(35.71)	7(20.58)
Neck	4(9.5)	1(2.9)	1(2.3)	1(2.9)	1(2.3)	1(2.9)
Chest	17(40.47)	13(38.23)	12(28.57)	7(20.58)	2(4.76)	0(0)
Abdomen	7(16.66)	7(20.58)	3(7.14)	2(5.8)	2(4.76)	1(2.9)
Upper Limb	19(45.23)	20(58.82)	2(4.76)	2(5.8)	1(2.3)	2(5.8)
Lower Limb	18(42.85)	15(44.11)	1(2.3)	2(5.8)	6(14.28)	5(14.7)

() states the percentage.

Most accidents involving two wheelers took place between 6PM–12 Mid Night which comprised 23 cases (30.26%) followed by 19 cases (25%) between 6 AM- 12 Mid Day. The reason could be inefficient lights, traffic rush as most people return home from work in the evening, alcohol abuse late at night etc. These findings were consistent with Sevitt S. (8) and Kumar A et.al⁷.

Highest number of victims with accidents in the urban areas accounting for 42%(55.26%) cases, followed by 18(23.68%) cases in rural areas. This was in agreement with Willard Ned⁹, Kumar A et.al⁷ and Central Road Research Institute¹⁰. Rush of traffic and faster speed of vehicles because of better roads may predispose to more accidents in urban areas.

Fracture of the skull was present in 22(64%) pillion riders, while it was seen in only 16(38%) riders. Devi et al³. found fracture of the skull in 71.43% pillion riders; while it was seen in 47.31% motorcyclist. Wearing of helmets might have prevented skull fracture in riders, a practice rarely practiced by pillion riders and thus predisposing them to more head injuries. Rib fractures were seen in 33.3% of riders and 17.64% of pillion riders.

Fracture of the long bones of the extremities was more commonly seen in riders; 4.76% in the upper limbs and 14.28% in lower limbs, compared to 2.9% in the upper extremities and 5.8% in lower extremities of pillion riders. Devi et al³. reported that none of the pillion riders had fracture of bones of the lower limbs that was seen in 10.28% of the motorcyclists. From the study they concluded that injuries to the limbs and rib fractures are more common in motorcyclists than in pillion riders, and skull fracture occurs more commonly in the later.

Amongst internal injuries, laceration to the brain was most commonly seen in 16(38%) riders and 18(52.94%) pillion riders. Again wearing of protective head gear may be the cause of slightly low incidence of brain injury in riders. However, contusion to the brain was seen in 9.5% in riders and only

Table IV: showing injuries to the organs among riders and pillion riders:

Viscera	Riders		Pillion Riders	
	Contusion	Laceration	Contusion	Laceration
Brain	4(9.5%)	16(38%)	1(2.9%)	18(52.94%)
Lungs	4(9.5%)	9(21.4%)	3(8.82%)	4(11.76%)
Pleura	3(7.14%)	10(23.8%)	4(11.76%)	5(14.7%)
Heart	1(2.3%)	3(7.14%)	0	2(4.76%)
Pericardium	2(4.76%)	3(7.14%)	0	1(2.9%)
Great Vessels	0	1(2.3%)	1(2.9%)	1(2.9%)
G.i Tract	1(2.3%)	0	1(2.9%)	5(14.7%)
Liver	0	8(19%)	1(2.9%)	5(14.7%)
Spleen	0	8(19%)	0	6(17.64%)
Mesentery	0	6(14.28%)	1(2.9%)	2(5.8%)
Kidney	0	0	2(5.8%)	1(2.9%)
Urinary Bladder	1(2.3%)	2(4.76%)	0	1(2.9%)

2.9% in pillion riders. Laceration to the liver and spleen constituted 19% each in riders while it was 14.7% and 17.6% respectively in pillion riders. Contusion and laceration of the lungs was seen in 13 (30.9%) riders and 7 (20.5%) pillion riders. Laceration of the heart was seen in 3 riders (7.14%) and 2 pillion riders (4.76%). Devi et al³. found contusion and laceration of the lungs in 32(29.91%) cases and laceration of the heart in 2(1.87%) cases among both riders and pillion riders combined.

Conclusion

The study thus highlights the importance of:

- Setting and enforcing speed limits on roads as appropriate.
- Strict enforcement of laws requiring riders and pillion riders of two wheelers to wear helmets.
- Requiring day time running lights for two wheeled vehicles.
- Managing existing road infrastructure to promote safety, through provision of safer routes for pedestrians and cyclists.
- Last, but not the least, to strengthen the health facilities for the victims.

References

1. WHO. South East Asia Regional Office Report 2001.
2. Ghosh PK. A study of complications contributing to death in vehicular accident death victims. In: a scientific article in the XIV Annual Conference (10-1-92 to 12-1-92) of Indian Association of Forensic Medicine.
3. Devi TM, Momonchand A, and Fimate L. Pattern of injuries in two wheeler fatal road traffic accidents. Journal of Medical Society RIMS, Imphal 1998; Vol. 12: 33-36
4. Ghosh PK. "A critical observation from the pattern of injury vis-à-vis vehicle and the road users in the city of Delhi" a scientific article XIV annual conference (10-1-92 to 12-01-92) of Indian Association of Forensic Medicine.
5. Salgado MSL, Colombage SM. Analysis of fatalities in road accidents. Forensic Science International 1988; 36: 91-96.
6. Adeyemo AO, et al; Thoracic injuries in road traffic accident; Injury 1984 July; Vol.16, No.1: 30-34.
7. Kumar A, Qureshi GU, Aggarwal A, Pandey DN. Profile of thoracic injuries with special reference to road traffic accidents in Agra. Journal of Indian Association of Forensic Medicine 1999; Vol.21, No. 4:104-109.
8. Sevitt S. Fatal road accidents in Birmingham, time to death and their causes. Injury: The British Journal of Accident Surgery 1973 May; 4(4):281-293.
9. Willard Ned. World Health 1969 Feb; 693.
10. Central Road Research Institute. New Delhi, Annual Report. 1997.

Pattern of homicidal deaths in children in Bangalore (2003-2009)

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Abstract

Pattern of Homicidal Deaths in children differs from that in adults in many ways like method of homicide, motive and victim-offender relationship. The present study is a retrospective analysis of 7 years data from January 2003 to December 2009, regarding homicidal deaths in children below 12 years. This study analyses the pattern, circumstances, autopsy findings in these cases and probable reasons for these crimes. An attempt is also made here to study the pesticide – suicide cases.

Key words

Homicidal deaths, Pesticide, Pesticide-Suicide

Introduction

Homicide is the most serious crime as old as civilization and reported as early as in the Bible¹. Homicide is defined as killing of one human being by another human being². The global rate of homicide is about 7.6 per 1 lakh population³. About 80 – 100 cases of homicides take place everyday in India⁴.

Homicidal death in children are known as pesticides and constitutes special category since pattern of homicidal deaths in children differs from that in adults in many ways like method of homicide, motive and relationship of victim with the offender etc.

Homicide-suicide describes a situation in which a homicide has been followed by the suicide of the perpetrator⁵.

Pesticide-suicide describes a situation in which homicide of child/children has been followed by the suicide of the perpetrator.

Homicide –suicide events commonly involve husband and wife, wife being the victim usually. Children comprise second most common victims of homicide- suicide events⁶.

Unfortunately the investigation of homicide suicides involving the children (Pesticide – Suicide) is often hampered by the death of the perpetrator who might have thrown more light on the motive. Parents killing their own children and then committing suicide afterwards are often seen⁷. In such cases

Table 1: Year wise distribution of cases

Year	No. of Pesticides			Total No. of Homicides
	Male	Female	Total	
2003	0	3	3	17
2004	1	2	3	19
2005	4	1	5	23
2006	2	3	5	30
2007	1	0	1	22
2008	1	6	7	26
2009	1	3	4	32
Total	10	18	28	169

Pesticides constituted 16.5% (28 Cases) of the total homicides deaths (169 Cases). There was spurt in such cases in the recent years with significant sex variation (males: female = 1: 1.8).

mothers tend to use non violent methods where as father tend to use violent methods⁸.

In spite of increasing incidence of pesticide and pesticide-suicide events very limited studies have been conducted in this part of the world about pesticide and pesticide – suicides. Hence the aims and objectives of this study were to ascertain the circumstances of crime in Pesticides, Pattern of pesticides with an emphasis on Pesticide – Suicides.

Materials and methods

This 7 years retrospective study was taken up from January 2003–December 2009. Data was collected from the postmortem reports of all homicidal deaths amongst autopsies conducted at M.S. Ramaiah Medical College, Bangalore and information/history was obtained from police/relatives of victim regarding the circumstances of crime. All cases of homicides aged up to 12 years were included. Unknown neonaticides were excluded as data regarding circumstances; motive and victim-offender relationship cannot be established in such cases.

Results and discussion

Probable reasons for Pesticide-Suicide

1. The perpetrator in 95% Pesticide - suicide events was victim's mother. The thought of leaving their children orphaned after their demise might have led to the act.
2. The cause/reason in 72% of pesticide - suicide cases was due to dowry harassment, by husband and in laws.
3. Socio economic constraint: - Most of the mothers were widows or were estranged from their husbands due to family disputes, unemployed or employed but unable to make ends meet thus resorting to the pesticide – suicide.

Table 2: Age distribution of Victims

Age groups	No of cases		
	Male	Female	Total
0 to 04 years	08	07	15
05 to 08 years	01	06	07
09 to 12 years	01	05	06
Total	10	18	28

Majority of the victims (15) belonged to 0-4 years age group, 7 of them were aged 5-8 years and 6 of them were in the age group 9-12 years.

Table 3: Pattern of pesticides

Sl. No	Pattern	No. of Cases
01	Hanging	06
02	Drowning	06
03	Poisoning	05
04	Sharp weapon injury	04
05	Blunt weapon injury	02
06	Strangulation	02
07	Smothering	01
08	Rape and murder	02
Total		28

Out of 28 Pedicide 6 were due to homicidal hanging, 6 were due to homicidal drowning, 5 cases were due to homicidal poisoning. In all these 17 cases the perpetrator was mother using a nonviolent method to kill her children; this is similar to observation made by Dayne J⁸. 6 cases were due to injuries where the perpetrator was an acquaintance or father.

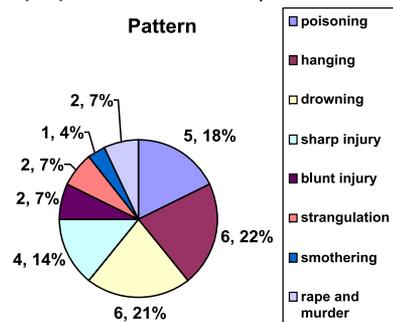


Table 4: Victim-offender relationship

Sl.no	offender	No. of Cases
01	Mother	17
02	Father	04
03	Grandmother	01
04	Acquaintance	03
05	Stranger	01
06	Not known	02
Total	28	

Victim – Offender Relationship: - Mother was the perpetrator in 17 cases which were also pedicide-suicide events. In 4 cases perpetrator was father of which only one was a case of pedicide-suicide. In 3 cases acquaintances were the perpetrators and in 2 cases of unknown victims offenders could not be traced.

Pedicide – Suicide (Offender - Mother) (PM No 279,280,281/06)



Pedicide – Suicide (Offender - Mother) (Ligature Material in Situ)(PM No 176,177/06)



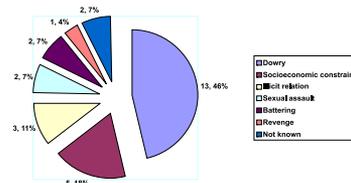
Table 5: Pedicide Suicide

Sl.No	Perpetrators suicide events	No of Pedicide -	No. of Pedicides
01	Mother	13	17
02	Father	01	01
Total		14	18

Out of 28 cases of Pedicide 18 were due to Pedicide-suicide involving 14 events (In 4 events the mother killed 2 children). In 95% of Pedicide-suicide cases (17 cases) the offender was mother.

Table 6: Motive for pedicide

Sl. No	Motive	No. of Cases
1	Dowry harassment of mother	13
2	Socio economic constraint of parents	05
3	Illicit relationship of mother	03
4	Sexual assault	02
5	Battering	02
6	Revenge on parents	01
7	Not known	02



13 cases (46%) were due to dowry harassment, 5(18%) were due to socioeconomic constraint of parents, and illicit relationship of mother was a reason behind 3 pedicides. In 2 cases the sexual assault was followed by homicide. Two deaths were the end results of child battering.

Pedicide – Suicide (PM No 679,680/05), the father after smothering his 4year old son with a pillow had committed suicide by hanging due to financial reasons.



Drowning (Pedicide – Suicide), Offender-mother, Pm No 695/05



Conclusion

Homicidal deaths in children have increased by the years alarmingly and the most common age group being 0-4 years more so among females. In our society mother is thrust upon the responsibility to bring up the child and to look after the family, hence the offender in all, but one case of Pedicide – suicide. The pattern commonly being employed by her is homicidal hanging, drowning followed by poisoning - the easily available methods.

References

- Gupta Avnesh et al. 'Study of homicidal deaths in Delhi' Medicine, Science and Law, 2004, 44(2), 127- 132.
- Narayana Reddy K.S, 'Essentials of Forensic Medicine and

- toxicology' Medical book company, Hyderabad, 25th Edition 2006, 250.
3. "Global burden of armed violence report, Geneva Declaration on armed violence and development" accessed on <http://www.geneva.declaration.org/resources-armed-violence-report.html> on 16/2/2010
 4. Mohd.S.I., Subramanyam BV, 'study of homicides in Surat with special reference changing trend'. J. Forensic Medicine and Toxicology, 1995; 12; 8 – 15
 5. Milroy C.M. 'The epidemiology of homicide – suicide' Forensic Science International, 1995; 71; 117 – 172
 6. Chan C.Y., Beh S.L., Broad Hurst R.G., 'Homicide – suicide in Hongkong, 1989 – 1998'. Forensic Science International, 2003; 137, 165 - 171
 7. Byard R.W., Knight D., James R.A., Gilbert J. 'Murder – suicide involving children a 29 year study' American Journal of Forensic medicine and Pathology 1999; 20(4); 323 – 327
 8. Dayne J., James, Busutil.A., Smock.W., 'Forensic Medicine –Clinical & Pathological Aspects' 2003 edition.

Thin layer chromatographic and spot test detection of carbosulfan by alkaline fast Blue-B reagent

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Abstract

Thin layer chromatographic and spot test detection of carbosulfan, a carbamate pesticide by alkaline fast blue-B reagent is described. Carbosulfan on alkaline hydrolysis forms a phenolic product which further condenses with fast blue-B and forms orange coloured complex on silica gel-G TLC plate. Sensitivity of this reagent to detect the carbosulfan was found to be 5µg. The method can be applied to detect this carbamate in various samples including forensic samples.

Key Words

TLC, Spot Test, Carbosulfan, Alkaline Fast Blue-B Reagent.

Introduction

Carbosulfan [2, 3-dihydro-2, 2-dimethyl-7 benzofuranyl {(dibutylamino) thio} methyl carbamate] is a carbamate pesticide which is largely used against variety of pests on cereal, fruit and vegetable crops. Easy availability and low price of carbamate pesticides particularly of carbaryl, propoxur, methomyl, carbofuran and carbosulfan have resulted in misuse for poisoning purpose in India and large number of cases related to their poisoning received in Forensic Science Laboratories across the country. Therefore, their detection and identification is important in frequently encountered forensic samples.

Owing to insecticidal properties of carbosulfan some investigators have carried HPLC¹, GC², LC-MS³, and Spectrophotometric methods^{4,5}, for determination of carbosulfan but these analytical methods are not considered good for initial screening of complex forensic samples. On the contrary TLC and spot test are the method of choice for initial screening and detection because of their simplicity, rapidity as well as ability for screening and detection of compounds from complex matrices. However, some chromogenic reagents such as diazophenol after alkaline hydrolysis⁶, tollens' reagent⁷, p-nitrobenzene diazonium fluoborate⁸, zinc chloride-diphenylamine⁹, diazotized p-nitroaniline and diazotized p-aminoacetophenone¹⁰, phenylhydrazine hydrochloride in alkaline media¹¹, ceric ammonium nitrate¹², diphenylamine-formaldehyde¹³, are reported for thin layer chromatographic detection of some carbamate and particularly of carbaryl but none of the above reagent is reported for detection of carbosulfan and further no other reference was found for TLC or spot test detection of carbosulfan.

This study describes use of alkaline fast blue-B reagent for detection of carbosulfan by TLC and spot test producing orange coloured spot. These methods can be used for initial screening and detection of carbosulfan in various types of matrices including biological matrices.

Material and methods

All chemicals of analytical grades were used.

1. Preparation of Standard Pesticide Solutions

Authentic reference standard carbosulfan was obtained from E.I.D. Parry (India) Limited as gift sample. Standard

solution of 1mg/ml (1µg/µl) strength of carbosulfan was prepared in methanol for thin layer chromatography and spot test.

2. Preparation of chromogenic reagent for TLC and spot test

Alkaline Fast Blue-B Reagent: 10g of sodium hydroxide dissolved in 100 ml of distilled water, then 0.1g of fast blue-B salt was added to it and the solution shaken well. Alternatively, 10% sodium hydroxide solution and 0.1% fast blue-B solution can also be sprayed one after another. Reagent should be prepared just before its utilization during thin layer chromatography and spot test.

TLC method

Thin layer glass plates coated with slurry of silica gel-G in water (1:2) to a uniform thickness of 0.25mm were activated at 110°C for 30 minutes just before use. A standard Desaga template was used for marking the position of sample application on activated chromatoplate and solvent front was kept at 10 cm. Reference standard solutions (5ml) of carbosulfan [1ml @ 1mg] along with solution of other commonly available carbamate pesticides (carbaryl, propoxur, carbofuran, methomyl, thiophanate methyl and cartap) were spotted on chromatoplate 1.5 cm above from bottom. The spotted chromatoplate plates were then developed in ascending way in a chromatographic chamber with 30 minutes prior chamber saturation with mobile phase toluene-acetone (9:1 v/v). After migration of mobile phase to the mark of solvent front the TLC plate was taken out from chromatographic chamber, dried in air and sprayed with alkaline fast blue-B reagent and development of colour was noted.

Spot test

Standard solution (5ml) of carbosulfan and other carbamate pesticides were applied on activated silica gel-G chromatographic plates. A drop of alkaline fast blue-B reagent was put over the spots of different pesticides and development of colour was noted. A blank test was also carried out to differentiate the colour so obtained by the compounds and the reagent reaction.

Detection of carbosulfan from spiked biological specimens

50g of biological tissues such as liver, lungs, spleen, kidney, stomach and intestine were cut into small pieces, macerated in small amount of water and transferred in a conical flask. Then 10 mg carbosulfan was added in conical flask and plugged with cotton. The contents of flask kept digested overnight and then subjected to extraction procedure. 25g sodium sulphate, 100 ml acetone and 100 ml n-hexane were added in conical flask and kept on water bath for about 1 hour. Then the contents of flask were filtered using whatman filter paper and washings of the residues were also taken with n-hexane and acetone and then combined with filtrate. The filtrate was taken in separating funnel and 25ml of 10% aqueous sodium sulphate solution (w/v) added and shaken for 15 minutes and the organic layer was collected. The remaining aqueous phase was again extracted thrice with aliquots of 100ml solvent (50ml acetone + 50ml n-hexane). All extracts were combined with previously

collected organic layer and taken in a porcelain basin and then kept in open air to evaporate the solvents at room temperature. Methanol (10ml) was added in the porcelain basin to solubilise the left over residue to prepare the test solution. Thin layer chromatography and spot test methods as mentioned earlier were carried out by spotting 10ml of this test solution ie biological extract for detection of carbosulfan.

Results and discussion

Thin layer chromatographic and spot test detection of sulphur containing carbamate namely carbosulfan with alkaline fast blue-B reagent was carried out. Alkaline fast blue- B reagent produced orange coloured spot for carbosulfan at its R_f value (0.94) during TLC against a white background of silica gel-G. The sensitivity of this reagent was found to be 5mg for detection of carbosulfan. This reagent was equally found suitable for detection of carbosulfan by spot test method.

This reagent did not react with carbamates namely methomyl, thiophanate methyl and cartap. However, this reagent produced violet coloured spot with carbaryl and orange coloured spot with carbofuran and propoxur. Alkaline fast blue-B reagent is previously reported for the detection of three carbamates namely carbaryl, propoxur and carbofuran, as well as cannabinoids and phenolic compounds^{14,15}, but these compounds give the positive reaction with this reagent at different R_f values; so they can be differentiated from each other by their respective R_f values. Moreover, the ability of this reagent to detect carbosulfan was also not studied by earlier researchers which may lead mistaken identity of a carbamate by this reagent in lieu of carbosulfan when present in forensic samples. But the present study will overcome this problem by extending the range of this reagent for detection of one more carbamate pesticide namely carbosulfan and will certainly enable the forensic toxicologists in screening and initial detection of carbosulfan in forensic samples along with other carbamates (carbaryl, propoxur and carbofuran) since they form coloured spots at different R_f values. The suggested reagent also responded positively for the detection of carbosulfan, extracted from spiked biological samples.

The described chemical test is simple, sensitive, and make use of readily available chemicals in a laboratory hence can be used for initial screening of carbamates namely carbosulfan in forensic samples and its further confirmation can be made by instrumental techniques.

References

1. Brooks M, Barrows A. Determination of carbosulfan in oranges by high-performance liquid chromatography with post-column fluorescence. *Analyst* 1995; 120(10): 2479-81.
2. Bruce CL, James CM, Robert CH, Glenn HF. Determination of carbosulfan and carbofuran residues in plants, soil, and water by gas chromatography. *J Agric Food Chem* 1983; 31(2):220-3.
3. Carla S, Jordi M, Yolanda P. Determination of carbosulfan and its metabolites in oranges by liquid chromatography ion-trap triple-stage mass spectrometry. *J Chromatogr A* 2006;1109 (2): 228-41
4. Harikrishna V, Prasad B. Spectrophotometric determination of propoxur and carbosulfan with 2,4,6- trichloroaniline as the coupling agent. *Asian J Chem* 2003; 15(2): 1013-9.
5. Naidu DV, Naidu PR. A simple spectrophotometric method for determination of carbosulfan and propoxur. *Talanta* 1990; 37(6): 629-31.
6. Randerth K. *Thin Layer Chromatography*. New York: Academic Press; 1965, p.176.
7. Kawale GB, Joglekar VD. Tollens's reagent for the detection of carbamate and organophosphate insecticides. *Curr Sci* 1976; 45(2): 57-8.
8. Sherma J, Kovalchick AJ, Mack R. Quantitative determination of carbaryl in apples, lettuce and water by densitometry of thin layer chromatograms. *J Assoc Off Anal Chem* 1978; 61(3): 616-20.
9. Sevelkar MT, Patil VB, Katkar HN. Zinc-chloride-diphenylamine reagent for thin layer chromatographic detection of some organophosphorus and carbamate insecticides. *J Assoc Off Anal Chem* 1991; 74(3):545-6.
10. Raju J, Gupta VK. A Thin layer chromatographic method for the detection of carbaryl with diazotized p-nitroaniline and diazotized p-amino acetophenone. *J Anal Chem* 1991; 339: 897.
11. Patil VB, Shingare MS. Thin-layer chromatographic detection of carbaryl using phenylhydrazine hydrochloride *J Chromatogr A* 1993; 653(1): 181-3.
12. Patil VB, Shingare MS. Thin layer chromatographic spray reagent for the screening of biological materials for the presence of carbaryl. *Analyst* 1994; 119(3):415-6.
13. Daundkar BB, Mavle RR, Malve MK, Krishnamurthy R. Detection of carbaryl insecticide in biological samples by TLC with a specific chromogenic reagent. *J Planar Chromatogr* 2006; 19(112): 467-8.
14. Tiwari SN, Singh R. *Brochure of the Autumn School of Forensic Science*, 1979. Chandigarh, India.
15. *Working Procedure Manual Toxicology*. New Delhi: Bureau of police research & Development; 2001, p. 130.

Aluminium and Alzheimer's disease – An overview

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Abstract

Alzheimer's disease is characterized by progressive loss of short term memory followed by general loss of cognitive and other brain functions, the need for constant care and eventual death. Researchers believe that in majority of those affected, Alzheimer's disease results from combination of different risk factors rather than one single cause. Such factors which vary from person to person may include age, genetic predisposition, other diseases and environmental factors. Researchers have claimed number of circumstantial links between aluminium and Alzheimer's disease. Aluminium, being a highly reactive metal, is known to cross-link hyperphosphorylated proteins, which may play an active role in the pathogenesis of critical neuropathologic lesion in Alzheimer's disease and other related disorder. Present article condenses existing scientific evidence about the relationship between aluminium exposure and development of Alzheimer's disease evaluating its long term effects on public health.

Key words

Aluminium, Alzheimer's disease, Risk factor.

Introduction

Alzheimer's disease (AD) is a neurodegenerative disorder prevalent in senile population. It is clinically characterized by progressive loss of memory and other cognitive abilities and pathologically by severe neuronal loss, glial proliferation and amyloid plaques composed of β amyloid protein surrounded by degenerated nervous terminations and neurofibrillary tangles^[1]. One of the problems in establishing the link between aluminum toxicity and Alzheimer's disease is difficulty in diagnosing Alzheimer's disease. Unless the autopsy is performed, the symptoms are often diagnosed as dementia or cognitive impairment. As a result, many studies in this area use dementia or cognitive impairment as the diagnosed disease and not Alzheimer's disease².

AD is probably the result of the multifactorial process in which genetic and environmental components are included. It is supposed that individual genetic characteristics modulate the environmental exposures. Environmental risk factors related to development of AD include exposition to Aluminium, one of the most studied potential environmental risk factor^[3]. This article reviews the role of aluminium in the etiology and pathogenesis of AD.

Sources of aluminium

Aluminium is a common metal in the environment and one the most abundant in terrestrial crust. Aluminium is liberated in the environment by natural process of soil eruption and anthropogenic actions. Main sources of aluminium in our diet include tea, beer, baked products, drinking water, toothpaste, aluminium based antacids, aluminium cookware and some canned beverages⁴. Aluminium uptake from body diets is usually very low with more than 99% passing through the digestive system unabsorbed. Absorption increases significantly in the presence of acidic food such as orange juice.

The small amount of aluminium that is absorbed into body is rapidly excreted by the kidneys in urine except in individuals with impaired kidney functions where aluminium retention within the body is responsible for dialysis dementia.

Even though food is important source of aluminium ingestion, it is the water which presents the higher bioavailability to be absorbed by the intestine⁵. Aluminium salts are largely used as coagulants to reduce organic matter, turbidness and microorganisms present during treatment of superficial water, which presents the largest quantity of particles in suspension. This use although useful for water treatment in many cities, can increase the concentration of aluminium at final point of consumption⁶.

Experimental evidences

The hypothesis that there is a link between aluminium and AD was first put forward by in the 1960's. They found that intracerebral inoculation of aluminium phosphate in rabbits resulted in neurofibrillar degeneration significantly similar to neurofibrillar degeneration of Alzheimer's disease^{7,8}. This led to assumption that there is relationship between aluminium and Alzheimer's disease. In 1973, the first article that evidenced increase in aluminium concentration in patients with AD was published⁹. Since then, researchers have claimed that number of other circumstantial links between aluminium and AD. Aluminium has been shown to be associated both with plaques and tangles in the brains of people with AD¹⁰. Some have claimed that people with AD have a higher than average level of aluminium in their brains¹¹.

Role of aluminium in pathogenesis of Alzheimer's disease

Alzheimer's disease is associated with general reduction of cerebral tissue with localized loss of neurons mainly in the hypofield and basal forebrain. An experimental study performed with mice treated with aluminium demonstrated a statistically significant reduction in their brain weight¹². Two microscopic aspects are typical in AD, the extracellular amyloid plaques, also called senile plaques, which consist of extracellular deposits of A β protein, and neurofibrillar intraneuron tangles that consist of filaments of phosphorylated form of a protein associated to microtubules (Tau). Alteration in the processing of A β protein from its precursor, APP (amyloid precursor protein), is recognized as an essential characteristic in the AD pathogeny¹³.

There are two types of A β protein, A β 40 and A β 42. A β 40 protein is normally produced in small quantities, though the A β 42 presents a super production due to genetic mutations. Both proteins aggregate to form amyloid plaques. However A β 42 presents a higher tendency to do this than A β 40, constituting the main responsible in the formation of amyloid plaques. The A β 40 and A β 42 are produced by proteolytic cleavage of a precursor amyloid protein, the APP, a protein of larger membrane and normally expressed by many cells, including neurons of the central nervous system¹³. The APP mutations of genes ease the formation of A β , especially the A β -42, with consequent increase in the formation of amyloid plaques. It has been observed that aluminium increases the

Ab protein neurotoxicity, the degeneration of neurons exposed to it and also aggregation of A β protein¹⁴.

Tau protein becomes abnormally phosphorylated in AD and is deposited intracellularly under the form of paired helical filaments with a characteristic microscopical aspect. When the cell dies, these filaments aggregate as neurofibrillar extracellular tangles. There is strong influence of aluminium ions on phosphorylation which can be the cause, because neurofibrillar tangles contains hyperphosphorylated microtubules associated to the Tau protein. It is possible that Tau phosphorylation is intensified by the existence of amyloid plaques. Its phosphorylation compromises rapid axonal transport, a process that depends on the microtubules¹³.

The increased concentration of aluminium favors the formation of Tau protein and consequently, the formation of neurofibrillar tangles¹⁵. Studies have shown that amyloid plaques are surrounded by glial reactive cells. Thus, exposition to aluminium can activate the oxidative processes of glial cells, which in turn can indirectly damage the neuronal integrity¹⁶.

The neuronal degeneration observed in AD can also occur due to oxidative stress. Oxidative stress refers to conditions like as hypoxia, characterized by compromised protection mechanisms, as the neurons become more susceptible to excitotoxic lesion. Oxidative stress is induced in brains exposed to aluminium¹³.

Conclusion

Alzheimer's disease is most common cause of senile dementia accounting for 50-60% of the total cases. It is present in about 17% of the population aged 65-69, but its incidence increases steadily with age, and in those who are 95 and older, the incidence is 40-50%. Most cases of AD are sporadic but some are familial. Thus, AD plus the other forms of senile dementia are major medical problem¹⁷.

In this review article, the need to understand the importance of environmental factors is highlighted, especially exposure to aluminium, as determinants in the population's health-disease process, stressing its potential to affect positively and negatively, natural aging processes. It is demonstrated in an epidemiological study that individual who use to ingest food with high aluminium contents presented a two times higher risk of developing AD¹⁸.

Thus it can be well substantiated from the different studies of research group that aluminium affects several neurophysiological processes, responsible for degeneration characteristic of AD. Therefore, scientific evidence has shown that, in the last years, aluminium has been associated with development of AD. Thus, preventing exposure to certain environmental factors like AD, which in recent years has acquired great importance for collective health all over the world.

References

1. Yokel RA. The toxicology of aluminum in the brain: a

- review. *Neurotoxicology* 2000; 21(5):813-28.
2. Flaten, Trond P. "Aluminium as a Risk factor in Alzheimer's Disease, With Emphasis on Drinking water." *Brain Research Bulletin* 2001; 55: 187-196.
3. Crapper DR, Krishnan SS and Quittkat S. Aluminium, neurofibrillary degeneration and Alzheimer's disease. *Brain* 1976; 99; 67-80.
4. Rao JKS and Rao VG. Aluminium leaching from utensils- a kinetic study. *International Journal of Food Sciences and Nutrition* 1995; 46:31-38.
5. Martyn CN, Coggan D, Inskip H, Lacey RF, Young WF. Aluminium concentrations in drinking water and risk of Alzheimer's disease. *Epidemiology* 1997; 8(3): 281-6.
6. Bates AJ. Water as consumed and its impact on the consumer-Do we understand the variables? *Food Chem Toxicol* 2000;38(1) suppl: 29-36.
7. Terry RD and Pena C. Experimental production of neurofibrillary pathology: electron microscopy, phosphate histochemistry and electron probe analysis. *Journal of Neuropathology and Experimental Neurology* 1965; 24:200-210.
8. Klatzo I, Wisniewski H and Streicher E. Experimental production of neurofibrillary pathology: 1. Light microscopic observations. *Journal of Neuropathology and Experimental Neurology*; 24; 187-199.
9. Alfrey AC. Aluminium and renal disease. In: Bourne E, Mallick NP, Pollak VE. *Moving points in nephrology. Contrib Nephrol* 1993;102: 110-124.
10. Crapper DR, Krishnan SS and Quittkat S. Aluminium neurofibrillary degeneration and Alzheimer's Disease. *Brain* 1976;99:67-80.
11. Trapp GA, Miner GD, Zimmerman RL, Mastro AR, Heston LL. Aluminium levels in brain in Alzheimer's disease. *Biological Psychiatry*; 13(6):709-718.
12. Tanino H, Shimohama S, Sasaki Y, Sumida Y, Fujimoto S. Increase in phospholipase C- α 1 Protein levels in aluminium treated Rat Brains. *Biochem Res Commun* 2000; 271(3): 620-625.
13. Hang HP, Dale MM, Ritter M. *Farmacologia*, 4a ed. Rio de Janeiro RJ: Guanabara Koogan: 2001.
14. Kawahara M, Midori K, Kuroda Y. Effects of aluminium on the neurotoxicity of primary cultured neurons and on aggregation of β -amyloid protein. *Brain Res Bull* 2001; 55(2); 211-217.
15. Campbell A, Kumar A, La Rosa FG, Prasad KN, Bondy SC. Aluminium increases levels levels of beta-amyloid and ubiquitin in neuroblastoma but not in glioma cells. *Proc Soc Exp Biol Med* 2000; 223(4): 397-402.
16. Campbell A, Prasad KN, Bondy SC. Aluminium-increases levels of beta-amyloid and ubiquitin in neuroblastoma but not in glioma cells. *Proc soc Exp Biol Med* 2000; 223(4): 397-402.
17. William F Ganong. *Review of Medical Physiology*. 22nd edn. New Delhi. Mc Graw Hill 2005: 271-72.
18. Rogers MA, Simon DG. A preliminary study of dietary aluminium intake and risk of Alzheimer's disease. *Age Ageing* 1999; 28(2): 2205-2209.

Study of electrical injuries in fatal cases at Victoria Hospital, Bangalore (Nov 2003 to Oct 2005)

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Abstract

Back ground and objective

With wide spread use of electricity at home and work place the amount of electrical related injuries are on the rise. The present study attempts to know distribution pattern of electrical injuries and Histo Pathological changes in skin with electrical contact mark and in internal organs.

Methods

Study was conducted on deaths due to electrocution brought to Victoria hospital mortuary during the period of two years. Specimen of skin with contact mark and internal organs like heart, kidney was sent for Histo Pathological examination. Data will be collected from police inquest and photographic evidence from scene of occurrence.

Result

Total number of 61 cases was studied in two-year period. High voltage electrocution constituted 54% of cases. Low voltage electrocution constituted 46%. High voltage electrocution mainly presented as flash burns, while low voltage electrocution presented with contact mark. Typical Histo Pathological changes were seen in skin and heart specimen sent for examination. Histo Pathological changes in kidney were nonspecific.

Conclusion

Electric contact mark is diagnostic criteria for electrocution. Histo Pathological changes were used as supportive evidence in determining cause of death.

Main reasons for electrocution deaths were found to be human negligence, faulty electrical equipments and connections and lack of protective measures.

Key words

High voltage current; Low voltage current; Electric contact mark; Flash burns; Histo Pathological examination

Introduction

The uses of electricity put to the service of man have no limitations. Electricity is the basic need for the economic development of the country.

Supply of electricity is of two types:

Alternating Current (AC)
Direct current (DC)

AC is called, if current periodically changes its direction and magnitude. At present large percentage of the electrical energy, being used for domestic and commercial purpose is generated as AC current.

DC current are steady and in one direction. DC current is absolutely necessary such as for electro plating, charging of

storage batteries, refining of copper and aluminum, production of industrial gases by electrolysis, metal rolling mills, high speed gearless elevators.

The normal supply of AC current to domestic purposes in India is between 220 -250 v, whereas for industrial purposes is 440 v

The quantum of danger exists as much as quantum of electricity is in use. Accidents and fatal deaths are inevitable due to negligence and non-application of proper guidelines. Thus electricity has become essential, inevitable, and invaluable but dangerous part of our lifestyle¹.

Effect of Electrotrauma on human body

Skin

Most important indication of electric contact is current mark also called 'Joule burn'. Skin is relatively thin layer of tissue but highly resistant to passage of current, resistance is not homogenous. At points of poor insulation ion mobilization of cellular fluids takes place, resulting in damage to cell membranes. The diminished tissue resistance allows current to penetrate the skin. Higher the voltage greater is chance of skin penetration. Greater length of contact greater is the penetration. Current marks are not likely to occur if contact of current is over a large surface of body and if time of impact is only a few seconds and the amperage is less than 1.5amp. Reduced cutaneous resistance and low density of current are two factors that facilitate the traceless passing of current through skin. It is convenient and of practical importance to consider the injuries in two groups.

1. Due to low or medium current up to 400v.
2. Those due to high-tension circuits.

1. Electric mark

An electric mark is found at the point of entry of current where the electric energy is converted into thermal energy. The electric mark is specific and diagnostic of contact with electricity. The finding of an electric mark gives strong indication of death by electrocution. Characteristically these marks are round or oval, shallow crater, bordered by a ridge of skin of about 1 to 3 mm height. The crater floor is lined by pale flattened skin. In some marks there will be breach of skin within or near margin of crater, resembling that of a broken blister. The skin of the mark as a whole is distinctly pale, but there may be hyperemia of skin immediately beyond it. The shape of the mark is determined by shape of conductor or that part of it, which is in contact with skin, burnt mark is usually circular or oval but when conductor is rod like structure or wire then the mark acquires shape of patterned wire.

Joule burn (Endogenous burn)

When the contact is more prolonged the skin acquires a brown tint and with further contact mark there will be charring. These changes are due to burning, so called Joule burn, a term, which distinguishes it from exogenous thermal heat, following contact with high voltage i.e.; flash burn. Electric marks and Joule burns are most often found in exposed part of body and in particular on hand, more frequently on flexor aspect of hand.

Joule burn is also termed endogenous burn where electric energy is converted to thermal energy within tissue.

A current mark gives useful information on path pursued by current. Current marks may be limited to epidermis and characterized by grayish white parchment like alteration. It may expand to corium or even deeper giving risk to crater shaped ulcer with curling borders. These marks are usually found at site of entry and exit of electrical current.

Exit marks are variable in appearance but have most features of entrance marks. There will be more disruption of tissue and instead of presenting as cracks they are often seen as split.

2. High tension circuit injuries (Exogenous burns)

Injury by high tension current is either by direct contact or an indirect result of arcing or flashes over. There is risk of grave thermal burns because of considerable heat generated in flash and knock down by sudden and appreciable increase in local atmospheric pressure. Risk of arcing arises when the person comes close to the high-tension cables.

The survival of victim implies that the current had passed by and not through the body. Flash burns may be extensive and severe and may be exaggerated by burns from ignited clothing. There will be gross destruction of soft tissues, charring of bones or fusion into pearl like bodies.

Heart and circulatory system

On brief exposure to low voltage current cardiac action may momentarily stop and start again with after withdrawal of current. Exposure to current of greater intensity following contact with longer duration leads on to ventricular fibrillation and cardiac arrest. With high voltage current ventricular fibrillation do not occur.

Pericardial petechiae may be only finding at autopsy apart from skin lesion.

Kidneys

Myoglobinuria occurs due to extensive tissue damage, which leads to multiple myoglobin casts in renal tubules.

Electrolyte imbalance, proteinuria, myoglobinuria, reddish brown color urine, myoglobin casts and renal failure are a common complication of high voltage shock².

Microscopic features in electrocution

Skin

small, multiple craters like defects or massive degeneration of epithelium and collagen with typical micro blisters in epidermis. The skin mark consists of vacuolation in epidermis caused by gas spaces from heated tissue fluids splitting cells apart. The affected tissues become more eosinophilic. Cap of epidermis may be detached and raised into a blister with large space beneath. The Cells of epidermis are often elongated with

Table 1: Distribution of Electrocution Deaths Based on Voltage

Sl.no	Voltage	No of Cases	Percentage
1	High Voltage	33	54%
2	Low Voltage	28	46%
	Total	61	

Table 2: Distribution Pattern of Injuries in Electrocution Deaths

Sl.no	Injuries	No of Cases	Percentage
1	Contact Mark	35	44%
2	Flash Burns	29	37%
3	Contact Mark With Flash Burns	9	11%
4	No Contact Mark/ Flash Burns	6	8%

nuclei of lower layers oriented and stretched. Heat generated causes formation of spherical vacuoles in horny layer (stratum corneum) of skin with splitting of layers of epidermis. Honey comb or Swiss cheese appearance may be produced if more heat is generated by blister formation in epidermis. Elongation of cell nuclei and cells in epidermis and formation of approximately parallel cellular sheets (pallisading), particularly basal and spindle cell layers are observed. This cellular arrangement often produces whorled appearance. Denaturation of dermal connective tissue and subcutaneous muscle cell may occur with altered staining reaction (increased basophilia)³.

Heart

Myocardial fibers show fragmentation, twisting of myofibres, interstitial edema and foci of colliquative necrosis, which are interpreted as direct manifestation of passage of electric current. Wavy appearance of myocardial fibers and thin fragmentation may be suggestive of electrocution. Contraction bands within fibers, especially bark like appearance have been described⁴.

Kidneys

congestion, Disseminated Intravascular Coagulation, lower nephron nephrosis in case of muscle degeneration⁵.

In cases of sudden death due to electrical shocks, the authorities will depend mostly or completely on medical evidence in establishing the cause of death. The doctors are the chief source of evidences upon which legal decisions are made.

The precise total number of electrical accidents is difficult to ascertain, as number of non-fatal accidents are not recorded. The present study is limited to fatal cases of

electrocution. The characteristic of electrocution being electric contact mark over the body is absent in many of the cases, which leads to the quest for diagnostic criteria of electrocution.

It is often difficult to prove whether a man has been electrocuted, since death from electrocution can occur with no marks on the body, hence it is important for forensic pathologist to be aware of histopathological changes other than contact mark to prove electrocution and aid in investigation of such cases.

Objectives

To know the pattern of injuries in deaths due to electrocution.

i.e.; pattern of injuries in low voltage and high voltage currents and to study Histo Pathological changes in skin, where contact mark is present and Histo Pathological changes in organs such as heart and kidney in fatal electrocution cases.

To suggest concrete preventive measures to make house and work place safer so that electrocution deaths could be prevented in future.

Table 3: Pattern of Electrical injury with respect to Voltage

Sl.no	Voltage	Total Cases	Flash Burns	Percentage	Contact Mark	Percentage
1	High Voltage	33	26	78%	10	30%
2	Low Voltage	28	3	11%	25	89%

Table 4: No of Cases with Histo Pathological Changes In organs

Sl.no	Organs	Total Specimen	HpeChanges Seen	Percentage
1	Skin	34	34	100%
2	Heart	34	16	47%
3	Kidney	34	21	62%

Methodology

The present study involves study of electrical contact marks in fatal electrocution cases brought for autopsy to Victoria Hospital, Bangalore. Study period is for two years. Skin with contact mark along with portion of normal skin as control is dissected and sent to Department of Pathology for Histo pathological examination. Sections were observed under low power and high power Microscope. Observations were made, recorded and photographed. Internal organs such as heart and kidney were sent for histopathological examination, sections from those organs were examined and observations were made, recorded and photographed. Retrograde study involves collection of data from the inquest report, relatives of deceased and police regarding the circumstances of electrocution. Photographic evidences from site of occurrence were collected.

Subjects included for the present study includes all age group, sexes, incidents occurring at home and work place.

Exclusion criteria are lightning cases and cases where electrocution is indirect cause of death for example a person after getting electrocuted falls from height and dies due to head injuries.

Results

A total number of 61 cases were received for autopsy in deaths due to electrocution in a period of two years. High voltage electrocutions were 33 in number. Low voltage electrocutions were 28 in number.

Among all the cases, typical electric contact mark was found in 35 cases, while flash burns were found in 29 cases. 9 cases showed both contact mark and flash burns. 6 cases showed neither electric contact mark nor flash burns.

All the cases reported were accidental in nature.

Flash burns were present in 78%, while contact mark were present in 30% of cases of high voltage accidents. Contact marks were present in 89%, while flash burns were present in 11% of cases of low voltage accidents.

Except for petechial hemorrhages over surface of heart, no specific gross internal finding of organs was observed at autopsy.

Following changes in skin were observed at gross examination of electric contact mark. Skin is yellowish or light yellow in colour, with the area hardened, pale area at the center, with margins everted surrounded by zone of erythema.

In all the cases with contact mark, Histo Pathological changes were seen in the sections of skin sent for examination.

Typical Histo Pathological changes were seen in 16 cases of sections of Heart sent for examination.

Non-specific Histo Pathological changes were seen in 21 cases of sections of Kidney sent for examination.

Histo Pathological changes in Skin

Sections from skin show breach or break in epidermis along with separation, with shrinkage of epithelial cells adjacent to breach. Nuclei of epithelial cells along the breach are pyknotic and oriented vertically – "Nuclear streaming".

Histo Pathological changes in Heart

Sections from heart show stretching of myocardial fibers along with elongation and wavy appearance of fibers at places.

Histo Pathological changes in Kidney

Sections from kidney show congested vessels and foci of hemorrhage.

Discussion

Number of cases being 61 in a span of 24 months is a

significant number compared to earlier studies, where 220 cases were reported in 22 years⁶ and 36 cases were reported in 3 years⁷ Electrical accidents involving high voltage form 54% and Low voltage form 46% of total electrocution.

Flash burns are present in 78%, while contact mark is present in 30% of cases of high voltage accidents. Contact marks are present in 89%, while flash burns are present in 11% of cases of low voltage accidents.

The above statistic indicates that high voltage electrocution invariably present as flash burns and low voltage electrocution present with contact mark in correlation with findings of Wright and Davis⁸, Vincent D Maio⁹.

Except for petechial hemorrhages over surface of heart, no specific gross internal finding of organs was observed at autopsy, which is in correlation with Bernard Knight's observation⁴.

Six cases of electrocution presented with neither contact mark nor flash burns. Death due to electrocution was confirmed by supportive Histo Pathological changes in heart and kidney. Histo Pathological changes were seen in the, entire skin specimen, which had contact mark.

Textbook description of vacuolation and honeycomb¹⁰ appearance in the layers of skin was not observed. Instead, breach or break in epidermis along with separation of epithelial cells adjacent to breach is seen with vertical orientation of nuclei, which is in correlation with findings of Tedeschi,² Bernard Knight⁴, Ludwig¹¹, Herbert Fischer³. In India voltage that is supplied for domestic purpose being 220v is higher when compared to that in western countries where it is 110v. This may be cause for separation of epithelial cells and absence of honeycomb appearance.

Histo Pathological finding in heart, which includes wavy appearance of myofibrils, is in correlation with the literature available⁴. Sections showed stretching of myocardial fibers along with elongation and wavy appearance in 47% of specimen sent. Histo Pathological finding in kidney is non-specific and suggests disseminated intravascular coagulation, present in 61% of cases, correlates with findings of Bernard Knight⁴.

In cases where contact marks were evident, Histo Pathological findings were used as supportive evidence for cause of death. In cases where no contact marks were found, positive Histo Pathological findings were used to give the cause of death as electrocution.

Causes of electrocution were investigated and found that human negligence was main cause of electrical accidents (41%), faulty electrical equipments and connections contributed to 31% of accidents and lack of protective measures caused 28% of electrocution, which agrees with study done at Armed Forces Institute of Patology¹².

Employees of electrical work even though provided with protective gloves, harness, belts were negligent of not using the same and they should ensure that main line should be devoid of current supply while they are working.

Domestic accidents are due to faulty electrical equipments or connections and human negligence. One fourth of domestic accidents occur in bathrooms involving boiler switches and immersion coils. Another important cause observed was contact with live wire while drying of wet cloths over metal wire (one fourth of total domestic cases) in correlation with Theodore Bernstein study¹³.

Houses constructed illegally with close vicinity to high-tension wire lead to electrical hazard with the dwellers. Short circuit with television cable wire has lead to electrical accidents to people who tried to connect the cable wire.

Following suggestions may be helpful in preventing electrical accidents:

- Ensure electrical appliances at home and workplaces are in proper working condition with effective non-leaking connections.
- All electrical wiring should have proper insulation.
- All electrical installation should be effectively earthed as per standard earthing practice.
- Houses or extension of houses should not be built underneath, overhead lines or in proximity of the lines.
- Balconies or windows of house should not be facing bare power lines. Power lines in such cases should be effectively insulated.
- Tying of wire to electrical pole for drying of cloths should not be allowed.
- Underground cables with in the house should not be laid.
- Over loading of switches should not be done.
- Open heating immersion coils should not be used.
- Electrical equipments or switches should not be operated with wet hands.
- Crisscrossing of television cable wire with live wire should not be done.
- Electrical employees should be given proper education regarding electrocution.
- Protective thick rubber gloves should be provided to the workers.
- Harness should be provided to the electrical employees working at heights.
- Helmets should also be provided.
- All electrical establishments should have Miniature circuit Breakers (MCB) to limit the load.
- Installation of Residential Current Circuit Breaker (RCCB) to avoid fatal shocks.

Conclusion

From the present study, following conclusions can be derived:

- ❖ Total number of 61 fatal electrocution cases was reported for autopsy in two years period.
- ❖ All cases were accidental in nature..
- ❖ High voltage electrocution formed 54 % of cases.
- ❖ Low voltage electrocution formed 46 % of cases.
- ❖ Most of High voltage electrocution present as flash burns.
- ❖ Most of Low voltage electrocution present with contact marks.
- ❖ Electric contact mark is pathognomonic of electrocution.
- ❖ Histo Pathological changes were used as supportive evidence in giving cause of death in presence of contact mark.

- ❖ Histo Pathological changes were used to give cause of death where no contact marks were found.
- ❖ Histo Pathological changes are present in all the specimen of skin with contact mark.
- ❖ Histo Pathological changes in Heart specimen were observed in 47 % of cases.
- ❖ Histo Pathological changes in kidney were nonspecific.
- ❖ Main cause of electrocution was determined to be human negligence followed by faulty electrical equipments, connections and finally lack of protective measures.
- ❖ Electrocution can be prevented by educating people about the equipments, precaution to be taken while working with electrical equipments, replacing old electrical installations with new one, use of protective measures like gloves and harness to avoid fatal electrocution.

References

1. J.B. Gupta, Basic electrical engineering, Third edition: 2002; pg325.
2. Endre Somogyi, C.G. Tedeschi, Injury by electrical force, 1977; 17: pg 645 750.
3. Herbert Fischer, James Kirk Patrick. A colour Atlas of Trauma Pathology, 1991:pg 72-74..
4. Bernard Knight, Forensic Pathology, Electrical fatalities, 1991; 12: pg. 302
5. Wright R.K, Death or injury caused by electrocution, Clinics of Laboratory Medicine, 1983; 3 (2): pg 343-353
6. John D. De Haan. B.S. A review of electrical Fire analysis 1987: pg513.
7. Electrocution deaths in North East Delhi, Journal of Indian Academy of Forensic Medicine. 1997; 19(1): pg 4-7
8. R.K.Wright; J.H.Davis. Investigation of electrical deaths, A report of 220 fatalities. Journal of Forensic Sciences; 1980; 25(3): pg. 514-521
9. Vincent J.M.Di Maio, Suzanna.E.Dana, Hand book of Forensic Pathology; 1998: pg199-208
10. Cyril John Polson. D.J. Gee. Bernard Knight. The Essentials of Forensic Medicine, 1985; 4: pg 271-315.
11. Jurgen Ludwig, Hand book of Autopsy practice, III Edition, pg 363.
12. Mellen P.F. Weedn V.W, Kao G. Electrocution: A review of 155 cases with emphasis on human factors. Journal of Forensic Sciences, 1992; 37; (4): pg. 1016-22
13. Theodore Bernstein, Effects of electricity and lighting on Man and Animals, Journal of Forensic Science, pg 3-11.

Effect of chlosite (xanthan gel with chlorhexidine) on clinical & microbiological parameters in smokers- A case series

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Abstract

Background & objectives

Prevention of the periodontal disease progression is the primary goal of periodontal therapy. When conventional therapy is found inadequate to attain periodontal health in Chronic Periodontitis, local antimicrobial agents have been used as an adjunct with scaling and root planing which have reproduced encouraging results. This case series evaluates the newly released sustained local drug Chlosite clinically & microbiologically in smokers.

Method

The patients were grouped into experimental group A treated with scaling and root planning plus Chlosite (SRP + CHL), experimental group B treated with Chlosite alone (CHL) and control group C treated only with scaling and root planing (SRP) alone. A total number of 74 sites from 3 chronic smoker patients participated in this study. The clinical parameters, Plaque index (PI), Gingival index (GI), Bleeding index (BI) and Relative attachment level (RAL), were recorded and microbiological samples taken before scaling and root planning.

Results

All the groups, showed statistically significant reduction in relation to Plaque index, Bleeding index & relative attachment level. Combination of scaling and root planning with chlosite resulted in added benefits as compared to the two treatment groups. All the groups showed reduction in the microbial count of fusobacterium nucleatum, Porphyromonas gingivalis and tannerella forsythesis.

Interpretation and conclusion: In this study all treatment groups were found to be efficacious in the treatment of periodontal disease as demonstrated by improvement in PI, GI, BI and RAL. Combination of SRP and Chlosite resulted in added benefits compared to the two treatment groups.

Key words

Chlosite, Chronic periodontitis, xanthan gel, Smokers.

Introduction

Traditionally periodontal disease therapy has been directed to altering the periodontal environment to one that is less conducive to the retention of bacterial plaque in the vicinity of gingival tissues. With the increasing awareness of the bacterial etiology of periodontal diseases^{1,2}, and in particular the hypothesis that specific bacteria are involved³, a more direct approach, using antibacterial agents has become an integral part of the therapeutic armamentarium. Recently a new sustained local drug delivery chlorhexidine with xanthan gel, Chlosite (1.5% of chlorhexidine in 0.5ml of xanthan gel) has been introduced. Therefore it was deemed important to evaluate the efficacy of this drug clinically and microbiologically in smokers.

The aim of our case series was to determine the effect of chlosite as a monotherapy, chlosite compared to scaling and root planning, chlosite with scaling and root planing (combination therapy) and to determine the efficacy of chlosite on periodontopathogens.

Inclusion & exclusion criterias

Patients who were diagnosed as suffering from chronic generalized periodontitis (AAP-1999). Patient had periodontal pocket measuring 5-7mm in different quadrants of the mouth on clinical examination and radiographic evidence of bone loss was seen. Patients who had not received any periodontal therapy for past 6 months⁴. Patients free from any systemic diseases. Patients who were excluded were pregnant women and nursing mothers, patients with known hypersensitivity to chlorhexidine and teeth with furcation involvement were excluded. (Fig. 1,2,3,4,5,6,7)

Clinical & microbiological parameters

Prior to scaling and root planing each selected site was subjected to assessment of the following clinical parameter. Plaque index (Silness and Loe, 1964)⁵, Bleeding index (Ainamo and Bay, 1975)⁶, Gingival index (Loe and Silness, 1963)⁷, Relative attachment level using UNC-15 periodontal probe, Sub-gingival microbiological plaque samples. The clinical parameters were assessed on day '0', 30th and 90th day. Relative attachment level was assessed only on '0' day and 90th day.

Subgingival microbiological samples

Subgingival microbial examination was performed at baseline and on 30th day. After removing supragingival plaque, two fine endodontic paper points were inserted to the depth of each periodontal pocket for 10 sec and then transferred to 1ml Thioglycollate broth (transport medium) and sealed tightly to avoid contamination. Samples were processed within 2 days of collection. Once it was received in the laboratory the sample was mixed thoroughly and 5 microliter each was inoculated using sterile loop onto the following mediums: Enriched blood agar (Porphyromonas gingivalis), Brewer's anaerobic agar (Fusobacterium nucleatum), Bacteroides bile esculin agar (Tannerella forsythesis)⁸.

Case 1

A 45 year old male patient visited the department of Periodontology and Implantology, complaining of pain in the right and left upper and lower back teeth region of jaw since 2 to 3 months. Medical history revealed no systemic problem and had not undergone any periodontal therapy in the past. He was a chronic smoker smoking 5 to 6 cigarettes per day since past 20 years.

Comprehensive periodontal examination revealed generalized periodontal pockets measuring between 5 to 7 mm and radiograph revealed generalized bone loss in relation to upper and lower arches, with deeper pockets present in relation to posterior teeth as compared to anterior teeth.



Plaque samples were collected from periodontal pockets of patients and transported in thioglycollate broth and sent to microbiology lab for evaluation. Clinical parameters were recorded as mentioned above. 25 sites were evaluated for microbiological evaluation of *Porphyromonas gingivalis*, *Fusobacterium nucleatum* and *Tannerella forsythensis*⁸. Out of these 25 sites, 10 sites were selected for only scaling and root planning group, 7 sites for scaling root planning with chlosite group and 8 sites for only chlosite group.

Case 2

A 60 year old male came to the department of Periodontology and Implantology with the chief complaint of sensitivity in relation to left upper back teeth region since 15 days. On examination, generalised periodontal pockets were present and the patient was smoking 6 to 7 cigarettes per day from past 12 to 15 years. His medical history revealed no systemic problem and had not undergone any periodontal therapy before.

Clinical and radiographic examination revealed generalized bone loss and generalized pockets ranging between 5 to 7 mm. All the clinical and microbiological parameters were recorded as in case-1 and the patient was recalled for examination on 1 month and 3 months respectively.

17 sites were evaluated for microbiological evaluation of *Porphyromonas gingivalis*, *Fusobacterium nucleatum* and *Tannerella forsythensis*⁸. Out of these 17 sites 6 sites were selected for only scaling and root planning group, 6 sites for scaling root planning with chlosite group and 5 sites for only chlosite group

Case 3

A 37 year old male complained of pain in the right upper and lower back teeth region since past 1 year. He was a chronic smoker smoking 9 to 10 cigarettes per day and his medical history revealed no systemic problem.

Periodontal examination revealed generalized periodontal pockets and radiograph revealed generalized horizontal bone loss. Clinical and radiographic examination revealed generalized bone loss and generalized pockets ranging between 5 to 7 mm. All the clinical and microbiological parameters were recorded as in case-1 and the patient was recalled for examination on 1 month and 3 months respectively

32 sites were evaluated for microbiological evaluation out of which 9 sites were selected for only scaling and root planning group, 11 sites for scaling and root planning with chlosite group and 12 sites for only chlosite group.

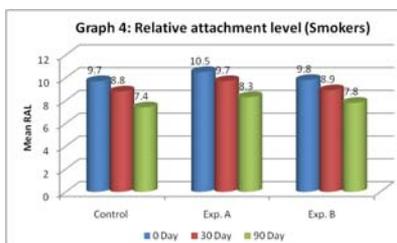
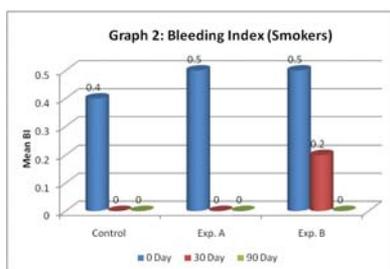
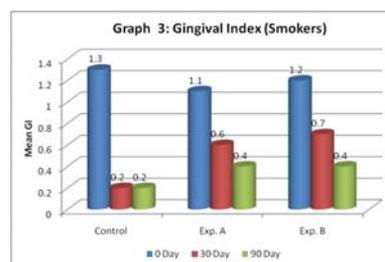
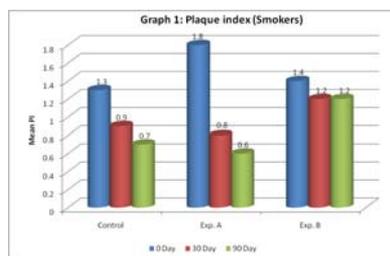
Results

All the groups, showed statistically significant reduction in relation to Plaque index, Bleeding index & relative attachment level. Combination of scaling and root planning with chlosite resulted in added benefits as compared to the two treatment groups. All the groups showed reduction in the microbial count of *fusobacterium nucleatum*, *Porphyromonas gingivalis* and *tannerella forsythensis*. (Graph 1,2,3,4,5,6,7)

Discussion

Chlorhexidine (CHX) is a widely used broad-spectrum antimicrobial agent to inhibit bacterial growth and, thus, an adjunctive mean to control oral hygiene in patients with periodontal disease^{9,10}. Attempts to prolong the subgingival application of chlorhexidine by incorporation of an antiseptic in a gel have not resulted in improved treatment outcomes¹¹. However with the use of Chlosite effective subgingival concentration of chlorhexidine can be maintained for several days. The physical properties of xanthan render it an optimum substrate for the formation of a stable gel that is easily extruded from a syringe needle; therefore xanthan appears to be the best biocompatible vehicle for clinical application¹².

The mean reduction in plaque score (plaque index) from '0' to 90th day was 46.2%, 66.7% and 14.3% in SRP, SRP + CHL and CHL respectively which was statistically highly significant in SRP and SRP + CHL and significant in relation to



CHL. The mean reduction in bleeding score (bleeding index) from '0' to 90th day was 100% in SRP, SRP + CHL and CHL, and it was statistically highly significant in all the above-mentioned three treatment modalities. The mean reduction in gingival score (gingival index) from '0' to 90th day was 84.5%, 63.6% and 66.7% which was highly significant for SRP and SRP + CHL, whereas significant for CHL. The mean gain in relative attachment level from '0' to 90th day was 23.7%, 21% and 20.4% for SRP, SRP + CHL and CHL respectively which was highly significant.

Prevalence of various microorganisms at different intervals of study period

Fusobacterium nucleatum showed 100% reduction which was statistically not significant. These findings are similar to that of Daneshmand et. al¹³. *Porphyromonas gingivalis* showed 100% reduction which was statistically not significant. *Tannerella forsythensis* showed 100% reduction which was statistically not significant.

Conclusion

In this case series all treatment groups were found to be efficacious in the treatment of periodontal disease as demonstrated by improvement in PI, GI, BI and RAL. Combination of SRP and Chlosite resulted in added benefits compared to the two treatment groups. Locally applied chlosite gel may partly counteract the negative effect of smoking on periodontal healing following non-surgical therapy.

References

1. Socransky SS. The relationship of bacteria of the etiology of periodontal disease. *J Dent Res* 1970;49(Suppl. 2):203-222.

2. Slots J. Subgingival microflora and periodontal disease. *J Clin Periodontol* 1979;6:351-382.
3. Loesche WJ, Syed SA, Schmidt E, Morrison EC. Bacterial profiles of subgingival plaques in periodontitis. *J Periodontol* 1985;56:447-456.
4. Daneshmand N, Jorgensen MG, Nowzari H, Morrison JL, Slots J. Initial effect of controlled release chlorhexidine on subgingival microorganisms. *J Periodont Res* 2002;37:375-379.
5. Silness J, Loe H. Periodontal diseases in pregnancy (2) correlation between oral hygiene and periodontal condition. *Acta Odontol Scand* 1964;24:747-759.
6. Ainoma J, Bay I. Problems and proposals for recording gingivitis and plaque. *Int Dent J* 1975;25:229-235.
7. Loe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontol Scand* 1963; 21(6): 533.
8. E.W. Koneman, S.D. Allen, W.N. Janda, P.C. Shreeckenberger and W.C. Winn. *Color atlas and textbook of diagnostic microbiology*. 5th Ed. 1997: 709-775.
9. Briner WW, Grossman E, Buckner RY et al. Effect of chlorhexidine gluconate mouthrinse on plaque bacteria. *J Periodont Res* 1986;21:44-52.
10. Banting D, Bosma M, Bollmer B. clinical effectiveness of a 0.12% chlorhexidine mouthrinse over two years. *J Dent Res* 1989;68:1716-1718.
11. Oosterwaal PJM. Comparison of the antimicrobial effect of the application of chlorhexidine gel, amino fluoride gel in debrided periodontal pockets. *Journal of clinical periodontology* 1991;18,245-251.
12. Needleman IG, Smales FC, Martin GP : An investigation of bioadhesion for periodontal and mucosal drug delivery. *J clin periodontal* 1997;24:394-400.
13. Daneshmand N, Jorgensen MG, Nowzari H, Morrison JL, Slots J. Initial effect of controlled release chlorhexidine on subgingival microorganisms. *J Periodont Res* 2002;37:375-379.

Guidelines for collecting forensic samples in casualty

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Abstract

The Casualty Department is the gateway to a medicolegal environment for both victims and perpetrators of violence. It is essential for certain personnel in the department to have the basic skills to gather evidence in a proper manner consistent with recommended standards of forensic science, and without overriding existing hospital policy. Local and state guidelines for collection and preservation of physical evidence must be kept in mind by those involved in this activity. Legal problems in this regard can be avoided if the Casualty Department is following established hospital policies and procedures based on the recommendations of a qualified Forensic Scientist or Expert.

Key words

Casualty Department, Forensic Science, guidelines, physical evidence

Introduction

"Forensics is the silent witness.....it does not speak for or against the victim. It merely speaks the truth." – Jeff Miller, Forensic Expert, Fairfax, Virginia.

Locard's Principle of Exchange states that whenever there is contact between two objects, there is a mutual exchange of material between these two surfaces. This "theory of evidence exchange" provides the basis for linkage between the victim, the perpetrator, and the scene of crime.

Forensic science usually begins at the crime scene, but when victims of trauma are quickly transported to the nearest hospital for treatment, the Casualty Department presents the first opportunity to collect physical evidence. This highlights the need to educate relevant Casualty staff, such as residents and nurses, in the scientific methods of preserving forensic evidence with a view to serving the cause of justice.

Physical evidence

Any matter, material or condition, large or small, solid, liquid or gas, which may be used to determine facts in a given situation, e.g. clothing, hair, nails, bullets, wounds, etc. The evidence has the capacity to link a victim to a suspect, suspect

to a crime scene, identify a perpetrator, establish an element of crime or corroborate an alibi. Casualty Department personnel should understand the legal procedures required in handling physical evidence, the types of physical evidence and the value of that evidence. They should be conversant with the proper methods for collecting, documenting and preserving forensic evidence.

Forensic cases in the casualty Department

Forensic cases are those that potentially involve criminal or civil liability. A medicolegal case is defined as a treatment situation with legal implications.

Methods of preserving physical evidence

1. Clothing

- It is a universal precaution for the person collecting evidence to wear gloves before examination of the patient
- Allow the patient to undress if he/she is able to do so. Place each item of clothing in a separate paper to prevent breakdown of the integrity of biological evidence. Seal, label and secure the package. Sign on the label and seal.
- Wet or damp clothing should be air-dried thoroughly in a secured area and then packed into paper bags labeled appropriately. Never place clothing in plastic bags as it could lead to accumulation of moisture and destruction of possible evidentiary material.
- Try not to fold, as far as possible. Pieces of paper may be placed between materials that must be folded against each other to prevent cross-contamination
- Do not dispose any items collected from the patient as they may have evidentiary value.
- In unconscious, severely ill or injured patients, the forensic examiner must remove clothing meticulously to preserve the integrity of the garment. Cut along seams and away from the injured area to reduce interference with physical evidence.
- The Forensic Analyst treats all objects placed in the same container as one evidentiary item. Hence, each item of clothing or physical evidence must be placed in its own separate container to maintain its forensic identity.

2. Bullets and Missiles

- During the treatment of a gunshot wound victim, bullets, gunshot residue, projectiles or fragments may be present on the patient's skin or clothing
- Remove from the body or clothing with rubber-tipped forceps to avoid damage, alteration or destruction
- Small items can be placed in rigid plastic containers with a few holes punched in the lids for ventilation. Pad with cotton or soft material within the container. Seal and label and sign on both. Place the container in a paper bag. Seal and label the bag and sign them.
- Large items are placed in appropriately-sized cardboard boxes padded with cotton or soft material. They should be handled with care. Seal, label and sign both.

3. Body fluids

- Blood Alcohol Estimation - Clean the venepuncture site with Povidone Iodine and saline. Do not use Alcohol for

Forensic cases in casualty

- Accident, due to Road Traffic or a Fall
- Victims of violent crime, assault, etc.
- Poisoning, snakebite and drug overdose
- Injuries due to firearms or other weapons
- Attempted suicide or homicide
- Occupational Injuries
- Domestic violence, child abuse and neglect
- Cases of criminal abortion
- Cases of drunkenness
- Anyone in police custody for any reason
- Sudden, unexpected and suspicious death
- Cases "brought dead" to Casualty
- Sexual offences

cleaning. A written request from the police is a must. Informed consent should be taken from the subject before obtaining blood for alcohol analysis. The blood can be stored in tubes, vials or screw-capped bottles with NaF as preservative and K-oxalate as anticoagulant. The container is labeled, sealed, signed and handed over to the Investigating Officer along with a requisition for analysis by the Forensic Science Laboratory and sample seals. Chain of custody is maintained in such cases.

- Urine – About 20-30 ml of urine is collected in a clean plastic container half an hour after the subject is made to empty his bladder. No preservative is required if the sample is sent to the laboratory immediately, otherwise 50 mg of Sodium Fluoride per 50 ml of urine may be used.
- Gastric contents – This can be stored in clean plastic containers such as empty saline bottles, sealed, labeled and signed. The first gastric aspirate should be preserved as it is most likely to yield a positive result.
- Vomitus- A sample may be sent to the laboratory in a clean glass jar or plastic tub with a tight-fitting lid.

4. The Body as evidence

- When a person is "brought dead" to Casualty, the body itself becomes the evidence.
- Casualty Department personnel should be instructed not to cut lines or remove tubes prior to releasing the body
- The hands of the deceased may be enclosed in small paper bags secured at the wrist with tape to preserve evidence such as gunshot residue, hair, fragments of skin and other trace evidence.
- Send all collected specimens and clothing with the body

5. Collection of evidence from a victim of sexual assault

- Pubic hair combings – Place a clean paper or towel below the buttocks and comb the area for loose or foreign hair
- Pubic hair control samples – Clip hair close to the skin line. Two dozen full-length hairs are sampled from this area
- Genital and thigh swabs – Two lightly moistened applicators are used for swabbing the genital and thigh areas. The swabs are air-dried for 5-10 minutes before placing in a swab box or test tube with holes for air circulation. Swab boxes can be placed in a paper bag or envelop for transfer to the laboratory.
- Vaginal swab and smear – Swabs are made with two moistened applicators, air-dried and packaged. Two other swabs are made and smeared onto separate microscopic slides, allowing them to air-dry before packaging in a swab box. Swabs must never be packaged while moist.
- Rectal swabs and smear when relevant
- Oral swabs and smear when there is oro-genital contact
- About 50 head hair controls are clipped at the skin line
- Fingernail scrapings are gathered by scraping the undersurface of the nails with dull object over a piece of clean paper to collect debris. A separate paper should be used for each hand.

6. Collection of evidence from a suspect in sexual assault

- Clothing, believed to have been worn at the time of the assault
 - Pubic hair combings
 - Pulled head and pubic hair controls
 - Blood/Buccal swab sample
 - Penile swab (within 24 hours of the assault)

7. Collection of evidence for DNA Profiling

- Blood samples can be collected in sterile vacuum tubes containing EDTA, which inhibits enzyme activity that degrades DNA. This sample can be used for both DNA Profiling and toxicological analysis, where necessary.
- DNA Evidence by buccal swab can be done by making two buccal swabs using applicators lightly moistened with

distilled water. The inside of the cheek and gum line are swabbed vigorously to transfer buccal cells to the swab.

8. Evidence from cases of poisoning or drug overdose

- Body fluids such as blood, urine, gastric aspirate and vomitus
- Head hair in cases of heavy metal poisoning may be cut or pulled at the roots and packed in a clean folded paper or envelop.
- Faeces can be collected in suspected heavy metal poisoning. A sample of 20-30 gm is placed in a plain screw-capped jar or plastic container with a lid.
- Nail clippings in poisoning with Antimony, Arsenic or thallium

Chain of custody

- It is a link established between each person who handles a piece of evidence
- Transfer of evidence from one person to another must be accompanied with written documentation
- The chain of custody forms a trail which records where the evidence was from the time it was collected to the time it is produced in Court
- A minimum record shows the collector's signature, location of evidence and date of collection
- Transfer must be recorded in notes or forms
- The chain of custody should be kept to a minimum to avoid confusion
- In a clinical setting, the evidence may be secured by keeping in a locked cabinet with a key maintained by a responsible nurse on duty.

Documentation of evidence

- Proper documentation of evidence forms the initial process of evidence collection
- It should be done as soon as possible after the patient's arrival in Casualty
- If feasible, take photographs of injuries prior to treatment
- The doctor's chief concern is for the health and well-being of the patient
- No forensic protocol should stand in the way of patient care
- The patient's informed consent is obtained before photographs are taken

Problems in collecting evidence

- Casualty Department personnel are often not aware of the presence and potential value of physical evidence
- Failure to recognize and preserve fragile and perishable evidence
- Lack of forensic education on the part of the Casualty staff

Summary

The Casualty Department is not always controlled and organized. It is a fact that lifesaving treatment and interventions for the patient do take precedence over forensic collection of evidence. A properly documented chain of custody should be maintained while collecting evidence to render it valid and admissible in a Court of Law. Relevant Casualty Department personnel, like residents and nurses, can become active participants in the evidence collection process.

References

1. Fulton DR, Assid P- Evidence Collection in the Emergency Department. In: Lynch VA- Forensic Nursing. St.Louis,

- Missouri: Elsevier Mosby, 2006: 570-577
2. Knight B- General Aspects of Poisoning. In: Knight B-Simpson's Forensic Medicine. 11th edition. London: Hodder Arnold Publishers, 1997: 174-175
 3. Cavelus NB, Spangler K- Evidence Collection and Documentation. In: Hammer RM, Moynihan B, Pagliaro EM- Forensic Nursing. Sudbury, Massachusetts: Jones and Bartlett Publishers, 2006: 490-518
 4. Mestri SC- Manual of Forensic Medicine for doctors, lawyers, police officers and nurses. 2nd edition. New Delhi: Jaypee Medical Publishers, 2003
 5. Nandy A- Identification from Trace Substances and their other Evidential Values. In: Nandy A- Principles of Forensic Medicine. 2nd edition. Calcutta: New Central Book Agency Pvt Ltd., 2000: 112-132
 6. Sturt PA- Medicolegal Evidence. In: Fultz J, Sturt PA- Mosby's Emergency Nursing Reference. 3rd edition. Missouri: Elsevier Mosby Inc., 2005: 865-868
 7. Holleran RS- Preservation of Evidence. In: Proehl JA- Emergency Nursing Procedures. 3rd edition. Missouri: Elsevier Saunders, 2004: 816-820
 8. Meghal SK, Mokashi RH- Forensic Evidence of Biological Fluids, Stains and other Materials. In: Parikh CK- Textbook of Medical Jurisprudence and Toxicology. 5th edition. Bombay: Medicolegal Centre, 1990: 606-658
 9. Dogra TD, Gupta S- Emergency Medical Services – Legal and Ethical Issues. In: Dave PK, Gupta S, Parmar NK, Kant S- Emergency Medical Services and Disaster Management: A Holistic Approach. 1st edition. New Delhi: Jaypee Brothers Medical Publishers, 2001: 180-203
 10. Reddy KSN- Forensic Science Laboratory. In: Reddy KSN- The Essentials of Forensic Medicine and Toxicology. 21st edition. Hyderabad: K. Suguna Devi, 2002: 420-424

Medico-legal and socio-demographic profile of rape cases in district hospital, Adilabad (Andhra Pradesh)

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Abstract

Rape is a weapon that distorts a woman's sexuality, restricts her freedom of movement and violates her human rights. A rapist not only violates the victim's privacy and personal integrity, but also causes serious physical and psychological damage. Total 93 rape cases admitted to district hospital, Adilabad were studied retrospectively over the period of five years from October 2004 to September 2009. 78.5% of the victims were found to be in 11-20 years age group. Majority of the victims (88.17%) were unmarried. Most of them (92.5%) were Hindu by religion. 70% of perpetrators were known to the victims. 14% of the victims were kidnapped and 4.3% cases were raped under threat of great bodily harm or death. Twenty (21.5%) victims became pregnant after the act.

Keywords

Rape, victim, age, relation, kidnapped

Introduction

"Rape is the vilest form of violence against a woman. Perhaps, the only one where the victim, the woman, has to carry the stigma of the society for the rest of her life in spite of the fact that she is not a consenting party to it"¹.

The victim faces degradation and social unacceptability. "It is a deathless shame or living with death"². Rapist does not only victimise her, but it also leaves a lifelong stigma on the character and dignity of a woman, causing her and her relatives, pain and agony. The mental torture is so deep that it hardly heals and if it heals at all, it takes a very long time to heal.

Violence on women is an extension of 'patriarchy', which means 'male rule'. The two main features of patriarchy are sexual power and supremacy. Rape is not like murder to rapist, though in actuality he murders the life of a woman. In the case of an unmarried woman, the stigma acts as a hurdle for a married life and she is looked as an outcaste. The married woman loses the love of her husband and her restoration in the family is jeopardised. The family members never show a positive approach to her. Even children lose the trust and security they reposed in her. The victims are destined to lead a painful life, irrespective of their age and circumstances³.

Some rape hits headlines, a few are reported but most remain covered up, hidden in the deep recesses of the scared minds of the victims. Women preferred to suffer the crimes rather than setting the law in motion⁴.

In this context, it is important to understand the magnitude and the reality of the rape cases and to propose measures not only to alleviate the agony of the victims but also to modify systems to support the victims and to prevent rape incidents. It is also important to strengthen the existing structures like police, judiciary, and health care in dealing with such a sensitive social issue.

Materials and methods

The present study was carried out in District Hospital, Adilabad which is a tribal area in Andhra Pradesh. The study

was conducted over the period of five years from October 2004 to September 2009. A total of 93 case records of admitted rape cases were retrieved retrospectively from the Medical Record Section of the hospital and the data was analyzed.

Results

Among 93 cases of rape studied maximum number of victim i.e., 73 (78.5 %) belonged to 11-20 years of age while number of pediatric victims (0-10 years) were 3 (3.23 %). Table 1 Majority of victims (88.17%) were unmarried and 2 cases were widow. Religion wise, Hindus were found to be most affected having 86 cases (92.5%) and 21.5% victims were from Schedule Caste/Schedule Tribe category. Regarding the residence of victim, 80 cases (86.02%) were from rural background and 13 cases (13.98%) were from urban setting. Table 2

In 65 cases (70%) the offender was previously known to the victim. In one case the father of the victim girl was the offender and in one case Police Constable was the offender. Table 3

In our study, 14% of victims were kidnapped by accused and in 4(4.3%) cases victims were threaten by accused of dire consequences if they reported the case to police or informed their relative.

Twenty victims (21.5%) became pregnant after the act, out of these 3 victims had given birth where two children died after birth. Septic abortions were done in 2 cases out of twenty pregnant victims. 15 victims (16.93%) were given false promises of marriage which is one of the causes of their continuation of pregnancy.

An overwhelming majority (75%) of the cases presented late for the examination i.e., 24 hours after rape and revealed only old tear of hymen on examination. Intact hymen was noted only in 7 cases.

In 90 cases (96.77%) the perpetrator of rape was alone, but in 3 cases two or more than two persons were involved in the act. In 60 cases (64.52%) only one sexual act was done where as in 33 cases (35.48%) multiple sexual acts were observed.

One of the victims made unsuccessful suicidal attempt by consuming pieces of glass and one victim was found to be lunatic.

Discussion

The fact that the growing tendency of the deviant and defiant behaviour of man in society is manifested as aggressive forms, irrespective of the age of the woman, is clear from the distribution of the rape victims by age groups (Table 1). In our study 78.5% victims were from 11-20 years age group. The more involvement of adolescent age group can be explained by exploitation of the younger girls by the opposite sex coupled with inquisitiveness, less maturity and less resistance on the part of the victim. In similar study, Grover Namita et al⁵ also found 62% victims were in adolescent age group.

Majority of victims (91.4%) were unmarried denotes that how girl children are oppressed right from their birth both inside and outside their families. Same observation was noticed by Roy Chowdhury et al⁶ in their study.

Table 1: Age-wise distribution of victims

Age in years	No. of cases	Percentage
0 to 5	2	2.15
5 to 10	1	1.08
10 to 15	36	38.71
16 to 20	37	39.78
21 to 25	11	11.83
26 and above	6	6.45
Total	93	100.00

Table 2: Socio-demographic profile of victims

	Number of cases	Percentages
Religion		
Hindu	86	92.47
Muslim	7	7.53
Category		
Schedule Caste	4	4.30
Schedule Tribe	16	17.20
Other	73	78.50
Marital Status		
Married	9	9.68
Unmarried	82	88.17
Divorced/Widowed	2	2.15
Residence		
Village	80	86.02
City	13	13.98

Religion wise Hindu outnumbered Muslims. This can be explained by the predominating population of Hindu in study area. As this study was conducted in tribal District of Andhra Pradesh and Schedule Tribe constitutes more than 17% of total district population⁷, we found that 17.2% victims were from Schedule Tribe category.

Number of rural victims (86%) dominated those from urban background and this can be explained by that rural population is more in study area⁷ and most of the cases were brought from surrounding villages.

70% of the offenders are previously known to the victims. As per prevalent Indian traditions and culture a girl or women usually does not go out alone with unknown person or entertain them when they are alone at home. In such acquaintance rape, victims may not feel fear, but also feel that someone she trusted betrayed her. In one case the father of the victim girl was the offender. Though society, culture and law detest sexual intercourse between close blood relations within prohibited degrees of relationship, it is disconcerting to note that incest rape has become a common phenomenon. According to Crime in India 2007⁸, there were total 20,737 reported Rape cases in the country. Offenders were known to the victims in as many as 19,188 (92.5%) cases. Parents/close family members were involved in 2.1% of these cases, neighbours were involved in 36.0% cases and relatives were involved in 7.5% cases.

In our study, 14% of the victims were kidnapped by accused and in 4 cases victims were threaten by accused of dire consequences if they reported the case to police or informed their parents. This shows that rape is not so much about sex; it is about other issue such as power and aggression.

In this study we found that 21.5% victims became pregnant after the act, out of which 3 victims had given birth to child and septic abortions were done in two cases. This higher percentage of pregnancy can be explained by the fact that in 33% cases multiple sexual acts were done even for months and about 15% victims were given false promises of marriage. This shows that women became victims of a carefully planned seduction and sexual exploitation. Some people revel in exploiting the innocence of tribal women by their empty promises of a few bangles or of marriage. This is near to the

Table 3: Details of relationship of an offender with victim

Offenders	No. of cases	Percentage
Parent/close family member	1	1.08
Relatives	4	4.30
Neighbours	2	2.15
Friends	2	2.15
Other known persons	56	60.22
Strangers	28	30.10
Total	93	100.00

study by Sukul Biswajit et al⁹ who reported that 16.09% victims became pregnant after the act.

In 96.77% case the perpetrator of rape was alone but in 3 cases two or more than two persons were involved in the act. Rahul Jain et al¹⁰ reported in his study that in 75% of cases the offender was alone but in 13% cases three or more than three persons were involved.

In the present study, 75% of the cases presented 24 hours after rape for medical examination. Medical evidence is most important in a rape case. Once the case is reported in the police station, the victims are immediately sent for medical examination. But women fear that crimes against women, if reported, may get reported by the print and the audio-visual media resulting in undue publicity. They prefer to 'suffer in silence' rather than 'publicise the incident'.

In our study one of the victim made suicidal attempt by consuming pieces of glass. For fear of blame, rape victims often remain reticent and withdrawn. Many a time, they attempt to commit suicide out of grief and self-contempt.

Conclusion

While a murderer destroys the physical frame of the victim, a rapist degrades and defiles the soul of a helpless female. Rape leaves a permanent scar on the mind and body of the victim. Rape victims, looked down upon by society, lead the rest of their lives in ignominy in most of the cases, unmarried and desolate. The society shuns contacts and interactions with the family. Majority of rape victims were in the age group of 11 to 20 years. Most of the victims were unmarried and from rural areas. The biggest threat came not from strangers but from known persons, near relations, friends, and neighbours. Pregnancy was seen in victims who were subjected to multiple sexual assaults. In Three-fourth cases, victim was brought for medical examination 24 hours after rape.

Systematic efforts should be made to organise large-scale awareness programmes and campaigns in order to disseminate gender-just values that should counter the ideology of patriarchy and conservative thinking about sex and sexuality. Delay in getting medico legal care in rape cases is common and all efforts must be made to reach comprehensive services to them. There is sufficient evidence to show that early and good quality documentation of evidence is associated with positive legal outcome. Special courts may be set up for speedy trial of rape cases. Such courts should be constituted of judges, lawyers, doctors, psychologists, and representatives of NGOs. Rape victims are in dire need of support by Govt. and NGO s in the form of counselling and rehabilitative measure.

References

1. R. L. Gupta. Medico legal Aspects of Sexual Offenses. Eastern Book Company, Lucknow; 1991
2. Sharma O.C. Crime against Women: Ashish Publishing House, New Delhi; 1994
3. Usha Venkitakrishnan, Sunil George Kurien. Rape Victims in Kerala: Kerala Research Programme on Local Level Development, Centre for Development Studies,

- Thiruvananthapuram; 2003,9-12
4. Krishan Vij. Textbook of Forensic Medicine and Toxicology, 4th edition, Elsevier, New Delhi, 2008, 402
 5. Grover Namita, Suneja Amita, Vaid N.B., Pandit Upasana. Female sexual assault: A study from east Delhi. Indian Internet Journal of Forensic Medicine & Toxicology. 2008, Vol 6(4). Available from URL: <http://www.indianjournals.com/ijor.aspx?target=ijor:iijfmt&volume=6&issue=4&article=001>
 6. U.B. Roy Chowdhury, T.K Bose and R. Prasad. Rape: Its medicolegal and social aspect. Journal of the Indian Academy of Forensic Medicine. 2008 April-June, 30(2), 69-71.
 7. <http://adilabad.nic.in/population.html>
 8. National Crime Records Bureau, Ministry of Home Affairs, Government of India. Crime in India – 2007. Available from URL : <http://ncrb.nic.in/cii2007/cii-2007/CHAP5.pdf>
 9. Sukul Biswajit, Chattopadhyay Saurabh, Bose T.K. A study of victims of natural sexual offence in the Bankura district of West Bengal. Journal of the Indian Academy of Forensic Medicine. 2009, 31(1). Available from URL: <http://www.jiafm.com/jiafm.aspx?target=ijor:jiafm&volume=31&issue=1&article=005>
 10. Rahul Jain, P N. Mathur, N.S. Kothari, Phulvanti Mathur. Medicolegal Evaluation of Sex Assault Cases Admitted at Sardar Patel Medical College & P.B.M. Hospital, Bikaner, India. Medico-Legal Update. 2008, Vol. 8, No. 1. Available from URL: <http://www.indmedica.com/journals.php?journalid=9&issueid=119&articleid=1591&action=article>

Prophylactic and antidotal effects of L-carnitine administration against valproic acid-induced hepatotoxicity in high risk children - A clinical study

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Abstract

Objective

To study the possible antidotal and protective effects of L-carnitine in acute and chronic valproic acid-induced hepatotoxicity, respectively, in high risk children (age under 24 months)

Material and methods

The current study was divided into 2 parts: Part I (Acute study): 47 children, aging between 9 months to 24 months of both sexes, were divided into 2 groups as follows: Group A: 20 normal healthy children, Group B: 27 children presented with acute VPA intoxication who were received at El-Minia poisoning control centre during the period from the 1st of January 2005 to the 31st of December 2009 and treated with L-carnitine administration at a dose of 600 mg/kg/day L-carnitine intravenously. Part II (Chronic study): 131, children aging between 9 months to 24 months of both sexes, who were received at the outpatient clinic of pediatric diseases in Suzan Mubarak university hospital of Obstetrics, Gynecology and Pediatrics during the period from the 1st of July 2007 to the 30th of June 2009. They were divided into 4 groups as follows: Group I: 20 normal healthy children, Group II: 54 children recently diagnosed as epileptics and were not started any medications yet, Group III: 57 epileptic children treated with VPA for 6 months or more and Group IV: the same 54 children who were diagnosed as epileptics after 6 months of treatment with VPA with L-carnitine supplementation at a dose of 100 mg/kg/day, up to a maximum of 2 g/day. ALT, AST and serum VPA levels were measured in all children underwent the current study.

Results

Results of the acute study revealed that on admission, there was a significant increase of the all measured parameters when compared to the control group and that these parameters were still rising during the 1st 24 hours despite of the starting of L-carnitine administration. Serum transaminases and VPA levels showed a significant decrease after 48h and, more or less, return to the normal ranges after 72h. Results of the chronic study revealed that epilepsy per se has no significant alterative effects on the liver transferases. In the group III, it has been found that the serum VPA level of those patients (87.23 + 7.69 ug/ml) was within the permitted therapeutic serum concentrations range and liver transaminases were significantly increased when compared to those groups I and II. On addition of L-carnitine, a significant decrease of the serum VPA and liver transaminases was reported with no statistically significant difference when compared to those of either the groups I and II.

Conclusion

Our results study suggest that L-carnitine may be a potential antidote for VPA toxicity and a safe prophylactic medical supplementation during VPA therapy in high-risk pediatric patients. It is advised to carry out further controlled,

randomized, and multicenter trials to better delineate the therapeutic and prophylactic roles of L-carnitine and the optimal regimen of administration in the management of VPA toxicity either acute or chronic.

Keywords

L-Carnitine, Valproic Acid, antiepileptic drug, ALT, AST Hepatotoxicity.

Introduction

Drug-induced liver injury associated with antiepileptic drugs (AED) is well recognized⁴. Valproic acid (VPA, 3 2-n-propyl-pentanoic acid) is a broad-spectrum antiepileptic drug (AED) that has been used for more than 30 years and is effective in the treatment of many different types of partial and generalized epileptic seizure. It is also prescribed to treat bipolar and schizoaffective disorders, social phobias and neuropathic pain, as well as for prophylaxis or treatment of migraine headache¹³. However, it shows important potential adverse effects including hepatotoxicity²⁷.

It is reported that about 44% of long-term treatment with VPA may be associated with significant elevation in transaminases during the first months of therapy⁶. Usually, it resolves completely when the drug is discontinued. Severe valporate-induced hepatotoxicity (VHT) in association with hepatic failure is rare, but it may develop as an idiosyncratic reaction that is often fatal. It usually occurs during the first 6 months of VPA therapy and is commonly but not always preceded by minor elevations in transaminases¹.

VPA-induced hepatotoxicity has been suggested to be a consequence of carnitine deficiency²³ and also to be an oxidative effect. Furthermore, depletion of glutathione has been observed in animals treated with VPA^{15,24}. Risk factors include age under 24 months². Although the overall incidence is estimated at 1/5000 to 1/50,000, the occurrence of fatal hepatotoxicity could be as high as 1/800 to 1/500 in this high-risk group¹⁹.

As it does not appear to be harmful, L-carnitine is commonly recommended in severe VPA poisoning, especially in children, although the clinical benefit in terms of liver protection has not been established clearly. Prophylactic carnitine supplementation is also advocated during VPA therapy in high-risk pediatric patients¹⁶.

As acute VPA intoxication occurs as a consequence of intentional or accidental overdose and its incidence is increasing²⁸, and epilepsy affecting 13.6/1000 individuals in Upper Egypt, most of them are children who need AEDs-therap [22], it is necessary to lessen the occurrence of such serious VHT. Accordingly, the present study is carried out to evaluate the effect of L-carnitine administration in valproic acid-induced hepatotoxicity in high risk children (age under 24 months).

Subjects and methods

Subjects

The current study was carried out on 158 children aging between 9 months to 24 months of both sexes

Table 1: Liver enzymes and serum VPA levels of the different investigated groups of the acute study (Mean \pm SD).

Group	Parameter						
	AST (mg/dl)		ALT (mg/dl)		VPA (ig/ml)		
	Mean	SD	Mean	SD	Mean	SD	
A	24.63	2.37	25.76	1.89	—	—	
B	B ₁ (On admission)	87.26	3.47	98.34	2.11	332.54	13.22
	B ₂ (24 h)	112.34	4.56	123.61	3.47	523.47	18.26
	B ₃ (48 h)	52.37	2.16	53.56	2.43	223.61	8.59
	B ₄ (72 h)	32.12	1.12	34.37	1.64	118.43	4.77

ALT: Alanine transaminase, AST: Aspartate transaminase, SD: Standard deviation.

Study design

The current study was divided into 2 parts:

Part I: Acute study: aiming at studying the antidotal effect of L-carnitine administration in acute valproic acid-induced hepato-toxicity; 47 children aging between 9 months to 24 months of both sexes who were received at the poisoning control centre of El-Minia University hospital during the period from the 1st of January 2005 to the 31st of December 2009. They were divided into 2 groups as follows:

Group A: consists of 20 normal healthy children.

Group B: consists of 27 children presented with acute VPA intoxication that were received at El-Minia Poisoning control centre and were treated with intravenous L-carnitine administration at a dose of 600 mg/kg/day L-carnitine¹⁴.

Part II: Chronic study: aiming at studying the protective effect of L-carnitine supplementation in chronic valproic acid-induced hepatotoxicity; 131 children aging between 9 months to 24 months of both sexes who were received at the outpatient clinic of pediatric diseases in Suzan Mubarak university hospital of pediatrics during the period from the 1st of July 2007 to the 30th of June 2009. They were divided into 4 groups as follows:

Group I: consists of 20 normal healthy children.

Group II: consists of 54 children recently diagnosed as epileptics and were not started any medications yet.

Group III: consists 57 epileptic children treated with VPA for 6 months or more.

Group IV: consists of the same 54 children whom were diagnosed as epileptics after 6 months of treatment with VPA with oral L-carnitine supplementation at a dose of 100 mg/kg/day, up to a maximum of 2 g/day¹⁰.

Investigations

All children underwent the following investigations:

1. Liver enzymes: Alanine transferase and Aspartate transferase (ALT and AST): were measured spectrophotometrically using Spekol II Carl-Zeiss spectrophotometer.
2. Serum VPA level: It was measured in El-Minia poisoning control centre laboratory using an automated computerized analyzer (EXPRESS PLUS, CHRION-DIAGNOSTICS, USA).

Statistical analysis

Data were checked, coded, entered and analyzed using SPSS (version 17.0 software). Results were expressed as Mean + S.D. and percentage (%). Comparisons were done using the unpaired Student's t-test with statistical significance assured at $p < 0.05$.

Results

Part I (Acute study): On admission, it was found that the all measured parameters (ALT, AST and VPA levels) increased significantly on admission when compared to the control group. Moreover, it has been noticed that these parameters were still rising during the 1st 24 hours despite of the starting of L-carnitine administration and the significant decrease in VPA serum level. Serum transaminases and VPA levels showed a significant decrease after 48h and, more or less, return to the normal ranges after 72 h (Table 1-2 & Fig. 1).

Part II (Chronic study): Results of the newly diagnosed patients as epileptics and had not received any medications yet revealed that epilepsy per se has no significant alterative effects on the liver transaminases. In the chronically treated patients with VPA alone, it has been found that the serum VPA level of those patients (87.23 + 7.69 ug/ml) was within the permitted therapeutic serum concentrations range (50 to 125 ug/ml). It was reported that the liver transaminases were significantly increased when compared to those of either control group or the newly diagnosed patients as epileptics and had not received any medications yet. On addition of L-carnitine, a significant decrease of the serum VPA and liver transaminases was reported

Table 2: Comparison of the liver enzymes and serum VPA levels of the different investigated groups of the acute study (Student t-test).

Group	Parameter					
	AST		ALT		Serum VPA	
	t-value	P-value	t-value	P-value	t-value	P-value
B ₁ (On admission)	2.41	0.037*	2.12	0.032*	1.914	0.021*
	5.657	0.000***	5.372	0.000***	3.542	0.004**
	2.36	0.042*	2.26	0.041*	1.325	0.029*
	1.593	0.133	1.175	0.259	1.958	0.071

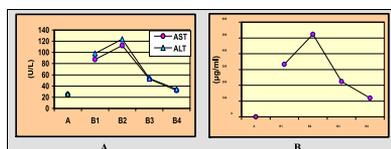
ALT: Alanine transaminase, AST: Aspartate transaminase, *: significant when compared to control

Table 3: Liver enzymes and serum VPA levels (Mean \pm SD) and comparison between the different investigated groups of the chronic study.

Group	Parameter								
	AST (U/L)			ALT (U/L)			VPA (ig/ml)		
	Mean	SD	P	Mean	SD	P	Mean	SD	P
I	24.63	2.37	—	25.76	1.89	—	—	—	—
II	24.58	2.44	0.24	26.13	1.93	—	—	—	—
III	36.12	2.58	0.023*	38.52	2.18	0.031*	87.23	7.69	—
IV	25.37	1.83	0.34	27.41	2.21	0.41	72.58	6.27	—
	0.019†	—	—	0.012†	—	—	0.023†	—	—

ALT: Alanine transaminase, AST: Aspartate transaminase, *: significant when compared to control

Fig. 1: Liver enzymes [A] and serum VPA levels [B] of the different investigated groups of the acute study (Mean \pm SD).



with no statistically significant difference when compared to those of either the control group or the recently diagnosed patients as epileptics and had not received any medications yet (Table 3 & Fig. 2).

Discussion

The results of the current study revealed that in the acutely intoxicated patients, the liver transaminases were significantly high on admission when compared to the control group. Moreover, it has been noticed that these parameters were still rising during the 1st 24 hours despite of the starting of L-carnitine administration and the significant decrease in VPA serum level. This finding indicates the time-dependent hepatotoxic effect of VPA, which is in agreement with which stated that the alteration of liver enzymes take time to be evident²¹. The following results of the serum transaminases reported significant decrease of their levels after 48h and, more or less, return to the normal ranges after 72h. This supports the opinion that L-carnitine may be a potential antidote for VPA toxicity^{24,25}. This antidotal effect may be induced by the ability of L-carnitine to correct the wrong metabolic pathway induced by a deficit in carnitine in valproic acid intoxications³. [My paper].

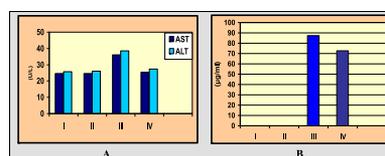
Analysis of the results of the newly diagnosed patients as epileptics and had not received any medications yet revealed that epilepsy per se has no significant alterative effects on the liver enzymes, which in accordance with the previous studies¹¹. On the other hand, in the chronically treated patients with VPA alone, it has been found that the serum VPA level of those patients (87.23 + 7.69ug/ml) was within the permitted therapeutic serum concentrations range (50 to 125 ug/ml) [19]. In addition, it was reported that the liver enzymes were significantly increased when compared to those of either control group or the newly diagnosed patients as epileptics and had not received any medications yet. This alterative effect could be referred to the use of VPA^{4,24}.

Studying the effect of L-carnitine supplemented group showed a significant decrease of the serum VPA when compared to that of the VPA-alone treated subjects. This finding indicates the corrective effect of L-carnitine on the metabolic pathway of VPA. Moreover, it was reported that the serum transaminases were nearly normal with no statistically significant difference when compared to those of either the control group or the recently diagnosed patients as epileptics and had not received any medications yet. Accordingly, it could be concluded that L-carnitine supplementation during chronic VPA therapy could protect the liver in the high risk children, which is in accordance with Lheureux and Hantson, (2009), who advocated L-carnitine supplementation during VPA therapy in high-risk pediatric patients as a prophylactic measure¹⁶.

Studying the mechanisms underlying the toxic effects of VPA and antidotal and protective effects of L-carnitine was not the concern of the current study. However, to explain the results of this research, a highlight on these possible mechanisms should be done.

The mechanisms underlying the toxic effects of VPA are unclear and have been investigated in different studies. Hepatotoxicity has been suggested to be a consequence of

Fig. 2: Liver enzymes [A] and serum VPA [B] levels of the different investigated groups of the acute study (Mean \pm SD).



carnitine deficiency and also to be an oxidative effect²³. Furthermore, depletion of glutathione has been observed in animals treated with VPA^{15,24}. In addition, it has been believed, based on limited experimental and clinical evidence, that hypocarnitinaemia, subsequent imbalance between α -oxidation and ω -oxidation, and accumulation of 4-en-VPA are involved. Additionally, carnitine deficit may result in disruption of mitochondrial functions due to depletion in CoA-SH^{6,9}.

To understand this, the metabolic pathway of VPA will be discussed. VPA is extensively metabolized by the liver via glucuronic acid conjugation, mitochondrial β - and cytosolic ω -oxidation to produce multiple metabolites, some of which may be biologically active. Some of them may be involved in toxic effects of VPA, either in patients on chronic dosing or after an acute overdose. For example, 2-propyl-4-pentenoic acid (4-en-VPA), a byproduct of ω -oxidation, have been incriminated in the development of hepatotoxicity of VPA⁸. Mitochondrial β -oxidation of VPA involves its transport within 2 the mitochondrial matrix, using the same pathway as do long-chain fatty acids. This pathway consists of several steps and is sometimes called the 'carnitine shuttle'. First, in the cytosol, VPA is activated and links with reduced acetyl coenzyme A (CoA-SH) to form valproyl-CoA. This process is achieved by the ATP-dependent medium-chain acyl-CoA synthetase, located on the outer side of the mitochondrial membrane. Valproyl-CoA then crosses the outer mitochondrial membrane. Under the effect of the palmitoyl carnitine transferase 1(PCT1), valproylcarnitine is formed; this step is needed because the inner mitochondrial membrane is not permeable to acylcarnitines. Valproyl-carnitine is then exchanged for free carnitine by carnitine translocase. In the mitochondrial matrix, PCT2 transforms valproylcarnitine into valproyl-CoA, which is able to enter a slow β -oxidation process¹⁸. Carnitine also helps to prevent valproyl-CoA accumulation⁹.

The ω -oxidation is normally responsible for only a small component of VPA metabolism. However, during long-term or high-dose VPA therapy, or after acute VPA overdose, a greater degree of ω -oxidation occurs, potentially increasing the risk for toxicity¹⁷. Carnitine (3-hydroxy-4-trimethylamino-butyric acid or β -hydroxy-gamma-N-trimethyl-amino-butyrate) thus appears essential to ensure proper metabolism of VPA⁵. With respect to VPA, it depletes carnitine stores, especially during long-term or high-dose therapy, through various synergistic mechanisms^{12,23}. First, as a branched chain fatty acid, VPA combines with carnitine to form valproylcarnitine, which is excreted in urine²⁰. Second, a reduction in tubular reabsorption of both free carnitine and acylcarnitine has been reported during VPA treatment⁷. Third, VPA reduces endogenous synthesis of carnitine by blockade of the enzyme butyrobetaine hydroxylase. Fourth, valproylcarnitine inhibits the membrane carnitine transporter, thereby decreasing the transport of extracellular carnitine into the cell and the mitochondria. VPA also induces reversible inhibition of plasmalemmal carnitine uptake in vitro in cultured human skin fibroblasts²⁶. Fifth, VPA metabolites combine with mitochondrial CoA-SH. The pool of free CoA-SH decreases, so that free mitochondrial carnitine stores cannot be restored from acylcarnitine (including valproylcarnitine) under the action of CPT2. Finally, the mitochondrial depletion of CoA-SH impairs α -oxidation of fatty acids (and VPA) and ATP production. ATP depletion further

impairs the function of the ATP-dependent membrane carnitine transporter. Accordingly, it seems that L-carnitine administration is essential for the normal metabolic pathway of VPA and in turn, VPA leads to carnitine depletion. Thus, it seems logic that external L-carnitine administration will correct the metabolic pathway of VPA and restores the depleted carnitine stores.

Our results suggest that L-carnitine may be a potential antidote for acute VPA toxicity and, as it does not appear to be harmful, prophylactic L-carnitine supplementation is recommended during VPA therapy in high-risk pediatric patients. It is advised to carry out further controlled, randomized, and multi-center trials to better delineate the therapeutic and prophylactic roles of L-carnitine and the optimal regimen of administration in the management of VPA toxicity either acute or chronic.

References

- Andersen GO and Ritland S: Life threatening intoxication with sodium valproate. *J Toxicol Clin Toxicol.* 1995; 33:279–284.
- Antoniuk SA, Bruck I, Honnicke LR, Martins LT, Carreiro JE and Cat R: Acute hepatic failure associated with valproic acid in children. Report of 3 cases [in Portuguese]. *Arq Neuropsiquiatr.* 1996; 54:652–654.
- [My paper]Bédry R.: New antidotes in toxicology. *Rev Prat.* 2008; 30; 58(8):844-8.
- [My paper]Björnsson E.: Hepatotoxicity associated with antiepileptic drugs. *Acta Neurol Scand.* 2008; 12: 1834-41.
- Borum PR, Bennett SG. Carnitine as an essential nutrient. *J Am Coll Nutr.* 1986;5:177–182.
- Bryant AE 3rd, Dreifuss FE: Valproic acid hepatic fatalities. III. U.S. experience since 1986. *Neurology.* 1996; 46:465–469.
- Camina MF, Rozas I, Castro-Gago M, Paz JM, Alonso C, Rodriguez-Segade S. Alteration of renal carnitine metabolism by anticonvulsant treatment. *Neurology.* 1991; 41: 1444–1448.
- Coulter DM.: Study of reasons for cessation of therapy with perhexiline maleate, sodium valproate and labetalol in the intensified adverse reaction reporting scheme. *N Z Med J.* 1981;93:81–84.
- Coulter DL: Carnitine, valproate, and toxicity. *J Child Neurol.* 1991; 6: 7–14.
- De Vivo DC, Bohan TP, Coulter DL, et al.: L-carnitine supplementation in childhood epilepsy: current perspectives. *Epilepsia.* 1998; 39(11): 1216-25.
- Hashim MA, Hanna MM, Khalaf MAM, Hussein GB.: Chronic biochemical and histopathological hepatotoxic effects induced by the most commonly used antiepileptic drugs in El-Minia governorate: clinical and experimental study. *ZUMJ.* 2002;VIII(5):1313-30.
- Ishikura H, Matsuo N, Matsubara M, et al.: Valproic acid overdose and L-carnitine therapy. *J Anal Toxicol.* 1996; 20: 55–58.
- Johannessen CU, Johannessen SI.: Valproate: past, present, and future. *CNS Drug Rev.* 2003;9: 199–216.
- Jung J, Eo E, Ahn KO.: L-Carnitine in the Treatment of Valproic Acid Overdose: A Case Study. *Am J Emerg Med.* 2008; 26(3): 388.
- Kin S, Basaran-Ku C, Kgergin C, Jdat MU.: Effect of acute and chronic administration of sodium valproate on lipid peroxidation and antioxidant system in rat liver. *Pharmacol. Toxicol.* 1999; 85: 294–298.
- Lheureux PER, Hantson PP.: Carnitine in the treatment of valproic acid-induced toxicity. *Clin Toxicol.* 2009;47:101-111.
- Lheureux PER, Penaloza A, Zahir S, Gris M.: Science review: Carnitine in the treatment of valproic acid-induced toxicity– what is the evidence?. *Crit Care.* 2005; 9(5): 431–440.
- Li J, Norwood DL, Mao LF, Schulz H.: Mitochondrial metabolism of valproic acid. *Biochemistry.* 1991;30:388–394.
- McNamara JO: Drugs effective in therapy of epilepsies. In; Hardman JG et al., (Eds.): *Goodman & Gilman's; The Pharmacological Basis of Therapeutics*, 9th edn., Ch. 20, pp. 461-186, 1996. New York, McGraw-Hill Co.
- Millington DS, Bohan TP, Roe CR, et al.: Valproylcarnitine: a novel drug metabolite identified by fast atom bombardment and thermospray liquid chromatography-mass spectrometry. *Clin Chim Acta.* 1985; 145:69-76.
- Piñero-Carrero VM, Piñero EO.: Liver. *Pediatr.* 2004; 113(4): 1097-1106.
- Raafat M: Epidemiology of Epilepsy. Paper presented in the 2th scientific meeting of Egyptian society for Neurology, 1991, Cairo, Egypt.
- Raskind JY, El-Chaar GM.: The role of carnitine supplementation during valproic acid therapy. *Ann Pharmacother.* 2000; 34:630–638.
- Raza M, Al-Bekairi AM, Ageel AM, Qureshi S.: Biochemical basis of sodium valproate hepatotoxicity and renal tubular disorder: time dependence of peroxidative injury. *Pharmacol Res.* 1997;35:153–157.
- Russell S.: Carnitine as an antidote for acute valproate toxicity in children. *Curr Opin Pediatr.* 2007; 19(2):206-10.
- Tein I, Xie ZW.: Reversal of valproic acid-associated impairment of carnitine uptake in cultured human skin fibroblasts. *Biochem Biophys Res Commun.* 1994; 204: 753–758.
- U' beda N, Alonso-Aperte E, Varela-Moreiras G.: Acute Valproate Administration Impairs Methionine Metabolism in Rats. *J of Nutrit.* 2002; 2737-2742.
- Watson WA, Litovitz TL, Klein-Schwartz W, et al.: 2003 annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *Am J Emerg Med.* 2004; 22: 335–404.

Sexual dimorphism of skull – morphological and mathematical facets

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Abstract

Determination of sex of an individual is the first step that should be undertaken in skeletal remains examination. In the living, we recognize two sexes, male and female. But, while dealing with skeletal remains, we frequently come across the bones possessing the features of both male and female sexes, in which it is best to look for the biologically most relevant traits. So instead of two sexes, we have the difficulty to choose from five sexes i.e. definite male, probable male, unknown, probable female and definite female, because of the continuum of spectra of dimorphic features. Therefore based on subjective traits, it is illogical to assign a particular sex to the skeletal remains. Present article aims at discussing some of the factors that are associated with making the sexual dimorphism a spectrum of sexual differentiation of human skull along with morphological and mathematical facets of sexing of skull.

Keywords

Identification, Skull, Sexual dimorphism, Forensic osteology, FORDISC.

Introduction

Identification of a missing person from recovered skeletal remains is a challenging as well as a complex task for any medico legal investigation. The sex of the bones has to be determined first, because it excludes half of the population from investigation. Furthermore, the determination of age and stature depends on the accurate diagnosis of sex as they are subjected to sexual variations. The accurate determination of sex depends on the availability of the number of bones. While it is recognized that the pelvis is the most reliable indicator for sexual traits, when bones are recovered for the purpose of medico legal investigation, all the bones need not be present. Frequently situations arise, when examination has to be performed from the available bones. Skull is generally considered next to pelvis in displaying the traits of sexual dimorphism. Sexing of human skull can be done by the morphological features and different mathematical indices. Both of these methods have their own advantages and disadvantages. No matter what method is used in sexing of skull, the final opinion must be appropriate since determination of sex is one of the pillars of the anthropological protocol and a key factor in construction of biological profile of human remains.

Discussion

Morphological Sexual Dimorphism:

Identifiable sex differences appear in the skull only after puberty. It is generally accepted that the male skull is larger in size, and of a more rugged architecture than that of female skull. The female skull retains the gracile attributes seen in prepubescent skull; whereas the male cranium becomes markedly rougher in adulthood at the site of muscular attachments.

Supraorbital ridges, glabella, mastoid processes, diagastric groove, muscular markings in the occipital area, etc are more developed in males. Forehead is steeper, less rounded in males

and rounded, full and infantile in females. Frontal and parietal eminences are more prominent in females. Occipital condyles and the foramen magnum are large in males. Orbit is squared, lower with rounded margins in males and rounded, higher with sharp margins in females. Fronto-nasal angulation is more prominent in males. Palate is larger and broader in males and the arch is more "U" shaped. In females the arch tends more towards a parabolic shape^{1, 2,3,4}.

According to Broca's hypothesis, when a skull is placed on a flat surface, the male skull rests on the mastoid process while the female skull rests on the occipital condyles^{5,6} Hoshi stated that, if the apex of the mastoid process is directed vertically downwards and there is a concavity immediately above the base of the process, it is more likely to be a male⁷. If the skull has a medially directed apex and the curvature of the skull above the base of the process is smooth and convex, it is more likely to be a female.

Mathematical Sexual Dimorphism:

Many studies were attempted to sex the skeleton on objective basis. Keen (1950) studied the colored population of South Africa, and showed that, the maximum cranial length, length of the base, horizontal circumference, cranial capacity, total face height, length of the mastoid process and the fronto-nasal angulation are the significant parameters from which sex can be calculated rather than estimated.⁸ But within 4 years after this work, Stewart proved that zones of these parameters shift with the population studies. Hanihara (1959) using Japanese skull developed discriminant functions to estimate the sex of the skull. But he stated that the validity of his discriminant functions must be limited to the Japanese population only, and should not be applied to other races⁹. Other indices that are studied to be useful for sex determination are¹⁰.

1. Total facial index = nasion-gnathion height / bizygomatic breadth X 100.
2. Upper facial index = nasion-prosthion length / bizygomatic breadth X 100.
3. Orbital index = maximum orbital height / maximum orbital breadth X 100.
4. Nasal Index = maximum nasal breadth / maximum nasal length X 100.
5. Palatal index = maximum palatal breadth / maximum palatal length X 100.
6. Gnathic index = basion prosthion length / basion- nasion length X 100.

Literature is scanty regarding the value of these indices for determining sex from skull.

In 1996, a team from Taiwan developed a method of sexing human skull by using cephalometric plots made from lateral telerradiography. Total of 114 dry skulls (59 men and 55 women) belonged to European population were examined. Cephalometric plots were made on lateral telerradiography with orthodontic software and 18 cephalometric variables were analyzed and sex was determined with 95.6% accuracy¹¹.

Morphological vis-à-vis mathematical sexing:

If the cranium alone is examined for determining sex, the difficulties encountered by the forensic expert are many. Traditionally, sexing of the skull was done by the descriptive

skeletal features or morphological traits rather than the dimensions (size and proportion). The problem with subjective sexing of skull is that, most of the traits in female are small to medium; and in males, medium to large. So any skeletal material with intermediate features is likely to be misinterpreted.

Another disadvantage of morphological sexing is inter-observer variations. However, this can be minimized by clear definition of traits rather than number of character traits.

Mathematical sexing when compared to morphological is more reliable since it is free from inter observer variation. Some crime laboratories are equipped with the data regarding these indices and when an unknown skeleton is sent for examination the data are fed to the databank 'FORDISC' which is used in the creation of a dead person's biological profile when only parts of the skull are available. The program compares likely profiles to data contained in a database of skeletal measurements of modern humans. Unfortunately, the applicability of these indices is very restricted as the value of these indices is confined to specific population.

In a study conducted at Germany, total of 98 skulls from recent forensic cases of known age, sex, and Caucasian ancestry from cranium collections in Frankfurt and Mainz (Germany) were analyzed, together by morphological method and by the statistical software solution FORDISC which derives its database and functions from the US American Forensic Database, to determine the accuracy of sex determination. In a comparison between metric analysis using FORDISC and morphological determination of sex, average accuracy for both sexes was 86 Vs 94% respectively. Possible explanations for this difference comprise different ancestry, age distribution, and socio-economic status compared to the FORDISC reference sample¹².

Determinants of sexual dimorphism:

Another aspect that is altogether ignored while sexing the skeleton is the factors that govern the expression of these traits. As will be discussed below, the expression of the sexual dimorphic trait is multi-factorial. Except pelvis, where the features are expressed due to the biological adaptability of females to child bearing, all the other features throughout the human skeleton are dependent on multitude of non-specific attributes.

The main factors affecting the expression of sexual dimorphism in skull are as follows:

1. Genetic make up of the individual: Berry RJ (1963) hypothesized that genetic make up of the individual governs the expression of sexual traits in the skeleton. This premise was based on the findings on laboratory mice¹³. Howe and Parsons (1967) confirmed the effect of genetic control. In human, suitable experiments to elucidate the degree of genetic control are obviously impossible¹⁴.
2. Heredity: Various studies done on the members of different families showed the role of heredity in the expression of sexual features in skeleton^{13,14}. It was concluded that some of the traits are likely to run in families. However while dealing with the unknown skeletal remains, a pathologist is bound to disregard the role of heredity.
3. Age: Sexual traits become prominent and reliable for sex determination only after puberty and the dividing line is around 15-18 years. Before this age sexing has been a 50-50 guess work except pelvis⁶. A study on the effect of age on cranial dimensions and indicated that there is a relationship between discriminant functions and age and concluded that older female and younger males are misclassified frequently. Towards old age, there occurs blurring of sex features in the skeleton^{6,15}. So it can be concluded that sex descriptions in the skull bones must be limited to 20-- 55 years.
4. Hormones: Though hormones play an important role in

the expression of secondary sexual characters in skeleton, literature is scanty regarding the hormone imbalance disorders and intersex conditions. However, it should be remembered that post-menopausal osteoporosis blurs certain features like the size of mastoid process and the foramen magnum^{6,7}.

5. Diet: It is an established fact that modeling of bones is affected by the nutritional status of the individual. Berry AC¹⁶ studied the association of rickets with the non-metrical variants of the skull. But, how the dietary influence affects the sexual traits is not studied in detail.
6. Habits: Most of the skeletal features used for sex determination are the points of muscular attachments. It is agreed that in males, due to bulkier muscle mass, the points of muscle attachments are more prominent and hence the masculine features, whereas in females the muscle bulk being less, they retain the gracile attributes seen in children. Conversely, females who are manual laborers, the expression of sexual dimorphic features will become more prominent. Therefore, sexing from the mastoid process and the external occipital protuberance frequently poses a challenge.
7. Diseases: Diseases that are known to affect the skeleton are many and beyond the scope of this article. But some common diseases that are more prevalent in a particular geographical area are to be kept in mind before sexing a skeleton.
8. Geographical variations: Most of the literatures we rely upon for sex determination are based on the studies done on archeological remains and museum samples. Since most of the parameters especially the metric indices are bound to vary in different races, the value of these indices and the discriminant functions can not be universally applied.

Accuracy of estimating sex from cranium varies from person to person and in different study groups. According to Krogman, given the skull alone, one can assess the sex within 92% accuracy^{5,6}. It may be emphasized here that his study comprised of 750 adult skeletons in a museum in which male to female ratio was 15:1. He observed in his book "Human skeletons in forensic medicine" that the accuracy should be lowered by 5-10%. So it will come to around 82-87%. In Stewart series, the accuracy was 80%; but in another study he could score only 77% along with mandible¹⁷. Keen could succeed in sexing 85% of skulls accurately⁸. His study was on adult skeletal materials, where juvenile and senile skulls were excluded. Giles and Eliot stated that one can sex accurately in 83-85 % of skull bones¹⁸. So the overall accuracy ranges from 77-87% by professional anthropologists of international repute using both the metric as well as the non-metric variants.

Conclusion

Although the pelvis is the most reliable indicator of the sexual traits among all the bones, a forensic expert while dealing with skeletal remains, is sometimes required to determine the sex from the skull alone. Since the morphological subjective traits are more vulnerable to inter-observer variations; and mathematical analysis is restricted to specific population, one should try to resort to the use of both methods for sexing of human skull accurately. Textbooks and literatures can not be always relied upon, however authentic they may be. Due to the wider racial intermingling in the present era, region wise and population specific studies must be undertaken. Therefore, this particular area is widely open for research.

References

1. Mathiharan K, Patnaik AK. Modi's Medical Jurisprudence and Toxicology. 23rd ed. New Delhi: Butterworths Lexis Nexis; 2006.

2. Parikh CK. Parikh's Textbook of Medical Jurisprudence and Toxicology. 6th ed. New Delhi: CBS Publishers; 2005 (Reprint).
3. Pillay VV. Handbook of Forensic Medicine. 14th ed. Hyderabad: Paras Publications; 2004.
4. Reddy KSN. The Essentials of Forensic Medicine and Toxicology. 27th ed. Hyderabad: Suguna Devi K; 2008.
5. Krogman WM. The Human Skeleton in Forensic Medicine. 1st ed. USA: Charles C Thomas; 1978.
6. Krogman WM, Iscan MY. The Human Skeleton in Forensic Medicine. 2nd ed. USA: Charles C Thomas; 1986.
7. Hoshi H. Sex difference in the shape of the mastoid process in norma occipitalis and its importance to the sex determination of the human skull. *Okajimas Folia Anat Jpn* 1962 Oct; 38:309-13.
8. Keen JA. Sex difference in skulls. *Am J Phys Anthropol* 1950; 8(1): 65-79.
9. Hanihara K. Sexual diagnosis of Japanese skulls and scapulae by means of discriminant functions. *Journal of Anthropological Society* 1959; 67(722): 21-7.
10. Berkovitz BKB. Skull and Mandible. In: Standring S, editor. *Gray's Anatomy* Edinburgh: Elsevier Churchill Livingstone; 39th ed. 2005. 455-91.
11. Veyre-Goulet SA, Mercier C, Robin O, Guerin C. Recent human sexual dimorphism study using cephalometric plots on lateral telerradiography and discriminant function analysis. *J Forensic Sci* 2008 Jul; 53(4): 786-9.
12. Ramsthaler F, Kreutz K, Verhoff MA. Accuracy of metric sex analysis of skeletal remains using Fordisc based on a recent skull collection. *Int J Legal Med* 2007 Nov; 121(6): 477-82.
13. Berry RJ. Epigenetic polymorphism in wild population of *Mus musculus*. *Genet Res* 1963; 4:193-220.
14. Howe WL, Parsons PA. Genotype and the environment in the determination of minor skeletal variants and body weight in mice. *J Embryol Exp Morphol* 1963; 17: 283-92.
15. Nandy A. Principles of Forensic Medicine. 1st ed. Calcutta: New Central Book Agency; 1995.
16. Berry AC. Factors affecting the incidence of non-metrical skeletal variants. *J Anat* 1975; 120(3): 519-35.
17. Stewart TD. Medicolegal aspect of skeleton- age, sex, race and stature. *Am J Phys Anthropol* 1948; 6 (3): 315-21.
18. Giles E, Elliot O. Sex determination by discriminant function analysis of crania. *Am J Phys Anthropol* 1963; 21: 53-68.

Anticonvulsant Induced Osteomalacia in Ghaziabad, Uttar Pradesh

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Abstract

Antiepileptic medications encompass a wide range of drugs including anticonvulsants, benzodiazepines, enzyme inducers or inhibitors, with a variety of effects, including induction of cytochrome P450 and other enzyme, which may lead to catabolism of vitamin D and hypocalcaemia and other effects that may significantly affect the risk for osteomalacia. With the current estimate of 50 million people worldwide with epilepsy together with the rapid increase in utilization of these medications for other indications, bone disease associated with the use of antiepileptic medications is emerging as a serious health threat for millions of people. Our results indicate that the prevalence of vitamin D deficiency in epilepsy patients under AEDs treatment is high, especially under polytherapy. Thus it is suggested that routine monitoring of serum 25-OH vitamin D and vitamin D supplementation on an individual basis should be considered.

Keywords

Antiepileptic (AEDs), Alkaline phosphate (ALP), Hypocalcaemia, Osteomalacia, Parathyroid hormone (PTH), Phenyoin (DPH), Carbamazepine (CBZ), Phenobarbitone (PB).

Introduction

Epilepsy is a common neurological disorder of childhood frequently requiring prolonged use of anticonvulsants. Most of the commonly used anticonvulsants particularly phenytoin, pheno-barbitone, and carbamazepine have the propensity to interfere with vitamin D metabolism. Though biochemical changes are frequent, clinically overt rickets or osteomalacia is rare¹. Since the signs and symptoms of osteomalacia are non-specific, the diagnosis is frequently delayed resulting in considerable morbidity². Anticonvulsants induced osteomalacia is uncommonly recognized complication of long term antiepileptic medication³. Majority of the studies have been done in institutionalized, non-ambulatory patients receiving high dose multiple anticonvulsants for several years with sub-optimal dietary intake of vitamin D as well as limited exposure to sunlight⁴. Biochemical evidence of osteomalacia, i.e., alteration in serum calcium, phosphorus and alkaline phosphates has been seen in 20-70% of these patients. Anticonvulsants exert their deleterious effects on mineral and bone metabolism by inducing hepatic microsomal mixed function oxidase enzyme systems, resulting in increased catabolism and excretion of vitamin D and its biologically active products⁵. In addition, drugs especially phenytoin may directly inhibit the intestinal calcium absorption⁶. Though all commonly used anticonvulsants have the propensity to cause osteomalacia, phenytoin and phenobarbitone have the greatest potential⁷. Most of the

patients, who develop anticonvulsant induced osteomalacia, usually are on these drugs for several years though osteomalacia has also been described to set in within 3-6 years of therapy⁸. Patients who receive more than one drug are at greater risk than those on monotherapy⁹. Prevention of anticonvulsant induced osteomalacia lies in avoiding polytherapy, using minimum doses, ensuring adequate vitamin D intake and encouraging physical activity. Role of prophylactic vitamin D is controversial¹⁰. Though beneficial effect of vitamin D supplementation on bone and mineral metabolism has been demonstrated in epileptic patients receiving various anticonvulsant drugs¹¹, there are several limitations. While some authors recommend prophylactic vitamin D at the beginning of anticonvulsant therapy¹² others advise an interval of at least 6 months⁶. There is a wide range of dose of vitamin D used for supplementation¹⁴. Additional costs, risk of inadvertent over dosage, and need for frequent monitoring are other potential objections to this approach¹⁵. More importantly, most of the epileptics are out-patients while majority of the reports on anticonvulsant osteomalacia have concerned institutionalized patients with multiple risk factors.

Material and method

Sixty cases of epilepsy both male and female which had been on various anticonvulsant drugs, attending all clinical departments of Saraswathi Institute of Medical Sciences, Hapur, Ghaziabad, U.P., India, from March 2009 to April 2010 were selected for the present study. Patients were excluded if they had known risk factor to influence metabolism of serum vitamin D and calcium. The patients were individually matched age and sex with randomly selected healthy controls. None of healthy woman had a history of oligomenorrhoea and menopause in the past or at the time of study, and none of the healthy participants were using medications known to affect bone density, including calcium or vitamin D supplements.

After an overnight fast of 14-16 hours, 5 ml blood samples of patient and control were collected in vacuum tubes and allowed to clot at room temperature for 60-120 minute followed by centrifugation at 3000 rpm for 10 min. at 40C. Serum was stored at -20C, for estimation of vitamin D, calcium, alkaline phosphate and parathyroid hormone. Analyses of calcium phosphorous, total alkaline phosphate were determined with standard automated equipment. Total Ca was corrected for variance in serum albumin by the following formula. Total Ca (mg/dl) = measured Ca (mg/dl) + 0.8X [4-Alb (g/dl)].

The intact PTH was measured using an immunoradiometric assay (Diasorin, Stillwater, MN; normal range 13-54 Pg/ml). Serum 25-OH vitamin D was determined by radioimmunoassay. (Biosource, Belgium).

Results

The study was conducted on 60 patients (42 male and 18 female) of different age group who were on various anticonvulsant drugs therapy for at least 3 years. 60 healthy age and sex matched individuals served as control.

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Table no-1 shows the distribution of patients according to age group. The result shows maximum patients (27) 45% were in the age group of 30-39 years followed by (20) 33.33% were in age group of 20-29 years, while the least (02) 3.33% were in age group of less than 10 years.

Table no-2 shows the distribution of patients according to duration of anticonvulsant therapy. The result shows maximum patients (36) 60% were on 3-6 years of anticonvulsant therapy followed by (18) 30% were on 7-10 years, while the least (06) 10% were on 11-15 years of anticonvulsant drugs therapy.

Table no-3 shows the distribution of patients according to various anticonvulsant drugs therapy used. The result shows maximum patients (18) 30% were on phenytoin alone or on phenytoin & phenobarbitone therapy followed by (15) 25% were on phenytoin & carbamazepine therapy, while the least (09) 15% were on alone carbamazepine therapy.

Table no-4 shows level of serum 25-OH vitamin D, Total calcium, Phosphorus in epileptic patients on antiepileptic therapy were significantly decreased 51.48 ± 08.34 ng/dl, 08.39 ± 0.6 mg/dl and 03.18 ± 0.51 mg/dl respectively as compare to control $p < 0.001$, while alkaline phosphate and parathyroid hormone were significantly increased 156.48 ± 20.49 IU/L and 32.96 ± 9.84 pg/ml as compare to control.

Table no-5 shows comparison between mono and poly antiepileptic therapy. The result showed level of serum 25-OH vitamin D, Total calcium, Phosphorus in epileptic patients on polytherapy were significantly decreased 48.36 ± 08.27 ng/dl, 07.18 ± 0.6 mg/dl and 02.86 ± 0.5 mg/dl respectively as compare to monotherapy $p < 0.001$, while alkaline phosphate and parathyroid hormone were significantly increased 164.82 ± 20.72 IU/L and 35.32 ± 9.93 pg/ml as compare to monotherapy.

Discussion

The influence of anticonvulsants drugs among others Phenobarbital, phenytoin, and also carbamazepine with a concentration of calcium in serum is quite well known. Hypocalcaemia which is very rare in those cases, comes from increase of metabolism of vitamin D as a result of induction

activity of hepatic microsomal enzymes¹⁶. In the liver vitamin D is converted to a compound of medium activity of 25-hydroxy vitamin D (25-OH-D3). The increased production of less active metabolite vitamin D, as well as increased of the excretion of the gall bladder with 1, 25-dihydroxy vitamin D, metabolism which is set to be most active kind of it, can cause serious disorder of calcium balance in people treated with anticonvulsant agent¹⁷.

Since the first reports of adverse bone effect of AEDs more than 3 decades ago, a number of biochemical abnormalities of bone metabolism have been reported with AEDs used including hypocalcaemia, hypo phosphatemia, low vitamin D levels and increased in PTH, ALP^{4,17,18}. This constellation of effects have been demonstrated both children and adults and commonly seen with AEDs that induced cytochrome P 450 enzymes, particularly DPH, CBZ, PB^{8,19}. These enzymes including AEDs may increase catabolism of vitamin D, resulting in hypophosphatemia and hypocalcemia²⁰. Moreover, AEDs may inhibit cellular response to PTH. Both mechanisms have been proposed and may increase bone remodeling. DPH decreases intestinal cation transport and calcium absorption. However, data are not consistent and many studies fail to observe any significant decrease in serum calcium and phosphate levels with the use of enzyme inducing AEDs²¹.

Decreased urinary excretion of calcium with the use of AEDs (CBZ, DPH, VPA) has been reported in both children and adults²². Surprisingly, in the latter study, decrease in urinary calcium excretion with CBZ and DPH was limited to female participants only²³. The mechanism underlying this hypocalcemic effects of AEDs is unknown and need further exploration. Although the evidence of newer AEDs is still limited, lamotrigine and topiramate have not been shown to cause significant effect on serum calcium and phosphate²³.

Table 2: Distribution of patients according to duration of anticonvulsant therapy.

Duration of therapy (Years)	No of patients	Percentage
3-6	36	60%
7-10	18	30%
11-15	06	10%
Total	60	100.0%

Table 1: Distribution of patients according to age

Age group (Years)	No of patients	Percentage
Less than 10	02	03.33%
10-19	08	13.33%
20-29	20	33.33%
30-39	27	45.00%
40 and above	03	05.00%
Total	60	100%

Table 3: Distribution of patients according to various anticonvulsant drugs therapy

Drugs	No of patients	Percentage
Phenytoin	18	30.0%
Carbamazepine	09	15.0%
Phenytoin & Carbamazepine	15	25.0%
Phenytoin & Phenobarbitone	18	30.0%
Total	60	100.0%

Table 4: Effect of antiepileptic on serum vitamin D, total calcium, phosphorus, alkaline phosphates & parathyroid hormone

Serum concentration	Antiepileptic (no=60) mean \pm S.D.	Control (no=60) mean \pm S.D.	P value
25-OH vitamin D (ng/dl)	51.48 ± 08.34	60.84 ± 09.28	<0.001
Total calcium (mg/dl)	08.39 ± 0.6	09.83 ± 0.4	<0.001
Phosphorus (mg/dl)	03.18 ± 0.5	03.86 ± 0.4	<0.001
Alkaline phosphates (IU/L)	156.48 ± 20.49	98.36 ± 23.37	<0.001
Parathyroid hormone (pg/ml)	32.96 ± 9.84	28.68 ± 8.43	<0.001

Table 5: Comparison between mono and poly antiepileptic therapy on serum vitamin D, total calcium, phosphorus, alkaline phosphates & parathyroid hormone

Serum concentration	Polytherapy (no=33) mean \pm S.D.	Monotherapy (no=27) mean \pm S.D.	P value
25-OH vitamin D (ng/dl)	48.36 ± 08.27	54.74 ± 08.38	<0.001
Total calcium (mg/dl)	07.18 ± 0.6	09.27 ± 0.5	<0.001
Phosphorus (mg/dl)	02.86 ± 0.5	03.42 ± 0.4	<0.001
Alkaline phosphates (IU/L)	164.82 ± 20.72	148.39 ± 20.18	<0.001
Parathyroid hormone (pg/ml)	35.32 ± 9.93	29.67 ± 9.72	<0.001

Low levels of biologically active vitamin D in patients on AEDs have been demonstrated in a number of studies, particularly with the use of hepatic enzymes including medications like PB, PB, DPH and CBZ²⁴. This effect has been attributed to metabolism of vitamin D to polar inactive metabolites by the hepatic microsomes²⁵. However data are not consistent and some studies have not shown significant reductions in vitamin D levels with the use of AEDS²⁶.

Elevation of serum parathyroid hormone have been reported with the use of AEDS in subjects with epilepsy^{8,27}. This rise in PTH levels likely represents a secondary response to low vitamin D levels. However, high PTH has also been demonstrated independent of vitamin D deficiency^{8,28}. High PTH levels may increase bone turnover and predispose to low bone mass²⁹. High bone turnover has also been demonstrated with AED use despite normal levels of PTH³⁰. Another proposed mechanism for the bone effects of AEDs is the inhibition of the cellular response to PTH. Animal studies have shown that the use of PB and DPH was associated with impaired PTH response although this effects has not been confirmed in human studies³¹.

In conclusion, epileptic patients on long term anticonvulsants should be closely supervised for the development of osteomalacia. In view of non-specific symptomatology, there is always a danger of the diagnosis being over-looked. A high index of suspicion is required to make the diagnosis. Since the treatment is easy and effective, the need for early diagnosis cannot be over emphasized.

Conclusion

Antiepileptic medications encompass a wide range of drugs including anticonvulsants, benzodiazepines, enzyme inducers or inhibitors, with a variety of effects, including induction of cytochrome P450 and other enzyme, which may lead to catabolism of vitamin D and hypocalcaemia and other effects that may significantly affect the risk for osteomalacia. With the current estimate of 50 million people worldwide with epilepsy together with the rapid increase in utilization of these medications for other indications, bone disease associated with the use of antiepileptic medications is emerging as a serious health threat for millions of people. Our results indicate that the prevalence of vitamin D deficiency in epilepsy patients under AEDs treatment is high, especially under polytherapy. Thus it is suggested that routine monitoring of serum 25-hydroxy vitamin D and vitamin D supplementation on an individual basis should be considered.

Acknowledgment

Authors are grateful to Dr. Bina Shukla, Professor & head, Department of Pharmacology, Dr. S. Nagtilak Professor and head Department of Biochemistry, Saraswathi institute of Medical Sciences, Hapur for the guidance, providing laboratory kits and time to time valuable suggestions.

Conflict of interest - None

References

1. Hahn TJ, Hendin BA. Serum 25-hydroxycalciferol levels and bone mass in children on chronic anticonvulsant therapy. *N. Engl. J. Med.* 1975; 292: 550-554.
2. Stamp TCB, Round JM, Haddad JG. Plasma levels and therapeutic effect of 25-hydroxycholecalciferol in epileptic patients taking anticonvulsant drugs. *Br. Med. J.* 1972; 4:9-12.
3. Wark JD, Larkins RJ. Chronic diphenylhydantoin therapy does not reduce plasma 25-hydroxy vitamin D. *Clin. Endocrinol.* 1979; 11:267-274.

4. Scott RA, Sander JW. The treatment of epilepsy in developing countries. *Bull World Health Organ.* 2001; 79:344-351.
5. Sheth RD, Bodensteiner JB. Effect of carbamazepine on bone mineral density. *J. Pediatr.* 1995; 127:255-262.
6. Richens A, Rowe DJ. Disturbance of calcium metabolism by anticonvulsant drug. *Br. Med. J.* 1970; 4:73-76.
7. Dent CE, Richens A. Osteomalacia with long term anticonvulsant therapy in epilepsy. *Br. Med. J.* 1970; 4: 69-72.
8. Weinstein RS, King W. Decreased serum ionized calcium and vitamin D metabolites levels with anticonvulsant drug treatment. *J. Clin. Endocrinol Metab.* 1984; 58:1003-1009.
9. Pack AM, Done S. Bone mass and turn over in woman with epilepsy on epileptic drug monotherapy. 2005; 57:781-786.
10. Ali Il, Schuh L. Antiepileptic drugs and reduce bone mineral density. 2004; 5:296-300.
11. Holick MF. Vitamin D, Photobiology, metabolism, mechanism of action, and clinical application. 5th Edition. American Society for Bone and Mineral Research. 2003; 129-137.
12. Nashef L, Lamb E. Vitamin D deficiency- Guidelines are needed for treating diseases of bone metabolism in epilepsy. *Br. Med. J.* 1999; 318: 1285.
13. Kulak CAM, Silvado CE. Bone mineral density and serum levels of 25-OH vitamin D in chronic users of antiepileptic drugs. *Arq. Neuropsiquiatr.* 2004; 62: 940-948.
14. Collins N, Maher J, Cole M, Baker M. A prospective study to evaluate the dose of vitamin D required to correct low 25-OH vitamin D levels, calcium and alkaline phosphates in patients at risk of developing antiepileptic drug induced osteomalacia. *Q. J. Med.* 1991; 286: 113-122.
15. Drezner MK. Treatment of anticonvulsant drugs induced bone disease. *Epilepsy Behav.* 2004; 5 Suppl. 2: S41-47.
16. Kulak CA, Borba VZ. Bone mineral density and serum levels of 25-OH vitamin D in chronic users of antiepileptic drugs. *Arq. Neuropsiquiatr.* 2004; 62: 940-948.
17. Sheth RD. Metabolic concerns associated with antiepileptic medications. *Neurology* 2004; 63: S24-29.
18. Kruse R. Osteopathies in antiepileptic long term therapy preliminary report. *Monatsschr Kinderheilkd.* 1968; 116: 378-381.
19. Ecevit C. Effect of carbamazepine and valproate on bone mineral density. *Pediatr Neurol.* 2004; 31:279-282.
20. Stephen L J, Harrison JH. Bone density and antiepileptic drugs. *Seizure.* 1999; 8:339-342.
21. Kfali G, Erseclan T. Effect of antiepileptic drug on bone mineral density in children between ages 6-12 years. *Clin. Pediatr (Phila).* 1999; 38:93-98.
22. Erbayat Altay E. Evaluation of bone mineral metabolism in children receiving carbamazepine and valproic acid. *J. Pediatr. Endocrinol Metab.* 2000; 13:933-939.
23. Lackner TE. Strategies for optimizing antiepileptic drug therapy in elderly people. *Pharmacotherapy.* 2002; 22: 329-364.
24. Gough H. A comparative study of the relative influence of different anticonvulsant drugs, UV exposure and diet on vitamin D and calcium metabolism in outpatient with epilepsy. *Q. J. Med.* 1986; 59: 569-577.
25. Morrell MJ. Reproductive dysfunction in women with epilepsy: antiepileptic drug effect on sex-steroid hormones. *CNS Spectr.* 2001; 6: 771-772.
26. Tsukahara H. Bone mineral status in ambulatory pediatric patients on long term antiepileptic drug therapy. *Pediatr Int.* 2002; 44:247-253.
27. Valimaki MJ. Bone mineral density by dual energy x ray absorptiometry and novel markers of bone formation and restoration in patients on antiepileptic drugs. *J. Bone Miner Res.* 1994; 9: 631-637.

28. Yanase T. Aromatase in bone roles of vitamin D3 and parathyroid hormone. *J. Steroid Biochem. Molbiol.* 1995; 53: 165-174.
29. Sato Y. Decreased bone mass and increased bones turn over with epileptic drugs in adult's patients. *Neurology.* 2001; 57:445-449.
30. Feldkamp J. Long term anticonvulsant therapy leads to low bone mineral density. *Exp. Clin. Endocrinol. Diabetes.* 2000; 108:37-43.
31. Pack AM. Bone disease associated with antiepileptic drug. *Cleve Clin. J. Med.* 2004; 71 Suppl 2; S42-48.

Palatoscopy: A review

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Abstract

Palatoscopy, or palatal rugoscopy, is the name given to the study of palatal rugae in order to establish a person's identity. Palatal rugae have been shown to be highly individual and consistent in shape throughout the life. It is well-established fact that the palatal rugae pattern is unique to human, as his fingerprints. Though rugoscopy is highly individualistic, there are very few attempts which are documented in the literature. This suggests the scope for the study of rugae pattern in individuals.

Key words

Palatal rugae, human identification, forensic odontology.

Introduction

One of the main focuses of the forensic odontologist is human identification. Dental identification can be used as the sole method of identifying a deceased person. Dental identification is based on the comparison of antemortem and postmortem records. The records collected to identify a decedent should be accurate and totally inclusive of objective findings¹.

Palatal rugae, also called plicae palatinae transversae and rugae palatina, refer to the ridges on the anterior part of the palatal mucosa, each side of the median palatal raphe and behind the incisive papilla. As an entity they form the rugae pattern. Anatomically, the rugae consist of around 3 to 7 rigid and oblique ridges that radiate out tangentially from the incisive papilla².

The anatomical position of the palatal rugae inside the oral cavity is surrounded by cheek, lips, tongue, teeth and buccal pad of fat. All these afford some protection in case of fire and high impact trauma. Rugae are amongst the best protected, morphologically individualizing soft tissue structure in the body, which are preserved after death and also accessible during life³.

Palatal rugae have been shown to be highly individual and consistent in shape throughout the life. It is well-established fact that the palatal rugae pattern is unique to human, as his fingerprints⁴.

The antemortem records of palatal rugae are easily available with the dentist if patient had undergone any dental treatment such as orthodontic treatment, crown & bridge, dentures etc. If dentist maintains his/her patient's records in a proper manner it is very easy to access the antemortem record. Thus palatal rugae pattern of an individual may be considered as a viable alternative for identification purpose.

History

The earlier references to the palatal rugae occur in various works on general anatomy. Winslow (1732) seems to have been the first to describe them in an anatomy text⁵.

Palatal rugae was first illustrated by Santorini in 1775 in Tabula VII of his Septemdecim Tabulae, a drawing depicting

three continuous wavy lines which cross the midline of the palate⁵.

Since then the rugae have continued to interest anatomists but studies have been confined mainly to counting the rugae and making superficial observations about direction and prominence⁵.

Many researchers have studied the morphology and the racial differences of palatal rugae⁶. It seems that Kuppler, in 1897, was the first person to study palatal anatomy to identify racial anatomic features⁷.

Palatal rugoscopy was first proposed in 1932, by a Spanish investigator called Trobo Hermosa^{8,9}. In 1937, Carrea developed a detailed study and established a way to classify palatal rugae^{8,9}. One year later, Da Silva proposed another classification and, in 1946, Martins dos Santos presented a practical classification based on rugae location^{9,10}.

In 1983, Brinon, following the studies of Carrea, divided palatal rugae into two groups; fundamental and specific in a similar way to that done with fingerprints¹⁰. In this manner, dactiloscopia and palatoscopy were united as similar methods based on the same scientific principles and are sometimes complementary: for instance, palatoscopy can be of special interest in those cases where there are no fingers to be studied like burned bodies or bodies in severe decomposition¹⁰.

Even in the present century the rugae have received scant attention and until Lysell's (1955) study, attempts at classifying the pattern have been unsatisfactory⁵.

Classifications of palatal rugae

A study of the rugae pattern cannot be undertaken without a classification, because they have an intricate and complex formation and are absolutely individual to all persons. Direct comparison between two or more rugae patterns is impossible unless each is reduced to a set of common denominators, that is, a classification⁵.

Researchers have found the task of classification as a difficult aspect of rugae studies. The subjective nature of observation and interpretation within and between observers poses a problem².

A classification is compiled by noting as many common features of the rugae as possible and giving each a symbol or number which can then be used for recording and, if desired, statistical analysis. The record thus obtained makes comparison with other patterns possible⁵.

Nowadays, there are several known palatal rugae classifications. However, according to several authors, Lysell, in 1955, developed the first classification system for palatal rugae pairs¹¹.

Lo'pez de Le'on classification (1924)⁸

Dating from 1924, this classification has only historic relevance. The author proposed the existence of a link between a person's personality and palatal rugae morphology. In this manner, there were four known types of palatal rugae:

- B— Bilious personality rugae;
- N— Nervous personality rugae;

- L— Lymphatic personality rugae.
- S— Sanguinary personality rugae;

The letters B,N,L, and S, stand for the different personalities. The letters l and r stand for the left and right side of the palate, and are followed by a number, which specifies the palatal rugae number on each side. For instances, a possible rugogram would be Br6; Bl8.

Carrea classification (1937)⁹

This classification divides palatal rugae into four different types, as shown in Table 1. Palatal rugae are classified only according to their form and no formula (rugogram) is developed.

Da Silva classification (1938)^{8,12}

In this classification, palatal rugae are divided into two groups: simple, from 1 to 6 (Table 2) and composed, resulting from two or more simple rugae. They are named according to each rugae number. It is possible to classify each ruga individually (describing its form), but also to describe all the palatal rugae system (describing each ruga type number), making this a difficult classification to use.

Martins dos Santos classification (1946)^{9,10}

This is based on the form and position of each palatal ruga, this classification indicates and characterizes the following:

- One initial rugae; the most anterior one on the right side is represented by a capital letter
- Several complementary rugae; the other right rugae are represented by numbers;
- One subinitial rugae; the most anterior one on the left side is represented by a capital letter
- Several sub complementary rugae; the other left rugae are represented by numbers. The numbers and letters given to each ruga relate to its form and can be seen in Table 3.

Trobo classification⁸

This classification also divides rugae into two groups: Simple ruga classified from A to F (Table 4) and composed rugae, classified with the letter X. Composed rugae result from two or more simple rugae unions. The rugogram is made from right to left, beginning with the principal ruga (the one closest to the raphae), which is classified with a capital letter (Table4). The following rugae are classified with small letters. Finally, the left side of the palate is described using the same criteria

Basauri classification (1961)^{8,12}

A classification by Basauri (1961) consists of two groups: single and compound which in turn are subdivided into a total of ten types. Types 0-5 are Punto, Recta, Curva, Angulo, Sinuosa and Circulo, types 6-9 are Griega, Caliz, Raqueta and Rama. These names all describe a particular shape of a ruga and are mostly self-explanatory⁵.

Like the Trobo classification, this is a very easy classification to use. It distinguishes between the principal rugae which is the more anterior one, labeled with letters and the accessory rugae which concern all the remaining rugae, labeled with numbers (Table 5).

The rugogram is elaborated beginning from the right side of the palate.

Cormoy System^{8,12}

This system classifies palatal rugae according to their size, in:

1. Principal rugae (over 5 mm);
2. Accessory rugae (ranging from 3 to 4 mm);
3. Fragmental rugae (with less than 3 mm length).

The form (line, curve, and angle), origin (medial extremity) and direction of each ruga are also described. Possible

ramifications are also pointed out. Rugae that share the same origin, interrupted rugae and the incisive papilla are described as well. It is a very complete system. However, its use does not lead to rugogram elaboration, which makes the managing and processing of data difficult.

Correia classification¹²

Rugae are labelled with numbers or letters, according to their form (Table 6). The rugogram is formed like a fractional equation. The right side is the numerator and the left side is the denominator. The first right and the first left palatal rugae (initial and subinitial rugae) are classified by a letter and the other right and left (complementar and subcomplementar rugae) are assigned numbers.

Lima (1968)⁵

Classifies rugae into four main types:

1. Punc-tate,
2. Straight,
3. Curved
4. Composite.

Each has a numerical and an alphabetical symbol, one denoting shape and the other position. The author states that the classification is fully usable in forensic work and is particularly useful when it forms part of the identification tetralogy: dactiloscopia, odontoscopia, rugoscopia and haematography.

Caruso (1969)⁵

Caruso (1969) in his classification subdivided rugae morphology into lineomorphism and configuration. Volume and direction are noted and the number of rugae and the relationship between their distal margins and the teeth is observed.

Tzatscheva and Jordanov (1970)⁵

Categorize rugae according to direction, branching, symmetry and radially. The number of rugae is counted but if they form a network this is noted as such.

Thomas and Kotze (1972)⁵

Thomas (1972) used Lysell's classification with minor variations. It was found necessary for example to add a feature labeled "cross-link" which could not be accommodated otherwise.

Having determined the length of all the rugae, three categories were formed:

1. Primary rugae: (A-5 to 10 mm; B-10 mm or more)
2. Secondary rugae: 3-5 mm
3. Fragmentary rugae: 2-3 mm.
Rugae under 2 mm were disregarded.

The shapes of individual rugae were classified into four major types²².

1. Curved,
2. Wavy,
3. Straight and
4. Circular

Straight types ran directly from their origin to termination. The curved type had a simple crescent shape which curved gently. Evidence of even the slightest bend at the termination or origin of a ruga led to a classification as curved. The basic shape of the wavy rugae was serpentine; however, if there was a slight curve at the origin or termination of curved rugae it was classified as wavy. To be classified as circular, a ruga needed to display a definite continuous ring formation.

The direction of each primary ruga was determined by measuring the angle between the line joining its origin and termination and a line perpendicular to the median raphe. Forward-directed rugae were associated with positive angles, backward-directed rugae with negative angles, and

perpendicular rugae with angles of zero degrees.

Unification occurs when two rugae are joined at their origin or termination. Unifications in which two rugae began from the same origin but immediately diverged were classified as diverging. Rugae with different origins which joined on their lateral portions were classified as converging.

Brinon (1983)¹¹

In 1983, Brinon, following the studies of Carrea, divided palatal rugae into two groups: fundamental and specific in a similar way to that done with fingerprints.

Lysell (1955)⁵

The most important among the recent classification is that of Lysell (1955) which has since its conception featured prominently in research involving the rugae (Peavy and Kendrick, 1967; Thomas, 1972; Comoy, 1973; Bamberadeniya, 1978; Van der Linden, 1978). It is comprehensive and includes the incisive papilla. Rugae are measured in a straight line between origin and termination and grouped into three categories:

1. Primary rugae: 5 mm or more
 2. Secondary rugae: 3-5 mm and
 3. Fragmentary rugae: 2-3 mm.
- Rugae under 2 mm are disregarded.

The rugae of each side are numbered separately from anterior to posterior and classified according to shape, position or origin in relation to the median palatal raphe and unifications.

Three categories of unification are recognized:

1. Common origin diverging laterally,
2. Separate origins converging laterally
3. Same as the latter but involving one primary and one secondary ruga.

Branchings, breaks, papillations, annular formations and spirals are counted while the directions of the rugae are measured in degrees relative to the median palatal raphe.

The distribution of secondary and fragmentary rugae is observed by noting their proximity to the nearest primary ruga while the posterior border relationship with the teeth is observed. The incisive papilla is measured and classified according to one of seven shapes.

Individuality of rugae

Palatal rugae have been shown to be highly individual and consistent in shape throughout the life. It is well-established fact that the palatal rugae pattern is unique to human, as his fingerprints⁴.

The anatomical position of the rugae inside the oral cavity, surrounded by cheek, lips, tongue and buccal pad of fat, also afford some protection in cases of trauma or incineration³.

Palatal rugae are used in human identification not only due to their singularity and unchangeable nature, but also due to other advantages, namely their low utilization costs¹¹.

Once formed, they do not undergo any changes except in length, due to normal growth, remaining in the same position throughout an entire person's life. Not even diseases, chemical aggression or trauma seem to be able to change palatal rugae form¹¹.

It is also concluded that changes that occurs from orthodontic movement, extraction, aging and palatal expansion do not modify the rugae enough to hamper identification¹.

Paternity determination

In the search for genetic markers for use in paternity

determination, it was considered a possibility that a trait(s) in the rugae could be identified as heritable¹³.

As early as 1955 Lysell suggested that the palatal rugae might possess unique characteristics that could be used in paternity identification¹⁴. Lysell investigated 50 pairs of monozygotic twins and found no clear evidence of hereditary influence and said that it was impossible to identify a child's father from the rugae¹⁵.

Keil reported that a higher degree of similarity exists in monozygotic than in dizygotic twins and that the rugae may be useful in a multivariate discriminant analysis for proving paternity¹⁶.

Bamberadeniya subjected five families, including two sets of twins, to rugae pattern comparison and came to the conclusion that there is no evidence that rugae features are inherited¹⁷.

It can thus be concluded that the palatal rugae cannot be used in a practical procedure to determine paternity. Considering the polygenic nature of the inheritance of these structures, the pattern can never predictably contain characteristics that appear in both generations. This is a further confirmation of the closely equivalent natures of dermatoglyphs and palatal rugae¹³.

Ritter¹⁸ studied the rugae of twins and found that the pattern was similar but not identical.

Thomas et al.¹³ have worked on the possible use of palatal rugae patterns in paternity determination. However, there were no findings to link the two aspects.

Identification using denture

Any dentures which may be present, if they are not expelled from the mouth by concomitant violence at the time of accident or assault, also tend to remain unharmed and sheltered by tongue and cheeks. Such dentures could have a proper mark in which case the identity of the victim would not be in any doubt. As denture marking is still spasmodic however other marks such as imprints of palatal rugae can be of use if an ante-mortem record exists. This is the principle which is so important in the identification of dentate individuals whose dental records are extant and obtainable⁴.

Sometimes the dentures possess a mark which is usually meaningless to a casual observer but to a dental laboratory or dentist may be part of an administration system which, if traceable to its origin, can be useful.

The palatal rugae patterns can be regarded as such a mark but again is meaningless unless it can be traced back to an ante-mortem record. This forms the basis of the vitally important and often applied procedure for identifying dentate individuals.

A method for marking and storing ante-mortem records of palatal rugae has been speculated upon^{19,20}. It could take the form of a classification, in which symbols and codes are recorded, it could be a direct imprint on paper or in a plastic material, an electronic imaging and storage in some form (perhaps a Chernoff face)²¹, a plaster cast holography or the simplest, but unfortunately usually incidental, another denture known to belong to the deceased which contains the same rugae pattern.

The quality of the imprint of the rugae is of course dependent upon the age and condition of the denture and the observer must be sensitive to, and experienced in observing form in general and the varied shape that palatal rugae can assume.

The shapes of alveolar ridges are more equivocal than the rugae and are subjected to change from resorption⁴.

Table 1: Carrea palatal rugae classification

Classification	Rugae type
Type I	Posterior-anterior directed rugae
Type II	Rugae perpendicular to the raphae
Type III	Anterior-posterior directed rugae
Type IV	Rugae directed in several directions

Table 2: Da Silva simple palatal rugae classification

Classification	Rugae type
1	Line
2	Curve
3	Angle
4	Circle
5	Wavy
6	Point

Table 3 : Matins dos Santos palatal rugae classification

Rugae type	Anterior position	Other positions
Point	P	0
Line	L	1
Curve	C	2
Angle	A	3
Circle	C	4
Sinuuous	S	5
Bifurcated	B	6
Trifurcated	T	7
Interrupt	I	8
Anomaly	An	9

Oral prostheses are often expelled in severe trauma and are lost, damaged, or melted. In these individuals, the palatal rugae could be the only means of making a positive identification⁶.

Regeneration of rugae

Coslet et al.²² reported the clinical removal of palatal rugae is not permanent and that when removed, the rugae returned several months later.

In a case study by Breault et al.²³ it showed that the rugae can be transplanted to different parts of the body. If transferred to other areas of the anterior palate, rugae can easily be confused in identification.

Camargo et al.²⁴ have referred that, in gingival graft surgery, the selection of the palatal donor site should avoid the rugae areas because they may persist in the grafted tissue.

Other studies on palatal rugae

In a study conducted, the impression of maxillary arch of 100 individuals were taken. Two casts were prepared for each individual using dental stone. Five evaluators including 2 dentists, 2 forensic medical experts and 1 general surgeon were asked to match the casts. Each evaluator was asked to match the 2 sets of 100 casts i.e. 100 casts which were designated by numbers and their 100 counterparts which were designated using codes. 4 evaluators got 100% accurate matching and 1 evaluator got 98% accurate result. This study supports the fact of individuality of the rugae. Manual method has given very good results to support the individualization of rugae²⁵.

Investigations have been carried out to study the thermal effects and the decomposition changes on the palatal rugae of burn victims with panfacial third degree burns, and have concluded that most victims did not sustain any palatal rugae

Table 4: Trobo palatal rugae classification

Classification	Rugae type
Type A	Point
Type B	Line
Type C	Curve
Type D	Angle
Type E	Sinuuous
Type F	Circle

Table 5: Basauri palatal rugae classification

Principal rugae Classification	Accessory rugae classification	Rugae anatomy
A	1	Point
B	2	Line
c	3	Angle
D	4	Sinuuous
E	5	Curve
F	6	Circle
X	7	Polymorphic

Table 6: Correia classification

Rugae type	Anterior position	Other positions
Point	P	0
Line	L	1
Curve	C	2
Angle	A	3
Circle	C	4
Sinuuous	S	5
Bifurcated	B	6
Trifurcated	T	7
Interrupt	I	8
Anomaly	An	9

pattern changes, and when changes were noted, they were less pronounced than in the generalized body state. Furthermore, the ability of palatal rugae to resist decomposition changes for up to seven days after death was also noted²⁶.

However, some events can contribute to changes in rugae pattern, including extreme finger sucking in infancy and persistent pressure due to orthodontic treatment²⁷.

Differences between genders have also been studied, without any conclusions. The relationship between hard palate measurements and dental arches has also been used to determine group ethnicity².

Kratzsch and Opitz²⁸ developed a study in cleft patients whose results suggest that palatal rugae, in combination with measuring points of the cleft palate, can serve to depict changes occurring in the anterior palate during various stages of therapy and growth. These findings suggest that some facial changes can be expected when studying specific rugae patterns.

Palatoscopy may be used as a necroidentification technique. In fact, the Brazilian Aeronautic Minister demands palatal rugoscopy of all its pilots, in order to ensure their identification in case of accident⁹. It will be in these particular circumstances that palatoscopy is most valuable. The possibility of finding antemortem data supports this idea. Nowadays, palatal rugae patterns are considered a viable alternative for identification purposes. Some investigators aim to assess its feasibility with the aid of a computer and a software program. The results so far are good, but expected to be better³.

Ways of analyzing the palatal rugae

There are several ways to analyse palatal rugae. Intraoral inspection is probably the most used and also the easiest and the cheapest. However, it can create difficulties if a future comparative exam is required^{15,20}. A more detailed and exact

study, as well as the need to preserve evidence may justify oral photography or oral impressions²⁹.

Calcorrugoscopy, or the overlay print of palatal rugae in a maxillary cast, can be used in order to perform comparative analysis. Other more complex techniques are also available.

By using stereoscopy, for example, one can obtain a three dimensional image of palatal rugae anatomy. It is based on the analysis of two pictures taken with the same camera, from two different points, using special equipment. Another technique is the stereophotogrammetry which, by using a special device called Traster Marker, allows for an accurate determination of the length and position of every single palatal ruga.

However, due to its simplicity, price and reliability, the study of maxillary dental casts is the most used technique⁸.

Kogon and Ling³⁰ described a photographic superimposition technique that can easily be adapted to conventional photographic equipment for rugae comparisons.

Comoy³¹ took elastomeric impressions of the entire maxillary arch of 200 subjects, poured them in dental stone, and then drew on the stone casts the outlines of the incisive papillae. Comoy also developed a classification system based upon the length of the rugae.

Sassouni³² devised a study using 100 casts in which he developed a print of the palate. He advocated the use of bubble gum and a wooden spoon for obtaining the impression of the rugae. He would ink the impression and transfer it to paper to make a palatoprint similar to fingerprinting procedures. This technique leaves doubt as to the accuracy of this procedure because of the inaccuracy of the impression material (bubble gum) and the formation of a negative impression to produce the palatoprint.

Lysell¹⁵ and others have used photographs of the entire maxilla with the teeth and alveolar ridges partially blocked out. The analysis of data in these investigations may not have taken into account other influencing factors, including

1. The inadvertent use of other features of the cast to aid in the identification, such as teeth, edentulous ridge morphology, muscle attachments, vestibular depth, or some combination of these;
2. The effects of growth, extractions, or palatal expansion upon the shape of the rugae; and
3. The possible distortion of the palatal rugae replicates as a result of poor duplicating materials and techniques.

Matching the palatal rugae pattern using the computer software

Limson K.S. and Julian R³ (2004) conducted a study on palatal rugae patterns for forensic identification with an indigenously developed computer software program. Comprehensive computerized antemortem records were constructed for 250 subjects and a comparison matching process performed using both recorded and unrecorded samples. The efficiency of this computer-based identification method was then assessed. The program proved to have an average sensitivity of 0.93 and specificity of 1 and had a success rate of 92-97% in matches with digitized rugae pattern samples.

Problems with palatoscopy

Palatoscopy is a technique that can be of great interest in human identification. In fact, contrary to lip prints, it is possible to have antemortem data established such as records found in dental practice in different forms (dental casts, old prosthetic maxillary devices and intraoral photographs). However,

palatoscopy might not be so useful in crime scene investigations in the linking of suspects to crime scenes. In fact, this kind of evidence is not expected to be found in such circumstances. Another aspect of palatoscopy that one must consider is the possibility of rugae pattern forgery¹¹.

Due to anatomical position, it is unlikely that the study of palatal rugae could be used in the process of linking a suspect to a crime scene¹¹.

In a case report, Gitto et al.³³ gives step-by-step instructions on how to add palatal rugae to a complete denture. The added rugae improve speech patterns by incorporating texture into the anterior denture region. Certain patients require a tactile sense to cue or orient their tongue. The addition of rugae to an existing denture takes about 30 minutes. This process can lead to false identity exclusion due to misleading antemortem data.

In forensic identification, each human is considered unique, and stable points of singularity are treasured. The forensic dentist should be aware that forgery of rugae patterns could easily be accomplished.

A concern about palatal rugae voiced by many researchers is the possibility of rugae patterns changing with age and other outside influences³³.

Orthodontic movement, extractions of adjacent teeth, cleft palate surgery, periodontal surgery, and forced eruption of impacted canines are only some of the concerns²⁷.

Conclusion

Rugae are in general unchanged throughout life and can thus be used to help in the identification of an individual. Many authors feel that the legal identification could be made solely on palatal rugae.

References

1. Stuart LS and Leonard G. Forensic application of palatal rugae in dental identification. *The forensic examiner* Spring 2005, 44-47.
2. Kapali S, Townsend G, Richards L, Parish T. Palatal rugae patterns in Australian Aborigines and Caucasians. *Aust. Dent. Journal.* 1997; 42 (2):129-133.
3. Limson KS and Julian R. Computerized recording of the palatal rugae pattern and an evaluation of its application in forensic identification. *J. Forensic Odontostomatol* 2004; 22 (1): 1-4.
4. Thomas CJ and van Wyk CW. The palatal rugae in identification. *J. Forensic Odontostomatol.* 1988; 6 (1):21-25.
5. Thomas CJ and Kotze T. The Palatal Rugae: a New Classification. *Journal of the Dental Association of South Africa* 1983; 38:153-7.
6. English WR, Robinson SF, Summitt JB, Oesterle LJ, Brannon RB, Morlang WM. Individuality of human palatal rugae. *J Forens Sci* 1988; 33: 718-26.
7. Carbajo IC, Identificacio ´n de cada ´veres y aspectos forenses de los desastres, Publicaciones de la Unidad de Investigacio ´n en Emergenciay Desastres, <http://www.desastres.org>.
8. Pueyo VM, Garrido BR, Sa ´nchez JAS, *Odontologı ´a Legaly Forense*, Masson, Barcelona; 1994. pp. 277-292.
9. Campos ML, *Rugoscopia palatina*. <http://www.pericias-forenses.com.br>.
10. Perrella M, Costa F, Vessecchi S, Moccelin E, Daruge E. Identificac ¢o por rugoscopia palatinae dactiloscopia, <http://www.ibemol.com.br/forense2000/071.asp>.
11. Caldas IM, Magalha ões T, Afonso A. Establishing identity using cheiloscopy and Palatoscopy. *Forensic Science*

- International 2007; 165:1–9.
12. Thomas CJ and van Wyk CW. The palatal rugae in identification. *J. Forensic Odontostomatol.* 1988; 6 (1): 21–25.
 13. Thomas C.J, Kotze TJWW, Nash JM. The palatal ruga pattern in possible paternity determination. *J. Forensic Sci.* 1986; 31 (1): 288–292.
 14. Bailey L J, Esmailnejad A, Almeida MA. Stability of the palatal rugae as landmarks for analysis of dental casts in extraction and nonextraction cases. *Angle Orthod.* 1996; 66:73-78.
 15. Lysell L. Plicae Palatinae Transversae and Papilla Incisiva in Man: a Morphologic and Genetic Study. *Acta Odontologia Scandinavica.* 1955; 13(18):112-116.
 16. Keil A. *Grundziige der Odontolog & Gebruder Borntraeger, Berlin, 1966; 118-119.*
 17. Bamberadeniya K. A Study of the Rugae Pattern and the Shape of the Incisive Papilla in a Sri Lankan Population, *Sri Lanka Dental Journal.* 1978; 9:11-23.
 18. Ritter R, Form U, den Verlauffunddie, Gaumenleisten T. *Zeitschrift fur Morphologie und Anthropologie.* 1943; 40: 367.
 19. Carrea JU. Gaumenfalten-Fotostenogramme, ein neues identifizierungs verfahren Dental. *Zahnartzl Zeischri.* 1955; 10: 10-17.
 20. Thomas CJ Incidence of primary O rugae in Bushmen juvenile. *J.Dent.Res.* 1972; 51: 676-679.
 21. Thomas CJ Palatal rugae as Chernoff face *J.Forensic Odontostomatol.* 1985; 3:31-38.
 22. Coslet JG, Rosenberg ES, Tisat R. The free autogenous gingival graft. *Dent Clin North Am.* 1980; 24:651-682.
 23. Breault LG, Fowler EB, Billman MA. Retained free gingival graft rugae: a 9-year case report. *J Periodontol.* 1999; 70: 438-440.
 24. Camargo P.M., Melnick P.R, Kenney E.B. The use of free gingival grafts for aesthetic purposes. *Periodontology.* 2001; 27: 72–96.
 25. Hemanth M, Vidya M, Nandaprasad, Bhavana V Karkera, Human identification using palatal rugae: - manual method, *Indian journal of forensic medicine & toxicology.* 2008; 3(1): 26-28.
 26. Muthusubramanian M, Limson K.S, Julian R. Analysis of rugae in burn victims and cadavers to simulate rugae identification in cases of incineration and decomposition. *J. Forensic Odontostomatol.* 2005; 23 (1): 26–29.
 27. Abdel-Aziz H.M, Sabet N.E. Palatal rugae area: a landmark for analysis of pre- and post-orthodontically treated adult Egyptian patients, *East Mediterr. Health J.* 2001; (1/2): 60–66.
 28. Kratzsch H, Opitz C. Investigations on the palatal rugae pattern in cleft patients. Part II: Changes in the distances from the palatal rugae to maxillary points, *J. Orof. Orthop.* 2000; 61 (6):421–431.
 29. Utsuno H, Kanoh T. O. Tadokoro, Inoue K. Preliminary study of postmortem identification using lip prints. *Forensic Sci. Int.* 2005; 149:129–132.
 30. Kogon SL and Ling SC. A New Technique for Palatal Rugae Comparison in Forensic Odontology. *Canadian Society of Forensic Science Journal.* 1973;6: 3-10.
 31. Comoy JP. La Rugoscopie. *Chirurgien: Dentiste de France.* Vol., No., 1973 43(180):59-60.
 32. Sassouni V. Palato Print, Physioprint and Roentgenographic Cephalometry as New Methods in Human Identification (Preliminary Report). *Journal of Forensic Sciences.* 1957; 2: 428-442.
 33. Gitto CA, Esposito SJ, Draper JM. A simple method of adding palatal rugae to a complete denture. *J Prosthet Dent.* 1999; 81: 237-239.

Biochemical changes in acute organophosphorus pesticide poisoning in Bijapur, Karnataka

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Abstract

Organophosphorus poisoning is a major global health problem with more than 200,000 deaths every year. Organophosphorus compounds are widely used worldwide in agriculture as well as in most household gardens. Unfortunately because of their easy availability and potent toxicity, there is a gradual increase in accidental poisoning and is commonly abused for suicidal purpose.

Toxicities of organophosphorus compounds cause oxidative damage of cell membranes and also result in disturbed biochemical and physiological functions. Hence we planned to study the biochemical changes in acute organophosphorus poisoning.

Blood samples were collected from the clinically diagnosed organophosphorus poisoning subjects admitted during the year 2007-2009, in Shri B.M. Patil Medical College Hospital and Research Centre, Bijapur. The samples were analyzed using standard methods for different biochemical parameters.

Progressive fall in plasma cholinesterase and increased serum malondialdehyde levels in correlation with the severity of organophosphorus poisoning were observed. There was significant decrease in serum total cholesterol and potassium levels without much change in serum sodium level. C-reactive protein levels were increased. Serum magnesium levels were decreased in organophosphorus poisoning cases as compared to control group.

Inhibition of cholinesterase leads to increased acetylcholine level which induces oxidative damage resulting in various biochemical changes during acute organophosphorus poisoning.

Key words

Pseudocholinesterase, Organophosphorus, Cholinesterase inhibition

Introduction

Hundreds of organophosphorus(OP) compounds are available which are used as pesticides. These compounds vary with great differences in their mode of action uptake by the body, metabolism and elimination from the body and toxicity to humans. The toxicity of these materials to insects and mammals is determined by number of factors as they are absorbed, translocated to the target site and as they inactivate the target leading to poisoning¹. OP poisoning remains one of the major issues in both developing and developed countries.

Primary biochemical effect associated with toxicity due to OP compounds is inhibition of acetyl cholinesterase (AChE). Hence, the classical laboratory tests for exposure to OP toxicants are inhibition of AChE and butyrylcholinesterase (BChE) activity in blood². Toxicities of OP pesticides cause adverse effects on many organs. It affects the immune system, liver muscles, urinary system, reproductive system, pancreas and hematological system³. BChE synthesized primarily in liver and detected mainly in plasma. It is genetically different from AChE although they share same important function such as

acetylcholine (ACh) hydrolysis⁴. OP compounds irreversibly bind to the AChE enzyme, inhibiting its activity; i.e. phosphorylation of the active site serine in AChE. BChE like other serine esterase reacts with OP compounds forming phosphorylated esterase. It normally scavenges anticholinesterase compounds such as OP compounds and thus prevents inhibition of AChE. However many OP compounds inhibit BChE faster than AChE and hence it is a more sensitive indicator of absorption⁵.

As per World Health Organization statistics three million people around the world consume the pesticides annually resulting in 2,20,000 deaths every year. In addition to being neurotoxic pesticides have been reported to induce production of reactive oxygen species and oxidative tissue damage; and disturb the biochemical and physiological functions of erythrocytes and lymphocytes. Hence we planned to study the potential correlation between plasma ChE inhibition and lipid peroxidation and also to investigate the biochemical changes in acute OP poisoning.

Material and methods

The present study was carried out in the Department of Biochemistry; B.L.D.E.A's Shri B.M. Patil Medical College Hospital, Bijapur. The study was approved by the ethical committee. The clinically diagnosed OP poisoning cases admitted in this hospital were studied. Diagnosis was based on information taken both from the patient or the patient's family and by considering the following criteria:

1. Characteristic clinical signs and symptoms.
2. Improvement in signs and symptoms after treatment with atropine and pralidoxime (PAM).

Immediately after admission to the hospital, 10 ml venous blood samples were collected from the subjects under aseptic conditions, before starting the atropine treatment. Serum and plasma were separated by centrifugation at 3,000 rpm for 10 minutes, at room temperature. Then all samples were immediately placed at 4°C until they were processed, to get accurate and reproducible results.

Toxicity marker

Plasma cholinesterase (ChE) was estimated by Butyrylthiocholine Kinetic Method, using standard kit of Agappe Diagnostics. Cholinesterase act on butyrylthiocholine to form thiocholine which acts on dithio-bis-nitro benzoic acid giving pink colored 2-nitro, 5-mercaptobenzoate^{6,7}.

Lipid peroxidation

Serum lipid peroxide was measured as lipid peroxidation product malondialdehyde (MDA) by precipitating lipoproteins with trichloroacetic acid and boiling with thiobarbituric acid which reacts with MDA to give pink color as the method of Kei Satho⁸.

Serum Cholesterol was assessed by CHOD-PAP method using a standard kit of Erba Diagnostics Mannheim⁹.

Serum C Reactive Protein was estimated by Turbidometric immunoassay based on the principle of agglutination reaction using a kit of Tulip Diagnostics Pvt.Ltd¹⁰.

Serum Magnesium based on colorimetric xylidyl blue method using Accurex biomedical kit¹¹.

Table 1: Shows levels of plasma ChE, serum MDA, serum Total Cholesterol and serum C - reactive protein in control and OP poisoning cases, (n=30).

	Plasma ChE (U/L)	Serum MDA (nmol/ml)	Serum Total Cholesterol(mg/dl)	Serum C-reactive Protein(mg/dl)
Control	6286±912	1.05±0.37	210.13±15.60	0.245±0.17
Grade I	4586±259*	1.9±0.39*	154.75±20.52*	1.84±1.55*
Grade II	3506±234*	2.1±0.55*	148.02±19.2*	1.46±1.24*
Grade III	2462±239*	2.16±0.51*	142.12±17.1*	1.69±1.37*
Grade IV	1390±254*	2.39±0.61*	132.42±20*	2.40±1.62*
Grade V	614±210*	2.52±0.64*	136±16.1*	2.40±1.66*

n=number of subjects selected; values are expressed as Mean ± SD. * indicates p<0.05 as compared to control.

Table 2: Shows levels of serum sodium, serum potassium and serum magnesium in control and OP poisoning cases, (n=30).

	Serum sodium(mEq/L)	Serum potassium(mEq/L)	Serum Magnesium(mg/dl)
Control	139.68±2.43	4.49±0.34	2.54±0.40
Grade I	139.97±3.17	3.84±0.63*	2.19±0.53*
Grade II	139.47±4.1	3.78±0.66*	2.09±0.64*
Grade III	138.22±4.17	3.85±0.57*	1.86±0.49*
Grade IV	138.3±4.94	3.57±0.52*	1.83±0.50*
Grade V	138.5±3.55	3.75±0.45*	1.70±0.47*

n=number of subjects selected; values are expressed as Mean ± SD. * indicates p<0.05 as compared to control.

Serum Sodium and Potassium were measured by flame photometer method¹².

Statistical analysis was performed using student't' test. P value less than 0.05 (P<0.05) was considered as significant.

The grouping of the OP poisoning cases was done depending upon signs and symptoms as per Balany and Moses method.

Grade I - OP poisoned with no signs and symptoms.

Grade II - Diarrhea, vomiting, abdominal pain, giddiness.

Grade III - Pupillary constriction with above symptoms.

Grade IV - Pulmonary edema.

Grade V - Unconsciousness

Results and discussion

Organophosphorus compounds are highly toxic to human beings. Poisoning due to OP compounds is steadily increasing in India due their easy availability and potent toxicity. Among the OP compounds the most commonly used were Dimethoate (Roger), Monocrotophos, Chlorpyrifos, Paraoxan, Mevinphos, Triazophos.

With an increase in the severity of poisoning there was a corresponding decrease in plasma ChE activities. Table-1 shows progressive fall in plasma cholinesterase and increased serum malondialdehyde levels in correlation (r = - 0.931) with the severity of organophosphorus poisoning. Our results are inconsistent with many workers^{13,14}. But Semir Nouria reported no correlation between serum ChE and severity of OP poisoning¹⁵.

The inhibition of ChE initiates the accumulation of free radicals leading to lipid peroxidation which may be the indicator of cell injury Toxic manifestations induced by OP compound may be associated with enhanced production of reactive oxygen species which induces the oxidative process and lipid peroxidative damage in cell membranes¹⁶.

Serum total cholesterol level was significantly decreased compared to control group. Direct relationship between low cholesterol level and suicidal behavior in mentally ill patient was reported¹⁷. Reduced total cholesterol and phospholipids level in human due to OP compound was also reported¹³. Lowered cholesterol may affect brain serotonin enough to trigger violence or suicide in susceptible adults prone to such behavior¹⁸. Hence we support that it can be used as a biological marker in identification of person prone to intentional OP poisoning.

C reactive protein (CRP) is an acute phase protein synthesized rapidly in liver in response to inflammatory stimulus. Hence increased serum CRP level may be due to inflammation caused because of OP compounds. Correlation between AChE suppression and immune responses by OP has also been suggested by B.D. Banerjee¹⁹. Jong Rung Tsai had strongly correlated the initial serum CRP level with the severity grading of OP poisoning. He further supported that the length of stay was significantly longer in high CRP group²⁰.

In this study we found significant decrease in serum potassium level compared to control while serum sodium remains unchanged as shown in table-2. Multiple and profound metabolic disturbances in carbohydrate, electrolyte and acid base metabolism was also reported by many workers The finding of hypokalemia suggests that OP intoxication may cause an alteration in the distribution of potassium between intracellular and extra cellular compartments²¹. The consequences of electrolyte imbalances may depend on state of hydration or concomitant changes in sodium, potassium, magnesium, calcium and phosphorus metabolism².

We found decrease in serum magnesium level in OP poisoning cases as compared to control group. Kin Sang Hui reported hypokalemia and hypomagnesaemia²¹. Magnesium was considered to counteract the direct toxic inhibitory action of OP on the enzyme Na⁺-K⁺-ATPase²². Magnesium sulphate inhibits acetylcholine release from motor nerve terminals and antagonizes the effect of OP compounds²³. Principal effect of magnesium was to decrease the amplitude of the end-plate potential. This is believed to occur as a result of inhibition of the release of ACh from motor nerve terminal by magnesium.

Conclusion

Plasma ChE estimation determines the severity of poisoning which can be helpful for predicting outcome in OP poisoning. Inhibition of cholinesterase leads to increased acetylcholine level which induces oxidative damage resulting in various biochemical changes during acute organophosphorus poisoning. Estimation of serum cholesterol can be used as a biological marker in intentional OP poisoning.

References

1. T Roy Fukuto. Mechanism of action of organophosphorus and carbamate insecticides. *Env.Hlth.Pers.* 1990; 87:245-254.
2. IPCS series Organophosphorus Insecticides: A General

Introduction. Environmental health criteria, No-63; World Health Organization, Geneva, 1986.

3. Fatemeh Teimouri, Nasim Amirkabirian, Hadi Esmaily, Azadeh Mohammadirad, Atousa Aliahmadi and M Abdollahi. Alteration of hepatic cells glucose metabolism as a non-cholinergic detoxication mechanism in counteracting diazinon induced oxidative stress. *Hum and Exp Toxicol.* 2006; 25: 697-703.
4. Darvesh S, Hopkins D A, Geulac. Neurobiology of butyrylcholinesterase. *Nat Rev Neurosc.* 2003; 4,131-138.
5. Elsa Reiner, Radic Zoran and Vera Simeon-Rudolf. Mechanism of OP toxicity and detoxication with emphasis on studies in croatia. *Arh Hig Rada Toksikol.* 2007; 58:329-338
6. Kendel M, Bottger R. A kinetic method for determination of the activity of pseudocholinesterase. *Klin. Wochenscher.* 1967; 45:325.
7. N W Tietz, editor. *Textbook of Clinical Chemistry.* 3rd edⁿ, 1986: 746.
8. Kei Satho. Serum lipid peroxide in cerebrovascular disorders determined by a new colorimetric method. *Clin. Chimica. Acta* 1978; 90: 37-43.
9. Allain C C, Poon L S, Chan C S G Richmond W and Fu P. *Clin Chem.* 1974; 20:470.
10. Fisher C L, Nakamura R. *Am J Clin Path.* 1976; 66:840.
11. Tietz NW editor. *Clinical guide to laboratory tests*, 3rd ed. W.B. Saunders Company, Philadelphia 1995:380-382.
12. Varley Harold. *Practical Clin Biochem.* 1969; 4th ed. CBC Publisher, New Delhi.
13. Vidyasagar J, Karunakar N, Reddy MS, Rajnarayana K, Surender T, Krishna DR. Oxidative stress and antioxidant status in organophosphorus insecticide poisoning. *Ind. J. Pharmacol.* 2004; 36(2): 76-79.
14. M.Dandapani, A Zachariah, MR Kavitha, L Jeyaseelan and A Oommen. Oxidative damage in intermediate syndrome of acute OP poisoning. *Ind.J.Med.Res.* 2003; 117:253-259.
15. Semir Nouira, Fekri Abroug, Souheil Elatrous, Rafik Boujdaria and Slah Bouchoucha. Prognostic value of serum ChE in OP poisoning. *Chest.* 1994; 106(6): 1811-1814.
16. Maryam Akhgari, Mohammad Abdollahi, Abbas Kebryaezadeh, Ruhollah Hosseini and Omid Sabzevari. Biochemical evidence for free radical induced lipid peroxidation as a mechanism for subchronic toxicity of malathion in blood and liver of rats. *Hum Exp Toxicol.* 2003; 22: 205-211.
17. Modai I, Valveski A, Dror S, Weizman A. Serum cholesterol levels and suicidal tendencies in psychiatric patients. *J Clin Psychiatry.* 1994; 55:252-254.
18. Hyman Engelberg. Low serum cholesterol and suicide. *The Lancet.* 1992; 339: 727-729.
19. B.D. Banerjee, V Seth, A Bhattacharya, ST Pasha, AK Chakraborty. Biochemical effects of some pesticides on lipid peroxidation and free radical scavengers. *Toxicol. Lett.* 1999: 33-47.
20. Jong-Rung Tsai, Chau-chyun Sheu, Meng-Hsuan Cheng, Jen-Yu Hung, Chau-Sheng Wang, Inn-Wen Chong et al. Organophosphorus poisoning: 10 years of experience in Southern Taiwan. *Kaohsiung J Med Sci.* 2007; 23:112-119.
21. Kin Sang Hui. Metabolic disturbances in organophosphorus insecticide poisoning. *Arch Pathol Lab Med.* 1983; 107: 154.
22. Abdolkarim Pajoumand, Shahin Shadina, Ali Rezaie, Mahboobeh Abdi and Mohammad Abdollahi. Benefits of magnesium sulfate in the management of acute human poisoning by organophosphorus insecticides. *Hum and Exp Toxicol.* 2004; 23: 565-569.
23. Del Castillo J, Kartz B. Action and spontaneous release of acetylcholine at an inexcitable nerve muscle junction. *J Physiol.* 1954; 126:27.

Comparative x-ray structure analysis of systemic fungicides β -4(-chlorophenoxy)- α -(1,1 dimethylethyl)1H-1,2,4-triazole-1-ethanol and 1-(4-chlorophenoxy) 3,3-dimethyl-1-H(1,2,4-triazole-1-Y-1)2-butanone

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Abstract

The unit cell parameters of β -4(-Chlorophenoxy)- α -(1,1 dimethylethyl)1H-1,2,4-triazole-1-ethanol are $a = 8.130(2)$ Å $b = 16.790(2)$ Å $c = 21.990$ Å. $\alpha = 90^\circ$ $\beta = 92.52(1)^\circ$ $\gamma = 90^\circ$. The space group is determined to be $P2_1/n$. The measured density is 1.3215g/cm^3 and calculated density is 1.3102g/cm^3 . The average bond distances of C-H and N-H types are $0.96(2)$ Å and $0.90(1)$ Å respectively. The Unit cell parameter of 1-(4-Chlorophenoxy) 3, 3-dimethyl-1-H(1,2,4-triazole-1-Y-1)2-butanone are $a = 8.16(10)$ Å, $b = 16.81(3)$ Å $c = 22.05(2)$ Å, $\alpha = 90^\circ$ $\beta = 92.37(1)^\circ$ $\gamma = 90^\circ$ and $Z = 8$ and space group is determined $P2_1/n$. The measured density is $1.291\mu\text{g/cm}^3$ and calculated density is $1.295\mu\text{g/cm}^3$. We can see that although there are different chemical groups attached with both the compounds (Ehanol group is attached with one systemic fungicides while butanone with another.) but their cell parameters and average bond distances and angles are nearly equal. Thus we determine the three-dimensional structure, molecular dimensions, molecular geometry, electronic structure and the conformation of fungicides and analyze their crystal structures also. Then correlate the chemical activity by substituting the chemically active groups at the crucial sites of the model fungicide to enhance chemical affinity and introduce conformational changes in the fungicides to make than more effective, active and to some extent cheaper.

Keywords

X-ray crystallography, Systemic fungicides, Triazole structure

Introduction

Recently it has been observed that some of these fungicides are losing their effects and becoming resistant to them. So that analogous compounds can be designed as substitute, if their structures are known. A rational approach to test these fungicides is to know the three dimensional structure of these compounds and their macromolecular receptor sites as well as their molecular complex. The interactions of proposed fungicides with the macromolecule of the parasite are dependent on the stereochemistry of these compounds. In order to design more effective synthetic fungicides, it is necessary to analyse the three dimensional structure of these compounds and if possible the receptor molecule. The structures of these compounds can be obtained by X-ray diffraction method in crystalline form and they will invariably be similar to their structures in solution.

Experimental: Colorless well formed crystals are grown by slow evaporation technique from a solution of cyclohexanone at 278°K of both the fungicides. The unit cell parameters are determined by automatic computerized 4-circled Enraf-Nonius CAD-4 Diffractometer. The crystal structure is solved using SHELXS-97. Bond Lengths of β -4(-Chlorophenoxy)- α -(1,1 dimethylethyl)1H-1,2,4-triazole-1-ethanol (Angstrom) involving Non-Hydrogen atoms is shown in table 1 and Bond Angles (Degrees) - involving Non-Hydrogen atoms is shown in table 2. Bond Lengths of 1-(4-chlorophenoxy)

3, 3-dimethyl-1-H(1,2,4 triazole-1-Y-1) -2 -butanone (Angstrom) involving Hydrogen atoms is shown in table 3 and Bond Angles (Degrees) - involving Hydrogen atoms is shown in table 4. The ORTEP Diagram of 1-(4-chlorophenoxy) 3, 3-dimethyl-1-H(1,2,4 triazole-1-Y-1) -2 -butanone is shown in figure 1 and the ORTEP Diagram of β -4(-Chlorophenoxy)- α -(1,1 dimethylethyl)1H-1,2,4-triazole-1-ethanol is shown in figure 2.

Result and discussion

In β -4(-Chlorophenoxy)- α -(1,1 dimethylethyl)1H-1,2,4-triazole-1-ethanol the average bond distances of C-H and N-H types are $0.96(2)$ Å and $0.90(1)$ Å respectively. In 1-(4-chlorophenoxy) 3, 3-dimethyl-1-H(1,2,4 triazole-1-Y-1) -2 -butanone the average bond distance of C-H is 0.95 Å. The bond lengths and angles in the benzene rings show regular features in both the molecules. The C(4)-Cl(1) and Cl(2)-C(18) distances are $1.748(1)$ Å and $1.747(2)$ Å in β -4(-Chlorophenoxy)- α -(1,1 dimethylethyl) 1H-1,2,4-triazole-1-ethanol. The Cl(1A)-C(4A) and Cl(1B)-C(4B) distances are 1.733 Å and 1.738 Å in 1-(4-chlorophenoxy) 3, 3-dimethyl-1-H(1,2,4 triazole-1-Y-1) -2 -butanone. These distances are short and shortening may be due to delocalization of electrons from the benzene rings. The whole molecules appeared to be twisted and folded and reason may be due to stacking constraints. The bond distances around C(7) and C(21) are as usual shorter than single bond values in β -4(-Chlorophenoxy)- α -(1,1 dimethylethyl)1H-1,2,4-triazole-1-ethanol. The bond distances around C(7A) and C(7B) are usual shorter than single bond values in 1-(4-chlorophenoxy) 3, 3-dimethyl-1-H(1,2,4 triazole-1-Y-1) -2 -butanone. They may also appear to bear a partial double bond character. In β -4(-Chlorophenoxy)- α -(1,1 dimethylethyl)1H-1,2,4-triazole-1-ethanol the C(7)-O(1) and C(21)-O(3) distances are $1.4028(1)$ Å and $1.4114(2)$ Å respectively. In 1-(4-chlorophenoxy) 3, 3-dimethyl-1-H(1,2,4 triazole-1-Y-1) -2 -butanone. the O(1A)-C(7A) and O(2B)-C(7B) distances are $1.423(2)$ Å and $1.411(2)$ Å. These distances do not change significantly in similar structures, despite variable intermolecular interactions through them. The bond distances in the triazol rings are comparable to corresponding distances in heterocyclic rings 1.339 (Å). The average set of data by Spencer is 1.377 Å and 119° respectively. The dimensions of the methyl groups are normal and comparable with those in *O*-methyl obtusaquinone and moscaline hydro bromide .

The molecule is found to adopt a conformation such that the triazolyl ring is inclined angle of $72.9(9)^\circ$ to the aromatic ring and at an angle of $61.5(9)^\circ$ O(1A), C(7A) grouping The resulting arrangement lead approach of the ortho-H, H(2A) to the triazol, atoms N(1A) and N(2A) such that both N...H distances lie within the sum of the Vander Walls radii of N and H. There was an accompanying distortion of the exocyclic angles at C(1A) with the C(2A)-C(1A)-O(1A) bond angle of $124.65(17)^\circ$ being considerably larger than the value found for O1(A)-C(1A)-C(6A) $114.97(17)^\circ$

The triazolyl ring is planar with C(7A) lying only $0.063(7)$ Å from the mean plane. Although the C(8A) and C(9A)-N(3A) distances are somewhat larger than C(8A)-N(3A) and C(9A)-N(2A), in keep With the uncharged canonical valance form. All four C-N distances are shorter than a normal single bond

Table 1: Bond Lengths of β -4(-Chlorophenoxy)- α -(1,1dimethylethyl)1H-1,2,4-triazole-1-ethanol (Angstrom) involving Non-Hydrogen atoms .

Cl(1)	-	C(4)	1.7484(1)
O(1)	-	C(1)	1.3889(2)
O(1)	-	C(7)	1.4028(1)
O(2)	-	C(8)	1.2070(1)
N(1)	-	N(2)	1.3456(1)
N(1)	-	C(14)	1.2955(2)
N(2)	-	C(7)	1.4360(1)
N(2)	-	C(13)	1.3410(1)
N(3)	-	C(13)	1.3084(2)
N(3)	-	C(14)	1.3239(1)
C(1)	-	C(2)	1.3757(2)
C(1)	-	C(6)	1.3951(1)
C(2)	-	C(3)	1.3732(2)
C(3)	-	C(4)	1.3881(2)
C(4)	-	C(5)	1.3696(2)
C(5)	-	C(6)	1.3636(1)
C(7)	-	C(8)	1.5399(2)
C(8)	-	C(9)	1.5157(1)
C(9)	-	C(10)	1.5361(2)
C(9)	-	C(11)	1.5022(1)
C(9)	-	C(12)	1.5219(1)
Cl(2)	-	C(18)	1.7473(2)
O(3)	-	C(15)	1.3751(1)
O(3)	-	C(21)	1.4114(2)
O(4)	-	C(22)	1.1995(2)
N(4)	-	N(5)	1.3349(1)
N(4)	-	C(28)	1.3032(1)
N(5)	-	C(21)	1.4402(2)
N(5)	-	C(27)	1.3342(1)
N(6)	-	C(27)	1.3096(2)
N(6)	-	C(28)	1.2971(1)
C(15)	-	C(16)	1.3851(1)
C(15)	-	C(20)	1.3947(2)
C(16)	-	C(17)	1.3720(2)
C(17)	-	C(18)	1.3606(1)
C(18)	-	C(19)	1.3670(2)
C(19)	-	C(20)	1.3835(1)
C(21)	-	C(22)	1.5470(2)
C(22)	-	C(23)	1.5043(2)
C(23)	-	C(24)	1.5154(2)
C(23)	-	C(25)	1.4884(1)
C(23)	-	C(26)	1.5587(1)

Table 2: Bond Angles (Degrees) of β -4(-Chlorophenoxy)- α -(1,1dimethylethyl)1H-1,2,4-triazole-1-ethanol (Angles are ordered on the middle label, left to right and top to bottom) involving Non-Hydrogen atoms with estimated standard deviations in parentheses:

C(1)	-	O(1)	-	C(7)	119.63(1)
N(2)	-	N(1)	-	C(14)	103.80(2)
N(1)	-	N(2)	-	C(7)	121.85(2)
N(1)	-	N(2)	-	C(13)	107.77(1)
C(7)	-	N(2)	-	C(13)	130.36(2)
C(13)	-	N(3)	-	C(14)	103.31(1)
O(1)	-	C(1)	-	C(2)	125.09(1)
O(1)	-	C(1)	-	C(6)	114.80(2)
C(2)	-	C(1)	-	C(6)	119.92(1)
C(1)	-	C(2)	-	C(3)	120.82(2)
C(2)	-	C(3)	-	C(4)	117.98(1)
Cl(1)	-	C(4)	-	C(3)	118.81(2)
Cl(1)	-	C(4)	-	C(5)	119.23(1)
C(3)	-	C(4)	-	C(5)	121.96(1)
C(4)	-	C(5)	-	C(6)	119.49(2)
C(1)	-	C(6)	-	C(5)	119.74(1)
O(1)	-	C(7)	-	N(2)	111.71(2)
O(1)	-	C(7)	-	C(8)	100.96(2)
N(2)	-	C(7)	-	C(8)	113.64(1)

O(2)	-	C(8)	-	C(7)	119.12(1)
O(2)	-	C(8)	-	C(9)	123.55(1)
C(7)	-	C(8)	-	C(9)	117.34(2)
C(8)	-	C(9)	-	C(10)	109.77(1)
C(8)	-	C(9)	-	C(11)	109.49(1)
C(8)	-	C(9)	-	C(12)	107.05(2)
C(10)	-	C(9)	-	C(11)	109.95(1)
C(10)	-	C(9)	-	C(12)	109.85(2)
C(11)	-	C(9)	-	C(12)	110.69(1)
N(2)	-	C(13)	-	N(3)	110.29(1)
N(1)	-	C(14)	-	N(3)	114.77(1)
C(15)	-	O(3)	-	C(21)	118.87(2)
N(5)	-	N(4)	-	C(28)	112.37(1)
N(4)	-	N(5)	-	C(21)	130.51(2)
N(4)	-	N(5)	-	C(27)	107.91(1)
C(21)	-	N(5)	-	C(27)	121.56(1)
C(27)	-	N(6)	-	C(28)	118.71(2)
O(3)	-	C(15)	-	C(16)	125.05(1)
O(3)	-	C(15)	-	C(20)	114.45(2)
C(16)	-	C(15)	-	C(20)	120.50(1)
C(15)	-	C(16)	-	C(17)	119.72(1)
C(16)	-	C(17)	-	C(18)	119.53(2)
Cl(2)	-	C(18)	-	C(17)	119.71(2)
Cl(2)	-	C(18)	-	C(19)	118.39(1)
C(17)	-	C(18)	-	C(19)	121.89(2)
C(18)	-	C(19)	-	C(20)	119.78(1)
C(15)	-	C(20)	-	C(19)	118.56(2)
O(3)	-	C(21)	-	N(5)	111.56(1)
O(3)	-	C(21)	-	C(22)	102.56(2)
N(5)	-	C(21)	-	C(22)	112.55(1)
O(4)	-	C(22)	-	C(21)	119.37(2)
O(4)	-	C(22)	-	C(23)	123.16(1)
C(21)	-	C(22)	-	C(23)	117.31(2)
C(22)	-	C(23)	-	C(24)	112.53(1)
C(22)	-	C(23)	-	C(25)	112.27(2)
C(22)	-	C(23)	-	C(26)	105.10(1)
C(24)	-	C(23)	-	C(25)	112.81(2)
C(24)	-	C(23)	-	C(26)	107.22(1)
C(25)	-	C(23)	-	C(26)	106.27(2)
N(5)	-	C(27)	-	N(6)	100.90(1)
N(4)	-	C(28)	-	N(6)	100.06(1)

Table 3: Bond lengths [Å] with estimated standard deviation in parentheses for 1-(4-Chlorophenoxy) 3,3-dimethyl-1-H(1,2,4-triazole-1-Y-1)2-butanone

C1(1A)-C(4A)	1.733 (2)
O(1A)-C(1A)	1.383 (2)
O(1A)-C(7A)	1.423 (2)
O(2A)-C(10A)	1.200 (2)
N(1A)-C(8A)	1.339 (3)
N(1A)-N(2A)	1.361 (2)
N(1A)-C(7A)	1.440 (2)
N(2A)-C(9A)	1.312 (3)
N(3A)-C(8A)	1.315 (3)
N(3A)-C(9A)	1.336 (3)
C(1A)-C(2A)	1.382 (3)
C(1A)-C(6A)	1.386 (3)
C(2A)-C(3A)	1.390 (3)
C(2A)-H(2AA)	0.9300
C(2A)-C(4A)	1.371 (3)
C(3A)-H(3AA)	0.9300
C(3A)-C(5A)	1.381 (3)
C(4A)-C(6A)	1.372 (3)
C(5A)-H(5AA)	0.9300
C(6A)-H(6AA)	0.9300
C(7A)-C(10A)	1.553 (3)
C(7A)-H(7AA)	0.9800
C(8A)-H(6AA)	0.9300
C(9A)-H(8AA)	0.9300

C (10A)-C (11A)	1.512 (3)
C (11A)-C (12A)	1.529 (3)
C (11A)-C (14A)	1.530 (3)
C (11A)-C (13A)	1.542 (3)
C (12A)-H (12A)	0.9600
C (12A)-H (12B)	0.9600
C (12A)-H (12C)	0.9600
C (13A)-H (13A)	0.9600
C (13A)-H (13B)	0.9600
C (13A)-H (13C)	0.9600
C (14A)-H (14A)	0.9600
C (14A)-H (14B)	0.9600
C (14A)-H (14C)	0.9600
C1(1B) -C (4B)	1.738 (2)
C1(1B) -C (1B)	1.388 (2)
O (1B) -C (7B)	1.411 (2)
O (1B) -C (10B)	1.201 (2)
N (1B) -C (9B)	1.337 (3)
N (1B) -N (2B)	1.356 (2)
N (1B) -C (7B)	1.443 (2)
N (2B) -C (8B)	1.316 (3)
N (3B) -C (9B)	1.314 (3)
N (3B) -C (8B)	1.331 (3)
C (1B) -C (2B)	1.381 (3)
C (1B) -C (6B)	1.386 (3)
C (2B) -C (3B)	1.381 (3)

Table 4: Bond angles [degree] with estimated standard deviation in parentheses for 1-(4-Chlorophenoxy) 3, 3-dimethyl-1- H(1,2,4-triazole-1-Y-1)2-butanone

C (1A) - O (1A) - C (7A)	119.69 (15)
C (8A) - N (1A) - N (2A)	108.92 (18)
C (8A) - N (1A) - C (7A)	130.80 (19)
N (2A) - N (1A) - C (7A)	120.28 (16)
C (9A) - N (1A) - N (1A)	101.36 (19)
C (8A) - N (3A) - C (9A)	101.62 (15)
C (2A) - C (1A) - O (1A)	124.65 (17)
C (2A) - C (1A) - C (6A)	120.37 (19)
O (1A) - C (1A) - C (6A)	114.97 (17)
C (1A) - C (2A) - C (3A)	119.45 (19)
C (1A) - C (2A) - C (2AA)	120.32(16)
C (3A) - C (2A) - C (2AA)	120.34(16)
C (4A) - C (4A) - C (2A)	119.81(12)
C (4A) - C (3A) - C (3AA)	120.12(12)
C (2A) - C (3A) - C (3AA)	120.14(14)
C (3A) - C (4A) - C (5A)	120.62 (12)
C (3A) - C (4A) - C (1A)	120.15 (19)
C (5A) - C (4A) - C (1A)	119.27 (18)
C (6A) - C (5A) - C (4A)	120.14 (12).
C (6A) - C (5A) -H (5AA)	120.02(15)
C (4A) - C (5A) - H (5AA)	120.03(14)
C (5A) - C (6A) - C (1A)	119.72 (12)
C (5A) - C (6A) - H (6AA)	120.14(16)
C (1A) - C (6A) - H (6AA)	120.13(15)
O (1A) -C (7A) - N (1A)	111.06 (16)
O (1A) -C (7A) - C (10A)	101.45 (15)
N (1A) -C (7A) - C (10A)	113.11 (16)
O (1A) -C (7A) - H (7AA)	110.34(18)
N (1A) -C (7A) -H (7AA)	110.32(12)
C (10A) -C (7A) -H (7AA)	110.33(12)
N (3A) - C (8A) -N (1A)	111.24 (12)
N (3A) - C (8A) -H (8AA)	124.42(18)
N (1A) - C (8A) -H (8AA)	124.43(19)
N (2A) - C (9A) -N (3A)	116.66 (12)
N (2A) - C (9A) - H (9AA)	121.54(12)
N (2A) - C (9A) -H (9AA)	121.53(15)
O (2A) - C (10A) -C (11A)	124.62 (12)
O (2A) - C (10A) -C (7A)	118.52 (19)
C (11A) -C (10A) -C (7A)	116.86 (18)

C (10A) -C (11A) -C (12A)	107.47 (18)
C (10A) -C (11A) - C (14A)	109.9 (20)
C (12A) -C (11A) - C (14A)	110.01(20)
C (10A) -C (11A) - C (13A)	110.3 (12)
C (12A) -C (11A) - C (13A)	109..7 (12)
C (14A) -C (11A) - C (13A)	109.4 (12)
C (11A) -C (12A) - H (12A)	109.5(14)
C (11A) -C (112A)- H (12B)	109.5(15)
H (12A) - C (12A) - H (12B)	109.5(14)
C (11A) - C (12A) - H (12C)	109.5(12)
H (12A) - C (12A) - H (12C)	109.5(14)
H (12B) -C (12A) - H (12C)	109.5(13)
C (11A) - C (13A) - H (13A)	109.5(12)
C (11A) - C (13A) - H (13B)	109.5(11)
H (13A) - C (13A) - H (13B)	109.5(10)
C (11A) - C (13A) - H (13C)	109.5(12)
H (13A) -C (13A) - H (13C)	109.5(14)
H (13B) -C (13A) - H (13C)	109.5(15)
C (11A) - C (14A) - H (14A)	109.5(14)
C (11A) - C (14A) - H (14B)	109.5(11)
H (14A) - C (14A) - H (14B)	109.5(15)
C (11A) - C (14A) - H (14C)	109.5(14)
H (14A) - C (14A) - H (14C)	109.5(12)
H (14B) - C (14A) - H (14C)	109.5(11)
C (1B) - O (1B) - C (7B)	119.10 (15)
C (9B) - N (1B) - N (2B)	109.03 (17)
C (9B) - N (1B) - C (7B)	130.16 (18)
N (2B) - N (1B) - C (1B)	120.79 (16)
C (8B) - N (2B) - N (8B)	101.42 (18)
C (9B) - N (3B) - C (8B)	101.8 (12)
C (2B) -C (1B) - C (6B)	120.65 (19)
C (2B) -C (1B) - O (1B)	124.74 (17)
C (6B) - C (1B) - O (1B)	114.60 (17)
C (3B) - C (1B) - C (1B)	119.35 (19)
C (3B) -C (2B) - H (2BA)	120.31(14)
C (1B) - C (2B) - H (2BA)	120.34(15)
C (4B) - C (3B) - C (2B)	119.9 (12)
C (4B) - C (3B) - H (3BA)	120.11(16)
C (2B) - C (3B) - H (3BA)	120.12(14)
C (3B) -C (4B) - C (5B)	121.11 (12)
C (3B) -C (4B) - C1(1B)	119.91 (12)
C (3B) - C (4B) - C1 (1B)	119.72 (18)
C (5B) - C (4B) -C (1B)	119.18 (17)
C (4B) - C (5B) - C (6B)	119.5 (12)
C (4B) - C (5B) - H (5BA)	120.22(15)
C (6B) - C (5B) - H (5BA)	120.21(16)
C (5B) - C (6B) - C (1B)	119.52 (12)
C (5B) - C (6B) - H (6BA)	120.25(14)
C (1B) - C (6B) - H (6BA)	120.22(13)
O (1B) - C (7B) - N (1B)	111.33 (16)
O (1B) - C (7B) - C (10B)	101.97 (15)
N (1B) - C (7B) - C (10B)	112.9 (16)
O (1B) - C (7B) - H (10B)	110.12(13)
N (1B) - C (7B) -H (7BA)	110.13(14)
C (10B) -C (7B) -H (7BA)	110.15(12)
N (2B) - C (8B) - N (3B)	116.71 (12)
N (2B) - C (8B) - H (8BA)	121.72(13)
N (3B) - C (8B) -H (8BA)	121.71(13)
N (3B) - C (9B) -N (1B)	111.13 (12)
N (3B) -C (9B) H (9BA)	124.54(14)
N (1B) -C (9B) H (9BA)	124.53(16)
O (2B) -C (10B) - C (11B)	123.6 (12)
O (2B) -C (10B) - C (7B)	119.35 (19)
C (11B) - C (10B) -C (7B)	117.01 (18)
C (13B) - C (11B) - C (10B)	110.3 (12)
C (13B) - C (11B) - C (14B)	112.8 (13)
C (10B) - C (11B) - C (14B)	111.5 (12)
C (13B) - C (11B) - C (12B)	107.7 (13)
C (10B) - C (11B) - C (12B)	106.50 (19)
C (14B) - C (11B) - C (12B)	107.8 (12)

C (11B) - C (12B) - H(12D)	109.5(12)
C (11B) - C (12B) - H (12E)	109.5(13)
H (12D) - C (12B) - H (12E)	109.5(14)
C (11B) - C (12B) - H (12F)	109.5(13)
H (12D) - C (12B) -H (12F)	109.5(15)
H (12E) - C (12B) -H (12F)	109.5(13)
C (11B) - C (13B) - H (13D)	109.5(14)
C (11B) - C (13B) - H (13E)	109.5(15)
H (13D) - C (13B)- H (13E)	109.5(14)
C (11B) - C (13B) - H (13F)	109.5(12)
H (13D) - C (13B) - H (13F)	109.5(11)
H (13E) - C (13B) - H (13F)	109.5(11)
C (11B) - C (14B) - H (14D)	109.5(12)
C (11B) - C (14B) - H (14E)	109.5(13)
H (14D) - C (14B)- H (14E)	109.5(13)
C (11B) - C (14B) - H (14F)	109.5(14)
H (14D) - C (14B) - H (14F)	109.5(14)
H (14E) - C (14B) - H (14F)	109.5(14)

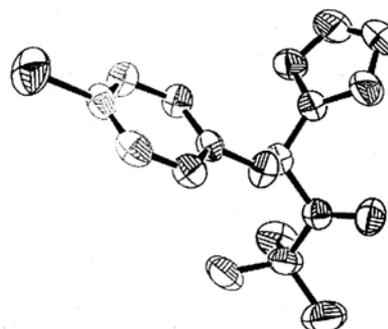
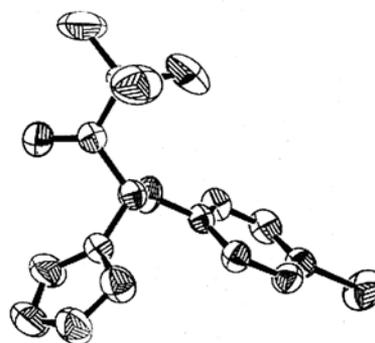
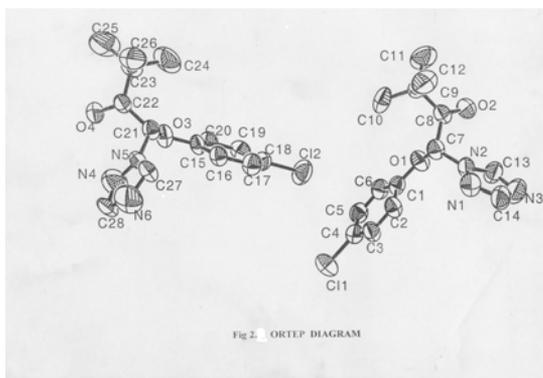


Fig. 1. : ORTEP Drawing at 50% probability level

(1.47Å). The N(1A)-N(2A) bond is also shorter than a normal single bond (1.45Å). The three atoms bonded to N(1) are almost co planar with it. Taken together these data indicate extensive delocalization within the heterocyclic ring. The most noteworthy feature of the heterocyclic ring is the asymmetry of the exocyclic angles at N (1A) [$\approx 30.80^\circ$]. We have observed a similar pattern in related triazole systems and it appear to be a function of a triazolyl ring itself rather than the influence of any inter or intramolecular interactions.

The C(11A), C(10A), C(7A), O(1A), C(1A) backbone is rather compressed resulting in the main from the orientation of the tert-butyl group, the C(1 1A)- C(10A)-C(7A)-O(1A) torsion being only $99.17(19)^\circ$. From the least square plane equation by Blow's method, the benzene and triazolyl rings are partially planner since the atomic displacements are much less than their e.s.d's. The triazolyl ring is inclined to the aromatic ring at an angle of $72.9(9)^\circ$

Thus we study the structure of variety of such compounds and correlate their structure with biological activity, so that more safer and effective fungicides at reasonable price can be developed.

Acknowledgement: The Financial assistance provided by Deptt of Science and Technology (D.S.T), New Delhi is gratefully acknowledged.

References

1. Kolbe, W. (1976), Pflanzenschutz - Nachrichten Bayer 31, 163-180.
2. Clark, T., D.R. Clifford, A.H Deas, P. Gendle and D.A.M. Watkins (1978), Pestic.Sci.9, 497506.
3. Sheldrich, G.M. (1997), SHELXS-97, Program for the solution of crystal structure.
4. Sheldrich, G.M. (1997), SHELXL-97, Program for crystal structure determination.
5. Jolmson, C.K. (1965), ORTEP, Report ORNL-3794. Oak Ridge National laboratory, Tennessee, U.S.A.
6. Nowell, I.W. and Walker, P.E. (1982) Acta Cryst. B38, 1857-1859
7. Bucheauuer, H. (1976). Z. Pflanzeskar. Pflanzenschutz. B3, 368-367.
8. Martin, T.J. & Morris, D.B. (1979) Pflanzenschutz Nachr. Am. Ed. 32, 3 1-79
9. Senger, Jyotsna, Ph.D. Thesis, Jiwaji University, Gwalior India (2002).
10. Haridus, M., Kulkarni, N.R., Tiwari; R.K. and Singh T.P. (1982), Curr. Sci. (India) 51(23), 1111.
11. Spencer, M. (1959), Acta Cryst. 12, 50.
12. Palmer. K.J., Wang, R.Y. and Jurd, L. (1973), Acta Cryst. B29, 1509.
13. Ernst, S.R. and Cogle Jr. F.W. (1973), Acta Cryst. B29, 1543.
14. Blow, O.M. (1960), Acta Cryst. 13, 168.

Jatropha poisoning (Bio-diesel Poisoning) - A case report

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Abstract

Jatropha curcas commonly known as bio-diesel plant can cause poisoning on ingestion. It mainly leads to gastro enteritis and dehydration. Mortality is rare. In a group of patients we also encountered manifestations like frothing at nostrils and papillary dilatation.

Key words

Jatropha poisoning, bio-diesel

What is already known?

Jatropha poisoning can cause gastroenteritis and dehydration.

What this study adds ?

Jatropha poisoning can also produce pupillary dilatation and frothing from nostrils.

Introduction

Jatropha curcas commonly known as bio-diesel plant is being cultivated in many parts of our country. Many parts of the plant can cause poisoning on ingestion. There is a lack of studies on Jatropha & its poisoning effects in text books and journals as well. One previous article reported gastrointestinal effects¹. We report mass poisoning in a group of 6 children in which we also encountered manifestations like nasal frothing and dilated pupil.

Case report

6 Children (4 male and 2 female) with age group 2-10yrs were admitted to pediatric emergency ward of BLDEA'S Shri B.M.Patil medical college hospital and research centre, Bijapur with history of consumption of seeds of a plant fruit of bio diesel plant. Time of consumption was 12 pm on the same day and symptoms started after 2 to 2.30 hrs. The consumption was accidental while playing, attractive nature of the seeds was the probable cause for ingestion. Average number of seeds consumed was 4 to 5. Presenting complaints were vomiting and loose stools. Two of them had moderate dehydration and drowsiness and one had frothing from nostrils at admission. A 10 year old boy with drowsiness developed pupillary dilatation and sluggish reaction to light 2 hours after admission, which resolved over next 8 hours. Serum electrolytes in two patients with dehydration were normal. Stomach wash was done in all children and dehydration was corrected with Ringers lactate. Milk

of magnesia and intravenous Ranitidine was given. All children improved in 6-8 hours and were discharged after 24 hours.

Discussion

Jatropha curcas is an evergreen plant found commonly in the southern parts of the India. Species include *J. cuneata*, *J. curcas*, *J. gossypifolia*, *J. integerrima*, *J. multifida*, *J. podagrica*. *J. curcas* is a small tree about 5 meters tall. The leaves are green smooth and lobed. The flowers are small, yellow and are mostly hidden by the leaves. The stem is thick, glabrous becoming woody at the base. The fruit is ovoid, oblong and contains 3 lobes with each lobe containing a seed. The fruit looks attractive and closely resembles cashew fruit though it bears no relationship to the latter¹. The seed extract oil is being used as the Bio-Diesel. Government is encouraging its cultivation for this purpose.

The poisonous properties of the plant are mainly due to the presence of toxalbumin called crucin and cyanic acid². Though all the parts of the plant are poisonous, seeds have the highest concentration of toxin. The seeds yield 40% oil known as hell oil or purgative oil, which contains small amounts of an irritant curcanoleic acid which is related to ricinoleic acid and crotonoleic acid, the principal active ingredients of castor oil and croton oil respectively³.

The adverse effects following consumption of seeds result primarily from gastroenteritis as in the present study. Depression and circulatory collapse are common in children⁴. Toxic dose ranges from consumption of as few as 3 seeds to 50 seeds. Human deaths are not noticed so far but animal deaths have occurred. Atropine like effects which are seen in the present report have not been reported earlier but are said to be common with *J. multifida*⁵.

Despite the above mentioned harmful effects, the seeds are used for various medicinal purposes. The yellow oil extracted from seeds is used for many medical conditions like eczema herpes, indolent ulcers, chronic rheumatic pain etc. In homeopathy it is used for cold sweats, colic, cramps, diarrhea etc. Recently the toxin (crucin) is also shown to have antitumour property⁶.

In spite of its ubiquity and propensity to cause many adverse effects on accidental consumption, not much information is available in leading toxicology and forensic medicine books of India^{7,8,9}. The present case report is a yard stick to measure the importance of the clinical problem due to Jatropha poisoning.

Table showing clinical manifestations.

AGE/SEX	TEMP...°C	PULSE	RESP.RATE	BP	PUPILS	OTHERS
10 Y M	36.8	120	26	84/48	Dilated /Sluggish	Dehydration
9 Y M	37	116	24	80/52	Normal	Dehydration
7 y M	36.5	92	20	102/64	Normal	Nasal Frothing
7 Y F	36.4	90	22	100/60	Normal	Normal
3 Y M	36.2	90	22	100/60	Normal	Normal
2 Y F	36.2	92	24	96/60	Normal	Normal

References

1. Kulkarni ML, Sreekar H, Keshavamurthy KS, Shenoy Nivedita. *Jatropha Curcas* – Poisoning. *Indian Journal of Pediatrics*; Vol 72, Jan 2005:75-76.
2. Perri LM. *Medicinal Plants of East and South East Asia*. Cambridge; MIT press, 1980: ([www- Hort. Purdue. Edu / new crop/ duke_energy / jatropha_curcas.html](http://www.Hort.Purdue.Edu/newcrop/duke_energy/jatropha_curcas.html)) .
3. Joubert P H, J M M Brown, I T Hay, P D B Sebata. (May 1984). Acute poisoning with *Jatropha curcas* (purging nut tree) in children. *South African medical Journal*, 65:729-730.
4. Watt JM, Breyer, Brandwijk MG. *The Medicinal and Poisonous Plants of Southern and Eastern Africa*. 2nd ed. London; E and S Livingstone, 1962: 1457.
5. Aplin T E H. (May 1976) Poisonous Garden plants and other plants harmful to man in Australia. Western Australia Department of Agriculture , Bulletin 3964.
6. Langdon KR. Physic nut, *Jatropha curcas* Nematology (Botany) circular 1977; No.3 ([www.doacs.stat.fl.us/npi/empp/botany/boteire/nem Botaro 30.htm](http://www.doacs.stat.fl.us/npi/empp/botany/boteire/nemBotaro30.htm)).
7. Reddy KSN. *The essentials of Forensic Medicine and Toxicology* 22nd ed. Hyderabad; Medical Book Company Publishers, 2003: 408-529.
8. Parikh CK. *Parikh's Text Book of Medical Jurisprudence and Toxicology* 6th ed. New Delhi; CBS Publishers 1999: 8.1-10.77.
9. Subramanyam BV. *Modi's Textbook of Medical Jurisprudence and Toxicology*, 22nd ed. New Delhi; Butterworths, 1999: section II 1-513.

Hopelessness, self-esteem and reaction to frustration in patients with alcohol dependence syndrome and normal controls

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Abstract

The objective of the present study is to compare hopelessness, self-esteem and reaction to frustration between alcohol dependent and normal controls. The control group was screened by General Health Questionnaire-5 (GHQ 5) and Alcohol Use Disorder Identification Test (AUDIT). Hamilton Depression Rating Scale (HDRS) was administered on the alcohol dependent group to rule out depression. Socio-demographic and clinical data were collected from the alcohol dependent group and Beck's Hopelessness Scale, Rosenberg's Self-esteem Scale and Rosenzweig's Picture Frustration study were administered to all 60 subjects. Result showed that alcohol dependent individuals are more hopeless and they possess lower self-esteem than their normal counterparts and their aggression is mostly directed toward external direction.

Key Words

Alcohol Dependence, Hopelessness, Self-Esteem

Introduction

Hopelessness is a psychological construct that has been observed to underlie a variety of mental disorders. Hopelessness expresses a way feeling (affective component), a way of thinking (cognitive component) and a way of acting (behavioural component). Hopelessness constitutes an essential experience of the human condition. It functions as a feeling of despair and discouragement, thought process that expects nothing, and a behavioural process in which the persons attempts little or takes inappropriate action¹. It was found that hopelessness were more prominent in drug abusers who attempted suicide². Alcohol provides a transient sense of power and adequacy to a particular person who lacks these two properties³. But to the person it is short-lived so that he has to take it again and again.

A person's self structure, self esteem is an important component of psychological health. To 'esteem' something is 'to admire, be fond of, cherish, honour, like, love, prize, regard highly, (or) respect' it⁴. Thus, in ordinary language, self-esteem refers to the extent to which one approves of and likes oneself. High self-esteem refers to a favourable evaluation of self⁵. Low self esteem has a greater tendency towards alcoholism and that college students with greater dissatisfaction are more likely to use a variety of psychoactive drugs⁶. Self-esteem is a reflection of both of his level of aspiration and of his self confidence. A person may be quite competent but sets his goals too high or a person may have realistic goals but feels unsure to protect his poor self image. This usually results in feeling of inferiority which are frequently compensated by his behaviour. These compensatory behaviour generally create further problems in interpersonal relations and increase inferiority which was often found in substance dependent individual⁷. Psychodynamic theories indicate that use of alcohol is protection against low self-esteem or for achievement of feeling of power⁸.

The term frustration refers to the blocking of behaviour directed toward a goal⁹. Frustration aggression hypotheses

states that frustration always leads to aggression. Although aggression is one of the outcome of frustration other response are also possible¹⁰. Alcohol consumption increases in situation involving stress conflict anger and frustration. The present study intended to see the nature of self-esteem, amount of hopelessness and reaction to frustration in patients with alcohol dependence syndrome and normal control¹¹.

Materials and methods

The study was a prospective cross sectional comparative hospital based study. The study was conducted in the Deaddiction Centre of CIP, Ranchi, Jharkhand. All the subjects were recruited by purposive sampling technique.

30 male between 18-55 years of age diagnosed as mental and behaviour disorder due to use of Alcohol Dependence Syndrome currently abstinent but receiving treatment, according to ICD-10 constituted the patient group. Exclusion criteria included present or past history of organic mental disorders, other substance misuse or dependence, co-morbid psychiatric disorder. The patients who are unable to comprehend the instruction of the study were also excluded from the experimental groups. The non-psychiatric control group consisted of 30 healthy male volunteers, matched in age, education, occupation, marital status and socio-economic status. Normal subjects who scored more than 1 in GHQ-5 were excluded from the study and other criteria were same.

Data were taken from the patient after their detoxification. Socio-demographic and clinical details were recorded on a specially designed pro forma by obtaining relevant information from case recode file and clinical interview. Hamilton Depression Rating Scale¹² was administered on the patient group to rule out depression. Mainly the people residing in and around Kanke were included in the study as a control group.

They were screened by using GHQ-5¹³ and Alcohol Use Disorder Identification Test¹⁴. After the Screening Beck's Hopelessness Scale¹⁵, Rosenberg's self-esteem Scale¹⁶ and Rosenzweig Picture Frustration Study, Adult version¹⁷ were administered on both the groups.

Results

Discussion

Both the groups were well matched with respect to various socio-demographic variables except residence. Most of the patients of the Alcohol Dependence group were from urban background. This is in accordance with the findings¹⁸ where it was found that level of frustration and hopelessness with substance consumption was mostly prevalent in urban areas than the rural areas. The significant findings that emerged from this study was both hopelessness, self-esteem and reaction to frustration could differentiate alcohol dependent patients from normal controls. The mean score of hopelessness in case of Alcohol dependence group was higher than normal control group and this was statistically significant. Hopelessness accompanied by addiction to alcohol was found to be a very common association by all the previous researchers^{2,2,19}. Beck and other researchers have also found that the degree of

Table 1: Socio-demographic characteristics of the patients and control group

Variable		Patients' group Mean ± SD(n%)	Control group Mean ± SD(n%)	t / ÷2	df	p
Age		33.80 ± 7.22	33.13±12.26	0.257	58	0.798NS
Education	Primary	1(3.3%)	4(13.3%)	2.11	2	0.348NS
	Secondary	8 (26.7%)	6 (20.0%)			
	Higher	21 (70%)	20 (66.7%)			
Occupation	Non working	5 (16.7%)	2 (6.7%)	6.876	2	0.076NS
	Student	9 (26.7%)	10 (33.3%)			
	Working	56.6 %(17%)	18 (60%)			
Marital status	Single	17 (56.7%)	19 (63.3%)	.278	1.00	.998NS
	Married	13 (43.3%)	11 (36.7%)			
Monthly income in Rs.	Upto 5000	16 (53.3%)	18 (60.0%)	0.313	2	0.855NS
	5000-10000	10 (33.3%)	9 (30.0%)			
	Above10000	4 (13.4%)	3 (10.0%)			
Residence	Urban	18 (60.0%)	8 (26.7%)	23.746	2	0.000**
	Semi urban	3 (10%)	21 (70.0%)			
	Rural	9 (30.0%)	1 (3.33%)			

NS Not Significant **Significant at 0.01 level

Table 2: Comparison of mean between two groups (alcohol dependent and normal control) on Beck Hopelessness Scale

Variable	Group (N=30)	Normal control	(t) (df)	P
	Alcohol dependent			
Hopelessness on Beck Hopelessness Scale	4.77±3.20	2.37±1.63	3.659 (58)	0.001**

**Significant at .01 level

Table 3: Comparison of Mean between Two Groups (Alcohol Dependent and Normal Controls) on Rosenberg's Self Esteem Scale

Variable	Group (N=30)		(t) (df)	p/p
	Alcohol Dependent	Normal Control		
Self Esteem	2.23±0.90	1.67±1.12	21.57	0.035*

hopelessness appeared to be an important predictor of suicide^{19,20}. Loss of self-esteem and negative self image in alcohol dependence and hopelessness seen in both depression and alcoholism has been found to speed up the suicidal process²¹.

Here self-esteem could differentiate alcohol dependent patients from normal controls. Alcohol dependent patients possess lower self-esteem than their normal counterparts. These findings are quite consistent with other observations. It has been emphasized by previous researchers that the problem of alcoholism results from lowered feelings of self-esteem^{6,22}.

Building of self-esteem is helping the alcoholic person to recognize assets and to accept himself as worth while person²³. So it is seen that reaction pattern of the person changes with the nature of self esteem and hopelessness that he posses. In the present study result showed that extra aggression (aggression in turned on the environment) score differ significantly in case of alcohol dependent group i.e. alcohol dependent peoples aggression are more prone to be directed externally. They are more aggressive than the normal control group. This finding is also supported by the findings of previous researchers. It was concluded that alcohol abusers are more aggressive and impulsive than their normal counter parts²⁴. It was also found in a study that alcoholic offenders possess irritability and impaired impulse control with increased aggression, monotony avoidance, and sensation seeking suspiciousness and reduce socialization²⁵.

It is also evident that alcoholics and normal people differed significantly in terms of their imaggression (Aggression is evaded in an attempt to gloss over the frustration) score. In imaggression response subject's aggression is not directed internally or externally but they totally gloss over the situation. Here result indicted that usually normal counter parts have a greater tendency to gloss over the situation than the alcoholic

persons. This finding is supported by another finding²⁶. They concluded that in case of substance abusers, aggression and hostility were reported more in situations that triggered their use of substances, and they had less confidence that they could avoid such situations in the future. As aggression and hostility is predominant in them, they are totally unable to gloss over the frustrating situation.

In the present study no significant difference was found in type of aggression between the two groups on Rosenzweig Picture Frustration (Obstacle dominance, ego defence, Need persistent) study.

Conclusion

Present study reveals that alcohol dependent individuals are more hopeless than their normal counterparts. Their self esteem is low and aggression is mostly directed toward external direction than their normal counterparts. So, it is important to provide them proper counselling and therapy to overcome all these problems; these factors are not only important in their treatment but also for improvement of their quality of life.

References

1. Farran CJ, Herth KA, Popovich JM. Hope and hopelessness: critical clinical constructs. New Delhi: Sage Publications; 1995.
2. Whitters AC, Cadoret RJ, Widmer RB. Factors associated with suicide attempts in alcohol abusers. *J Affect Disord.* 1985;9(1),19-23.
3. McClelland DC, Davis WN, Kalin R, Wanner Eric. The drinking man. New York: The Free Press; 1972.
4. Maslow AH. Motivation and personality. New York: Harpur and Raw; 1970.
5. Corsini RJ, Auerbach AJ, Anastasi A, Allen M. Concise

- Encyclopaedia of Psychology. New York: John Wiley; 1996. p. 792-795.
6. Wolman BB. International encyclopedia of psychiatry, psychology, psychoanalysis and Neurology. Aesculapius Publishers; 1977.
 7. Steffenhagen RA. An adlerian approach toward a self-esteem theory of deviance: a drug abuse model. *J Alcohol Drug Educ.* 1978;24,1-13.
 8. McCord J. Etiological factors in alcohol, family and personal characteristics. *J Stud Alcohol.* 1972;33,1020-1027.
 9. Morgan CT, King RA, Weisz JR, Schopler J. Introduction to psychology. New York: McGraw Hill; 1994.
 10. Dollard J, Doob LW, Miller NE, Maurer OH, Sears RR. Frustration and aggression. New Haven: Yale University Press; 1939. p. 105-112.
 11. Davison GC, Neale JM. Psychological Theories of Mood Disorder: Abnormal Psychology. New York: John Wiley & Sons; 1997. p. 231-238.
 12. Hamilton, M. A rating scale for depression. *J Neurol Neurosurg Psychiatry.* 1960;23(1),56-62.
 13. Shamsunder C, Sriram TG, Muraliraj SG, Shanmughan V. Validity of a short 5-items version of the General Health Questionnaire. *Indian J Psychiatry.* 1986;28,217-219.
 14. Babor TF, de la Fuente JR, Saunders J, Grant M. Alcohol use disorder identification test (AUDIT): guidelines for use in primary health care. Geneva: World Health Organization; 1992.
 15. Beck AT, Weissman A, Lester D, Trexler L. The measurement of pessimism: The Hopelessness Scale. *J Consult Clin Psychol.* 1974;42(6),861-865.
 16. Rosenberg M. Society and the adolescent self-image. New Jersey: Princeton University Press; 1965.
 17. Pareekh U, Devi RS, Rosenzweig S. Manual of the Indian adaptation of the Rosenzweig Picture-Frustration Study: Adult Form. Varanasi: Rupa Psychological Corporation; 1968.
 18. Hall, MD. Suicide risk assessment: A review of risk factors for suicide. *Psychol Rev.* 2002;90,124-133.
 19. Beck AT, Brown G, Steer RA. Prediction of eventual suicide in psychiatric inpatients by clinical ratings of hopelessness. *J Consult Clin Psychol.* 1989;57(2),309-310.
 20. Hewitt PL, Norton R, Flett GL, Callander L, Cowan T. Dimension of perfectionism, hopelessness and attempted suicide in a sample of alcoholics. *Suicide, Life Threat Behaviour.* 1998; 28(4), 395-406.
 21. Cornelius JR, Salloum IM, Mezzic J, Cornelius MD, Fabrega Jr H, Ehler JG, et al. Disproportionate suicidality in patients with comorbid major depression and alcoholism. *Am J Psychiatry.* 1995;152(3):358-364.
 22. Neeliyara T, Nagalakshmi SV, Ray R. Self esteem and psychopathic characteristics of individuals with alcohol dependence. *J Pers Clin Stud.* 1988;4(1),7-10.
 23. Estes NJ, Heinemann ME. Development perspective of alcoholism: Alcoholism development consequences and intervention. London: C.V. Mosby Co.; 1977. p. 112-121.
 24. Evans CM. Alcohol and aggression. *Alcohol and Alcoholism.* 1980;15,104-117.
 25. Virkkunen M, Kallio E, Rawlings R, Tokola R, Poland RE, Guidotti A, et al. Personality profiles and state aggressiveness in Finnish alcoholic, violent offenders, fire setters and healthy volunteers. *Arch Gen Psychiatry.* 1994; 51(1), 28-33.
 26. McCormick RA, Smith M. Aggression and hostility in substance abusers: the relationship to abuse patterns: coping style and relapse triggers. *Addict Behav.* 1993; 20, 555-562.

Trends of sex related crime in india and its quality management

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Abstract

Like other developing countries, in India also Sexual assault is a neglected public health issue. According to crime clock there is one rape case every 29 minutes and one molestation every 15 minutes¹.

A total of 1,64,765 incidents of crime against women were reported in country during 2006 which was a 0.6% increase than the previous year. Tripura has accounted for maximum 28.1% of total incidents although it shares only 0.31% of country's population. On second place is Delhi which has 1.44% share of country's population and has accounted for 28% of total crime¹.

Therefore this alarming situation needs prompt actions to be taken to prevent it along with an effective quality clinical management for the traumatized victims.

The essential components of medical care after the assault are²

- Documentation of injuries
- Collection of Forensic evidence
- Treatment of injuries
- Evaluation for STIs (HIV) and preventive care
- Evaluation for risk of pregnancy and prevention
- Psychological support, counseling and follow up.

All the concerned persons like physicians, Forensic experts, social worker, police etc. should coordinate with each other so that the patient is not disturbed and made to feel vulnerable and ashamed again and again.

Introduction

Despite the frequent usage of jargons like women empowerment gender based equality etc. women still remain the most vulnerable victims of crime. The fact is that society still remains dominated by men who tend to exploit and torture females whenever and wherever they can.

Broadly crime against women can be classified as follows according to the Indian Penal Code (IPC)¹.

1. Rape (u/s. 376 I.P.C.)
2. Kidnapping and abduction for different purposes (u/s. 363-373 I.P.C.)
3. Homicide for dowry, dowry deaths or attempts (u/s. 302/304-B I.P.C.)
4. Torture both mental and physical (u/s. 498-A I.P.C.)
5. Molestation (u/s. 354 I.P.C.)
6. Sexual harassment (u/s. 509 I.P.C.)
7. Importation of girls of more than 21 years (u/s. 366-B I.P.C.)

A total of 1,64,765 incidents of crime against women were reported in country during 2006 which was a 0.6% increase than the previous year. Tripura has accounted for maximum 28.1% of total incidents although it shares only 0.31% of country's population. On second place is Delhi which has 1.44% share of country's population and has accounted for 28% of total crime¹.

Discussion

Crime head wise analysis (IPC)-

1. Rape cases-

Rape cases have reported mixed trends over last five years

but a substantial increase of 0.6% in the year 2006 over 2005. Madhya Pradesh has reported the highest number of rape cases accounting for 15% of total such cases reported in the country. However Mizoram has reported the highest crime rate 7.5% as compared to national average of 1.7%.

2. Incest rape-

As compared to 5.4% increase in overall rape cases, incest cases have increased by 42.5% from 2005 to 2006. Chattisgarh has accounted for highest 22.5% of total such cases in country.

Rape victims- of the total victims of rape 8.2% were girls under 15 years of age while 17.4% were teenaged girls (15-18 years). Nearly two third women were in age group 18-30 years 15.5% women were in 31-50 years. Women victims of age group over 50 years were 0.5% of the total rape victims. Offenders were known to victims in as many as 75.1% of cases.

3. Molestation-

Incidents of molestation in the country have declined by 7.1% over the previous year. A maximum of 17% cases were reported from M.P, which also has the highest rate (9.3%) as compared to national average of 3.3.

4. Sexual harassment-

Numbers of such cases were marginally declined by 0.2% over the previous year. U.P has reported 27.2% of cases followed by Andhra Pradesh (24.2%).

Among 35 cities Delhi has accounted for 18.9% of total crimes followed by Hyderabad (8%). The crime rate was significantly higher in Vijay Wada and Vishakhapatnam at 74.3 and 54.3 respectively as compared to national average of mega cities at 20.3 only¹.

These statistics refer to the reported cases whereas if the unreported cases were to be included it would be a matter of seconds rather than minutes. Many times molestation and other cases are not reported because of family pressures, the attitude of police, unreasonable inquest process and application of law and resulting consequences.

It is quite paradoxical that sexual offences are not limited to a particular class of women; all the sections of women are equally vulnerable. Educated – uneducated, urban-rural, rich-poor, young-old or local-foreigner.

Thus the problem is so much wide spread and yet its eradication ineffective. All of us are familiar with Rupan Deol Bajaj vs. KPS Gill case³. A senior IAS officer was harassed by the then chief of police in 1998 and it took 10 years to seek justice. In 1997 in the famous case of Vishakha Vs. state of Rajasthan and others⁴ for the first time, sexual harassment had been explicitly legally termed. It was defined as an unwelcome sexual gesture or behaviour whether directly or indirectly as

1. Sexually colored remarks
2. Physical contacts and advances
3. Showing pornography
4. A demand or request of sexual favors
5. Any other unwelcome physical, verbal, nonverbal conduct being sexual in nature

It was in this landmark case that the sexual harassment was treated as a separate illegal behavior⁴.

According to Justice Saghir Ahmad, women in our country

belong to that class or group of society unfortunately which are at disadvantaged position on account of several social barriers and impediments. Therefore they have been victims of tyranny at the hands of men with whom they should enjoy equal status under the constitution.

According to S C Sarkar et al⁵, Sexual assault is a neglected public health issue in most of the developing countries and there is likely to be an even smaller percentage reporting of sexual assault. Only 10.55% of female victims report sexual assault. The under reporting of cases of sexual assault is mainly due to social stigma and prejudice with regard to the chances of marriage. They are often considered promiscuous and responsible for incident. Often there is attendant's humiliation and shame, embarrassment caused by cross examination in court, publicity in press, risk of losing the love and respect of society- friends and that of husband if married which leads to underreporting.

In their study they found that no age is safe from rape. It is comparatively easy for assailant to rape children as they can not resist much. Further in India as in many other countries, rape of children is due the superstitious belief that gonorrhoea and syphilis can be cured by sexual intercourse with a virgin.

Rape is a traumatic experience both emotionally and physically². It is a form of sexual violence, a public health problem and a human rights violation².

Quality management-

The quality management programme is designed to assure the quality of clinical care as well as its availability, accessibility and coordination. The quality management is based on the principle of continuous quality improvement⁶.

The sexually assaulted patient who might be an adult or child presents special medical, psychological, legal and safety needs. The medical system is involved in the sexual assault investigation for two reasons- therapy involving the physical

and emotional consequences of the assault and evidence gathering to corroborate the initial charges or to be used in the adjudication of the complaint⁷.

All the concerned persons involved in the matter- e.g. medicine, prosecution, crime investigation, NGOs need to proceed in a coordinated manner. Interview should be coordinated so that duplication of effort does not become another harassment and infringement on the patient's rights. The patients should not be made to feel vulnerable again⁸.

Thus with the cooperative efforts of local government, law enforcement agencies, hospitals, courts and relevant organization, a community plan should be established to deal with sexually assaulted patient. The plan should ensure that capable trained personnel and appropriate equipments are available for treating sexually assaulted patients⁷.

Quality clinical management

Clinical management of such patients can be described in following steps²-

1. First of all necessary preparations should be made so that medical care can be offered to the sexually assaulted patients in a comprehensive, confidential and nonjudgmental manner. A written medical protocol should be followed and health care professionals should be trained. All sorts of essential supplies like drugs, consent forms, paper bags for collection of samples and other equipments should be present.
2. The person who has experienced the trauma may be in an agitated or depressed state so the patient should be prepared for the examination with compassion and trust. She often feels fear, guilt, shame or anger. Consent form should be signed only after she is convinced thoroughly. The patient should not be forced for any thing.
3. The patient's history should be taken without letting her

Table 1(A)

Crime Head-wise Incidents of Crime Against Women during 2002-2006 and Percentage variation in 2006 over 2005

Sl.No.	Crime Head	Year					Percentage variation in 2006 over 2005
		2002	2003	2004	2005	2006	
1.	Rape (Sec. 376 IPC)	16373	15847	18233	18359	19348	5.4
2.	Kidnapping & Abduction (Sec. 363 to 373 IPC)	14506	13296	15578	15750	17414	10.6
3.	Dowry Death (Sec. 302/304B IPC)	6822	6208	7026	6787	7618	12.2
4.	Torture (Sec. 488A IPC)	49237	50703	58121	58319	63128	8.2
5.	Molestation (Sec. 354 IPC)	33943	32939	34567	34175	36617	7.1
6.	Sexual Harassment (Sec. 509 IPC)	10155	12325	10001	9964	9966	-0.2
7.	Importation of Girls (Sec. 366-B IPC)	76	46	89	149	67	-55.0
8.	Sati Prevention Act, 1987	0	0	0	1	0	-100.0
9.	Immoral Traffic (P) Act, 1956	6598	5510	5748	5908	4541	-23.1
10.	Indecent Rep. of Women (P) Act, 1986	2508	1043	1378	2917	1562	-46.5
11.	Dowry Prohibition Act, 1961	2816	2684	3592	3204	4504	40.6
	Total	143034	140601	154333	155553	164765	5.9

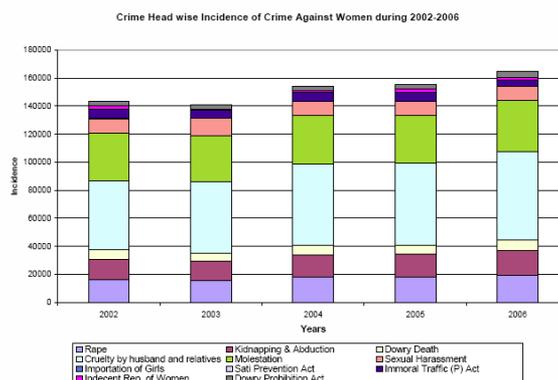


Table 1(B)

Proportion of Crime Against Women (IPC) towards total IPC crimes

Sl.No	Year	Total IPC Crimes	Crime Against women (IPC cases)	Percentage to total IPC crimes
1	2002	17,80,330	1,31,112	7.4
2	2003	17,16,120	1,31,364	7.6
3	2004	18,32,015	1,43,615	7.8
4	2005	18,22,602	1,43,523	7.9
5	2006	18,78,293	1,54,158	8.2

feel offended. She should be allowed to describe the incident at her own pace. It should be determined whether the patient has bathed, urinated, defecated, vomited, douched or changed her clothes as these may affect forensic evidences. Also existing health problems allergies etc. should be noted. Probability for pregnancy should also be evaluated.

4. Forensic evidences should be collected as soon as possible to help patient to pursue legal redressal. Documenting injuries and collecting samples such as blood, hair, saliva and sperm within 72 hours of incident may help to support the patient's story and might help identify the aggressor. To avoid multiple examination forensic evidences should be collected at the time of medical examination. The case should be documented properly in an objective and non judgmental way.
5. Then thorough physical and genital examination is conducted to determine what care should be provided. It is ascertained whether the patient has been admitted before or after 72 hour of incident and then examination is done accordingly.
6. After examination patient is prescribed treatment to prevent sexually transmitted infections or HIV transmission, pregnancy, tetanus and hepatitis B. The patient is also provided with wound as well as mental health care.
7. Along with treatment patient should be counseled properly as they are likely to be extremely distressed. Their psychological and emotional problems should be very carefully dealt with. They might undergo common mental disorders, stigma and isolation, substance abuse, risk taking behaviour or family rejection thus they should be given full support and encouragement.
8. The patient should be followed up accordingly whether they have received post exposure prophylaxis or not. If required, again history, examination and treatment should be considered.

Conclusion

Apart from the above steps rape crisis workers or NGOs should meet the families of sexually assaulted patients³. Often there is a tendency to blame victims for the assault. The assault is often attributed to the behaviour or manner of dressing of victim. So the family of victims should also be given proper counseling to support patient mentally for early recovery of patient⁸. In this context justice Arijit Pasayat had observed that while a murderer destroys the physical frame of the victim, a rapist degrades and defiles the soul of helpless female³.

References

1. National Crime Record Bureau. Ministry of Home Affairs, New Delhi, Chapter 5, 2006, p. 241-246.
2. Clinical management of rape survivors- Developing protocols for use with refugees and internally displaced persons- revised edition. WHO Report, www.who.int/reproductive-health/index.htm 2005, p. 1-76.
3. Dhruv Desai Sexual harassment and rape laws in India. 4th year law student Symbiosis society of law college, Pune. www.legalserviceindia.com. p. 1-10.
4. Vishaka Vs State of Rajasthan and other AIR, S.C., 1997, p. 3011.
5. Sarkar SC, Lalwani S, Rautji SR, Bhardwaj DN, Dogra TD. A study of victims- sexual offences in south Delhi. *Journal of Family Welfare*. June 2005 51(1), p. 60-6.
6. SFMHP Provider Manual of Quality Management Programme, 2006, p. 1-4.
7. American college of emergency physicians. Management of patient with the complaint of sexual assault. - revised and approved by ACEP board of directors. October, 2002, p. 1-2.
8. Robert RH, Ann Wolbert Burgess. Practical aspects of rape investigation – a multidisciplinary approach. 1st edition, 1990, p. 315-325.

Psychological aspects behind dating

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Abstract

Dating is a form of courtship which has been prevalent in the society in the past and still exists, but with continuously changing forms. This paper focuses on different types of dating, the various reasons behind the same and its consequences depending upon the psychology of the persons involved in the dating.

Keywords

Dating, Different systems, Reasons, Consequences, Date Rape

Introduction

Dating is a form of courtship, and may include any social activity undertaken by, typically, two people with the aim of each assessing the other's suitability as their partner in an intimate relationship or as a spouse. The word refers to the act of meeting and engaging in some mutually agreed upon social activity. Traditional dating activities include entertainment or a meal¹.

In many cultural traditions, a date may be arranged by a third party, who may be a family member, acquaintance or professional matchmaker. Recently internet dating has become popular.

Although dating etiquette has become more relaxed during the twentieth century, there are considerable differences between social and personal values. For example, when an activity costs money (for example, a meal), traditionally the man was expected to pay; but in recent times the practice of "going Dutch" (splitting the expenses) has become more common and more acceptable.

The average duration of courtship before proceeding to engagement or marriage varies considerably throughout the world. The dating² patterns have been continuing in the society with different twists since time immemorial. Dating and going out were still central, but the frequency decreased and the ages increased. Even the age of initial dating has been changing. It has been noted that dating changed in three major ways: greater opportunity for informal opposite-sex interaction, dating became less formal, and there was no set progression of stages from first meeting to marriage. This means it became more acceptable for women to initiate a dating relationship and to be responsible for paying at least a portion of the cost. While not entirely equal, dating was moving away from a strictly patriarchal ideology. It started with knowing the person with whom life has to be spent to have fun, dancing, movies and nowadays to even sexual experiences.

Different Systems for organizing dates¹

- a) **Online dating:** Instead of using a traditional matchmaker, online dating uses specifically targeted websites to meet new people.
- b) **Speed dating:** Where a group of people get together for several hours in public; one is given a set amount of time to sit and talk to a particular person before moving on to

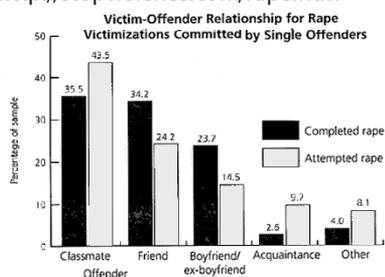
the next.

- c) **Mobile dating/Cell phone dating:** Where text messages to and from a mobile/cell phone carrier are used to show interest in others on the system. Can be web-based or online dating as well depending on the company.
- d) **Virtual dating:** A combination of video game playing and dating, where users create avatars and spend time in virtual worlds in an attempt to meet other avatars with the purpose of meeting for potential dates.
- e) **Singles events:** Where a group of singles are brought together to take part in various events for the purposes of meeting new people. Events can include such things as parties, workshops and games.
- f) **Blind date:** Where the people involved have not met each other previously. The match could have been arranged by mutual friends, relatives or by a dating system.
- g) **Group dating:** Group dating² is a modern pattern for dating where a group of single men and a group of single women organise a night out, with the hope of forming romantic partnerships. It is most popular in Japan where it is called "compa" or "gōkon". It has become popular because many Japanese find it difficult to find a partner. Group dating can also be found in many other countries, however, compa is very ritualized and unique. Generally, a single guy and girl who know each other organize the compa in advance, each agreeing to bring 3 or 4 eligible friends. The venue is usually a restaurant or anywhere people can eat, drink and make a bit of noise. In other cultures, group dating is becoming more popular as a safe alternative to single dating (especially blind dating), also helping to ease tension, since both parties will feel more comfortable having the company of their friends.

Reasons for dating in the Present Scenario

- i) **Fun and Overcome loneliness:** In the competitive era, every one is busy trying to prove him/her better than the other. So they try to find out the ways by which they can be relieved of the day to day stresses. In order to so they sometimes choose dating as one of the modes.
- ii) **Impression in friend circle:** Sometimes people take their partner or any friend for date just to show to their friend circle that s/he also has a partner. Sometimes in order to impress they even take more than one partner for date at different times.
- iii) **Affection towards a person/ physical intimacy:** Human beings being a social animal are always dependent on the other. They are always in search of a person with whom they can share all their thoughts and problems. So they select a person as a friend towards whom they are more affected or are feeling more intimated and take them for a date.
- iv) **Dissatisfied with their present relationship:** Sometimes because of the dissatisfaction in their existing relationship with their friend/spouse, the persons go on a date with another person/s searching for happiness.
- v) **Revenge:** Human brain is one of the complicated brains of all the living beings on this earth. It can do wonders if used in right direction but if used in wrong path can also do harm that no one might have thought. Sometimes a person comes close with whom they have to take revenge or who is associated with the person with whom they

Courtesy: <http://stopviolence.com/rape.htm>



have to take the revenge. In both the cases, after befriending they go out on date so that the person with whom they wanted to take revenge is pained directly or indirectly.

- vi) **Bindings of Society:** Human behavior even sexual, like many other kinds of activity engaged in by human beings, is generally governed by social rules that are culturally specific and vary widely. These social rules are referred to as sexual morality (what can and can not be done by society's rules) and sexual norms (what is and is not expected). These restrictions are often determined by religious beliefs or social customs.
- vii) **Sexual attraction/behavior:** Sexual attraction/behavior¹ is natural instinct to seek gratification, gifted to us by nature. For the life form it is the pleasure of release but for nature it is the purpose. Purpose of nature influences life form to react to the attraction. Reaction to attraction to act for the purpose of carnal appetite; we can call as sexual behavior.

There are two purposes of carnal attraction: 1st is passing gene and conceive to create new generation. 2nd is reaction of fluid during relief, as medication to retain body functioning just right. Youthful healthy body produces essence regularly and fast, so release of essence becomes essential objective that incites to act, is an essential taste of survival and tonic for body.

Our existence is because of this carnal activity. This activity is for release and release is for relief. Relief is the ultimate ecstasy that influences for action. Nature created a rule of gratification in order to achieve its purpose, which pulls our mind to act. Sexual behavior is under control of the negative and positive feature of our genetic material. Reaction of gene is by dominance of either; whichever is dominant will react and incite us to act for its objective. In the same time, gene also modifies self by interacting with the environment in order to maintain or survive. The negative and positive environment too play role to shape the gene. Dominant environment drags towards its characteristics to shape the gene character alike. Again the force of gene character if is stronger than existing environment, will dominate and act on its terms to assist to strengthen opposite environment. Sexual violence is the result of the dominance of negative behavior and the environment. Such characters are psychologically treated by influencing the opposite dominant environment to what one was undergoing before.

Consequences of dating

Positive consequences

- a) **Soul mate/better half:** Generally the people who go on date, find their soul mates/ better halves with whom they can spend the rest of their life tying nuptial knot.
- b) **Long time open relationship:** If the persons went on date are not interested in tying the nuptial knot, then they will be in a long time open relationship, which will last till the end of their lives.

Negative consequences

- a) **Broken Heart/ Post-traumatic stress disorder:** If the date turns out to be a one time affair and is not materializing, the way any one of the involved party is wishing, then it will lead the person to depression and anxiety.
- b) **Physical abuse:** Hitting/punching, kicking, slapping, biting, shoving, throwing objects, restraining another, or preventing them from leaving a room or residence may take place.
- c) **Emotional abuse and verbal abuse:** Name calling; controlling, humiliating or jealous behaviors, humiliation; attempts to undermine self-esteem.
- d) **Threats:** Threatening violence, or threatening to leave the relationship.
- e) **Isolation:** Attempts to prevent partner from spending time with others in order to isolate them from friends and family.
- f) **Harassment:** Repeated, unwanted and/or excessive phone calls, text messages or other communication.
- g) **Stalking:** Following and/or surveiling a would-be, current, or former partner.
- h) **Sexual Abuse/Date Rape:** When people think of rape, they might think of a stranger sexually attacking someone¹. But it's not only strangers who rape. In fact, about half of all people who are raped know the person who attacked them as depicted in the below figure.

Girls and women are most often raped, but guys also can be raped. Most friendships, acquaintances, and dates never lead to violence, of course. But, sadly, sometimes it happens. When forced sex occurs between two people who already know each other, it is known as date rape or acquaintance rape. Even if the two people know each other well, and even if they were intimate or had sex before, no one has the right to force a sexual act on another person against his or her will. Although it involves forced sex, rape is not about sex or passion. Rape has nothing to do with love. Rape is an act of aggression and violence. Rape is always the fault of the rapist. And that's also the case when two people are dating – or even in an intimate relationship. One person never owes the other person sex. If sex is forced against someone's will, that's rape.

Healthy relationships involve respect – including respect for the feelings of others. Someone who really cares about you will respect your wishes and not force or pressure you to have sex.

During date rapes generally date rape drugs¹, also known as "predatory drugs" or "club drugs", are used which are particularly dangerous when combined with alcohol. Common date rape drugs include Rohypnol, Ketamine, and GHB, a central nervous system sedative also known as "Liquid Ecstasy". The use of the drugs assists the attacker in a rape by altering the physique or mind. Drugs used to facilitate rape may have sedative, hypnotic, dissociative, and/or amnesiac effects, and may be added to a food or drink without the victim's knowledge².

Most date rape drugs are virtually undetectable, as they are colorless, odorless, and usually tasteless. Common side effects of the drugs are relaxation, drowsiness, loss of muscle control, nausea, blackout, coma, and sometimes death. They also have been known to lower blood pressure, cause stomach problems, confusion, amnesia, and hallucinations³.

Approximately 90% of rape victims, who knew their attacker, do not report the attack to authorities because of fear of guilt/responsibility, self blame, embarrassment, helplessness, denial, and a number of other emotional factors. There is also often confusion of whether or not consent was given, further causing the attack to remain anonymous. A person who has been a victim of rape may become distant, quiet, and may lack motivation. Some become depressed,

anxious, or suffer from Post Traumatic Stress Disorder (PTSD), while others may become promiscuous. Some of the victims choose to seek help through counseling.

It is imperative that any investigation into the suspected use of date rape drugs involve an immediate blood test, as waiting too long to test for the presence of drugs may cause false negatives, because these drugs are quickly metabolized and eliminated by the body. Trying to deduce from the symptoms whether or not date rape drugs have been used can cause false positives.

Conclusion

Dating which started with a good objective of knowing the other person with whom one wishes to spend his/her life in a better manner is changing drastically in the current scenario. The negative consequences of the dating are very drastic and may make a person's life hell and may also even leave him/her on the verge of death. So whenever one goes on a date with a person, it should be with proper care and only after properly understanding the psychology of the person, so that no untoward incident happens.

A rare case of elongated styloid process

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Key words

Elongated Styloid process, Eagle's Syndrome, Skull bone.

Abstract

Many studies have been done on the elongated styloid process. -The close proximity of styloid process to vital neurovascular structures in the neck makes it clinically significant. Eagle's syndrome is characterized by craniofacial and cervical pain due to an elongated styloid process. The normal length of styloid process is between 20 mm-25 mm¹. However, the length of styloid process varies between 15.2mm-47.7mm according to Moffat et al² study. The present case report of a female skeletal remains sent for expert opinion from Medical Officer of Primary Health Center Kalahangaraga, Gulbarga district, highlights the skull bone with bilateral styloid process measuring 40 mm on right side and 55 mm on left side.

Introduction

The normal average length of styloid process is 25 mm³. Different studies have shown variation in the length of styloid process. An elongated styloid process or calcified stylohyoid ligament is seen in Eagle's syndrome which is characterized by craniofacial or cervical pain. Patients with Eagle's syndrome may present various signs and symptoms. Eagle first described clinical findings of this syndrome in 1937. As the diagnosis is not very easy, it is reported that the number of cases are underestimated in the population. Eagle's syndrome when suspected clinically is confirmed radiologically with the help of Three Dimensional- Computed Tomography (3D-CT).

Materials and methods

Nine cases of skeletal remains, we received from January 2007 to December 2009 for expert opinion regarding the identification of age, sex, time since death and cause of death from various primary health centers from Gulbarga and Bidar district to Department of Forensic Medicine and Toxicology, M. R. Medical College, Gulbarga. During the examination of the sent skull bone, length of styloid processes, the distances between the bases, mid points and the tips of the two styloid processes were recorded. The distance of base of the styloid processes from the external acoustic meatus were also noted on both sides. The procurement of clinical details from relevant sources was tried but proved unfruitful.

Case report

The present case with elongated styloid process is concerned with expert opinion No-04/2009. This case was sent

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by Medical Officer of Kalahangaraga Primary Health Center, Gulbarga district for identification of age, sex, time since death and cause of death. The deceased met with an accident (RTA) at 2pm on 23/05/08 in Jewargi. Then the deceased was taken to Government Hospital Jewargi from there the deceased was referred to Higher Center in Gulbarga, on the way to Gulbarga the deceased died. Later the deceased was buried in Gulbarga. On 19/06/08 the son of the deceased filed a complaint in Jewargi Police Station against the alleged Auto Driver. Exhumation was done on 02/08/08 and Postmortem was conducted by Medical Officer of Kalahangaraga Primary Health Center, Gulbarga district. Medical Officer after conducting the Postmortem sent the Skeletal remains for Expert Opinion to the Department of Forensic Medicine M R Medical College, Gulbarga in the form of the sealed and labeled cardboard box containing Skull with few other bones.

On examination of skull, the right sided styloid process measured 40 mm and left sided styloid process measured 55mm. The distance between the bases of styloid processes was 75mm, distance between mid points of styloid processes was 70mm and the distance between the tips of styloid processes was 61 mm. The distance from the base of the styloid processes to the external acoustic meatus measured 9mm on both the sides.

Discussion

In the present study of the skull of a female about 60 years of age, the styloid process measured 40mm on right side and 55mm on the left side. On comparing with other reported studies, it was concluded that both the styloid processes were markedly elongated. Kaufman et al⁴, reported that the length of 30mm is the upper limit for normal styloid process. Moreover, Monsour and Young⁵ concluded that the diagnosis of an elongated styloid process when it was longer than 40mm. Keur et al⁶ stated that, if the length of the process or the mineralized part of ligaments which appeared in radiography was 30mm or more, this could be considered an elongated styloid process. Jung et al⁷ suggested that the styloid process should be considered to be elongated, when its length exceeds 45mm. Thot et al⁸ reported that the length of the left side styloid ranged from 07mm to 16mm, and on the right side, from

Fig 1: Showing base of the Skull with bilateral elongated Styloid Processes.



Fig 2: Showing measurement of the left Styloid Process.



Fig 3: Showing measurement of the right Styloid Process.



08mm to 24mm. The average lengths for the left and right styloids were 15.2mm and 15.9mm, respectively, in Indian subjects. Thot et al⁸ stressed that length in isolation is not a risk factor, but that its combination with increased acuity in deviation from the norm, both anteriorly and medially, makes the elongated styloid process the sole cause of Eagle's syndrome. Many studies were done on the length of the styloid process and found rare case of elongated styloid process. A study done by G Rath and C Anand⁹, found elongated styloid process in one skull out of 232 Indian human skulls. Since an elongated styloid process is very rarely seen, it can aid in the identification of the person. Rizzatti- Barbosa¹⁰ reported that an anatomical variant of stylohyoid ligament complex was more frequent in the elderly female population, although this abnormality was present in both sexes. There was a greater tendency for the abnormality to be present in patients between 60 and 79 year of age.

Conclusion

The present study emphasizes the uncommon occurrence of elongated styloid process of the Skull and this finding is usually seen in the elderly females. This fact can be used as an aid in the identification of a person, and gives an idea about the age of the person and sex of the individual.

Reference

1. Prabhu, Kumar A, Nayak S R, Pai M M, Vadgaonkar R, Krishnamurthy A, Madhan Kumar S J. An Unusually Lengthy Styloid Process, Singapore Med J 2007; 48(2):e34-36.
2. Moffat DA, Ramsden RT, Shaw HJ. The styloid process syndrome: Etiological factors and surgical management. J Laryngol Otol 1997; 91:279-294.
3. Gray's Anatomy, The Anatomical Basis of Clinical Practice. 39th Edition, Elsevier Limited 2005: 470.
4. Kaufman SM, Elzay RP, Irish EF. Styloid process variation: radiologic and clinical study. Arch Otolaryngol 1970; 91:462-463.
5. Mansour P and Young W J. Variability of styloid process and stylohyoid ligament in panoramic radiographs. Arch Otolaryngol 1986; 61:522-526.
6. Keur JJ, Campbell JP, McCarthy JF, Ralph WJ. The clinical significance of the Elongated styloid process. Oral Surg Oral Med Oral Pathol 1986; 61:399-404.
7. Jung T, Tschernitschek H, Hippen H, Schneider B, Borchers L. Elongated styloid process: when is it really elongated? Dentomaxillofac Radiol 2004; 33:119-24.
8. Thot B, Revel S, Mohandas R, Rao AV, Kumar A. Eagle's syndrome, Anatomy of the styloid process. Indian J Dent Res 2000; 11:65-70.
9. G Rath and C Anand. Abnormal styloid process in a human skull, Surg Radiol Anat (1991) 13: 227-229.
10. Rizzatti-Barbosa CM, Ribeiro MC, Silva-Concilio LR, Di Hipolito O, Ambrosano GM. Is an elongated stylohyoid process prevalent in the elderly? A radiographic study in a Brazilian population. Gerodontology 2005; 22: 112-5.

Burns and burning issue in Gulberga, Karnataka

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Abstract

Deaths due to burns though rare in the western context are frequently encountered in the Indian scenario. A study was conducted at Government General Hospital Gulbarga over a period of five years to find out the incidence and influence of different factors leading to fatal burns injury. Out of the total 525 cases studied maximum were found in the age group of 20-30 years. 115 patients were brought dead and the remaining 410 succumbed later after the admission. Among these 161 cases survived for five days after the injury. Most common cause of death in our study was septicemia which accounted for 343 cases. Maximum number of deaths i.e., 450 was accidental in nature and least common was homicidal deaths accounting for only 23 cases. The paper also describes the comparison between our study and studies conducted by different authors across the country to find out the similarities and dissimilarities in various factors affecting the burns victims.

Key words

Burns; Death; Society;

Introduction

Thermal burns are more common causes of unnatural deaths. Incidence of death due to burns stands next to road traffic accidents in India. Social evils like dowry deaths also contribute significantly to the number of deaths by burns. On an average 1/4th of the deaths constitute death due to burns among all postmortem examinations conducted. Mortality rate due to burns is much more in India than any other developed countries.

Considering the magnitude of the problem, this study was conducted to know the incidence rate as well as influence of various factors on burns cases in this part of the country.

Materials and methodology

The present study was conducted over a period of 5 years from January 1998 to December 2002 at Mortuary, Government General Hospital, Gulbarga. Information was collected from relatives / friends/ neighbours at the time of occurrence of the incident, inquest report and relevant documents.

Information from case history papers and relevant hospital documents of the victims, along with the autopsy findings, were tabulated and statistically analysed.

Results

Discussion

The result / information obtained is compared with the studies conducted in different parts of our country.

Incidence

The incidence of death due to burns is 25.41% which is the second commonest cause of death next to road traffic

accidents in the present study. This finding is comparable with the other studies done by V.N. Ambade¹ Rao.N.G² in which the incidence was 21.6% and 22.73% respectively.

Age

Maximum number of deaths i.e., 44.84% occurred during this study. In the age group 21-30 years, and second highest deaths occurred in age group of 11-20 years. Incidence of death in the age group above 61 years was minimum i.e., 8 cases and 43 cases were children under 10 years. Mohanty MK et al³ observed that majority of deaths (46%) occurred in 21-30 years of ages group. Ambade VN et al¹ in a study on trends of burn deaths it was found that most of the victims were between 11-40 years with peak at 21-30 years (47.1%).

So, the present series is in accordance with the above studies.

Sex distribution

Females outnumbered males by about 3-times i.e., the incidences being 73.20% in females and 26.80% in males. In fact by close observation more number of deaths occurred in the young females in the age group 15-30 years. This study clearly depicts the magnitude of the problem in young females, particularly in the early married life. Mohanty MK et al³ in a study on death by burning found that female preponderance was more with 79.5%. Ambade VN et al¹ observed female predominance (74.2%) in burning with male-female ratio equal to 1:2.9. Ashish K. Jaiswal⁴ found that the incidences was more in females as an absolute number (70.3%). Mago V⁵ found that female outnumbered males with a ratio of 1.17:1.

So, the present study is in consistent with the above studies conducted by Mohanty³, Ambades¹, Ashish⁴ and Mago⁵

Place of death

The cases brought dead includes the cases which either dies just before admission and treatment or those who died on the spot, died in other hospitals, nursing homes and brought dead directly to the mortuary. Apart from the cases brought dead directly to the mortuary for above said reasons, the other major contributing factors being lack of transportation facilities, illiteracy, awareness are among the major contributions to brought dead cases.

The hospital deaths and brought dead ratio is 3:1 approximately.

Period of survival

Period of survival is directly related to surface area of body involved in burns. In this study maximum number of cases survived for more than 5 days period i.e., 214 cases (30.66%). In a study, Jairama et al⁶ showed that maximum survival period was less than 5 days i.e., in 91% cases. Dalbir Singh⁷ found that in 86% cases, period of survival was within one week.

The analysis made by Jairaman⁶ and Dalbirsingh⁷ and the present study's duration of survival is approximately 5 days, which is coherent.

Table 1: Incidence of Burns.

Year	Autopsy in case of burns
1998	106
1999	096
2000	121
2001	105
2002	97
Total	525

Table 2: age wise distribution

Age group	No. of cases
Upto 10 years	33
10-20 years	123
20-30 years	235
30-40 years	74
40-50 Years	32
50-60 Years	22
60 and above	06
Total	525

Table 3: Sex wise distribution

Year	Male	Female	Total
1998	26	80	106
1999	20	76	96
2000	31	90	121
2001	32	73	105
2002	35	62	97
Total	144	381	525

Table 4: Hospital deaths Vs Brought dead.

Year	Brought dead	Hospital death	Total
1998	20	86	106
1999	9	87	96
2000	57	64	121
2001	19	86	105
2002	10	87	97

Cause of death

The findings of the present series noting the sepsis as the most important factor for the cause of death, as the period of survival in maximum number of cases is more than 5 days. In the present study, out of 698 cases, maximum deaths i.e., 65.54% were due to septicemic shock and minimum i.e., 2.57% were due to Neurogena shock.

This is because lot of the victims of burns, who survived the initial 24 hours after burns, succumbs to infection of the burnt area and its complications. Burns cause devitalization of tissue leaving extensive raw areas, which usually remain moist due to the outflow of serous exudate. The exposed moist area along with the dead and devitalized tissue provides the optimum environment favoring colonization and proliferation of numerous microorganisms, which is further enhanced by the depression of the immune response. All these factors contribute towards sepsis in a burns victim⁸.

Tripathi CB et al⁹ found 3.92% death due to septicemia, while Nageshkumar Rao² found that 53% of cases died due to septicemia.

Manner of death

Maximum death were due to accidental burns (85.67%), followed by suicidal burns (9.89%) and the minimum number of cases were of homicidal burns (4.44%).

But the above facts remain controversial, when we actually looked into the real history and circumstantial evidences. History in such cases was debatable, because of common age

Table 5: Period of survival.

Period of survival	No. of cases
Upto 12 hours	91
1 st day	102
2 nd day	80
3 rd day	27
4 th day	64
5 th day	161
Total	525

Table 6: Cause of death.

Cause	No. of cases
Neurogenic	14
Hypovilemic	113
Toxicemic	55
Septicemia	343
Total	525

Table 8: Manner of death.

	1998	1999	2000	2001	2002	Total
Accidental	94	83	104	88	81	450
Suicidal	9	8	12	12	11	52
Homicidal	3	5	5	5	5	23
Total	106	96	121	105	97	525

group and allegations of dowry deaths.

In many cases of alleged suicide and accidental deaths, when questions were put to the relatives or the attendants of deceased, a hostile attitude was often noticed which arouse suspicion of foul play.

In some of these cases, the fact is that the relatives of the deceased were forced to give false account of cause of death, to make it appear accidental though in all probability a clear case of suicide.

Further highly selective factors, such as socioeconomic conditions, domestic quarrels, disturbed domestic life, chronic disease, mental disorder, disappointment in love or failure in examination etc, may determine the number of suicidal cases.

Conclusion

Accidental burns are mostly preventable by adequate safety measures and safety education. "Bride burning" is a social evil unmatched in its cruelty and cynicism in today's civilized society. Any discussion on its etiopathogenesis and remedial measures must take into account the socio-cultural and economic ramifications underlying this scourge. Legal measures however, harsh or deterrent, cannot suffice to combat this scourge due to complete dependence of the woman on her husband and in-laws. There is a need for more stringent laws for possession and use of explosive and inclusive and inflammable, materials to prevent accidents.

References

1. Ambade VN et al. Study of burn deaths in Nagpur, Central India. Burns. 2006 Nov; 32(7): 902-8.
2. Nageshkumar G Rao. Study of fatal female burns in Manipal. JFMT. 1997 Jul-Dec XIV (2).
3. Mohanty MK et al. Self inflicted burns fatalities in Manipal, India. Med Sci Law. 2005 Jan;45(1):27-30.
4. Ashish K Jaisal. Epidemiological and sociocultural study of burn patients in MY hospital, Indore, India. Med Sci Law. 2007; 40(2); 158-163.
5. Mago.V. Epidemiological and mortality of burns in JNMC Hospital, AMU Aligarh: A 5 year study. Indian Journal of

- Community. 2004; 29(4): 10-12.
6. Jairaman V et al. Burns in Madras, India: An Analysis of 1368 patients in one year. Burns. Aug 1993; 19(4): 339-44.
 7. Dalbir Singh et al. Recent trends in mortality in North – West India and its preventive aspects. JAFM. 1997; 19(4).
 8. Sharma SR. Study of Postmortem findings in burns. JFMT. 1984; 45(1).
 9. Tripathi CB et al. Burnt wives: A Sociological study. Int J Med Toxicol & Legal Med. 1999 July – Dec; 2(1).

Pattern of appearance and age from sesamoid bones in hand In Ahmedabad population

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Abstract

Sesamoid bones (Arabic word meaning seed like small bone arising in tendons at the point of maximum friction) were studied in the present study conducted on 592 subject of Ahmadabad. The commonest site at which sesamoid bone observed in hand was the ulnar side of head of first metacarpal bone (in 1001 boys and girls). The other sites in descending order of frequency were the radial side of head of 1st metacarpal bone (in 36% cases), head of 5th metacarpal bone (in 30% cases), head of 2nd metacarpal bone (in 14% cases), ulnar side of head of 1st phalanx of thumb (in 12.46% cases), The unusual sites were the head of 3rd and 4th metacarpal bones (in 3 girls only). The only sesamoid bone which followed definite age pattern was the one on the ulnar side of head of 1st metacarpal bone. The appearance of sesamoid bones was earlier in girls than boys by 3 to 4 years,

Introduction

Various workers inside as well as out side India worked on assessment of age from epiphyscal union in different bones on the body. Only few workers worked out age from the appearance of patella which is considered to be bigger sesamoid bone in the body. According to Joseph (1951) the sesamoid bones in hand appear in teen age and earlier in girls by three years. Except this study we did not come across any work on the age pattern of appearance of sesamoid bones. In the light of this an effort has been made to work out the age pattern of appearance of sesamoid bones in hand in the present study and its possible contribution towards age assessment in medico legal cases.

Materials and methods

The present study was conducted on 592 subjects (313 boys and 279 girls) from the schools and colleges of Ahmadabad. The criteria for selecting the cases were as given below:

- 1) Bonafide residents of Ahmadabad or the families settled in Ahmadabad for the last three generations.
- 2) Cases whose data of birth could be verified either from parents, horoscope, or hospital record were included in the present study.
- 3) Only subjects of sound health were included in the present study. Clinical screening Of the cases for the diseases known to affect the epiphysis union was also done.
- 4) Factors like dietary habits (Veg/Non-Veg.), economic status, Rural/Urban habitat, eruption of teeth, height of the subjects and physical exercise were also studied to find out any correlation to epiphysis union.

The age range covered in the present study was between 10 years to 21 years in boys and 10 to 20 years in girls. Groups were made at an interval of every six months e.g. the age of 10 years as group I, 10.1/2 years as group II and 11 years as group III and so on. In each group 10 to 15 subjects were included. In each case skia gram of wrist joint including hand was taken in P.A view.

Observations and discussion

The different sites at which the appearance of sesamoid bones observed were (in descending order of frequency):

- 1) Ulnar side of Head of 1st Metacarpal bone.
- 2) Radial side of head of 1st Metacarpal bone.
- 3) Head of 5th Metacarpal bone.
- 4) Head of 2nd Metacarpal bone.
- 5) Head of 1st Phalanx of Thumb.
- 6) Heads of 3rd and 4th Metacarpal Bones.

1. Ulnar side of head of 1st metacarpal bone:

Girls: The youngest case showing appearance was 10 years, 2 months, 20 days old. 100% girls showed appearance by the age of 13 1/2 years. Average age of appearance 12 1/2 years. The acceptable range: 11 1/2 years to 13 1/2 years.

Boys: The youngest case showing appearance was 11 year 11 months, 06 days old. 100% boys showed appearance by the age of 17 years. Average age of appearance: 16 years. Acceptable range: 15 years to 17 years.

This was the only site at which sesamoid bone followed definite age pattern both in boys and girls. The appearance of this bone was earlier in girls by 3 to 4 years than boys. It may be fused to the head of metacarpal or lie separately. It was also interestingly observed in few cases in both sexes that there were two sesamoid bones at this site instead of one.

2. Radial side of head of 1st metacarpal bone

Girls: Youngest girl showing appearance was 10 years, 8 months, 7 days old- After this age only 84 girls out of 261 cases showed appearance i.e. the appearance was in 32.18% cases.

Boys: Youngest boy showing appearance was 13 years, 6 months, 15 days old. After this age only 82 boys out of 206 cases showed appearance i.e. the appearance was in 39.18% cases. Its frequency of appearance was 2nd in descending order.

3. Head of 5th metacarpal bone

Girls: Youngest girl showing appearance was 12 years, 5 months, 18 days old. After this age 70 girls out of 218 cases showed appearance i.e. the appearance was: 32.11%.

Boys: Youngest boy showing appearance was 15 years, 7 months 26 days old. After this age 40 boys out of 142 cases showed appearance i.e. the appearance was 28.16%. The frequency of appearance of this bone was third in descending order.

4. Head of 2nd metacarpal bone

Girls: Youngest girl showing appearance was 12 years, 5 months 26 days old. After this age only 34 girls out of 218 cases showed appearance i.e. the appearance was only in 15.5% cases.

Boys: The youngest boy showing appearance was 16 years, 3 months, 03 days old. After this age only 15 boys out of 113

Age at which Nil cases showed	Age at which 10% cases showed	Age at which 25% cases showed	Age at which 50% cases showed	Age at which 75% case showed	Age at which 100% cases showed
appearance 10.5 years 13.5 years	appearance 14 years	appearance 11 years 14.5 years	appearance 11.5 years 15 years	appearance 12.5 years 16 years	appearance 13.5 years 17 years

cases showed appearance i.e. the appearance was only 12.7% cases

It was fourth in descending order of frequency in its incidence of appearance.

5. Head of 1st phalanx of thumb

Girls: Youngest girl showing appearance was 12 years, 5 months 26 days old. After this age 25 girls out of 218 cases showed appearance i.e. the appearance was in 11.46 % cases.

Boys: Youngest boy showing appearance was 17 years, 01 month, 23 days old. After this age 14 boys out of 104 cases showed appearance i.e. the appearance was in 13.46% case. The frequency of appearance of this bone was fifth in descending order.

6. Head of 3rd metacarpal bone

The appearance of sesamoid bone at this site was occasional. It was seen only in 3 girls. The earliest case showing appearance was 12 years 5 months 21 days old. It was not seen in boys.

7. Head of 4th metacarpal

The sesamoid bone at this site was observed in only one girl aged 15 years 5 months and 9 days old. It was not seen in boys.

Conclusion

The sesamoid bone on the ulnar side of head of 1st metacarpal bone appeared in 100% cases and also followed a specific age pattern and hence it is of use in age estimation. As the other sesamoids are not appearing in 100% cases, they are only of limited use by providing the lower age limit before which they never appeared. Their appearance earlier in girls by 3-4 years is in accordance with the finding of Joseph (1951). In cases of mutilation where only a hand is coming for examination these sesamoid bones along with carpals, metacarpals and phalanges may prove useful in age assessment.

References

1. Basmajian, J.V. (1960), Grant's Method of anatomy, 10th ed, pp-5-6, Baltimore, U.S.A.
2. Frazer, J.E. (1965), Anatomy of the Human Skeleton, 6th edr pp - 33, Gloucester Place, London.
3. Joseph, J. (1951), The sesamoid bones of hand and time of fusion of the epiphyses of thumb, J. Anat. Lond., Vol: 85, pp- 230-41.
4. Warwick, R, and Williams, P.L. (1980), Gray's Anatomy, 36th ed, pp- 417-418, Edinburgh, London.

A study on victims and accused of sexual offences in South Delhi

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Abstract

Sexual offences are the most heinous crimes against women. It is the most barbarous and humiliating and the women and children remain the most vulnerable group of this crime. Find out and preserving the evidences of sexual offences are the importance task for doctors and poor medical evidences are often responsible for low conviction rate of criminals. Careful analysis of preserved biological specimens are yielding the vital evidences in criminal investigations. Victims 11- 20 years and accused 21- 25 years of age groups are most vulnerable and dangerous for sexual assault.

Key words

Sexual assaults, poor evidences, low conviction rate, younger groups are vulnerable.

Introduction

Sexual offences are acts of sexual intercourse with a second person or an animal to obtain sexual gratification. The Law and customs of the society normally permit heterosexual intercourse between a man and his own wife as provided by the nature. The crime of rape requires that the perpetrator used force and the victim actively resist to the last moment.

All the crime, sexual offences are the most barbarous and humiliating and the women and children remain the most vulnerable group of this crime. The alarming rise in the rate of sexual offences worldwide represents a major health problems¹.

In USA, an estimate of one in every four women and children², in Nigeria four out of every ten women are victims of sexual assaults³. In South Africa, the incidence of rape is approximately 300 per 100,000 women⁴. In India over the last five years, rape cases have been shown alternate increasing and decreasing trends with increase of 6.6% in the year 2000 over 1999 and a decline of 2.5% in the year 2001. In the year 2001, a total of 16,075 (1.6 per 100,000 population) cases of rape against women and 2,113 (0.2 per 100,000 Population) cases of child rape were reported all over the India. It shows that 11.2% and 19.5% of total crimes are against women and children respectively. The Delhi has been ranked 5th for the incidence of rape cases against women and 1st for the rape on children (0.8 per 100,000 Population)⁵.

Find out and preserving the evidences of sexual offences is an important task for a Doctor⁶. Poor medical evidences are often responsible for low conviction rate of the criminals⁴. Therefore, proper and correctly perform physical examination in sexual offences cases is of crucial importance. Careful analysis of preserved biological specimens such as blood, semen, saliva often yields vital evidence in the cotemporary criminal investigation⁶. In the present article, the incidence and pattern of sexual offences in South Delhi along with the demographic variables, findings of physical and genital examination of victims and accused and results of medical evidences collected are presented.

Material and methods

During the study period (January 2001- September 2002)

a total 207 victims and accused of sexual offences from South Delhi were brought to the emergency of All India Institute of Medical Sciences, New Delhi for medical examination. Female victims were examined by the doctors of the Department of Obstetrics and Gynaecology and male victims and accused were examined by the doctors of the Department of Forensic Medicine. Details pertaining to the age, sex, religion, literacy, socioeconomic status, site of incidence, time interval between incidence and medical examination, number of assailants, relationship with assailants, findings of physical and genital examination, results of evidence collected during examination were noted.

Results

A total of 207 sexual offences cases (including victims and accused) 90 (43.47%) were victims, 117 (56.53%) were accused. Out of 90 victims, 88.9% were females 11.1% were males (all were sodomy victims). The age of the victims ranged from a 4years old child to a 60 years old women. The most effected group was victims between 11-20 years (68.9%) followed by 0-10 years (12.2%). Two cases were above 50 years of age. The female victims were mostly in the age group of 16-20 years while 5 male victims were of the age group of 6-10 years. But the assailants, the 21-25 years (39.31%) of age group are the dangerous to attack the victims (Table-1).

Sixty victims were Hindu (75.5%), while 95 assailants were Hindu (81.19%), 73 victims (81.11%) and 101 assailants (86.32%) were unmarried (Table-2)

Less educated upto class V (33.3%) in case of victims and the assailants (24.78%). Lower socioeconomic background of victims 83 (92.22%) and 104(88.88%) of assailants (Table-3)

The vast majority of victims were knew the assailants . In 52 (44.4%) cases there were acquaintance between victims and assailants , in 30% cases they were closed friends while in 22 (18.8%) cases assailants were stranger. The common sites of offences were the houses of victims (41.1%) followed by the accused houses (28.9%) (Table-4)

Maximum of the victims were brought for medical examination after 5-7 days of the incident (25.5%) while assailants were 28(23.93%). Only 9(10%) victims and 5(4.27%) assailants were examined in the same day of incidence (Table-5).

In 39(43.3%) cases there was invalid consensual sexual intercourse followed by forcible rape in 23(25.5%) cases and statutory rape in 18(20%) cases. Ten (11.11%) cases were unnatural sexual offences (Table-6).

There were 7(7.77%) cases of gang rape, with a maximum of 5 assailants were involved in one case. Four (4.4%) victims were killed following the rape, among those, the youngest rape-murder victim was of a 6 years old girl. The swabs collected from 73 victims and 25 assailants showed positivity of spermatozoa in 5(5.5%) cases in victims and only one (0.85%) case in assailants (Table-7) .

Discussion

Sexual offences one of the most heinous crimes against

women. Various social Organizations and Women Welfare Organizations, legal and administrative steps have been taken by the Government to give protect to women against such sexual crimes. Sexual assault is a neglected public health issue in most of the developing countries and there is even smaller percentage of reporting of sexual assaults⁷. Only 10-50% female victims report such assault⁸. The under reporting of sexual assault is mainly due to social stigma, prejudice because of marriage being considered promiscuous and responsible for incidence, humiliation, shame, embarrassment caused by appearance and cross examination in the court of law. Publicity in the press, risk of losing the love and respect of the society, friends, her husband, if she married⁹.

The present study was carried out on 90 victims and 117 assailants of sexual offences in South Delhi from January 2001 to September 2002. In the previous studies, Sagar et al¹⁰ reported 38 victims in 1991 and Bhardwaj et al¹¹ reported 80 victims in the years 1993 -1994 in South Delhi population.

In this study, the majority of victims were females. The results are in agreement with the study of Sagar et al¹⁰, Bhardwaj et al¹¹, Grossing et al¹², Riggs et al¹⁵ and Fimate et al.¹⁶ In keeping the study of Bhardwaj et al¹¹, all males were victims of sodomy and only one female victim was found anal intercourse in this study. In the study of Riggs et al,¹⁵ 17% victims were assaulted with anal intercourse. Bhardwaj et al¹¹ and Malhotra et al⁷ found that 40.70% victims of sexual assault were in the age group of 13-20 years. But in DuMunt et al¹³ study, majority of victims were of age group of 15-20 years, while in the study of Islam et al¹⁴ 33.5% of victims were between 12-15 years. In this study, 31 victims were between 16-20 years of age. Malhotra et al⁷ reported that the majority (76.9%) of victims were adolescents. From this study, it can be concluded that no age is safe from rape. It is comparatively easy for assailants to rape children, as they are innocent and cannot physically resist and defend themselves. In India and also some other countries, rape of children is due to the superstitious belief that venereal diseases can be cured by sexual intercourse with a virgin¹⁷.

In this study, the majority of victims and assailants were Hindu. The findings are in agreement with the study of Fimate et al¹⁶ (57%), Majority of the victims and accused were poorly educated (upto class V) (n=30,33.33% of victims, 24.78% of accused) and from a low socioeconomic background (n= 83, 92.22, n=104, 88.88% respectively) with a monthly income below 5000 rupees. Islam et al¹⁴ reported that majority of victims were illiterate.

In this study, half of the victims were known to the assailants, only 19% of the cases the assailants were stranger. Similar findings have been observed by Fimate et al¹⁶ of the victims and assailants relationship, acquaintance (69.7%) and strangers (25.6%). While Islam et al¹⁴ reported that in majority of cases the victims knew the assailants. The National data of India⁵ shows that in majority of cases the assailants were the neighbour. Strangers have been reported as the common assailant in the study of Okonkwo et al¹³ (34.8%), Riggs et al¹⁵ (39%) and Dumont et al¹³ (49.2%). Malhotra et al⁷ reported that rape by person acquainted with victims is common for girls less than 10 years of age. Rape or assault by strangers increased significantly with age. Grossin et al¹² reported that in half of the cases of victims examined within 72 hours, the assailant was a stranger, while in those examined after 72 hours the assailant the mainly the father (30%).

The most commonest site of offences was the victims houses (41%) as reported by Grossin et al¹² and Okonkwo et al¹³. A quarter of victims in this study were brought for medical examination 5-7 days and in a third of the cases within 72 hours of the incident. In the study of Grossin et al¹² of the victims were presented for medical examination within 72 hours. In the study of DuMont et al¹³ (40.1%) the victims were

reported to the hospital within 2 -6 hours after incident, while Islam et al¹⁴ reported that 23.7% of victims reported within 72 hours.

There was invalid consensual sexual intercourse in 39(43.33%) cases as found in this study. According to the section 375 IPC, a woman above 16 years is capable of giving consent to an act of sexual intercourse, but the consent must be free and voluntary and giving while she is in full possession of her faculties. Under section 114 A of Indian Evidence Act 1872 if a woman in her statement before the court states that she did not consent, the court presumes that she did not consent¹⁷. In this study found that seven cases (7.8%) cases there was more than one assailants (gang rape). In the study of Riggs et al 15 more than one assailants was involved in 20% cases. Grossin et al¹² reported that in 15% of cases examined within 72 hours and 10% of cases examined after more than one assailant was involved.

Killing after sexual act could be either in panic or to destroy a witness of crime¹⁷. Only 10–20% cases murder is committed by the urge of sexual gratification. 18 Four victims (44.4%) were killed following rape as found in this study. Victims being under influence of alcohol and drugs was reported by Okonkwo et al¹³ (39% and 12% respectively) and DuMont et al¹³ (41.7% of alcohol). Though it was not found in this study.

A quarter of victims had a few simple to grievous injuries on there bodies. Six victims had extra genital, 10 victims had genital and 7 victims had combined genital and extra genital injuries. All of them were involved in forceful rape. Rupture of hymen was found in 85% cases of victims and maximum cases were of old rupture. Islam et al¹⁴ reported extra genital violence in 91 cases. Rupture of hymen was observed in 38.9% cases with fresh tears in fourchette in 2-6% of cases. Malhotra et al⁷ reported injuries in 32.3%, extra genital injuries 21.5% cases. DuMont et al¹³ reported injuries on bodies of victims in 64.2% cases. Grossin et al¹² general body trauma and genital trauma in 39.1% cases and 35.1% cases examined within 72 hours and 6.3% and 19.5% cases examined after 72 hours respectively. Hymenal (1%), vulvovaginal (20%) and anal lesion (7%) cases were found. Riggs et al¹⁵ found general body trauma (67%) and genital injuries (53%). Absent of genital injuries in the victims examined could be due to various reasons. Majorities of the victims were adults with prior sexual activities. No hymenal lesions can be seen in such victims because they have residual hymen. Genital injuries are common in children and post menopausal women. The nature and time of assault determines whether injuries would normally be expected¹⁹. The absence of physical injuries may not contradict the allegation as absence of general body trauma could be explained by the vulnerability of the victims and by the fact that the assailant could have exercised authority over the victims, so that the victim offers minimum resistance¹⁹. Rapidly healing injuries can be missed in cases with delayed examination or there may be false allegation²⁰. Similarly, value of examination immediately following an alleged incident is limited by the fact that bruises may not become apparent for at least 48 hours. It is widely acknowledged by the medical profession that absence of injuries or abnormality of an anal area in sodomy cases does not refute a history of anal intercourse as these are very few abnormalities which provide conclusive evidence of anal intercourse²¹.

Swabs collected 73 cases of victims and 25 cases of accused show positivity for spermatozoa in 5(5.55%) and acidphosphatage in 3.33% cases of victims. But in one case spermatozoa was found positive in case of accused. In the study of Grossin et al¹² spermatozoa were positive in 30.3% cases while Riggs et al¹⁵ reported that evidence of sperm and semen in 48% cases. Devis and Wilson (1974)²² observed that seminal blood group antigens could be detected on swabs collected within 48 hours, acid phosphatase upto 2 days,

Table 1: Age of victims and accused of sexual offences

VICTIMS				ACCUSED			
Age (Years)	Female N= 80	Male N=10	Number	%	Age (Years)	Number	%
0 – 10	6	5	11	12.2	15 -20	33	28.19 %
11 – 20	58	4	62	68.9	21 – 25	46	39.31
21 – 30	8	0	8	8.9	26 – 35	26	22.12
31 – 40	2	1	3	3.3	36 – 45	7	5.97
41 – 5	4	0	4	4.4	46 – 50	-	0
50+	2	0	2	2.2	50+	5	4.27

Table 2: Religion and marital status of Victims and Accused sexual offences

VICTIMS			ACCUSED		
Religion	No.	Percent	Religion	No.	Percent
Hindu	68	75.5	Hindu	95	81.19
Muslim	20	22.2	Muslim	22	18.80
Christian	1	1.1	Christian	-	0
Sikh	1	1.1	Sikh	-	0
Married	15	16.7	Married	16	13.67
Unmarried	73	81.1	Unmarried	101	86.32
Widow	2	2.2			

Table 3: Literacy and Socio economic status of Victims and Accused of sexual offences

VICTIMS			ACCUSED		
Education (Class)	No.	%	Education (Class)	No.	%
Not literate	26	28.9	Not literate	20	17.09
Class I-Class V	30	33.3	Class I-Class V	29	24.78
Class VI –Class IX	22	24.4	Class VI –Class IX	52	44.44
Class X-Class XII	5	5.5	Class X-Class XII	8	6.83
Above Class XII	7	7.8	Above Class XII	8	6.83
Low socioeconomic	83	92.22	Low socioeconomic	104	88.88
Middle socioeconomic	6	6.67	Middle socioeconomic	11	9.40
Upper socioeconomic	1	1.1	Upper socioeconomic	2	1.70

Table 4: Relationship of assailants with victims and places of sexual offences

Types of relationship	No.	%	Places of Incidence	No.	%
Acquaintance	52	44.1%	Victim's house	37	41.1%
Stranger	22	18.8%	Accused house	26	28.9%
Closed friends	36	30.8%	Relatives house	9	10.0%
Neighbour	2	1.8%	Roadside/ Isolated places	7	7.8%
Student of same school	2	1.7%	Jungle/Field	7	7.8%
Master and servant	2	1.7%	Guest house/Hotel	3	3.4%
Teacher and student	1	0.8%	School Madarsa	1	1.1%

Table 5: Distribution of cases according to time of examination of victims and accused of sexual offences

VICTIMS			ACCUSED		
Days/Weeks	No. (N= 90)	%	Days/Weeks	No. (N= 117)	%
Same day	9	10	Same day	5	4.27
Second day	13	14.4	Second day	15	12.82
Third day	8	8.9	Third day	5	4.27
Fourth day	5	5.9	Fourth day	5	4.27
5th day -7th day	23	25.5	5th day -7th day	28	3.93
1st week -2nd week	15	16.7	1st week -2nd week	27	3.07
2nd week -3rd week	4	4.7	2nd week -3rd week	12	10.25
3rd week – 4th week	4	4.4	3rd week – 4th week	8	6.83
> 4th week	9	10.0	> 4th week	12	10.25

Table 6: Distribution of types of offences and pattern of injuries on victims of sexual offences

Type of offences	No. (N = 90)	%	Pattern of injuries on victims	No. (N = 90)	%
Forcible rape	23	25.5	Extra genital	6	6.7
Invalid consensual rape	39	43.3	Genital	10	11.1
Statutory rape	18	20.0	Extra genital and Genital	7	7.8
Unnatural sexual offences	10	11.1	Hymen rupture	77	85.5

Table 7: Results of Laboratory tests of specimens of victims and accused of sexual offences

VICTIMS			
Test performed	Total No.	Positive No.	%
Microscopic examination of spermatozoa	73	5	5.5
Acidphosphatase tests	73	3	3.3
Florence test	73	2	2.2
Baberio's test	73	0	0
Hanging drop preparation	10	0	0
ACCUSED			
Microscopic examination of spermatozoa	25	1	0.85
Acidphosphatase tests	25	0	0
Florence Baberio's test	25	0	0
Hanging drop preparation	25	0	0
Lugol's Iodine Test	25	1	0.85

choline within one day, and complete spermatozoa within 3 days after sexual intercourse. The absence of sperm may be attributed to any of the following causes, erectile inadequacy, impotence, premature ejaculation before penetration and ejaculation incompetence²³. Other factors may include prolonged postcoital intercourse, oligospermic or orchidectomised assailants, penetration without ejaculation, digital penetration, use of condoms or spermicidal agents and menstruation and vaginal inflammation²⁴.

This study highlights the importance of addressing rape as a public health issue and focuses on the demographic profile of victims and accused in an urban area.

References

1. Waqlch, A.G., Broadhed, W.E. Prevalence of life time sexual victimization among female patients, *Journal Family Practitioner*, 35: 511-516, 1992.
2. Sexual Assault and Domestic Violence, Beckmann C.R>B> et al, 3rd edition.
3. Obstetrics and Gynecology, Lippincott Williams and Wilkins, 576-582, 1998.
4. Okonkwo J.E.N., ilbech.C. Female sexual assault in Nigeria, *International Journal of gynaecology and Obstetrics*, 83 (3): 325-326, 2003.
5. Martin L.J. Forensic evidence collection for sexual assault: A South Africa Perspective, *International Journal of Gynaecology and Ostetrics*, Supplement 1 S: 105-110, September 78, 2002.
6. Crime in India, National Crime Record Bureau, Ministry of Home Affairs, New Delhi.
7. Ansell R. Securing evidence after sexual offence is an importance task for physicians. Increasing severity of crimes and use of DNA analysis necessitate higher quality standards, *Lakartidningen*, October 14; 95 (42): 4626 -8, 4630- 1, 1998.
8. Malhotraet, N, Sood M. sexual assault, a neglected public health problem in developed world, *International Journal of Obstetrics and Gynaecology*, 71:257-258, 2000.
9. Beeba, D.K., Emergency management of the adult female rape victim, *American Family Physician*, 43:2041-2043, 1991.
10. Okonkwo, C.O., Rape (sexual assault), Society and child problems and papers of a symposium held at Enugu on May 7, 1986, FIDA (anambra Enugu). Family Law Publishers, 50-59.
11. Sagar, M. S. Sexual Offences in South Delhi, *Journal of Forensic Medicine and Toxicology*, Vol. IX:8-11, 1992.
12. Bhardwas D.N., Sharma R.K., Sagar M.S., Murty O.P., Study of sexual offence victims in South Delhi, *Journal of Forensic Medicine and toxicology*, Vol.XII, No. 3&4:33-34, 1995.
13. Grossin,C.,Sibille,I., Grandmaison G.L.D.I., Banars, A., Brion, F., Durigon, M., Analysis of 418 cases of sexual assault, *Forensic Science Int.*, 131: 125-130, 2003.
14. Du Mont, J., Parnis, D., Sexual assault and legal resolution: Querying the Medical collection of Forensic evidence, *Medical Law*, 19(4): 779-92, 2000.
15. Islam, M.N., Retrospective study of alleged rape victims attended at Forensic Medicine Department of Dhaka Medical College, Bangladesh, *Legal Medicine(Tokyo)*: Suppl 1:5351-3, March 5, 2003.
16. Riggs N., Houry, D., Long, G., Markovchick, V., Feldhaus K.M., Analysis of 1076 cases of sexual , *Ann Emergency Medicine*, 35: 358-362, 2000.
17. Fimate, L., Devi, M. An analytical study of rape in Manipurm International Journal of Medical Toxicology and Legal Medicine, Vol. 1: 1-2, 1998.
18. Subramanyam, B.V., Modi's Text Book of Medical Jurisprudence and Toxicology, Butterworth India Publication, New Delhi, 22nd Edition, 495-99, 1999.
19. Gee, D.J., Sexual offences, in Polson CJ, Gee DJ, Knight B., (eds). *The Essential of Forensic Medicine*, Pergamon Press, Oxford, 4th Edition: 485, 1985.
20. Rogers, D., Physical aspects of alleged sexual assaults, *Medicine, Science and Law*, April 36(2) :117-122, 1996.
21. Christian, C.W., Levelle, J.M., Dejong, A.R., et al, Forensic evidence finding in prepubertal victims of sexual assault, *Paediatrics*, 106: 100-104. 2000.
22. Royal College of Physicians . Physical signs of sexual abuse in children, London, RCP Publication, 1991.
23. Davis, A., Wilson, E., The persistence of seminal constituents in the human vagina. *Forensic Science*, 3: 45-55, 1974.
24. Haibert, D.R., Darnell Jones D.E., Medical Management of the sexual assaulted women. *Journal of Reproductive Medicine*, 20 : 265- 74, 1978.
25. Rao, P.N., Collins, K.A., Geinger, K.R. et al. Identification male epithelial cells in routine postcoital cervicovaginal smears using fluorescence in situ hybridization. *American Journal of Clinical Pathology*, 104 : 32-35, 1995.
26. Chan A., A review of outrage of modesty offenders remanded in a State Mental Hospital, *Med. Sci. Law*, 37(4): 349-352. 1997.
27. Copeland L J., The physician approach to sexual assault victim. *The Text Book of Gynaecology*, 8th Edition, 814-821.
28. Dolan M., The psychological characteristics of Juvenile sexual offenders referred to a adolescent Forensic service in UK, *Med. Sci. Law*, 36 (4) : 343-352, 1996.
29. Schiff AF, A statistical evaluation of rape, *Fore.Sci.*, 2 : 339-349, 1972.
30. West DJ., Rape a comparison of group offences and lone assaults. *Med. Sci. Law*. 21(1): 25-30, 1981.

A study of sexual offences in Tripura

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Abstract

Sexual offences are the most heinous crimes against women and the children. It is the most barbarous and humiliating and the women and children remain the most vulnerable group of this crime. Careful medical examination and preserving the biological evidences of sexual offences are the moral and ethical duties for doctors and poor medical evidences are often responsible for low conviction rate of criminals. Careful analysis of preserved biological specimens are yielding the vital evidences in criminal investigations. Victims of 11- 20 years age groups are most vulnerable for sexual assault.

Keywords

Sexual assaults, poor evidences , low conviction rate, younger groups are vulnerable.

Introduction

Among all crimes, sexual related crimes are the most heinous crimes and humiliating against women and children. The Law and customs of the society normally permit heterosexual intercourse between a man and his own wife as provided by the nature. The crime of rape requires that the perpetrator used force and the victim actively resist to the last moment. Women and children remain the most vulnerable group of this crime. The alarming rise in the rate of sexual offences worldwide represents a major health problems¹.

Material and methods

Age of the victims of sexual offences

Age(years)	Female Victims No=71	Male Victims No.0	Total No.	%
0-10	6	0	6	8.45
11-20	50	0	50	70.42
21-30	7	0	7	9.85
31-40	5	0	5	7.04
41-50	3	0	3	4.22
>50	0	0	0	0.00

Lowest age of the victim= 3 yrs child, Highest age of the victim= 4-6 yrs

Religion of the Victims of sexual offences

Religion	No.	%
Hindu	64	90.14
Muslim	7	9.85
Christian	0	0.00
Siks	0	0.00

Martial status of the victim of sexual offences

Martial status	Total No.	%
Married	13	18.30
Unmarried	57	80.28
Widow	1	1.40

Literacy status of victims of sexual offences

Education(Class)	Total No.	%
Not literate	33	46.47
Class I-Class V	15	21.12
Class VI-Class IX	11	15.49
Class X-Class XI	9	12.67
Above Class V	3	4.22

Relationship of assailants with victims of sexual offences

Type of Relationship	Total No.	%
Acquaintance	31	43.66
Stranger	11	15.49
Close friend	12	16.90
Neighbour	15	21.12
Teacher-students	2	2.81

Types of offences of sexual assault of sexual assault

Type of offences	Total No.	%
Forcible rape	17	23.94
Invalid adult consensual sexual offences	41	57.74
Statutory rape	11	15.49
Attempted rape	2	2.81
Unnatural sexual offences	0	0.00

Time distribution of alleged sexual offences of victims

Days/Week	No.71	%
Same day	1	1.40
2 nd day	5	7.04
3 rd day	9	12.67
4 th day	12	16.90
5 th day - 7 th day	8	11.26
1 st week - 2 nd week	11	15.49
2 nd week - 3 rd week	10	14.08
3 rd week - 4 th week	7	9.85
> 4 th week	9	12.67

Results of Laboratory Tests Performed of alleged rape victims.

Test performed	No.46	+ve	%
Microscopic Examination of spermatozoa	46	1	1.40
Acid phosphate test	46	0	0.00
Florence Test	46	0	0.00
Barberior's Test	46	0	0.00
Hanging drop preparation	1	0	0.00

Distribution of assailant & victims of sexual offences in gang rape

No. of victims	No. of assailant
1	3
1	3
1	2
1	2
4 (5.63%)	10 (Two assailant on each victims)

In the study showed that, in USA an estimate of one in every four women and children², in Nigeria four out of every ten women are victims of sexual assaults³The incidence rate in South Africa is approximately 300 per 100,000 women⁴. In India over the last five years, rape cases have been shown alternate increasing and decreasing the trends with increase of 6.6% in the year 2000 over 1999 and a decline of 2.5% in the year 2001. In the year 2001, a total of 16,075 (1.6 per 100,000 population) cases of rape against women and 2,113 (0.2 per 100,000 Population) cases of child rape were reported all over the India. It shows that 11.2% and 19.5% of total crimes are against women and children respectively. The Delhi has been ranked 5th for the incidence of rape cases against

women and 1st for the rape on children (0.8 per 100,000 Population)⁵.

Securing of evidence and preserving the medical evidences of sexual offences are the important task for a Doctor⁶. Poor medical evidences are often responsible for low conviction rate of the criminals⁴. Careful analysis of preserved biological specimens such as blood, semen, saliva often yields vital evidence in the cotemporary criminal investigation⁶. In the present article, the incidence and pattern of sexual offences in Tripura along with the demographic variables, findings of physical and genital examination of victims and results of medical evidences collected are presented.

A multidimensional and retrospective analysis of female suicide deaths in Chennai

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Abstract

Background

Suicide is a multidimensional and multifactorial malaise. This study was to give a broad picture of all available information about the female suicide death cases dealt in Institute of Forensic Medicine and Toxicology, Government General Hospital, Chennai.

Objective

The aim was to collect data, make a broad analysis, interpret and arrive at conclusive opinion about female suicide deaths in Chennai.

Methodology

A retrospective analysis of female suicide deaths encountered in Institute of Forensic Medicine and Toxicology, Government General Hospital, Chennai in the year 2008 was done. This information was collected from the available records.

Results

A total of 43 female suicide deaths were dealt with. Among these cases maximum number of them belong to the age group of 21 to 40 years, was married and was unemployed. 25 cases of poisoning was reported which contributed to the maximum. Organophosphorus compounds were the most common poison consumed. Other methods include hanging and burns. Interestingly abdominal pain was reported to be most common reason for committing suicide as per the records available.

Conclusion

The cause of death supported by the autopsy findings has been elucidated in all the cases. Further on considering the reason for committing suicide, it is noteworthy to mention that in majority of cases abdominal pain has been the factor behind such acts as the records available. This reason could be a manifestation of psychosomatic illness. The object of the study would be more fulfilled if it throws open newer dimensions of studies like psychological autopsy to prevent such deaths in future.

Keywords

Government General Hospital, Chennai, poisoning, hanging, burns, organophosphorus compound, Hypoxic Ischemic Encephalopathy, asphyxia, dysmenorrhoea,

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Introduction

In the past two decades, the suicide rate in India is increasing, especially in southern India. Majority of studies note that around 90% of those who die by suicide have a mental disorder¹. In the year 2008, 2812 autopsies were conducted in the Institute of Forensic Medicine and Toxicology, GGH, Chennai. Out of these 43 female suicide death cases were recorded. This study was done to elaborate on various aspects of these suicide deaths. The methods adopted in these cases are poisoning (58.13%), hanging (37.2%) and burns (4.65%). In case of poisoning, the type of poison consumed were organophosphorus compounds (52%), inorganic acids (12%), carbamates (8%) and others which included organochlorides, Lysol, ammonium dichromate, hair dye and olanzapine constituted 4% each. In the remaining 8% of cases the type of poison consumed was unknown. The effect of individual poisons on the viscera were demonstrated in the autopsy and the cause of death was inferred and recorded accordingly. In other methods which include hanging and burns, the various changes in the viscera were also recorded and the cause of death confirmed. Age pattern observed in these deaths is given in illustrations 1. Among these cases 28 were married and 12 were unmarried. It was also seen that 29 were unemployed, 7 were students and 4 were employed.

Objective

The purpose of the study is to collect data, make a broad analysis, interpret and arrive at a conclusive opinion about:

- Methods adopted in suicide and cause of death in each case.
- Reason for committing suicide.

Methodology

The records pertaining to 43 female suicide cases recorded in the year 2008 in Department of forensic medicine & Toxicology, Government General Hospital, Chennai were studied. This retrospective analysis included collection of data under the parameters such as age, occupation, reasons for committing suicide, methods adopted in suicide and their postmortem findings. This information was collected from the available records submitted by the investigating agencies, enquiries conducted with the medical officer who has done the autopsy, the postmortem reports, chemical examiners report and the like. While gathering data on individual cases identity number was given in each case to maintain confidentiality.

Results

Poisoning: Out of the 43 cases reported, 25 cases were due to poisoning. Postmortem findings in all these cases were recorded in detail along with the chemical analysis report of the visceral contents. Viscera samples collected in all these cases were stomach and its contents, intestine and its contents, liver, kidney, brain and blood. These samples were then sent to forensic science laboratory to detect the presence of poison in them. The postmortem findings for individual poisons are given

in Table 1

Hanging: Out of the 43 cases reported, 16 were due to hanging. Analysis of the postmortem finding in all these 16 cases reveals the following details.

Ligature marks with various dimensions, directions and their positions were found in the neck in all these cases with or without fracture of the hyoid bone.

Central nervous system: On opening the cranial cavity, the brain showed congestion and engorgement of surface vessels. In all the cases brain appeared with varying degrees of edema and presence of petechial hemorrhages in the white matter of the brain except in 2 cases where an additional finding of subdural hemorrhage and subarachnoid hemorrhage were reported.

Cardiovascular system: On opening the thoracic cage, significant postmortem findings in the cardiovascular system were seen in only 1 case by way of presence of petechial hemorrhages in heart. The rest of the cases show no significant remarkable findings.

Respiratory system: On examination of the findings in the respiratory system, 16 cases showed congestion and edema of the trachea and larynx along with oozing exudates from lungs on cut section. 4 cases had interlobar surface petechial hemorrhage and in 2 cases the appearance was found to be rubbery in consistency and grayish white in color.

Other viscera: All the 16 cases showed visceral congestion of the abdominal organs namely stomach, liver, spleen and kidney.

Burns: Only 2 cases were reported as death due to burns and in both cases the pattern of injuries noted was identical in the form of dermoepidermal burns involving different body parts. In both cases the extent of burns were reported to be more than 70%.

Central Nervous System: On examination of brain no

significant findings were made out other than presence of edema, congestion and engorgement of the surface vessels.

Cardiovascular system: On examination of the findings in thoracic cavity there was presence of petechial hemorrhage on the surface of the heart along with clotted blood in the chamber in 1 case. There were no other significant findings.

Respiratory System: While examining the respiratory system in both cases there was presence of blackish gray soot particles in the mucosal surface along with edema of laryngeal and tracheal mucosa. The congestion and edema along with frothy white fluid exudate was reported in both cases.

Other Viscera: On examination of abdominal organs multiple hemorrhagic spots were found in the gastric mucosa in 1 case and it was pale in the other. Other visceral organs such as liver, spleen, and kidney were congested in both the cases.

Reasons for committing suicide: Reasons for committing suicide are represented in Illustration: 2

Discussion

On correlation of the postmortem findings with that of the cause of death, the following observations have been made with particular reference to the method of suicide adopted, the type of poison consumed and the underlying pathology.

- **Poisoning**
 - o **Organophosphorus compounds:** Organophosphorus, a pesticide, is one of the commonest agent used in suicidal poisoning mainly owing to its easy availability and accessibility to the common people^{2,3}. Being irreversible inhibitors of Acetylcholinesterase, they predominantly produce central nervous system toxicity. In all the cases of organophosphorus compounds poisoning, except one case, medical intervention was brought in within 24 hours (fatal period). It is seen to cause depression of respiratory centre and vasomotor centre⁴. As a result of depression

Table 1: post mortem findings:

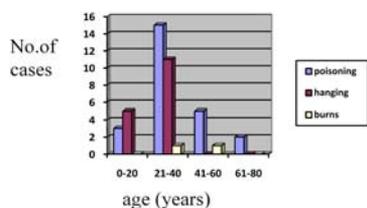
POISON	INJURIES	*CNS	†CVS	‡RS	§GIT	L	¶K	**S	Presence of poison in the Viscera
organo-phosphorus	Nil	Brain surface vessels congested, brain edema	nil	Lungs congested	Stomach- Yellow fluid-3cases Black fluid-7 cases Straw colored fluid-1case No fluid-2cases	congested	congested	congested	Present-5 cases Absent-8 cases
carbamates	Nil	Brain dural vessels congested, brain edema	Atheroma in root of aorta- 1 cases, Petechial hemorrhage-2 cases	congested	Stomach-Grey pungent fluid-1 case Greenish black fluid with black granules-1 case	congested	congested	congested	Present-2 cases Absent-0
organo-chlorides	nil	Brain edema	nil	nil	Nil	nil	nil	Nil	Present-1 case
lysol	Erosion of lips and buccal mucosa	Brain edema	nil	nil	Peritoneal Cavity - Reddish black fluid-1 case	congested	congested	congested	Absent - 1 case
Inorganic acid	Lips tongue gum darkened and leathery	Nil	nil	Trachea-multiple erosions, lungs-edema congested	Esophagus darkened and Eroded, stomach- multiple perforations greater curvature, duodenum-mucosa eroded.	congested	congested	congested	Absent-3 cases
Ammonium dichromate	Nil	Brain surface vessels cong	Heart clotted blood	nil	Stomach- dark fluid	nil	Nil	nil	Absent-1 case
Hair dye	nil	Brain edema, surface vessels congested	nil	nil	Stomach- mucosa black and congested	nil	Enlarged and congested	Nil	Absent - 1 case
olanzapine	nil	Brain edema, surface vessels congested	Heart-Atheroma in root of aorta, coronaries narrowed, ventricular walls thickened.	Lungs- edema and congestion	Nil	congested	congested	congested	absent- 1 case
Unknown 1	nil	Brain edema	Heart- clotted blood	Lungs-Adherent to chest wall, apices with cavities, oozing of greenish yellow pus	Stomach- mucosa congested.	congested	congested	congested	Absent-1 case
Unknown 2	nil	Nil	nil	Lungs- congested and edema	Stomach- dark fluid	congested	congested	congested	Absent-1 case

Foot notes

*Central Nervous System, †Cardiovascular System, ‡Respiratory System, §Gastrointestinal System (||Liver, ¶Kidney, **Spleen

Illustration 1:

Age: On analysis of the above suicide cases, the age pattern in each method of suicide is elucidated as a pictorial representation. Age is represented along horizontal axis where each block corresponds to 20 years. The number of cases in each of the methods (poisoning, hanging, burns) is represented along the vertical axis where each block corresponds to 2 cases.

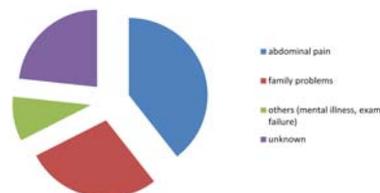


of these vital centers, there is acute cardiac decompensation and respiratory failure leading to severe generalized hypoxemia⁵. Since brain is the most susceptible organ to hypoxia, there is subsequent development of the fatal condition called hypoxic ischemic encephalopathy – the cause of death in organophosphorus poisoning^{3,6}.

- o **Carbamates** : These are the group of insecticides which produce reversible inactivation of enzyme acetylcholinesterase⁵. Therefore it shows milder manifestation as compared to that of organophosphorus compounds. Similar to organophosphorus compounds, carbamates cross the blood brain barrier and produce respiratory depression thus causing hypoxemia. Hence the cause of death is mainly due to respiratory failure⁵.
- o **Organochlorides** : These are yet another group of pesticides commonly used². They are highly lipid soluble and act as neurotoxins. They produce intense and persistent central nervous system stimulation⁵. This is followed by respiratory depression which is the major cause of fatality.
- o **Lysol**: Lysol is a proprietary preparation of 50% cresol in a mixture of linseed oil, potassium hydroxide and water. This is used as a household disinfectant. Lysol on ingestion is absorbed into the circulation producing methemoglobinemia and massive hemolysis⁷. This has its effect on kidney as acute tubular necrosis that results in acute renal failure. There is concomitant accumulation of toxic metabolites in the blood which further affects brain producing toxic encephalopathy which is the cause of death. On aspiration Lysol produces laryngeal edema and later aspiration pneumonitis which culminates in Acute Respiratory Distress Syndrome which has a contributory role in mortality.
- o **Inorganic acids** : Inorganic acids are a corrosive agent which on ingestion cause intense irritation of the skin and mucosa⁸. It produces ulceration followed by perforation of esophagus, stomach and duodenum thus resulting in chemical peritonitis. The acid fumes irritate respiratory passage resulting in edema of larynx, trachea and lung parenchyma producing aspiration pneumonitis and finally Acute Respiratory Distress Syndrome which is the cause of death⁹.
- o **Ammonium dichromate**: It is used in printing and dyeing industry and produces poisoning on ingestion. It produces severe and acute gastrointestinal bleeding which manifests as circulatory shock. The cause of death in dichromate poisoning is Acute Renal Failure¹⁰. This occurs by prerenal and renal reasons. The prerenal cause is the circulatory shock which compromises renal blood supply. The renal cause is the Acute Tubular Necrosis due to the toxic effects of metabolites affecting the kidneys¹¹.

Illustration: 2

Reasons for Committing Suicide: The reasons for committing suicide are depicted in the pie chart. The reasons in 17 cases were abdominal pain. 12 of them committed suicide due to family problems. Other reasons such as mental illness and exam failure were seen in 4 cases. The reason for committing suicide in 10 cases could not be elicited.



- o **Hair dye** : It contains active ingredient called paraphenylene diamine. It is marketed in India as Supervasmol. Immediately on ingestion, it produces cervico fascial and angioneurotic edema which is transient¹². Later, it produces Acute Tubular Necrosis and the resultant Acute Renal Failure which is the cause of death.
- o **Olanzapine** : This is an atypical antipsychotic drug, one of the most commonly encountered drug over dosage. Olanzapine over dosage produce intense central nervous system stimulation and hence extra pyramidal effects¹³. Isolated olanzapine poisoning will not cause fatality. In the case already mentioned, the autopsy findings showed atheromatous plaques in the root of aorta, narrowed coronaries and thickened ventricular walls. These are indicative of ante mortem cardiac decompensation which would have had a contributory role in producing death in olanzapine poisoning.
- **Hanging**: Out of 16 hanging cases, 9 were declared dead when brought to the hospital. When a minimal prolonged pressure is applied over the neck, there is compression of internal jugular veins which produces decreased venous return from brain, gradually backing up blood in the brain causing unconsciousness and depression of respiratory centre. Following loss of consciousness the full weight of the suspended part of body falls against the ligature creating enough pressure to restrict the air flow through trachea thus resulting in irreversible asphyxiation causing death¹⁴. In rare instances, when significant pressure is applied over the neck producing soft tissue injury, obstruction of internal carotid arteries occurs. This causes compromised blood flow to the brain. This will also cause baroreceptor stimulation and produce reflex bradycardia and cardiac arrest, all contributing to the cause of death. In the remaining 7 cases delayed effects of hanging caused death. In these, the cause of death may be Hypoxic Ischemic Encephalopathy or pulmonary edema. Following ischemic insult to the brain there is primary energy failure occurring in minutes. This is to an extent overcome by auto regulation and restoration of cerebral blood flow. The reperfusion caused sodium overload and excitotoxicity results in immediate necrotic cell death. Later during the secondary phase, there is mitochondrial dysfunction and caspases activation producing delayed apoptotic cell death. Thus Hypoxic Ischemic Encephalopathy ensues and causes death¹⁵. Cerebral hypoxia causes increased release of cytokines. This coupled with systemic hypoxia causes pulmonary vascular constriction, pulmonary hypertension and damage to the pulmonary vessel wall. Following sudden removal of airway obstruction there is abrupt fall in the intrapulmonary pressure, increased venous return and thus pulmonary hyperemia. All the above stated effect

culminates in pulmonary edema and death.

- **Burns:** Burns due to kerosene and flames produce severe pain & shock because of extensive stimulation to sensory nerves in skin. Death may occur quickly from immediate shock with vasomotor collapse or may be delayed to few hours^{16,17}. If injury is not sufficient to cause death within a few hours, the patient may subsequently succumb to secondary effects such as pneumonia, septicemia, Acute Tubular Necrosis etc.¹⁸

Reasons for committing suicide : Among the various reasons abdominal pain was the reason for committing suicide in majority of the female cases (17 cases-39.53%). Out of these 17 cases, 6 were due to hanging, 10 were due to poisoning and one of them adopted burning as the mode of suicide. When the marital status was noted it was found that 14 of them were married. On looking into the cause for the abdominal pain a range of reasons were noted. 12(70.58%) cases committed suicide due to dysmenorrhea which contributed to the maximum cause of abdominal pain. When the information about the cases where the suicidal deaths due to dysmenorrhea was explored it was seen that 9 of them were married and belonged to the age group 20-45 years. Of the 12 cases 3 of them were unmarried in the age group of 18-20 years. 3(17.64%) of the total 17 cases developed abdominal pain due to pregnancy related reasons (termination of pregnancy and retention of placenta). 1 case (5.88%) of age 34 years was reported to be chronic sufferer from abdominal pain due to appendicitis and renal calculi^{19,20}. Out of 17 cases, the reason for abdominal pain in 1 case was unknown.

Conclusion

Classification of the female suicide deaths reported at Government General Hospital, Chennai during the year 2008 has been recorded as above. The cause of death has been interpreted with reference to the mode of suicide adopted, i.e. hanging, poisoning or burns, in case of poisoning with particular reference to the type of poison consumed and their effects on various organ systems supported by the autopsy findings. It is interesting to note that in majority of cases (29.26%) the reason for committing suicide was reported to be abdominal pain due to dysmenorrhea as per records. Since there were no apparent stressors found in the records such as issues related to marriage, dowry, relationship affairs, alcoholism of spouse, financial debts, etc., in these cases, the abdominal pain could be considered as a manifestation of somatisation or depression. The object of the study would be much more fulfilled if it kindles interest and throws open newer dimensions in related studies like psychological autopsy in future.

Acknowledgement

We thankfully acknowledge the encouragement and guidance received from Dr.Vallinayagam M.D., director of Department Of Forensic Medicine and Toxicology, Government General Hospital, Chennai, India, during the collection of data and analysis. Our heartfelt thank goes to Mr.Anburaj, who has rendered invaluable help and assistance in preparation of the above paper.

References

1. Lakshmi Vijaykumar. Suicide and its prevention: the urgent need in India. *Indian Journal of Psychiatry*. 2007 Dec 10; 49(2): 81-4.
2. Gupta BD, Vaghela PC. Profile of fatal poisoning in and around Jamnagar. *Journal of Indian academy of forensic*

3. Ahmed M, Farial Naima Rahman. Overview of OPC poisoning in Bangladesh and medicoaspects related to fatal case. *Journal of Armed Forces medical college, Bangladesh*. 2009 June 1; 5(1): 57-9.
4. Donald J Ecobichon. Toxic effects of pesticides. In: Curtis D Klaassen, editor. Casarett and Doull's toxicology The basic science of poisons. 5th ed. United States: McGraw Hill; 1975. p. 643-683.
5. Pilay VV. Pesticides. In: Pilay VV, editor. Handbook of Forensic Medicine and toxicology. 13th ed. Hyderabad: Paras publications; 2003. p. 424-442.
6. Gannur PG, Prakash Maka, OPC poisoning in Gulbarga region-five year study. *Indian Journal of Forensic Medicine and Toxicology*. 2008 Oct 3; 2(1): 38-40.
7. Yao WC. A case of acute Lysol poisoning. *Chinese Journal of Industrial Medicine*. 2005 Apr 6; 18(6): 17-20.
8. Jelenko C, Story J, Ellison RG. Ingestion of mineral acid. PMID [4810637]. 1974 Feb 14; [cited 1974 Feb 24]; 40(2): [about 4 screens]. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/4810637>.
9. Pilay VV. Caustics. In: Pilay VV, editor. Handbook of Forensic Medicine and Toxicology. 13th ed. Hyderabad: Paras Publications; 2003. p. 181-200.
10. Meerut KL, Ellis J, Aronow R, Perrin E. Acute ammonium dichromate poisoning. PMID [8092606]. 1994 Oct 7; [cited 1994 Oct 12]; 24(4): [about 5 screens]. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/8092606>.
11. Job C. Ammonium dichromate poisoning: A case report. Anil Aggarwal's Internet Journal of Forensic Medicine and Toxicology [7018321]. 2005 Jan-June; [cited 2005 Jan-June]; 6(1): [about 3 screens]. Available from: http://www.geradts.com/anil/ij/vol_006_no_00/papers/paper003.html.
12. Sumeet Singla, Sanjeev Miglani. Paraphenylene diamine poisoning. *Journal of Indian academy of Forensic Medicine*. 2005 July 7; 6(3): 236-8.
13. Pierre Chue, Peter Singer. A review of olanzapine associated toxicity and fatality in overdose. *JPN* [165790]. 2003 July 8; [cited 2003 July 18]; 28(4): [about 5 screens]. Available from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=165790>
14. Pilay VV. asphyxial death. In: Pilay VV, editor. Handbook of Forensic Medicine and toxicology. 13th ed. Hyderabad: Paras publication; 2003. p. 181-200.
15. Bruce R Ranson, Stephen G Waxman, Robert Fern. Molecular pathophysiology of white matter anoxic and ischemic injury. In: Henry J M Barnett, Mohr JP, Bennett M Stein, Frank M Yatsu, editors. Stroke Pathophysiology, diagnosis and management. 3rd ed. Philadelphia: Churchill Livingstone; 1986. P. 85-100.
16. Werner U Spitz. Thermal injuries. In: Werner U Spitz, editor. Spitz and Fisher's Medicolegal investigation of death Guidelines for the application of pathology to crime investigation. 3rd ed. Illinois: Charles C Thomas; 1993. p. 413-443.
17. Lari AR, Alaghebandan R, Panjeshahin MR, Johatari MT. Suicidal behavior by burns in province of Fars, Iran. *Crisis*. 2009 Aug 10; 30(2): 98-101.
18. Pilay VV. Thermal injuries. In: Pilay VV, editor. Handbook of Forensic Medicine and Toxicology. 13th ed. Hyderabad: Paras publications; 2003. p. 162-171.
19. Spiegel B, Schoenfeld P. Systemic review- the prevalence of suicidal behavior in patients with chronic abdominal pain and irritable bowel syndrome. *Alimentary Pharmacology and therapeutics*. 2007 Nov 8; 26(3): 183-193.
20. Venkoba Rao A. Physical illness, pain, suicidal behavior. *Crisis*. 1990 Nov 14; 11(2): 48-56.

Care allowance for people in need of care in Turkey: An ethical and social evaluation

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Abstract

With economical and social developments, population structures of countries have changed and the rate of people in need of care and demands for care services have increased.

People may need help of their relatives to activities of daily living in order to survive at one stage of life. Both the foresight that the burden of care is too severe to cope with and the fact that all states have social responsibilities underlined the importance of regulations to support families although traditionally people are responsible for caring their ill relatives. In recent years, attempts to support people in need of care have increased in Turkey. The Disability Law was enacted in 2005 and it was modified in 2006. The statutes regulating selection of disabled people in need of care were also issued in 2007. The Disability Law and the statutes, for the first time, made it possible for people taken care of by formal and informal caregivers to receive care allowance.

Care allowance for formal and informal care at home has underlined ethical principles. It provides formal and informal caregivers with financial support. However, it is still debatable whether caregivers should be paid by governments.

In this article, the scope and effects of the Disability Law and the relevant statutes will be evaluated and ethical and medico-legal problems likely to appear in practice will be discussed.

Key words

Home care, people in need of care, care allowance and ethics.

Introduction and aim

In this article, recently enacted laws concerning being in need of care and home care in Turkey will be evaluated and social effects and medico-legal and ethical aspects of home care will be compared with those from other countries.

Being in need of care

People may need help of their relatives to perform activities of daily living (ADLs) necessary to lead a normal life at one stage of life (Seyyar., 2005). Traditionally, if one of the family members is ill, the rest of the family is responsible for the care of the ill member. However, the burden of care may become so severe that family members may not cope with it. Both this excessive burden of care and social responsibilities of states have resulted in new laws concerning support for families of people in need of care (Commonwealth of Australia., 2003).

Increased elderly and disabled population, changes in the family structure and increased care expenditures have forced countries to seek effective solutions to care for the elderly and disabled people throughout the world. In many developing and developed countries, home care services are financed by care insurance systems and predominantly by government funds. The governments have shared the burden of care and

started to create care insurance systems which support families of elderly and disabled people (Brodsky et al., 2000). In this setting, elderly and disabled people have been provided care at home by either family members or professional and semi-professional caregivers. The care insurance systems require that difficulties which prevent people in need of care from leading a normal life in their own places should be eliminated (Ođlak., 2007a). Care insurance systems provide long-term care and short-term preventive medicine, medical and social care and rehabilitation as well as professional health care either at home or in institutions and financial support in cases of irreversible conditions (Seyyar, 2005; Ođlak, 2006). Home care has been increasingly preferred in recent years in that it decreases health care costs, allows delivery of health care at home and offers an appropriate environment for maintenance of patient self-esteem.

Care allowance and ethics

Home care is offered by three types of organization: profit organizations, public institutions and non-profit organizations. This naturally causes differences in duration, monitoring and quality of home care. Home care should have a high quality in that a high quality home care increases quality of life and patient satisfaction and is an indication of respect for patient rights (Francis & Netten, 2004). Ethical principles are of great importance particularly in home care. It is quite difficult to monitor home care services compared to other health care and social services and care standards and ethical principles can be violated by caregivers. Respect for preferences of people in need of care and neglect, abuse and bad behaviour likely to arise in home care are the issues which attract attention at present (Garcia, 2006; Letizia&Casagrande., 2004).

So that patient rights are not violated, individuals receiving care and treatment at home should participate in the decision making processes for all interventions they undergo and should have the right to decline interventions. In other words, patient autonomy should be respected. Care given by caregivers unaware of patient rights may have undesirable effects. There are ethical concerns about home care given by family caregivers in that it may not be qualified enough, can be difficult to monitor and may cause such risks as abuse and mistreatment (Kondratowitz et al., 2002; Picard et al., 2003; Brodsky et al., 2000; Gross., 1994; Penhale., 2006; Özkara.,2003).

Care allowance is money paid monthly either directly to caregivers or to individuals in need of care depending on the degree of care needs. The primary goal is to decrease financial burden on people taking care of their ill relatives. Care allowance is preferable in that it offers the right to choose the person/institution which will provide care and flexibility in care plans (Stryckman & Nahmiash.,1994). In addition, it is agreed that care allowance encourages families to provide care for elderly and disabled people, provides compensation for financial losses resulting from work leaves of carers and is indicative of an appreciation of family caregivers' efforts although the amounts of payment are not so high (Horfmarcher & Riedel., 2001).

However, it is argued that care allowance for the care of elderly and disabled people may increase the number of

caregivers without relevant qualifications who work independently and that care services may not be sufficiently monitored. In addition, individuals may have difficulties in selecting caregivers and care providing institutions, caregivers may abuse care allowance and elderly and disabled people can be abused financially, exposed to violence and neglected (Picard et al., 2003).

It is difficult to determine criteria for quality standards of home care, a complicated and multi-faceted process, since it is difficult to monitor home care services, individuals offered home care cannot easily assess or criticise the value and quality of the care and care providing institutions offer a wide variety of care services. In addition, there is concern that uneducated staff and lack of standards for home care may result in neglect and mistreatment (Merlis., 2000). In some situations, not only concrete criteria but also moral values play an important role in home care quality. Irrespective of education, lack of mercy and compassion may cause neglect and maltreatment. In fact, American Bar Association reported that home care services may cause quality problems such as physical damages, insufficient time for patients due to caregivers' reluctance or incompetence, mistreatment, insufficient or inappropriate performance and behavioural problems such as insensitivity, disrespect, threats, psychological abuse and financial abuse (The Quality of Home Care., 2006). The fact that individuals who receive home care are usually old, disabled and alone and do not have strength to defend themselves makes the quality of home care important. In fact, if home care is not performed in accordance with predetermined standards, abuse of individuals in need of care is inevitable (Penhale., 2006).

The quality of life is directly related to the quality of home care among individuals who need home care. Therefore, determination of quality standards for home care is the primary responsibility of states. Netten et al. noted that there were six aspects of home care quality: reliability, flexibility, continuity, communication, behaviour and attitude of caregivers and knowledge and skills of caregivers (Netten et al., 2003; Francis & Netten., 2004;).

The WHO made the following recommendations to improve the quality of home care (Gibson et al., 2003):

- a) Regulatory systems should be created and regulations for fulfilment of minimum standards should be adopted.
- b) Knowledge and skills of caregivers should be improved and accordingly educational standards should be determined.
- c) Standard processes and substructure characteristics of education should be clearly described.
- d) Measurements concerning educational outcomes should be evaluated regularly.
- e) The obtained results should be used to improve the quality of home care.

It is important to monitor home care in order to assure the quality of care. Abuse and neglect are social and forensic problems (Wang et al., 2006; Penhale., 2006). Based on the statistics, abuse of elderly and disabled people is widespread (Wang et al., 2006).

There is public awareness in these problems in many countries. For example, many studies from Ireland have revealed that caregivers suffer from heavy workload and psychological stress. Another issue which strikes attention is abuse of individuals in need of care. It has been reported that individuals cared by family members are exposed to verbal and physical violence (Mc Cann & Evans., 2002). Ventura found in 1980 that 115 caregivers who offered home care had negative attitude towards the elderly and did not have sufficient knowledge about old age (The Quality of Home Care, 2005).

The WHO reported that 4-6% of the elderly were exposed to abuse in Europe. The highest rate of psychological abuse

was reported to be 54.1% in the USA followed by 21% in China and 5% in Britain and Holland (Tazuko et al., 2005).

A study from Japan revealed that home care related excessive stress caused mistreatment and neglect, which cause elder abuse (Tazuko et al., 2005).

It has been reported that women and individuals aged over 80 years are more frequently exposed to abuse and neglected. In Japan, it has been shown that women who look after their spouses' parents and disabled family members more frequently exhibited abuse and bad behaviour (Tazuko et al., 2005; The Administration for Children and Families and the Administration on Aging; 1998).

The elderly with Alzheimer's disease are more frequently exposed to abuse than the general elderly population. In fact, one study revealed that 33% of the caregivers who took care of family members with Alzheimer's disease abused and neglected the ill family members (Coyne et al., 1993).

Disabled people taken care of by family members such as mother, father, spouse, children and spouses' wives are most frequently abused and neglected. Excessive workload related stress, lack of support from other family members, insufficient time for personal things, insufficient knowledge and skills and conflict with the individuals who need care are factors which increase neglect, mistreatment and abuse (Tazuko et al., 2005).

In Turkey, the Disability Management Directorate affiliating with the Prime Minister (ÖZÝDA) has created programs to solve social problems of disabled people. These programs have increased sensitivity to and public awareness in abuse of disabled people. In fact, governments should give priority to prevention of abuse as much as solving social problems (Baþbakanlýk Özürlüler Ýdaresi Baþkanlýđý ÖZÝDA.,2006). In Turkey, the disability law numbered 5378 enacted on 1 July 2005 requires that disabled people are taken care of at home by caregivers authorized by Sosyal Hizmetler ve Çocuk Esirgeme Kurumu (SHÇEK) (Social Services and Child Protection Agency) and by health staff working at public institutions (Özürlüler ve Bazý Kanun ve Kanun Hükmünde Kararnamelerde Deđiþiklik Yapýlmasý Hakkýnda Kanun., 2005). The law, for the first time, provided poor disabled people with care free of charge either at home or in an institution (Oðlak., 2007b).

However, the law 5378, which stipulates that disabled people without health insurance should be provided with care, and the relevant statutes have created some inequalities in practice. Therefore, the law was changed on 10 February 2007. The new law, numbered 5579, requires that if disabled people and their family members have a total monthly income of less than two thirds of the minimum wage, those disabled people should be provided with care in public and profit organizations or at home when they need it (Sosyal Hizmetler ve Çocuk Esirgeme Kurumu Kanununda Deđiþiklik Yapýlmasý Hakkýnda Kanun, 2007).

According to regulations numbered 26430 and dated 23 October 2007 on determination of disabled people in need of care and the principles of care services:

- a) A two-month minimum net salary is paid when a disabled person is cared in an institution for 24 hours.
- b) A one-month minimum net salary is paid when a disabled person is offered care in an institution for 8 hours during day time.
- c) When a disabled person is offered home care by the staff of a care institution for 4 hours a day, a one-month minimum net salary is paid to the institution.
- d) When a disabled person is taken care of by his relatives, a one-month minimum net salary is paid to the relative taking care of the disabled person.

Despite its deficiencies, the law which requires that people who provide home care for their disabled relatives for 24 hours

should be paid a one-month minimum salary in Turkey seems promising. This has improved care for the disabled people. However, there are concerns about neglect and abuse likely to be caused by care allowance to people for caring their disabled family relatives. In fact, care strategies and goals which guarantee the quality of the given care and quality performance criteria have not been described yet and it is not obligatory for family members to attend trainings and to get a certificate in order to offer high quality care (Ođlak., 2007c).

High standards and monitoring which will prevent financial, verbal, psychological and physical violence towards vulnerable disabled people taken care of by their family members or caregivers who work for private care institutions should be formed. According to the results of many studies and news, the belief that there are strong ties between family members and that neglect and abuse are less frequently encountered in our country has been weakened. For example, in a study on 3500 elderly people from seven different regions of the country by Ulusal Sosyal ve Uygulamalı Gerontoloji Derneđi (Turkish Gerontology Society), it has been noted that the number of the elderly exposed to various forms of physical and psychological violence in family settings was striking and that out of 10 elderly people, 9 were exposed to psychological violence and 3 were exposed to physical violence (Ulusal Sosyal ve Uygulamalı Gerontoloji Derneđi., 2006).

In view of the literature, it can be suggested that both ÖZÝDA and SHÇEK shoulder great responsibilities for home care which is in its infancy in Turkey. The things which should be prioritized are as follows:

- Care quality standards should be determined.
- Individuals at a high risk of abuse and neglect should be followed closely.
- Caregiver education should be supervised and caregivers should be observed for neglect.
- Caregivers should be provided with emotional support.
- Caregivers should be offered training for communication and problem solving skills.
- Training for moral values and compassion should be offered and sensitivity to the care of elderly and disabled should be increased.
- Caregivers' workload should be decreased. Attempts to provide formal care should be increased.

Conclusion

Despite its deficiencies and limitations, the recently enacted law which requires the government should pay for the care of the disabled is an important and promising development and an indication of social welfare function of the state. Provision of care for disabled people and care allowance to caregivers taking care of their disabled relatives seem to be promising; however, quality care assurance systems, performance indicators, home care standards and grading systems for monitoring home care should be formed. In addition, it is imperative that home care services given in partly isolated places should be monitored and that caregivers should be provided with appropriate education and trained for communication and problem solving skills in order to avoid malpractices likely to result from the home care environment and personal characteristics of caregivers and to prevent elderly abuse.

References

- AC Coyne, WE Reichman and LJ Berbig; (1993) The relationship between dementia and elder abuse, *American Journal Psychiatry*, 150:pp.643-646
- Bařbakanlık Özürlüleri Ydaresi Bařkanlıđı, Ýstismarı Önlemek Devletin Önceliđidir.www.ozida.gov.tr

- Brodsky Jenny, Habib Jack, Mizrahi Ilana; (2000), Long-Term Care Laws in Five Developed Countries, A Review, JDC Brookdale Institute of Gerontology and Human Development, Jerusalem, World Health Organization.
- Commonwealth of Australia; (2003), Review of Pricing Arrangements in Residential Aged Care, Long Term Aged Care, International Perspectives, Background Paper, No.3. p.127
- Francis Jennifer, Netten Ann; (2004), "Raising the Quality of Home Care: A Study of Service Users' Views, *Social Policy and Administration*: 38:3;290-305, June.
- Garcia Teresa; (2006), Ethics in Home Care, *Home Health Care Management & Practice* Vol.18, Number 2, pp.133-137
- Gibson Jo Marry; Gregory R.Steven; Pandya M.Sheel; (2003) "Long-Term Care in Developed Nations:A Brief Overview", *AARP Public Policy*, October. pp. 25-26
- Gross M. Arnold; (1994) "Payments for Care: The Case of Israel", *Payment For Care, A Comparative Overview*, (Edit; Evers A; Pijl M; Ungerson C), European Centre Vienna, Avebury, s.296
- Horfmarcher M.Maria, Riedel Monika; (2001) "Development of age structure in the EU: The EU is ageing: even when expanding eastward. Focus: Acute care and long-term care: An interface according to analysis or chance?", *Health System Watch*, Supplement of the Journal Social Security by the Institute for Advanced Studies, IHS Health Econ, Edited by the Federation of Austrian Social Security Intuitions.
- J. Wang, J. Lin, F. Lee ; (2006) Psychologically Abusive Behavior by Those Caring for the Elderly in a Domestic Context, *Geriatric Nursing*, Vol:27, Issue 5, pp. 284-291
- Kondratowitz H.Joachim; Tesch Römer Clemens et al; (2002) "Establishing Systems of Care in Germany: A Long and Winding Road", *Aging Clinical and Experimental Research*, 14; 239-246
- Letizia M.John; Casagrande Kate; (2004) The Importance of Implementing a Code of Ethics for Home Health Agencies, *Home Health Care Management & Practice*, 16 (5), 412-413
- Mc Cann S; Evans D.S; (2002) "Informal Care: He Views of People Receiving Care", *Health and Social Care in the Community*, 10 (4): 221-228
- Merlis Mark; (2000) "Caring for the Frail Elderly: An International Review", *Health Affairs*, May-June Volume 19, Number 3, pp. 141-149
- Netten Ann; Francis Jennifer; Bebbington Andrew; Curtis Lesley; (2003) "Costs, Quality and Outcomes", *PSSRU Bulletin* 14, June, pp.12-13
- Ođlak Sema; (2006) "Evde Bakým Hizmetleri ve Bakým Sigortası", *IV. Ulusal Sađlık Kurululuđları Yönetimi Kongresi Bilimsel Kitabı*, 13-16 Nisan 2006, Fethiye-Muđla, s.102-109
- Ođlak Sema; (2007a) *Evde Bakým Hizmetleri ve Bakým Sigortası (Ülke Örnekleri ve Türkiye)*, Ýskenderun Belediyesi Kültür Yayınları No:6, Color Ofset Matbaası, Ýskenderun, Hatay, 1. Basým
- Ođlak Sema; (2007b) "Türkiye Evde Bakýma Hazır mı?" *Uluslararası Sađlık ve Hastane Yönetimi Kongresi Özetler Kitabı*, 01-03 Haziran 2007, Yakın Dođu Üniversitesi Lefkođa-KTTC, pp.103-104
- Ođlak Sema; (2007c) *Uzun Süreli Evde Bakým Hizmetleri ve Bakým Sigortası*, *Turkish Journal of Geriatrics*, 10 (2): 100-108
- Özkara Erdem, Yavuz Ý.C, (2003) "Acil Serviste ve genel hekimlik uygulamasında hasta otonomisi kavramının yansımaları" , *Acil Týp Dergisi*, 3(2),32-37
- Özürlüleri Kanunu (Özürlüleri ve Bazı Kanun ve Kanun Hükmünde Kararnamelerde Deđiřiklik Yapılması Hakkında Kanun), Kanun No: 5378, Kabul Tarihi:

- 01.07.2005, www.ozida.gov.tr
22. Penhale B. (2006) Elder Abuse in Europe: An Overview of Recent Developments. *J Elder Abuse Negl.*; 18(1): 107-16.
 23. Picard Linda, Comas-Herrera Adelina, Font Costa Joan et al; (2003) "Modelling an Entitlement to Long-Term Care in Europe: Projections for Long-Term Care Expenditure to 2050" 6th. European Sociological Association Conference, Murcia, Spain, September 23-27, Research Network on Ageing in Europe.pp. 1-52
 24. Seyyar, Ali; (2005) "Yaşlılarda Bakım Sorunlarına Çözüm Olarak Sosyal Politikalar"; T.C. Başbakanlık Aile ve Sosyal Araştırmalar Genel Müdürlüğü ve ÝSAV (Ýslami Ýlimler Araştırma Vakfı); Aile Sempozyumu; Ýstanbul; 02.-04.12.2005
 25. Stryckman Judith, Nahmiash Daphne; (1994) Payments for Care: The Case of Canada, Payment For Care, A Comparative Overview, Edit; Evers A; Pijl M; Ungerson C), European Centre Vienna, Avebury. pp.311-318
 26. Tazuko Shibusawa, Tazuko; Kodaka Manami; Iwano Shinji; Kaizo Kiyoko; (2005) Interventions for Elder Abuse and Neglect With Frail Elders in Japan, *Brief Treatment and Crisis Intervention* 5:203-211
 27. The Administration for Children and Families and the Administration on Aging; (1998)The National Elder Abuse Incidence Study, Final Report, September, pp.18
 28. The Quality of Home Care, <http://www.nih.gov/ninr/research/vol3/HomeCare.html>, 10.18.2006, p.12
 29. Ulusal Sosyal ve Uygulamalı Gerontoloji Derneği, (2006) Dünya Yaşlı Suistimali Farkındalık Günü Basın 5579 Sayılı; Sosyal Hizmetler ve Çocuk Esirgeme Kurumu Kanununda Değişiklik Yapılması Hakkında Kanun, Kabul tarihi: 01.02.2007, RG No: 26430, 10.02.2007

A study of duplication of the optic canals in human adult skulls of North Karnataka

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Abstract

Bilateral duplication of optic canal is considered to be a rare anomaly. Literature is available & cases are reported showing bilateral & unilateral duplication of the optic canal in dry human skulls. In the present study 2 cases (1 male & 1 female) bilateral duplicate optic canals were found out of 316 macerated adult skulls of both sexes. A bony septa separated the main canal from the duplicate canal. The larger canal usually carries the optic nerve with the meninges & the smaller one transmits the ophthalmic artery.

This study has been made & reported as a rare anomaly.

Key words

Optic canal, optic nerve, ophthalmic artery, duplication.

Introduction

The optic foramen or rather the optic canal leads from the middle cranial fossa to the apex of the orbit, it is formed by the body of sphenoid medially & the lesser wing of sphenoid laterally, directed forwards laterally & some what downwards. The canal is funnel shaped the mouth of which being the anterior opening. This opening is oval in shape with the greatest vertical diameter, the cranial opening, on the other hand is flattened from above downwards, while in the middle it is circular.

The lesser wing is connected to the body by an anterior root which is thin & flat, while the posterior root is thick & triangular in shape. between them the optic canal contains the optic nerve & ophthalmic artery., whose cranial opening may be duplicated. more often the division is incomplete.

Rarely the optic canal/foramen is duplicated. A bony septa of variable thickness separates the main canal/foramen from the duplicate.

In each duplicated optic canal, the main canal was in usual position & the accessory canal variable in size, but smaller than the main canal, was inferolateral to it in position photo 1&2.

In all these duplicated canals, the oval cranial end of main canal was continuous with the anterolateral end of Charismatic sulcus. The accompanying accessory canal, on it's cranial end was continuous with the anterior end of the sulcus for internal carotid artery. In all the skulls studied with duplicated canals, the lateral wall of the accessory canal was shorter than it's medial wall which formed lateral wall of the main canal.

Visconti (1885)¹, Zoja (1885)² & Le double (1903)³ have reported cases of bilateral duplicate canal. Choudhary et al (1988)⁴ & (1999)⁵ have also reported cases with unilateral & bilateral duplications of optic canal in both the sexes.

Materials & methods

Materials for study comprised of 316 dry human adult skulls of both sexes (182 male & 134 female between the age group of 25 to 80 yrs). This study was done in the dept of Anatomy at M. R. Medical College Gulbarga (Karnataka). These

were collected from the department's (Osteology section) & also from the students of First MBBS (2006-2007).

Observations

Of 316 dry adult human skulls of both sexes (182 male & 134 female between 25 & 80 yrs of age) studied, we found two skulls (1 male & 1 female) having bilateral duplication of optic canal. The measurement of these duplicate canals could not be done as they were pin head size. One of it was large which was the main optic canal & the other was smaller below it, separated from the main canal by the fibroosseous/bony septa.

Photo 1: shows the male skull with bilateral duplication of optic canal.

Photo 2: shows the female skull with bilateral duplication of optic canal

Table 1: shows the no of male & female skulls with bilateral duplication of optic canal & with probable age group.

Discussion

From the above observation we can say that it is a rare anomaly ,as there are only 2 skulls out of 316, showing bilateral duplication of optic canals. According to Choudhry & Anand (1888)⁴ worked on 435 skull found 13 duplications of optic canals of which 9 were male out of which 5 were bilateral & 4 unilateral. 4 were female out of which 2 was unilateral & 2 bilateral duplications. Choudhry R, Choudhry S, & Anand M, Tuli (1999)⁵ have reported cases of duplicate optic canals. Le double (1903)³ have reported cases having bilateral duplicate optic canals.

Orham magden A, Kaynak S (1996)⁶ who studied on 369 skulls found 1 bilateral & 1 unilateral duplication.

Keyes (1935)⁷ reported duplicate optic canals in 36 out of 2187 skulls

A case reported by Warwick (1951)⁸, a skull of male child of 21 months old, showed duplication of optic canals & division of supra-orbital fissure. White (1924)⁹ & Whitnall LE (1932)¹⁰ also reported 3 cases of new born fetuses showing duplication of optic canals. In our present study it is observed that bilateral duplication of optic canals is more common in female (0.74%) than male (0.54%) & at an earlier age group in females (30-40yrs) than that of the male age group (60-70yrs). The present study when compared with other's study it can be assumed that bilateral duplication to be more common than unilateral, & the duplication can occur at any age.

Wolff(1976)¹¹ speculates that the cause for duplication could be due to increased space between the optic nerve & ophthalmic artery, where the connective tissue between the two gave way to ossification during the period of normal ossification of that region of the skull. Thus ossification of the intervening space occupied by the connective tissue being responsible for the development of separating septum. Which formed the main & accessory canal.

When duplication of optic canal is present (which is a fibroosseous/bony septa separating the main canal from the

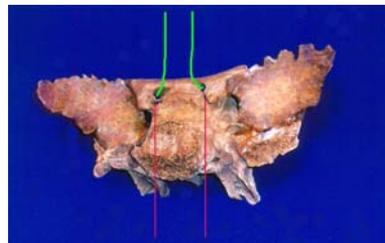
Table 1: shows number of male & female skulls with bilateral duplication of optic canals with probable age.

Total no.of Skulls	Total no of Duplication.	Percentage of Bilateral. of Optic Canal	Probable age of the Skulls Duplication. of Optic Canal
Skulls-316	Skulls-2	0.63%	25-80yrs
Male-182	Skull-1	0.54%	60-70yrs
Female -134	Skull-1	0.74%	30-40yrs

Photo 1: shows skull-1 male with bilateral duplication of optic canals.



Photo 2: shows skull-2 female with bilateral duplication of optic canals.



duplicate) it is observed that the larger canal usually carries the optic nerve with the meninges & the smaller one transmits the ophthalmic artery.

Conclusion

The present study highlights that duplication of optic canal is a rare anomaly. It is observed that bilateral duplications are more common than unilateral. At the same time it is also observed that sexual dimorphism is not statistically significant & to know this further study with more & equal number of male & female skulls has to be done.

Recent years have seen enormous advances in imaging techniques, the role of conventional radiography, C.T scans, MRI for detecting pathology or trauma to the orbit its contents & demonstration of optic canal form an integral part of investigation of a patient with loss of vision or upper cranial nerve involvement. Hence the study of duplication optic canal though a rare anomaly becomes essential.

Looking at the vast range of age (new born to 80 years), & the speculation done by Wolff¹¹ that the ossification of the connective tissue between the optic nerve & the ophthalmic artery being the main cause for duplication cannot be held true, as it is observed that the duplication can occur even in a new-born, where ossification is not possible.

Hence it can be postulated that duplication of optic canal could be due to the growth of a fibro-osseous septa during the early days of life in the posterior root of the sphenoid which is closely related to the narrowing of the canal, which gets ossified in latter days of life (normal period of ossification). But how & why it occurs still remains as a matter of question.

References

1. Visconti A. 1885 cited by Le -Double 1903 quoted by Warwick 1951
2. Zoja G. 1885. Spora il foro otticodoppio. Bollettino scientific, Pavia. 7 :65-69. Quoted by Warwick 1951.
3. Le-Double AF. Taite des variations des os du crane de l'Homme.Paris: Vigot Fre'res,:1935 ;1903 pp. 372
4. Choudhry R. Choudhry S, & Anand C. 1988. Duplication of optic canals in human skulls. Journal of anatomy. 159: 113-
5. Choudhry R, Choudhry S, & Anand M, Tuli A, Meenakshi A, Kalra A. Morphologic & imaging studies of duplicate optic canals in dry adult human skulls. Surgical & Radiological Anatomy. 1999 21(3): 201-
6. Orhan Magden A, Kaynak S. 1996 . Bilateral duplication of optic canals. Annals of Anatomy. 178(1): 61-4.
7. Keyes JEL. (1935). Observations on 4000 optic canals. Albrecht v Graefes Archiv fur ophthamologie
8. Warwick R. A juvenile skulls exhibiting duplication of optic canals & subdivision of superior orbital fissure. Journal of Anatomy 1951: 85: 289-291
9. White LE. An anatomic & X-ray study of optic canal. Boston Medical & Surgical Journal. 1942: 189: 741-748
10. Whitnall SE. 1932. The anatomy of the human orbit. 2nd edition. Oxford University Press, London. Pp. 53, 313.
11. Bron AJ, Tripathi RC, Tripathi BJ.(Eds) 1996. Wolff's Anatomy of eye & orbit, 8th edition. Chapman & Hall Medical, London, pp-11-12
12. Singh M. Duplication of optic canals in adult Japanese human skulls. Journal of Anatomical Society of India.: 54 (2): 2005-07-2005-12

Prognathism as race appraisal criterion in a study of 60 Indian crania of known sex using metric and non-metric modes – Assessment of methodology

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Abstract

Race assessment is one of the essential components in establishing identification of an individual from human skeletal remains. The skull stands out as the most reliable amongst skeleton for determining racial affinity, both morphologically and osteometrically. Inferring from observations related to morphological indicators depends largely upon the experience of the observer while adequate level of methodological standardization, is an essential precondition at the outset. Variability with regard to prognathism among members of all major racial groups is more a rule than exception. Assessment of race, using metric studies vis-à-vis morphological indicators was put to comparison and tested for reliability of methodology in the present study, subsequent to estimation of prognathism by both methods on skulls of 60 Indian individuals of known sex (30 of either). The prognathism was estimated (1) non-metrically – by inspection using casts prepared and standardized by Australian National University and (2) metrically- by calculating Gnathic Index (as defined by Breathnach, 1965). The results of both studies revealed that of a total of 60 Indian skulls studied, 50 skulls were found to have orthognathic profile (Gnathic Index < 98) by Metric Study in contrast to Non-Metric Study where only 16 skulls showed 'Small' Grade prognathism. Subsequent to chi-square test, the difference between Metric and Non-Metric study was significant ($X^2 = 38.922559$). Consequently it can be stated that determination of prognathism applying metric studies is superior in reliability compared to morphological indicators using casts.

Introduction

Skeletal remains contain an abundance of information which can lead to reliable determination of age, sex, race and stature of the individual in life (Buchner, 1985). The skull is the best part of the skeleton to use for the determination of racial affinity, both morphologically and osteometrically (Novotny et al., 1993). The areas of the face in the vicinity of the nose, mouth, and cheekbones are the most useful in determining race (Kerley, 1977).

Forensic identification often involves fragmentary remains. Indeed, it is in cases with incomplete remains that metrical rather than observational methods are most helpful (Burriss and Harris, 1998).

Ideally, both approaches should be utilized whenever possible (Novotny et al., 1993).

Kerley (1977) says that there is a great deal of variability with regard to prognathism among members of all major racial groups. As a result of variations in prognathism, there are differences in the shape of the palate as well.

The Negroid face is usually marked by strong alveolar prognathism- a particular bulging of the jaws in the subnasal

region. Mongoloid facial skeletons usually show some alveolar prognathism of both jaws, but not nearly also much as is usual in Negroid or Australoid skulls. Straight or orthognathous (having the front of the head, or the skulls, nearly perpendicular, not retreating backwards above the jaws) faces, devoid of prognathism, are most commonly diagnostic of Caucasoid (white) racial ancestry (Hooton, 1965) (Fig. 1a,b).

The skull of an Indian is Caucasian with a few Negroid characters (Modi, 1977).

Materials and methods

60 Indian skulls of known sex (30 of either) were studied to assess the reliability level of metric studies vis-à-vis morphological indicators with respect to race determination with prognathism as sole criterion (applying both methods). The samples for the study were drawn from the collection maintained by the Departments of Anatomy and Forensic Medicine, Government Medical College, Patiala.

Indian skulls of known sex in which spheno-occipital junction was synostosed and all required bony landmarks were intact were considered for study.

Prognathism

Prognathism refers to the anterior projection of the alveolar point relative to the nasion and basion (Glanville, 1969).

Metric study

For the metric study of prognathism, the craniometric points (as detailed hereunder) were located on each cranium and series of measurements were taken between the points. These measurements were obtained with Spreading caliper to the nearest millimeter (mm), as per standard anthropological conventions and then Gnathic index was calculated.

Bony landmarks (photograph 1)

1. **Basion** : The point where the anterior margin of the foramen magnum is intersected by the mid-sagittal plane. The point is located on the inner border of the anterior margin of the foramen magnum directly opposite of Opisthion (the point at which mid-sagittal plane intersects the posterior margin of the foramen magnum) (Moore-Jansen et al., 1994).
2. **Nasion** : The intersection of the fronto-nasal suture and the median plane (Howells, 1973). This does not refer to the internasal suture in any way.
3. **Prosthion (Alveolar point)** : The most anterior-inferior point on the maxilla between the upper incisor teeth (El-Najjar and McWilliams, 1978).

Metric measurements

1. **Basion-nasion length: (photograph 2)** : The direct length between nasion and basion (Howells, 1973). With the skull base up, one end of the caliper was placed at nasion and the other end at basion and length was recorded.

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2. **Basion-prosthion length: (Photograph 3)** : The direct distance from basion to prosthion (Moore-Jansen et al., 1994). With the skull base up, one end of the caliper was fixed at prosthion and the other end at basion and length was recorded.

3. **Gnathic Index** : (Breathnach, 1965) Index above 103: Prognathous; 98-103: Mesognathous; below 98: Orthognathous

$$\frac{\text{Basi - prosthion length}}{\text{Basi - nasion length}} \times 100$$

Non-metric study

Prognathism (Photograph 4)

Grades:

- Small
- Medium
- Large (Larnach and Macintosh, 1966)

The grading was done following visual inspection and comparison with casts prepared and standardized by Australian National University.

The data so obtained both by metric and non-metric study, was tabulated and statistically analyzed.

Results and discussion

Metric study : Insert Table 1, here

Non - metric study : Insert Table 2, here

In the present study, Indian skulls were used. The skull of an Indian is Caucasian with a few Negroid characters, as stated by Modi in 1977. The most prominent feature of Caucasians is

the completely straight profile (orthognathic profile) or the very prominent nose. There is generally rather little prognathism (Kerley, 1977). Thus, Indians have orthognathous jaws (with little prognathism).

The gnathic index below 98, indicates orthognathous jaws (Breathnach, 1965).

Table 1 showed that by Metric study of a total of 60 Indian skulls studied, 50 were found to have orthognathic profile (Gnathic Index below 98).

In the present non-metric study, casts prepared and standardized by Australian National University were used. Hooton in 1965, has mentioned that Australians (composite race)¹ are characterized by medium to pronounced facial protrusion (prognathism) in comparison to Whites/ European/ Caucasoid (primary race) in whom facial protrusion/prognathism is usually lacking. As stated by Modi in 1977, that the skull of an Indian is Caucasian with few negroid characters. Thus, most of the Indian skulls should show 'Small' Grade prognathism using Australian standardized casts.

Table 2 shows that by Non-Metric study of a total of 60 Indian skulls studied, only 16 were found to have 'Small' Grade prognathism.

The Metric and Non-Metric data was statistically analyzed by Chi-square test. The value of X² revealed (X² = 38.922559) that the difference between Metric and Non-Metric study is significant.

Thus, it can be stated from the present study that Metric study stands out as more reliable than Non-metric study at least with respect to race determination.

Many researchers have stressed the development of metric methods for race determination due to following reasons:¹ To

Photograph 2 : (Basion- Nasion Length)

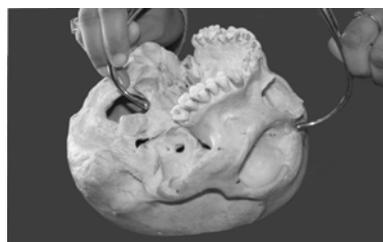


Table 1: Metric study

Sex	Gnathic Index		
	Orthognathous	Mesognathous	Prognathous
Male	24	3	3
Female	26	3	1
Total	50	6	4

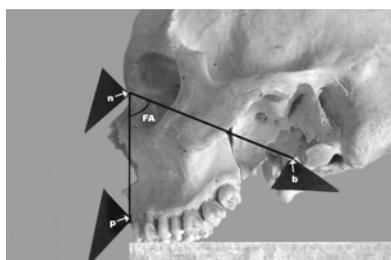
Table 2: Non-Metric study

Sex	Prognathism		
	Small	Medium	Large
Male	5	19	6
Female	11	15	4
Total	16	34	10

Footnote

Composite Races – Races representing stabilized blends of two or more primary races. They present mosaics of features from the different racial stocks involved and usually occupy definite geographical areas where the blend has been stabilized in isolation. Australian – composite race (Archaic White + Tasmanian + recent minor fraction of Melanesian – Papuan).

Photograph 1 : (b-basion; n-nasion; p-prosthion) (FA- Facial Angle)



Photograph 3 : (Basion-prosthion Length)



Photograph 4 : (Australian standardized casts)



Fig.1: (a) Caucasoid skull

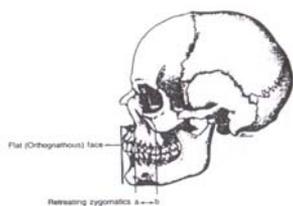
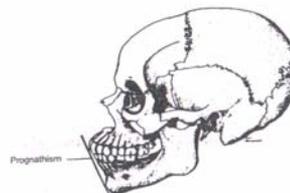


Fig.1: (b) Negroid skull



obviate the need for experience as assessment from non-metric study improves with the experience and are accurate in the hands of expert only², standardization of the methodology of scoring of non-metric traits also has significant role in modifying the end result³, lack of quantification of non-metric traits (quantification of trait means the frequency of occurrence of that trait in various populations).

The results of the present study support the fact that there is a need to develop Metric technique for race determination.

The race can be metrically determined by taking measurements and especially average measurements; ratio of two measurements; cranial, facial and mandibular indices; Discriminant function statistics.

Giles and Elliot in 1962, developed the first Discriminant function race formulae, but Birkby, in 1966 suggested that the Discriminant function analysis for race/ or sex determinations are not applicable to problems of human identification unless the crania are from populations on which these were established.

Gill et al. in 1984 found a reliable metric method by using simometer, useful in regions where Giles and Elliot technique was not very successful. Gill and Gilbert (1990) discovered that the formation of indices produces better results than discriminant function analysis. They were also easier to apply.

The present study has provided, in addition, a simple Metric technique for race determination, that is, estimation of Gnathic Index. Prognathism can be estimated by measuring Facial angle (Photograph 1). Thus, combination of Gnathic Index and measuring facial angle can serve as very useful guide for race determination

Acknowledgements

The authors are thankful to Dr.P.Raghvan, Senior Research Associate Scientist, Australian National University, for giving us training in Biometric methods and Dr.Avnish Kumar, Associate Professor, Department of Physiology, Government Medical College, Patiala for his valuable help in statistical analysis.

Literature cited

1. Birkby WH. An evaluation of race and sex identification from cranial measurements. *Am J Phys Anthropol* 1966;

- 24: 21-27.
2. Breathnach AS. The skull: general account. In: Frazer's anatomy of the human skeleton. 6th ed. London: J and A Churchill, 1965; p 180.
 3. Buchner A. The identification of human remains. *International Dental Journal* 1985; 35: 307-311.
 4. Burris BG, Harris EF. Identification of race and sex from palate dimensions. *J Forensic Sci* 1998; 43(5): 959-963.
 5. El-Najjar MY, McWilliams KR. Anthropometry. In: Forensic anthropology. The structure, morphology, and variation of the human bone and dentition. Springfield: Charles C Thomas, 1978; p 107.
 6. Giles E, Elliot O. Race identification from cranial measurements. *J Forensic Sci* 1962; 7: 147-157.
 7. Gill GW, Fisher JW, Jr and Zeimens GM. A pioneer burial near the historic Bordeaux Trading Post Plains Anthropologist 1984; 29: 229-238.
 8. Gill W, Gilbert BM. Race identification from the midfacial skeleton: American blacks and whites. In: Gill GW, Rhine JS(eds). Skeletal attribution of race. Maxwell Mus Anthropol Papers 4. Albuquerque: University of New Mexico, 1990; p 47-53.
 9. Hooton EA. Heredity and Race. In: Up from the ape, 1st ed. Delhi: Motilal Banarsidas, 1965; p 499-607.
 10. Hooton EA. The anthropology of the individual. In: Up from the ape, 1st ed. Delhi: Motilal Banarsidas, 1965; p 745-747.
 11. Howells WW. Cranial variation in man. Papers of the Peabody Museum of Archaeology and Ethnology, 67. Harvard University, Cambridge Massachusetts, 1973; p 169-171.
 12. Kerley ER. Forensic anthropology. In: Tedeschi CG, William G. Eckert, Luke G. Tedeschi, editors. Forensic medicine a study in trauma and environmental hazards. Vol. 2 Physical trauma. London: WB Saunders, 1977; p 1108-1111
 13. Larnach SL, Macintosh NWG. The craniology of the aborigines of Coastal New South Wales. The Oceania Monographs No.13, 1966; p 32..
 14. Modi NJ. Personal identity. In: Medical Jurisprudence and toxicology. 20th ed. Bombay: N.M. Tripathi, 1977; p 21.
 15. Moore Jansen PM, Ousley SD, Jantz RL. Data collection procedures for forensic skeletal material, 1994; p 45-51.
 16. Novotny V, Iscan MY, Loth SR. Morphologic and osteometric assessment of age, sex and race from the skull. In: Iscan MY, Helmer RP, editors. Forensic analysis of the skull. New York: Wiley- Liss, 1993; p 76-80.

Study of nature of ground, height of fall and injuries sustained

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Abstract

Fall from height results in unique pattern of injuries that depends on various factors such as inertia of the body, movement of the body, rigidity of stationary objects, height of fall and the nature of Ground nature against which body falls. In this present study effort is made to correlate the nature of ground, height of fall and to the injuries sustained.

Introduction

Deaths due to fall from height, constitute one of the major causes of unnatural deaths. In the present era of extreme industrialization and intense urbanization, deaths due to fall from height are alarmingly increasing¹.

Lack of space at the prime localities, gradual shift in social concept of isolated home towards more sophisticated apartments, newer generations inclination towards the well equipped flat culture, are giving rise to construction of more and more sky scapers which are in turn leading to more mishaps¹. Other causes contributing to deaths due to fall from height include fall from tree, fall of children while playing which is a most unfortunate happening in recent times.

During fall, the potential energy due to height is converted to kinetic energy under the influence of gravity. Fall from height, results in various types of injuries which are associated with rapid vertical deceleration²

Victims of fall from height tend to sustain a unique pattern of injuries that depends on various factors such as inertia of the body, movement of the body, rigidity of stationary objects, height of fall and the nature of Ground nature against which body falls³.

Thus in the present situation study of effort is made to correlate the nature of ground, height of fall and injuries sustained.

Materials and methods

The present study has been carried out in department of forensic medicine, M.S.Ramaiah medical college and hospital, Bangalore during the period October 2005 to May 2007, of all the cases of fall from height subjected for medico legal autopsy. A total number of 52 cases were studied after the ethical clearance was obtained. Detailed information regarding the deceased and a circumstance of death was collected from the police, relatives and visiting crime of occurrence.

Results and discussion

It is observed that majority of the cases falls were onto hard surface like cement/concrete, stone, hard soil and marble followed by falls on to soft surface like sand and mud. The surface on to which a body falls determines the pattern of deceleration and energy exchange^{4,5}.

On a relatively yielding surface, the energy is given up slowly, but on a relatively unyielding surface such as hard soil, concrete, time of deceleration is shorter and hence the forces on the body are much great. Multi organ involvement was noticed where the nature of Ground was hard surface and isolated fatal injuries were hall mark of falls over the soft surface⁶.

Fissure fracture of the skull and fracture of cervical spine was noticed when the fall occurred on to concrete from minimum height of 3 feet and on to hard soil from the height of 10 feet. Multiple rib fractures, fracture of clavicle and laceration of liver was noticed when the fall height was 7 feet on to concrete. Spleen and kidneys were damaged when height of fall was more than 20 feet on to hard surface. Fracture of upper limbs were seen in falls occurred on to concrete from height of 6 feet where as the fracture of lower limb was found when the fall height was more than 15 feet on to hard surface.

Conclusion

- Most cases of falls from height were accidental in nature. (88.46%)
- 21-40 years age group contributed the highest number (34.61%).
- Most cases of falls from height were due to accidental in nature. (88.46%)
- In 48.07% of cases the height of fall was 0-20 feet followed by 21-40 feet
- Among occupation, fall from height most frequently seen among construction workers (42.3%)
- Multi organ trauma was seen when fall was on to hard surface.
- Fissure fracture of the skull and fracture of cervical spine was noticed when the fall occurred on to concrete from minimum height of 3 feet and on to hard soil from the height of 10 feet.
- Multiple rib fractures, fracture of clavicle and laceration of liver was noticed when the fall height was 7 feet on to concrete.

Table 1: Distribution of study population according to Height of fall and body region involved.

Sl.No.	Height of Fall	Head	Neck	Thorax	Abdomen/pelvis	Upper /Lower Limb
01	0-20 Feet	18	06	12	10	03
02	21-40 Feet	15	00	16	16	09
03	41-60 Feet	01	00	01	01	02
04	61-80Feet	00	00	00	00	00
05	81-100 Feet	00	00	00	00	00
06	101-120Feet	02	01	03	03	02
07	121-140 Feet	00	00	01	01	01
Total	36	07	32	31	17	

Table 2: Distribution of study population according to nature of the Ground, Height of fall and injuries sustained

Height of fall	Hard surface					Soft surface		Total
	Hard soil	Cement / concrete	Stone	Tiles / Marble	Tar	Mud	Sand	
0-20 feet	13	04	02	02	02	02		25
21-40 feet	07	11	02	-	-	-	01	21
41-60 feet	-	01	-	-	-	-	01	02
61- 80 feet	-	-	-	-	-	-		-
81-100 feet	-	-	-	-	-	-		-
101- 120 feet	-	02	-	-	-	-		02
121- 140 feet	02	-	-	-	-	-		02
Total	22	20	04	02	02	02		52

- Spleen and kidneys were damaged when height of fall was more than 20 feet on to hard surface.
- Fracture of upper limbs were seen in falls occurred on to concrete from height of 6 feet where as the fracture of lower limb was found when the fall height was more than 15 feet on to hard surface.

Reference

1. Manson.J.K. The pathology of trauma 3rd editions Arnold publication, New York: 2000;313-326
2. Murthy O.P, "Pattern of injuries in fatal falls from height- a retrospective review" Journal of forensic medicine and toxicology; 1999:16(2):38-46
3. "Krishan Vij. The text book of forensic medicine and toxicology 3rd edition Elsevier publication, New Delhi: 2005:13-45
4. Albert Goonetillekke U.K.D, "Injuries caused by a falls from height" Medicine Science and Law; 1980: 20(4): 262-275
5. Dogra T.D, Chandra J, "Blunt force lesions related to heights of a fall" The American journal of forensic medicine and pathology; 1982: 3(1)
6. Dixit P.C. Forensic Medicine and Pathology 1st edition Peepee Publication, India; 2007:229-232

Irrational drug utilization in urology unit

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Abstract

Cases treated in urology unit are mainly infections of Urinary Tract (upper and lower) and cases of Acute Renal Failure and Chronic Renal Failure including complications during hemodialysis. A prospective cross sectional study was conducted for 15 months in Basaveshwar Teaching and General Hospital, Gulbarga. WHO prescribing indicators and patient care indicators were used. Cephalosporins were used maximally (80 to 90% including all the generation of Cephalosporins). Ciprofloxacin, Metronidazole and Ampicillin + Cloxacillin were next choice. NSAID's were commonly used in the Urology unit. Diuretics-Frusemide was used 70% in Urology unit. Average cost of drugs per day/patient, Urology unit =Rs. 100 to 150/- + 800 extra for patients undergoing hemodialysis.

Irrational prescriptions are harmful and may lead to number of problems like, Increased cost of therapy, Therapeutic failure, adverse drug reactions, Dangerous drug interactions and Inappropriate treatment.

Key words

Urinary Tract Infection; Cephalosporins; Cost;

Introduction

The first international study on drug use was undertaken by Dr.A.Engel of Sweden and Dr.P Siderius of Netherlands who visited six European countries on behalf of the WHO. Ultimately formed the Drug Utilization Research Group (DURG). A novel agency for Drug Utilization studies at international level.

Rational drug prescribing is defined as "the use of the least number of drugs to obtain the best possible effect in the shortest period and at a reasonable cost.^{1,2} Irrational prescription of drugs is of common occurrence in clinical practice³. Important reasons are:

1. Lack of knowledge about drugs.
2. Unethical drug promotion and
3. Irrational prescribing habit by clinicians.

Objectives

1. To study the drug utilization pattern in urology unit.
2. To analyze the prescription pattern of drugs in acute and chronic renal failure.
3. Hospital based studies aim to carry out a complete "Therapeutic audit" and to see what is prescribed, what is the intention and to analyze the cost effect benefit.
4. To study the adverse drug reaction and
5. To study the cost benefit ration.

Methods of data collection

The study was conducted after obtaining the permission of the ethical committee of our institution. The present study included patients who were admitted to the Urology unit and the Medical unit which deals with urinary tract infection and Acute Renal Failure and Chronic Renal Failure cases respectively.

Detailed history, chief complaints, physical signs, and investigations were recorded. The prescriptions were noted down for a period of 3 days patients were followed for adverse effect and prognosis until discharge or death. Total duration of study was over a period of 15 months i.e., from August to October.

Results

Among these 70 cases who presented with urinary symptoms 38 were females and 32 were males.

Out of 38 females 20 were culture positive and out of 32 males 10 were culture positive. The dominance of females with Urinary Tract Infection is maintained. The increased incidence of Urinary Tract infections was seen in the age group of 18 to 35 years, after 50 the increased incidence of males is maintained.

The incidence of infection due E-coli were in clear majority. E-coli were found in all age groups in higher frequency when compared to other organisms.

It was observed in over study that maximum number of patients in the Urology unit were in the age group of 25 to 50 percentage is 42.82%, 50 to 75 years is 35.71%, 1 to 25 years is 17.14% and 4.28% above 75 years. This correlates with much number of studies that increased risk of Urinary Tract Infection is seen females of age 20 to 30 and increased incidence in males is seen between the age group of more than 75 years.

In the Urology unit patients 35.71% had an average stay of 1 to 7 days, 50% of the patients stayed for 2 to 5 days, 14.28% stayed for 1 to 2 days. 50% of patients were discharged within 2 to 5 days encouraging early ambulation, 20% of them underwent surgeries for various grades of obstructive Urology 5.45% of patients stayed for more than 7 days because of complications like indwelling catheter and vascio ureteric reflux.

36.66% of patients were discharged after 10 days, this was to get treatment of complication inAcute Renal Failure and Chronic Renal Failure. In the Urology unit 71.42% of patients were discharged on advice, 11.42% of patients got discharged against medical advice, 4.28% were of absconding, 12.85% of patients expired during treatment and most of them expired due to Septicemia and Renal failure.

In the study of drug utilization of Urology unit NSAIDS were most commonly used. 85.71% of patients received Diclofenae, which was given parenterally and followed by oral route, 28.57% of patients received Paracetamol, 14.28% of patients received Nimuslide + Paracetamol, 28.57% of patients received Tramadol, 17.42% of patients received Brufen + Paracetamol combination.

These drugs were used to reduce pain, swelling and edema, which either acted as analgesic or ant-inflammatory

Age	Urology Cases	
	Number ofPatients	Percentage%
1 to 25	12	17.14
26 to 50	30	42.85
51 to 75	25	35.71
> 75	03	04.28

Adverse effects of drugs

Cases	Adverse Effects	No. of Patients	Percentage
Urology Unit	Gastritis	20	28 %
	Super infection	2	2.85 %
	Allergic reaction	2	2.85 %
	Vomiting	6	8.57 %

agents. 2.82% of patients developed allergic manifestations and were treated accordingly, 28.57% of patients reported gastritis this was due to over prescriptions of NSAIDS. 28.57% of patients received dicyclomine which correlates with the earlier studies the use of anti-spasmodic helped in reliving obstructive urology⁴.

In the Urology unit the average cost of drugs was as follows

- For 1 day = 100 to 150 Rs./day/patient
- For 3 days = 400 to 45 Rs./day/patient
- For 7 days = 1300 to 1400 Rs./day/patient

Conclusion

In our study Cefuroxime, Cefotaxime, Ampicillin & Cloxacillin, Metronidazole and Ciprofloxacin, and Prazocin, NSAID's were used irrationally. Irrational prescriptions are harmful and may lead to a number of problems such as

- 1) Increased cost of therapy

Average cost of Drugs used in the Treatment

Cases	Average cost per day per patient (Rs.)	
Urology Unit	Days	Amount (Rs.)
	One	100 to 150
	Two	200 to 300
	Three	300 to 400

- 2) Therapeutic failure
- 3) Adverse drug reactions
- 4) Dangerous drug interactions
- 5) In appropriate treatment
- 6) Health education
- 7) Patient education regarding drug use is needed to improve patient compliance.

References

1. WHO (1957), Technical Report, Series No.118.
2. WHO Drug Action Committee: Model Guide to Good Prescribing WHO 1994.
3. Bapna JS, Training Medical Professionals on the concept of Essential Drugs and Rational drug use, Br.J. Clin. Pharm., 1994; 37; 399-400.
4. Yu HT Progression of Chronic Renal Failure Arch.Intern. Med. 2003;163:1417-1429.

Estimation of time since death by gastric contents: An overview

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Abstract

Assessment of time since death is subjected to variable variations because in Biology, variation is a rule and stability is an exception, whereas in Science, stability is a rule and variation is an exception. The time of death estimation plays important role in solving both criminal and civil cases. Many factors contribute to great intra and inter individual variability of gastric emptying. This study is undertaken to collect available literature for reference and to form a common opinion that the doctor can depose safely. During medico-legal autopsy, if the doctor finds distinguishable full meal in the stomach, it would suggest that the victim died within 2 hours after the last meal and 4 hours if it is indistinguishable.

Key words

Gastric emptying, time since death, Cholecystokinin, Autopsy.

Introduction

The time since death is the time interval passed between the date and time of death to date and time of examination of the body. The determination of time of death has important role in solving problems in both criminal and civil cases. In criminal cases, it can set the time of the murder, eliminate or suggest suspects and confirm or disprove an alibi. While in civil cases, it might determine who inherits the property or whether an insurance policy was in force. There are various parameters like post mortem staining, rigor mortis, cooling of the body, bio-chemical changes in the body fluids, insect activities, degree of decomposition, etc., in use to determine the time of death. Among them, analysis of gastric contents is also one of the methods to determine the same. The time since last meal to death or time of death assessment during the medico legal post mortem examination can be done on the basis of study of gastric contents by their volume and state of digestion. Unfortunately, all the methods now in use are to a degree unreliable and inaccurate in fixing time of death, but only an approximate range of time of death can be estimated.

In the court of law, the prosecution as well as defense advocate shall make no stone remain unturned to prove or disprove their client's relation to the crime and it is the medical witness that can solve their problem. Many a times, the defense advocate is much more bent upon the time since last meal to death and compels to give conclusive opinion depending on state and quantity of stomach contents. The doctors have faced hard time to answer such defense questions, to be answered in 'Yes' or 'No' format, leading to miscarriage of justice.

Therefore, the present study is undertaken for generating collective information from recent and old studies and literature available till date, so as to form a comprehensive opinion that can be depose safely by the medical witness and as well to create ready made reference criteria for the honorable court of law.

Physiology of gastric emptying

Gastric emptying results from progressive waves of forceful contraction which sequentially involves antrum, pylorus and

proximal duodenum and all three function as a unit^{1,2}. The physiological mechanisms which regulate emptying of food from the stomach involve the complex integration of myogenic, neural and hormonal mechanisms. Long chain fatty acids and amino acids have been shown to be potent inhibitors of gastric emptying, since these foods stimulate the release of Cholecystokinin (CCK). In physiological concentration, the CCK delays the gastric emptying³.

Normal gastric emptying is the coordinated action of gastrointestinal tract and nervous system. The relaxation in the fundus is primarily regulated by a vaso-vagal reflex. The proximal part of the stomach is a primary location for storage of both liquids and solids. The distal portion of the stomach is primarily responsible for churning the solids and generating smaller liquid-like material which then exits the stomach in a manner similar to that of ingested liquids. Thus the gastric emptying of liquids and of solids is closely integrated^{4,5}.

Emptying of the stomach is controlled only to a moderate degree by stomach factors such as the degree of filling and the excitatory effect of gastrin on stomach peristalsis. Probably the more important control of stomach emptying resides in inhibitions feedback signals from the duodenum including both enterogastric inhibitory nervous feedback reflexes and hormonal feedback by CCK⁶.

The emptying of liquid from the stomach can be divided in to 2 phases, as an initial rapid phase followed by a slower mono-exponential decline. This early, rapid phase of emptying is reflected by the 5 minutes volume. The subsequent rate of emptying is expressed as a half life $t^{1,2}$ i.e. the gastric volume from 5 minutes after the ingestion of the meal until the subject's stomach has emptied to the less than 20ml, which declines mono-exponentially⁷. After a normal meal, stomach emptying time is 2 to 3 hours¹.

Methods to measure rate of gastric emptying

Many techniques have been used to measure the rate of gastric emptying in living but following are the few clinical methods which determine both the rate and pattern of gastric emptying⁸.

- 1) Radiological Methods.
 - Radio isotope technique using Radioactive Chromium.
 - Enteric-coated Barium granules and surface scanning.
- 2) ^{13}C Breath Test.
- 3) Real Time Ultrasound technique.
- 4) Serial test meal technique.
- 5) Dye dilution and double-sampling of test meal technique.

Factors affecting gastric emptying

All the methods that are used to assess gastric emptying show that the rate of digestion and gastric emptying is dependent several factors such as anatomical, physiological, pathological, psychological factors and kind of food such as the quantity, type, consistency, temperature, pH, chemical composition and nutritive content of food, along with gastrointestinal and autonomic conditions that were existing in the deceased at the time of death^{4,9}. The gastric emptying time of

fluids and solids in healthy subjects is significantly affected by age, sex and body mass index of the individual¹⁰.

The gastric emptying is regulated by various physiological factors like liquidity of the chyme, gastric factors, duodenal factors and others like emotions. The liquids empty much faster than solids. For any type of meal, gastric emptying is directly proportional to the volume, greater the volume of food in the stomach, greater is the stretching of stomach wall leading to strong peristalsis wave and increased rate of gastric emptying. The properties of a liquid meal which are the major determinants of the emptying of liquids from the stomach include volume, pH, osmolarity and nutrient contents. The liquids with nutrient value, particularly fats and proteins empty from the stomach at the rate that is slower than either the emptying of water or saline. The inert liquids leave the stomach rapidly. Water is emptied into intestine as soon as it is swallowed, but the solids move out of the stomach only after being converted into fluid or semi fluid. The carbohydrates leave the stomach more rapidly than the proteins, and the proteins leave rapidly than the fats. Thus, the fatty food remains in stomach for a longer period. The gastric chyme with low pH (acidic) leaves the stomach slowly. The gastric content which is isotonic to blood, leaves the stomach rapidly than the hypertonic or hypotonic content⁴.

The warm liquids resulted in greater relaxation of gastric muscle resulting in initial reduction of gastric emptying and the subsequent higher 5 minutes gastric volume, which suggests that warm drinks are more filling than cold ones. The meal temperature affects initial adaptive relaxation mechanisms, hence temperature of the meal affects only the first 5 minutes of gastric emptying, from this 5 minute time point the gastric emptying is not dependent on the meal temperature⁷.

The effect of alcoholic beverage on gastric emptying is the result of the interplay of the effect of ethanol itself and the non-alcoholic substances. Compared with water both red beer and red wine resulted in a longer t^{1/2} emptying phase. The pure ethanol and whisky result in the delaying of gastric emptying rates of solid meals¹¹.

The bulk of the meal leaves stomach within 2 hours. A light meal (small volume) usually leaves stomach within 1 to 2 hours; a medium sized meal requires 3 to 4 hours and heavy meal 5 to 8 hours after being eaten. The stomach empties gradually and the emptying rate increases directly with meal weight^{1,4,9,12}.

The emotions also have a strong effect on gastric motility as anger and aggressions increase gastric motility whereas depressions and fear decrease it¹. The head injury, any physical or mental shock or stress mediate through parasympathetic system, may slow or completely stop gastric motility and secretion of gastric juice. In such cases undigested food may be seen after more than 24 hours. The antecedent stresses due to domestic disputes or a developing altercation culminated in a strangling or stabbing would almost certainly affect gastric function. The stomach contents do not enter the duodenum after death, but digestion of the stomach contents may continue for some time after death^{9,12}.

Based on the results of test meals given to healthy persons, the 2 hour emptying time concept can be drawn i.e. if the presence of a pint or more of undigested or partly-digested food in the stomach indicates the intake of a meal shortly before death, probably less than 2 hours. Conversely, an empty stomach suggests that nothing has been eaten for not less than 2 hours before death¹³. A substantial variation in gastric emptying rates can be shown in normal subjects, this variation being made greater by disease, trauma or certain drugs¹⁴.

Conclusion

One established method of estimating time since death is by estimating the time interval between the consumption of

last meal to death and then finding out the time of last meal. However, the gastric emptying alone is of minor value in ascertaining the time since death but it can be taken as important corroborative evidence to narrow the post-mortem interval. The various factors related to food and persons contribute to great intra and inter individual variability of gastric emptying. Thus estimate of post mortem interval by this method is an opinion based upon probabilities and subjected to limitations. But, if due allowances are given to the individual variations, along with other methods of estimating the time since death; the gastric emptying can be a valuable evidence.

Examination of the gastric contents after death may indicate the nature of the last meal before death and provide an approximate estimate of the probable time since death. If at autopsy, one finds that the stomach is full and food is distinguishable, it would suggest that the victim died within 2 hours of taking the last meal and 4 hours if it was indistinguishable. Commonly, the bulk of the meal leaves the stomach within 2 hours and the stomach is emptied in 4 to 6 hours after intake of normal, medium quantity meal.

References

1. Indu Khurana. Gastric Emptying- Physiological Activities in Stomach: Text Book of Medical Physiology 2006: 1st Edition: Elsevier, pp: 612-613.
2. WF Ganong. Gastric Motility and Emptying-Regulation of Gastrointestinal functions: Review of Medical physiology 2006: 21st Edition: Lange-Mc.Graw Hill Publications; pp 498.
3. RA Liddle et al. Regulation of gastric emptying in humans by Cholecystokinin: Journal of Clinical Investigation (March 1986): Vol. 77; pp: 992-996.
4. K. Sembulingam, Prema Sembulingam. Filling and Emptying of stomach; Movements of Gastrointestinal tract: Essentials of Medical Physiology 2006: 4th Edition: Jaypee Brother's Publication, New Delhi; pp: 247-248.
5. WF Boron, EL Boulpaep. Filling and Emptying of the stomach- Gastric Emptying: Text Book of Medical Physiology 2009: 2nd Edition: Saunders Elsevier Publication; pp: 909-911.
6. AC Guyton, JE Hal. Motor Functions of the stomach; Propulsion and Mixing of food in Alimentary tract: Text Book of Medical Physiology 2006: 1st Edition: Saunders Elsevier Publishers; pp: 784-786.
7. DN Bateman. Effect of meal temperature and volume on the emptying of liquids from the human stomach: Journal of Physiology 1982: Vol.331; pp: 461-467.
8. JD George. New clinical methods for measuring the rate of gastric emptying-the double sampling test meal: Gut 1968: Vol.9, pp: 237-242.
9. K.S. Narayana Reddy. Gastrointestinal factors in Estimation of Post Mortem interval-Post Mortem changes: The essentials of Forensic Medicine and Toxicology 2008: 27th Edition: Medical Book Company, Hyderabad. pp: 153-155.
10. Stephan Hellming et al. Gastric emptying of fluids and solids in humans: Journal of Gastroenterology and Hepatology 2006: Vol.21 (12); pp: 1832-1838.
11. Anderson Franke et al. The effect of Ethanol and alcoholic beverages on gastric emptying of solid meals in humans. Journal of alcohol & Alcohol 2005: Vol-40(3); pp: 187-193.
12. Rekka Sankko, Bernard Knight. Stomach Emptying as a measure of time since a death- Pathophysiology of death: Knight's Forensic Pathology, 2004; 3rd Edition: Arnold – Hadder Headline Group, London: pp: 83-88.
13. CJ Polson, DJ Gee, Bernard Knight. The Stomach Contents and the time of Death- the signs of death: The Essentials of Forensic Medicine 1985: 4th Edition: Pergamum press, Oxford. pp: 32-34.
14. Horowitz M., Pounder DJ. Gastric emptying- Forensic implications of current concepts: Medical science and Law Journal, 1985: Vol-25; pp: 201-214

Consumption of super vasmol poison and its effects on human lungs: A case report

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Abstract

A case study of super vasmol poison was reported, 34 years aged women consumed few ml of super vasmol hair dye on 11.05.2009. at 7:00 A.M in her house. Supervasmol 33™ is an emulsion based hair dye commonly used in India. At autopsy External findings - there was significant neck swelling. On Internal examination Brain, Lungs, Liver, Kidneys were congested. In this paper the focus is mainly on lung manifestations.

Key words

Super Vasmol poisoning ,PPD, lungs, Congestion.

Introduction

Hair dye ingestion is very common in Africa and Asia^{1,2}. The main component of hair dye causing toxicity is a Para phenylene diamine (PPD), resorcinol, propylene glycol, liquid paraffin, sodium lauryl sulphate, EDTA sodium and herbal extracts. Accidental or suicidal ingestion of PPD causes systemic toxicity manifested by severe edema of neck and face with respiratory distress. Congestion in alveoli hinders the exchange of respiratory gases resulting in respiratory failure².

Case report

A 34 years aged women was admitted in emergency ward of R.I.M.S hospital Kadapa (Dt) India at 7:00 a.m., alleged to have consumed few ml of hair dye (P.P.D) couple of hours ago. On examination she was in drowsy condition and responding to verbal commands. Pupils were normal in size and responding to light, pulse rate 89/mit, B.P:110/70mmHg.

Signs & symptoms

Face and Neck swelling, difficult in breathing, haematuria, sweating.

Treatment

Inj. Avil 2ml I.M Stat.
Inj. Decadran 2ml I.M. Stat.
Inj. Ceftriaxone 1gm I.V/ Bid.

I.V.Fluids- 5% Dextrose, D.N.S

Inj. Rantac 2ml I.V

Pulmonary edema and Bronchospasm occurred. Due to respiratory arrest the Patient's condition became critical and shifted to I.C.U and put on ventilation.

Investigation reports

The following investigations have been reported. Hb% 10.06g/dl, TLC 18000, P 73%, L 23% E O2, M O2, Platelet count 300000, Blood urea 40 mg/dl, Serum creatine 1.1 mg/dl, Liver function test 1mg/dl, SGOT 450U/L, SGPT 200U/L, Alkaline Phosphate 150U/L, Total Protein 6.4, Serum albumin 4.2g%, Blood glucose 140mg/dl, Uric acid - Normal, Urine analysis PH - 6.5, Protein 24, No ketone bodies were found. Mechanical ventilation was continued, for a clonic - tonic seizures Injection. Midazolam was given IV. To alleviate metabolic acidosis NaHco₃ infuzer was given under arterial blood gas control. A central line was established Foleys catheter was inserted to monitor urine output. At the time of catheterization urine out put was 1100ml in the next 24hrs total urine was 800ml. The patient gradually developed oliguria reflected by the decreasing urine output on second and third day. In view of the gradually developing oliguria and increasing serum urea and creatinine levels the patient was put on peritoneal dialysis on the third day. The patient also developed pulmonary edema and hypotension and was supported with isotopes. Despite vigorous treatments, metabolic acidosis persisted; the patient could not maintain normal SPO₂ even after ventilation with 100% oxygen. Cardiac arrest was occurred despite cardiopulmonary resuscitation, the patient died on the early morning of 4th day i.e., on 15-05-09. Autopsy revealed diffused pulmonary edema, renal congestion, an eosinophilic substance in the renal cortical tubulae lumen and hyperemia in all abdominal organs. Congestion was observed in lungs. (Fig-1 & 2)

Discussion

Through hair dye poisoning is rare in western countries it is common in eastern Africa, Middle East countries and Indian sub continent. Here we have presented the key clinical manifestations of systemic hair dye poisoning.

Common hair dye ingredients used are, hydrogen peroxide, PPD, resorcinol, or aminophenol. Although there are a few studies that indicate systemic toxic effects of PPD on

Fig. 1:

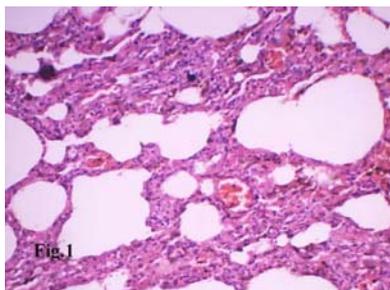
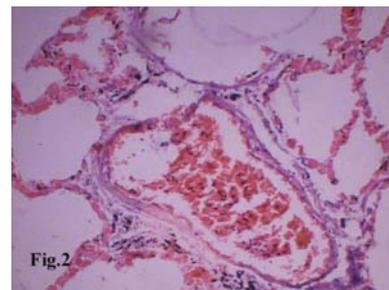


Fig. 2:



humans, information on the effect of resorcinol in acute poisoning after oral ingestion is limited. Resorcinol is of moderate acute toxicity. Studies have reported rapid absorption of resorcinol from GI tract following its oral ingestion. It is rapidly metabolized and excreted through urine. Resorcinol is also neurotoxic and its acute exposure effects range from seizures, followed by CNS depression to lethargy, coma and death³. Heart and Lung congestion was reported in PPD poisoning⁴.

In our report the patient developed symptoms common to both resorcinol and PPD poisoning, as shown by clinical presentations, lab and autopsy findings. Thus clinical manifestations of hair dye poisoning were associated with respiratory and renal symptoms.

References

1. Chug KS, Malik GH, Singhal PC. Acute renal failure following paraphenylenediamine (hair dye) poisoning: report of two cases. *J. Med* 1982; 13:131-137.
2. Ashraf W, Dawling S, Farrow LJ. Systemic paraphenylenediamine poisoning: A case report and review. *Hum Exp Toxicol* 1994;13:167-170.
3. Duran B, Gursoy S, Cetin M, Demirkopru N, Demirel Y, Gurelik B. The oral toxicity of resorcinol during pregnancy: a case report. *J. Toxicol Clin Toxicol* 2004; 42(5):663- 666
4. Abidi K, Himdi B, Cherradi N, Lamalmi N, Alhamany Z, Zeggwagh AA, Abouqal R. Mayo cardiac lysis in fetus induced by maternal PPD poisoning following intentional ingestion to induce abortion. *Human and Experimental toxicology*, 2008; 7 (5): 435-438

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