Editorial

MEDICO-LEGAL CARE FOR FEMALE VICTIMS OF SEXUAL VIOLENCE

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Abstract

The Ministry of Health & Family Welfare, Government of India has drafted detailed Guidelines & Protocols for Medico-legal care, treatment and rehabilitation of survivors / victims of Sexual Violence in India which recognize the positive role of the health sector towards providing empathetic support and rebuilding lives after assault. The health care system is the only institution that interacts with almost every woman at some point in her life and women living with violence are likely to visit health facilities more frequently than non-abuse women. Health providers need to be fully sensitized and trained to mitigate both the short term and long-term health effects of gender based violence on women and their families.

Keywords: Sexual Violence, World Health Organization. Sexual Assault, Rape, Sexual Harassment, Right to Health, Standard Operative Procedure, Informed Refusal, Two-Finger Test, Registered Medical Practitioner, LGBT persons, Psycho-Social Care.

Introduction

Sexual violence against women is a global and age-old problem. It is never a physical one alone but also violation of her dignity, self-esteem and confidence while inflicting severe mental injury to her. Once subjected to such violence, she has to further undergo lot of stress and strain in getting herself medically examined and reporting to police authorities. Many a times, medical evidence has not been recorded and documented in a proper fashion there by leading to a poor conviction in such cases. To bring about a certain degree of uniformity in approaching, treating and documenting cases of sexual violence, mainly against female victims and as a step towards sexual violence mitigation, the Ministry of Health and Family Welfare, Government of India has come up with certain guidelines which are being discussed in brief to sensitize the medical professionals dealing with such cases.

Definition of Sexual Violence:-

The World Health Organization (WHO) defines Sexual Violence as "any sexual act, attempt to obtain a sexual act, unwanted sexual comments/advances and acts to traffic, or otherwise directed against a person's sexuality, using coercion, threats of harm, or physical force, by any person regardless of relationship to the victim in any setting, including but not limited to home and work" [1]. Sexual assault, a form of sexual violence, is a term often used synonymously with rape. However, sexual assault could include anything from touching another person's body in a sexual way without the person's consent to forced sexual intercourse – oral and anal sexual acts, child molestation, fondling and attempted rape.

Forms of Sexual Violence include

- Coerced/forced sex in marriage or live in relationships or dating relationships.
- Rape by strangers.
- Systematic rape during armed conflict, sexual slavery.

- Unwanted sexual advances or sexual harassment.
- Sexual abuse of children.
- Sexual abuse of people with mental and physical disabilities.
- Forced prostitution and trafficking for the purpose of sexual exploitation.
- Child and forced marriage.
- Denial of the right to use contraceptive or to adopt other measures to protect against STIs.
- Forced abortion and forced sterilization.
- Female genital cutting.
- Inspections for virginity.
- Forced exposure to pornography.
- Forcibly disrobing and parading naked any person.

The health concerns of survivors/victims of sexual violence, and their right to health is an issue of importance. The Right to Health is not a fundamental right in India. However, the Supreme Court has interpreted the Right to Life as including Right to Health. The Right to Health is enshrined in a number of international instruments ratified by India, including the International Convention on Economic, Social and Cultural Rights (ICESCR), the Convention on the Elimination of Discrimination against Women (CEDAW), the Convention of the Rights of the Child (CRC), and the Convention on the Rights of Persons with Disabilities (CRPD). The Criminal Law Amendment Act 2013 has expanded the definition of rape to include all forms of sexual violence – penetrative (oral, anal. vaginal) including by objects/weapons/fingers and non-penetrative (touching, fondling, stalking etc.) and recognized right to treatment for all survivors/victims/victims of sexual violence by the public and private health care facilities.

Priorities of the Ministry of Health & Family Welfare

These include,

a. Provide medical assistance to the persons suffering from violence.
b. Provide psychosocial assistance to both victim and perpetrator of violence, if required. Health facilities can be instructed to handle all cases of violence/suspected Gender Based Violence compassionately and to encourage them to seek the help of psychologist / psychiatrist.
c. Help the law enforcing agencies to bring to book the perpetrators of Violence by conducting the necessary medico-legal examination.
d. Refer those women, who come to healthcare facilities for self or childcare, to appropriate agencies as stated above, if it is suspected that they may be suffering from any kind of violence.
e. Give parenting lessons to women coming to health care facilities for child care.
f. Provide information about the ill-effects of drug abuse and alcoholism in order to help people in abstaining from such activities.
g. Lay down standard operative procedures for the care, treatment and rehabilitation of survivors/victims of sexual violence.
h. Propose to use these guidelines and protocol in all the health care facilities under the Ministry of Health and Family Welfare.

1. Aim of the Protocol and Guidelines formulated by Ministry of Health & Family Welfare, Government of India:-
   1. Operationalise informed consent and respect autonomy of survivors in making decisions about examination, treatment and police intimation.
   2. Specific guidance on dealing with persons from marginalized groups such persons with disabilities, sex workers, LGBT persons, children, persons facing caste, class or religion based discrimination.
   3. Ensure gender sensitivity in the entire procedure by disallowing any mention of past sexual practices through comments on size of vaginal introitus, elasticity of vagina or anus. Further, it bars comments of built/height-weight/nutrition or gait that

perpetuate stereotypes about victims.

4. Focus on history by recognizing various forms and dynamics of sexual violence including activities that lead to loss of evidence.
5. Evidence collection based on science and history, with specific guidance for taking relevant samples and preservation of evidence.
6. Lay down Standard Treatment protocols for managing health consequences of sexual violence.
7. Lay down Guidelines for provision of first line psychological support.

The Guidelines

The Committee for drafting the guidelines was constituted by Government of India on 18th March, 2013 Vide order No. Z.28015/21/2013-H of the Ministry of Health & Family Welfare with relevant Terms of Reference and these guidelines were notified on 19th March of 2014(3). The guidelines focus on health consequences and role of health professionals, response to special groups including children, operational issues, medical examination and reporting for sexual violence, psycho-social care for survivors/victims, interface with other agencies such as police and judiciary with instructions for the doctors as follows:-

1. Informed Consent: Doctors shall inform the person being examined about the nature and purpose of examination and in case of child to the child's parent/guardian/person in whom the child reposes trust. This information should include:
    a. The medico-legal examination is to assist the investigation, arrest and prosecution of those who committed the sexual offence. This may involve an examination of the mouth, breasts, vagina, anus and rectum.
    b. To assist investigation, forensic evidence may be collected with the consent of the survivor. This may include removing and isolating clothing, scalp hair, foreign substances from the body, saliva, pubic hair, samples taken from the vagina, anus, rectum, mouth and collection of blood samples.
    c. The survivor or in case of child, the parent/guardian/person in whom the child reposes trust, has the right to refuse either a medico-legal examination or collection of evidence or both, but that refusal will not be used to deny treatment to survivor after sexual violence.
    d. As per the law, the hospital/examining doctor is required to inform the police about the sexual offence. However, if the survivor does not wish to participate in the police investigation, it will not result in denial of treatment for sexual violence. Informed refusal will be documented in such cases.
2. Per vaginum examination, commonly referred to by lay persons as 'two-finger test', must not be conducted for establishing an incident of sexual violence and no comment on the size of vaginal introitus, elasticity of the vagina or hymen or about past sexual experience or habituation to sexual intercourse should be made as it has no bearing on a case of sexual violence. No comment on shape, size, and/or elasticity of the anal opening or about previous sexual experience or habituation to anal intercourse should be made.
3. Injury Documentation: Examine the body parts for sexual violence related findings 9 such as injuries, bleeding, swelling, tenderness, discharge). This includes both micro mucosal injuries which may heal within short period to that of severe injuries which would take longer to heal.
4. Injuries must be recorded with details – size, site, shape, and colour.

5. If a past history of sexual violence is reported, then record relevant findings. Sexual violence is largely perpetrated against females, but it can also be perpetrated against males, transgender and intersex persons.
6. The nature of forensic evidence collected will be determined by three main factors – nature of sexual violence, time elapsed between incident of sexual violence and examination and whether survivor has bathed or washed herself.
7. Opinion: The issue of whether an incident of rape/sexual assault occurred is a legal issue and not a medical diagnosis. Consequently, doctors should not, on the basis of medical examination conclude whether rape/sexual assault had occurred or not. Only findings in relation to medical findings should be recorded in the medical report.
8. Drafting of provisional opinion should be done immediately after examination of the survivor on the basis of history and findings of detailed clinical examination of the survivor.
9. It should be always kept in mind that normal examination findings neither refute nor confirm sexual violence. Hence circumstantial / other evidence may also be taken into consideration.
10. Absence of injuries may be due to
    a. Inability of survivor to offer resistance to the assailant who is under intoxication or threats.
    b. Delay in reporting for examination.

Health consequences of Sexual Violence

In addition to being a violation of human rights, sexual violence is an important public health issue as it has several direct and indirect health consequences. Survivors of sexual violence may present to the health care services with varying signs and symptoms, both physical and psychological, prompting one to suspect the possibility of sexual abuse / assault. Section 164 (A) of the Criminal Procedure Code lays out following legal obligations of the health worker in these cases:-

- Examination of a case of rape shall be conducted by a Registered Medical Practitioner (RMP) employed in a hospital run by the government or a local authority and in the absence of such a practitioner, by any other RMP.
- Examination to be conducted without any delay and a reasoned report to be prepared by the RMP.
- Record consent obtained specifically for this examination.
- Exact time of start and close of examination to be recorded.
- RMP to forward report without delay to Investigation Officer (IO) and in turn IO to Magistrate (4).

The Criminal Law Amendment Act 2013, in Section 357 CrPC says that both private and public health professionals are obliged to provide treatment. Denial of treatment of rape survivors is punishable under Section 166-B IPC with imprisonment for a term which may extend to one year or fine or with both. Health professionals need to respond comprehensively to the needs of the survivors. It is also important to establish a rapport with the survivor.

Responding to Special Groups

The health professionals must be alert to the specific health care needs of different marginalized groups and be equipped to respond to them in an appropriate, comprehensive and sensitive manner in a difficult situation. Special guidelines for these groups stem from recognition of the historical stigmatization faced by the marginalized groups in accessing health services and include transgender and intersex persons, persons with altered sexual orientation, sex workers, persons with disability and people facing caste, class or religion based discrimination.

Responding to Children

The prevalence of child sexual abuse in India is known to be high. Children are considered soft targets for sexual abuse because they may not realize that they are being abused. While the principles of medical examination and treatment for children

remain the same as that for adults, it is important to keep some specific guidelines in mend:-

- In case the child is under 12 years of age, consent for medical examination needs to be sought from the parent or guardian.
- Children may be accompanied by the abuser when they come for medical treatment, so be aware and screen when you suspect abuse. In such situations, a female person appointed by the head of the institution / hospital may be called in to be present during the examination.
- Do not assume that because the child is young he/she will not be able to provide a history.
- Believe what is being reported by the child.
- Specific needs of the children must be kept in mind while providing care to child survivors.

Operational issues

Every hospital must have a Standard Operating Procedure (SOP) for management of cases of sexual violence:-

1. To provide comprehensive services.
2. For the smooth handling of the cases and clarity of roles of each staff.
3. To have uniform practice across all doctors in the hospital.

The SOP must be printed and available to all staff of the hospital.

Medical Examination and Reporting

The following guidelines are for health professionals when a survivor of sexual violence reports to a hospital:-

a. Initial resuscitation / first aid.
b. Informed Consent for examination, evidence collection, police procedures.
c. Detailed history taking.
d. Medical examination.
e. Age estimation (physical / dental / radiological)-if requested by the investigating agency.
f. Evidence collection as per protocol.
g. Documentation.
h. Packing, sealing and handing over the collected evidence to police.
i. Treatment of injuries.
j. Testing / prophylaxis for STIs, HIV, Hepatitis B & Pregnancy.
k. Psychological support & counseling.
l. Referral for further help (shelter, legal support).

Psycho-Social Care

Clinical guidelines for responding to IPV and sexual assault, W.H.O. 2013. (5) Health care providers should, as a minimum, offer first-line support when women disclose violence which included:

A. Ensuring consultation is conducted in private.
B. Ensuring confidentiality, while informing women of limits of confidentiality.
C. Being non-judgmental and supportive and validating what the woman is saying.
D. Providing practical care and support that responds to her concerns, but does not intrude.
E. Asking about her history of violence. Listening carefully, but not pressuring her to talk.
F. Helping her access information about resources, including legal and other services that she might think helpful.
G. Assisting her to increase safety for herself and her children, where needed.
H. Providing or mobilizing social support.
I. Health professionals have to interface with other agencies such as the police, public prosecutors, judiciary and the child welfare committees to ensure comprehensive care to survivors of sexual violence and they must be familiar with the same for smooth interagency coordination.

Conclusion

Taking into consideration the rise in the reported cases of violence against women and also the gaps in responding to the needs of survivors of sexual violence at various levels, the Ministry of Health and Family

Welfare, Government of India is committed to setting up of standardized protocols for care, treatment and rehabilitative services for survivors of sexual violence. These guidelines and protocols recognize the role of the health sector and is a positive way forward towards providing empathetic support and rebuilding lives after assault. The health care system is the only institution that interacts with almost every woman at some point in her life and women living with violence are likely to visit health facilities more frequently than non-abuse women. Interventions by health providers can potentially mitigate both the short term and long-term health effects of gender based violence on women and their families.

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4. Criminal Procedure Code Section 164 (A)
5. Clinical guidelines for responding to IPV and sexual assault, WHO 2013.

Original Research Paper

MORTALITY INDICATORS IN ALUMINIUM PHOSPIDE POSIONING

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Abstract

Aluminium phosphide (AlP) is a highly toxic pesticide and rodenticide which is commonly used in India esp. in North India for food grain preservation. Aluminium phosphide is very effective and thus its use is increasing as pesticide and rodenticide in the rural as well as sub urban regions for storage purposes of grains. In this study we have recorded multiple variables and observed that cardiac rhythm changes like arrhythmias, ST wave changes, circulatory changes like low Blood Pressure, low pH, Low bicarbonate levels and high CPK level at the time of admission are important mortality indictors and increasing efforts should be made to keep these parameters within normal range to reduce the mortality which is 50.72 % as per current study.

Keywords: Aluminium phosphide; Inotopic; Mortality

Introduction

Aluminium phosphide (AlP) is a highly toxic pesticide and rodenticide which is commonly used in India esp. in North India for food grain preservation. Aluminium phosphide is very effective and thus its use is increasing as pesticide and rodenticide in the rural as well as sub urban regions for storage purposes of grains. It is available in northern India under name of Celphos, Quickphos etc. Due to its commercial and home use, Aluminium Phosphide in the form of tablets is freely available as over the counter drug. Since it is easily available and cheap, it is commonly used as suicidal poison. Accidental poisoning with the aluminium phosphide is also not very uncommon, since people are using piece of old cloth to put two three tablets of Aluminium Phosphide in a knot and putting in the stored grains. At the time of usage of grains, they forget to remove the tablets and accidentally cook the grains along with Celphos tablets. This leads to accidental Celphos poisoning. Usually this type of accidental poisoning is not very fatal, since celphos tablets have already released phosphine gas by coming in contact with the moisture and air in the stored grains.

On coming in contact with moisture in the environment or hydrochloric acid in stomach, AlP liberates phosphine gas (PH3), which produces all the clinical features. Phosphine gas inhibits cellular oxygen utilization and can induce lipid peroxidation [1]. As per previous studies, two kinds of Celphos poisoning have been reported: indirect inhalation of phosphine released during approved use or direct ingestion of metal phosphides [2]. In the autopsy study of unnatural deaths in Northwest India, suicidal poisoning is the most common,

causing 68.4 % of total deaths due to poisoning between 1992 and 2002 [2].

It is a more common case in adults rather than teen agers [1]. In agriculture doing countries it is commonly used poison for storage to keep away insects and rodents. Till date, there is no effective antidote for aluminium phosphide poisoning. Also, some experimental results suggest that magnesium sulphate, N-acetyl cysteine (NAC), glutathione, vitamin C and E, beta-carotenes, coconut oil and melatonin may play an important role in reducing the oxidative outcomes of phosphine [1].

As per previous studies, phosphine mainly binds cytochrome oxidase and changes the valences of the haeme component of haemoglobin [3]. It also induces oxidative stress and produces oxygen free radicals [4] that results in lipid peroxidation and protein denaturation of the cell membrane [5, 6] in various organs. As per Abdollahi [7] oxidative stress is one of the main mechanisms of action of AlP toxicity that is somehow similar to that of organophosphate (OP) compounds. Furthermore, AlP reduces glutathione, which is one of the main antioxidant defences. In fact, AlP and OP alike cause a toxic stress that is accompanied by changes in glucose metabolism [8, 9]. Al-Azzawi observed [10] that in vitro exposure to phosphine leads to reduction of human serum cholinesterase activity, depending on the duration and phosphine concentration. On the other hand, some studies found no change in erythrocyte cholinesterase activity in accidental phosphine inhalation cases (11).

On ingestion of Celphos, phosphine gas is released which is rapidly absorbed into the entire gastrointestinal tract. It reaches the circulation and to liver through portal vein. On inhalation, it is rapidly absorbed through lungs. The phosphine is excreted unchanged in expired air; part of absorbed phosphine is oxidized to phosphite and hypophosphite ions and thus excreted in urine [12].

The purpose of this cross sectional study was to study the profile of patients presenting with Celphos poisoning in emergency and to deduce mortality indicators from Celphos poisoning.

Material and Methods

This is cross sectional study done in emergency cases coming to Dayanand Medical College Hospital, Ludhiana, India as Aluminium phosphide poisoning cases. Only those cases were considered where aluminium phosphide poison is taken in any form and confirmed by patients themselves and their relatives during the patient treatment and also cross checked with sign and symptoms presented. In this study, 69 patients of Celphos poisoning are considered and all other cases of non Celphos poisoning are discarded. Multiple parameters are studied (Table 1).

Table 1 showing different parameters studied.
Demographic Profile including Age, Sex
Reason for consumption
Type of chemical- Tablet, powder
Haemodyanamics
Laboratory parameters including CPK-MB , ABG etc.
ECG abnormalities
Treatment given

Observation & Result

In our study, a total of 69 patients of AlP were considered. The male patient outnumbered the female patient and is present in the ratio of 2:1 (46:23). On the basis of cases reported in emergency, the mortality rate is 50.72 % (35/69). On the other hand, mortality in males is more than females i.e. 56.53% vs. 39.13% (Table 2). Sex wise distribution is shown in table along with survivors and non survivors. The majority of patients were young in the age group of 21 to 30 years as 51 out of 69 [Figure 1]. Most of the cases involved suicidal consumption of the poison (53.6%) table 3.

It is also observed that patients whose systolic blood pressure is more than 90 mm of Hg at the time of admission, is having less mortality as compared to the patients having systolic blood pressure less than 90 mm of Hg. This is also showing positive correlation (Figure 2).

Table 2 showing Sex-wise distribution of cases

	Survivors	**Non- survivors**	**Total**
Males	20	26	46
Females	14	9	23
Total	34	35	69

Table 3 showing distribution of cases as per Manner of death

Sr. No	Manner	Cases
1	Suicide	37
2	Homicide	0
3	Accident	6
4	Unknown	26

Figure 1 showing distribution of patients age-wise

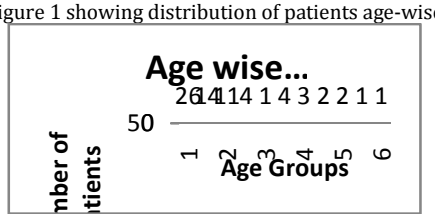
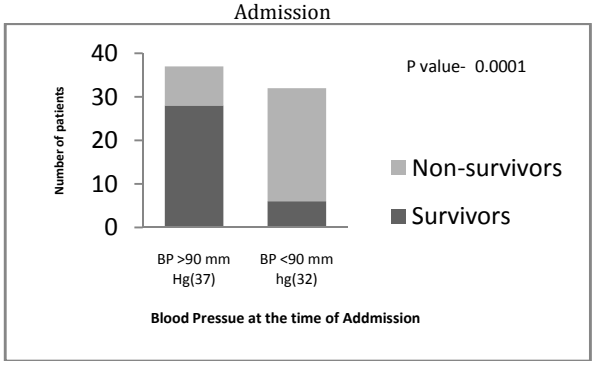
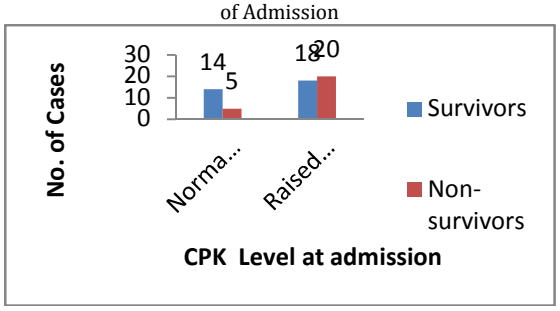


Figure 2 showing distribution of BP at time of Admission



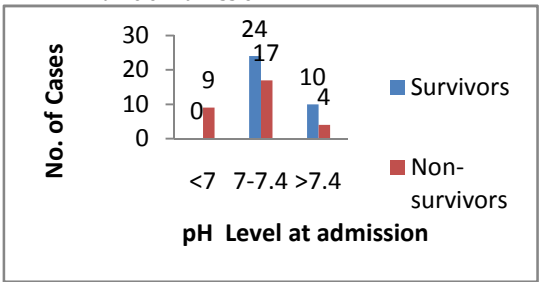
In our study, it is also observed that patients whose CPK level is within normal limits at the time of admission, are having less mortality as compared to the patients having raised levels (38 % vs. 52.63 %). This is also showing positive correlation (figure 3).

Figure 3 showing distribution of CPK level at time of Admission



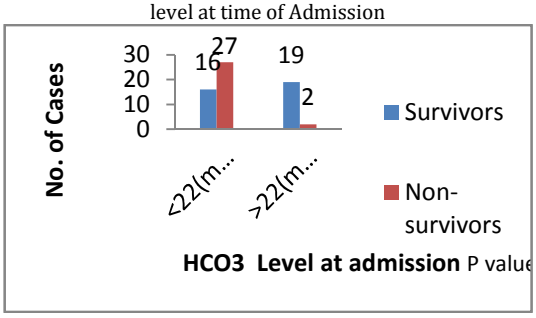
In our study, it is also observed that patients whose blood pH is less than 7.0 at the time of admission, are having more mortality as compared to the patients having blood pH more than 7.4 (100 % vs. 28.5%). In pH range 7 to 7.4, the mortality is 41.5 % (17 out of 41). This also suggests that metabolic acidosis should be corrected as early as possible to increase the survival rate by giving bicarbonate (Figure 4).

Figure 4 showing correlation of blood pH level at time of Admission



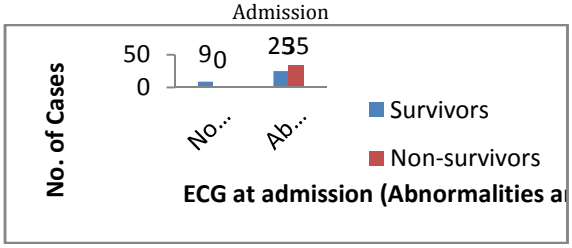
In our study, it is also observed that in patients whom blood bicarbonate level at the time of admission is less than 22.0 mEq/L, is having more mortality. This also suggests positive correlation (figure 5).

Figure 5 showing correlation of blood bicarbonate level at time of Admission



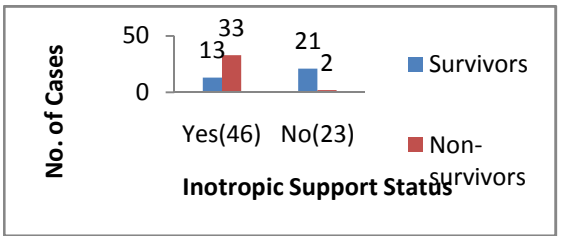
In our study, it is also observed that in patients who are showing ECG abnormalities at the time of admission, are having high mortality. Most common ECG abnormalities observed are arrhythmias, ST segment changes, QRS intervals prolongation etc. This also suggests positive correlation (Figure 6).

Figure 6 showing ECG pattern at time of Admission



In our study, it is also observed that patients who require inotropic support at time of admission have more mortality as compared to patients who didn't require inotropes. This also suggests positive correlation (Figure 7).

Figure 7 showing status of Inotropic support given during hospital stay



Discussion & Conclusion

As we already observed that aluminium phosphide is lethal poison. We have to give treatment to the patients very quickly. As there is no antidote available for the poisoning, we have to give the supportive treatment only. On lines with earlier study [13] it was observed that aluminium phosphide poisoning results in GIT symptoms like nausea, vomiting, abdominal discomfort, cardiac rhythm changes liker arrhythmias, ST wave elevation, circulatory changes like low Blood Pressure, low pH, Low bicarbonate levels and high CPK level which act as mortality indicators of the aluminium phosphide poisoning.

We have to correct all the indicators/parameter mentioned above to decrease the mortality due to the aluminium phosphide poisoning. We have to device a treatment protocol for correcting all these parameter, so that treatment for the aluminium phosphide poisoning can be started from the primary centre. In this way, patients can get the treatment at the primary level and thus undue delay for getting treatment by reaching the tertiary level is decreased.

Conflict of interest

None

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Original Research Paper

AUTOPSY BASED RETROSPECTIVE STUDY OF CAUSES OF DEATH IN VICTIMS OF ROAD TRAFFIC ACCIDENTS

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Abstract

Road Traffic Accidents (RTA) are increasing at an alarming rate all over the world and act as a major epidemiological as well as medico-legal problem. This autopsy based retrospective study of causes of death in road traffic deaths was conducted at Department of Forensic Medicine**,** S. S. Institute of Medical Sciences and Research Centre, Davangere, Karnataka, India, during the period 2011 to 2012. A total of 170 post-mortem cases that died due to RTA were included in the study. Objective of this study was to find out the cause of death in fatal road traffic accident. Most common cause of death was found to be cranio-cerebral injuries, followed by haemorrhage and shock, septicaemia, spinal cord injuries and pulmonary embolism. Strict enforcement of traffic rules with compulsory use of road safety measures like helmet wearing, using seat belts will help to reduce the incidence of fatal injuries to the body. Legal enforcement is not just enough to cut down such mortalities, this requires multi-dimensional approach to bring awareness among the public about road safety measures and to shift such victims immediately to the hospitals without waiting for the legal formalities.

Keywords: Road Traffic Accident (RTA); Cause of Death; Cranio-cerebral injury; Autopsy;

Introduction

Road Traffic Accident (RTA) is any vehicular accident occurring on the roadway that is, originating on, terminating on, or involving a vehicle partially on the roadway [1]. Accident can be sudden, unexpected and may occur inadvertently. Worldwide the number of people killed in road traffic crashes each year spastically estimated to be around 1.2 million, while the number of injured could be as high as 50 million [2] Road traffic accident represents 45 to 50 % causes of head injuries and young adult were the most common victim [3].

RTA is due to the tremendous increase in the number of vehicles, high-speed technology and also contributing factors like poor roads, intoxicating influence of alcohol or drugs, poor safety measures, ignorance or intentional violation of traffic rules. Fatality in RTAs can be due to immediate causes like haemorrhage, injury to vital organs, vagal inhibition, neurogenic shock, embolism etc. and late causes like infection, complications of

injuries, etc. The present study was conducted with the objective to know the cause of death due to RTA.

Material and Method

The present retrospective study material comprised of 170 cases that died due to RTA and subsequently autopsied at the S.S.I.M.S. & R.C., Davangere, Karnataka, during the period of two years from 2011 to 2012. Controversial RTA cases were excluded from the study.

Observation and Results

A total of 420 cases were autopsied during the two-year study period (2011-12). Out of which 170 cases were due to RTA. Males outnumbered the females in total number of deaths due to RTA, 143 (84.11%) male cases compared to 27 (15.88%) female cases (Table 1). The male to female ratio in the study was 5.29:1.

Age group most commonly involved in male sex was 21–30 years (50 cases), followed by 31-40 years (32 cases), 41-50 years (28 cases), 51-60 years (16 cases), 11-20 years (6 cases), 61-70 years (6 cases), less than 10 years (2 cases) and least in the age group of 71-80 years (1 case).

In females maximum deaths occurred in the age group of 31-40 years (11 cases), followed by 41-50 years (6 cases), 5 cases in the age group of 51-60 years, 2 cases in the age group of 21-30 years, and 1 case each in less than 10 years, 10-20 years, 61-70 years. In total most commonly involved age group in both sexes is 21-30 years (30.58%), followed by 31-40 years (25.29%), 41-50 years (20%), 51-60 years (12.35%), 11-20 years and 61-70 years (4.11%), and 2.94% in the age less than 10 years.

Out of 170 cases, cranio-cerebral injury which includes either intracranial haemorrhage, skull fracture and or cerebral damage, accounted for death in 108 cases, haemorrhage and shock was accountable in 45 cases, 11 cases died due to septicaemia, 5 cases due to Spino-vertebral damage, and one case due to pulmonary embolism (Table 2).

Discussion

In present study maximum number of RTAs victims were 143 male cases (84.11%), compared to 27 (15.88%) female cases. The male to female ratio in the study was 5.29:1. This is in agreement with the study done by Singh [4] wherein male to female ratio was 4.9:1. Age group most commonly involved in both sexes was 21–30 years (52 cases; 30.58%), 31-40 years (43 cases; 25.29%) and 41-50 years (20%).

The maximum numbers of victims were younger age group because of their ambulatory nature as compared to persons belonging to other age groups. Present study and its results are consistent with the study done by Singh H [4] showed that the commonest age group involved was 21-30years (27.3%), followed by 31-40 years (20.6%).

In our study cranio-cerebral injury was the commonest cause of death due to RTA, which accounted for 108 cases (63.5%), followed by shock and haemorrhage in 45 cases (26.4%), septicaemia in 11 cases (6.47%), spinal cord injury in 5 cases (2.94%) and one case (0.058%) due to pulmonary embolism. Cranio-cerebral injuries were the commonest cause of death due to RTA in this study is in consistent with the studies done by Singh [5], Sharma [6], and Moharamzad [7].

In an earlier study, [5] head injuries were accountable for about 80% of total death, followed by haemorrhage and shock accountable for about 16% Combined effect of cranio-cerebral damage, haemorrhage and shock were responsible for cause of death in 5.88% of cases. Spino-vertebral damage, Septicaemia and fat embolism were less common cause of death. Study conducted by Moharamzad [7] showed that most common cause of death was central nervous system injury (146 cases, 58.1%). The other causes were skull base fractures (10%), internal bleeding (8%), lower limb haemorrhage (8%), skull vault fractures (4%), cervical spinal cord injury (3.6%), airway compromise (3.2%), and multifactor cases (5.1%), respectively.

Conclusion

Road traffic accidents are causing the loss of valuable work force and

resources. Improvement of roads, streetlights and displaying of traffic signs needed. Cranio-cerebral injuries were the commonest cause of death due to RTA in our study. These fatal injuries can be avoided by strict enforcement of law of using helmets for both pillion and rider, compulsory wearing of seat belts in four wheelers. Public need to get more awareness of the road safety measures by mass media and display of hoardings at waiting signals.

Conflict of Interest

None declared.

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Table 1 showing age and sex wise distribution of RTA cases.

Age group	Male	Female
<10 years	04	01
11-20 years	06	01
21-30 years	50	02
31-40 years	32	11
41-50 years	28	06
51-60 years	16	05
61-70 years	06	01
71-80 years	01	--
Total	**143**	**27**

Table 2 showing causes of death due to RTA

Sl. No.	Cause of death	Year 2011	Year 2012	Total cases
1	Cranio-cerebral injury	56	52	108
2	Haemorrhagic shock	18	27	45
3	Septicaemia	05	06	11
4	Spinal injury	04	01	05
5	Pulmonary embolism	01	--	01

Original Research Paper

EPIDEMIOLOGY OF PEDESTRIAN FATALITIES IN MANIPAL: A RETROSPECTIVE STUDY

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Abstract

Road traffic accident (RTA) is a major price we pay for urbanization. Forecast suggests that by the year 2020, RTA will move to 6th place as a cause of death & disability. Pedestrians injured in automobile accidents constitute one of the most frequent serious problems in management for emergency room surgeon. With increasing RTAs, pedestrian injuries as well as fatalities are on a rise.

Factors responsible for pedestrian fatalities include pedestrian factors such as disobeying the traffic rules, walking or playing on the road, children darting or running on the road, failure to yield right of way and alcohol intoxication, while driver factors include failure to keep proper lane, alcohol intoxication and failure to yield right of way.

Our study of pedestrian fatalities was for a period of 5 years in Manipal, a coastal township in south of Karnataka state includes the study of pattern of injuries among pedestrians. The results of the study are compared with studies done elsewhere in India and abroad.

Keywords: Road traffic accident, Pedestrian, fatalities

Introduction

Accidents are the 4th leading cause of mortality around the world. Pedestrians share a major part of road traffic accidents. They are the most vulnerable road users. About 65 percent of 1.17 million annual traffic fatalities world-wide are pedestrians [1]. Human, vehicle and environmental factors play roles before, during and after trauma event. Both motorists and pedestrians are frequently observed committing road-rule violations at signalized. Intersections suggesting a potential human behavioural contribution to pedestrian injury.

Pedestrian factors include - Pedestrians wearing headphones, talking on a cell phone, eating & drinking, smoking or talking while crossing the roadway, children darting or running on the road. Motorist Factors include failure to keep proper lane, drunken driving, failure to yield right of way.

Materials & methods

This autopsy based retrospective research was conducted to determine the causes and the epidemiological aspects of Pedestrians fatalities in Manipal, a coastal township in South India. The study was conducted at the Department of Forensic Medicine, Kasturba Medical College, Manipal. All the cases recorded as pedestrian deaths from January 2001 to

December 2012 were included in the study. Autopsy case files, police requisition letters and hospital case files, if available of the same were studied in detail. The data was analyzed using statistical software, Statistical Package for the Social Sciences.

Results

Fig 1 showing percentage of Pedestrian fatalities

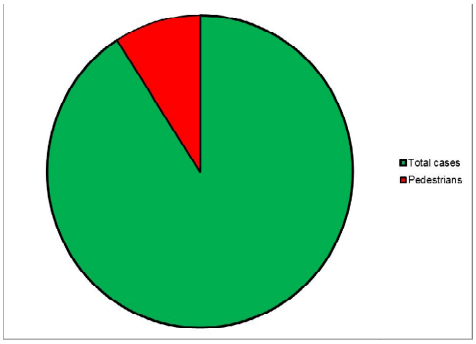
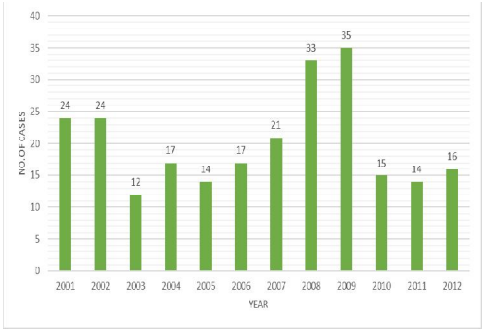


Fig. 2 showing yearwise distribution of cases



A total number of 2408 medico-legal autopsies were performed during this period. Out of these 242 cases were cases due to pedestrian fatalities as seen in Fig. 1. It accounted for 10.09% of the total cases. The year wise distribution of cases is shown in Fig. 2. Maximum no. of cases was recorded in 2008 and 2009 constituting 33 and 35 cases respectively. It shows gradual increase in no. of cases during study. Out of 242 cases 205 were males and 37 were females as seen in Fig. 3. The fifth decade had the highest number of cases followed by the sixth as seen in table 1. Mean age of the study population was 42.73 years and it varied from 1 to 83 years. Cases were reported highest in the winter season followed by rainy season as depicted in Fig. 4. The offending vehicle in maximum number of cases of pedestrian injury was a two wheeler, followed by a heavy motor vehicle as seen in Fig. 5. Head injury was the most common fatal injury in maximum no. of cases followed by the trunk. The 8 hour interval of 4:00 PM to 12:00 AM had the highest no. of cases, average being 6:43 PM as seen in table 2.

Fig 3 showing gender wise distribution of cases

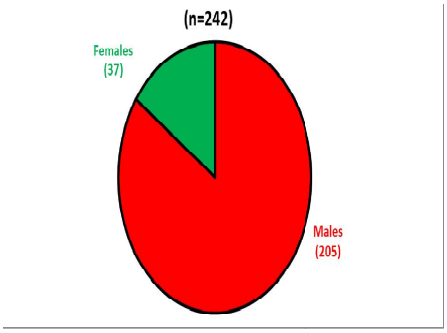


Fig 4 showing month wise distribution of cases

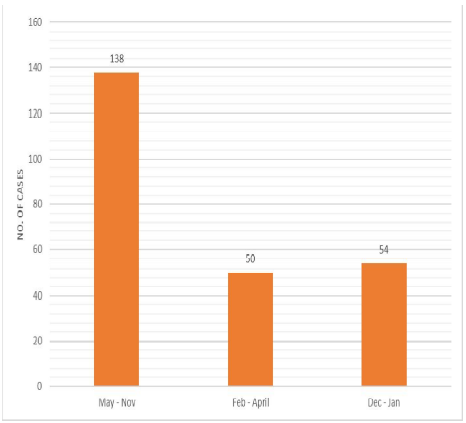
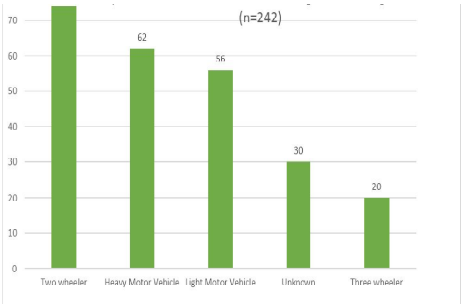


Fig 5 showing distribution of cases according to offending vehicle



Discussion

Pedestrian injuries contribute to majority of fatalities. This can be understood by the fact that, walking essentially remains an important mode of transport in south India. Majority of roads here lack sidewalks, crosswalks and traffic signals leading to increased pedestrian injuries as well as fatalities. Also pedestrian injuries may increase by hawking on the sidewalks and on the road, social and religious processions on the road which are very much prevalent in this region.

No. of male pedestrian fatalities predominated in our study. Similar findings were observed in various studies [2, 3, 4]. This can be explained by the fact that males being the working category use roads more for walking and transport from one place to another as compared to females.

Fifth decade had the highest no. of cases; this is because this age group prefers walking as a means of transport for short distances. Plus people in the fifth decade are the part of main working group in this area increasing their risk of exposure.

The highest no. of cases in our study was reported during the period from May to November which corresponds to the Rainy season i.e. (June to September), as rain starts early and continues till November in this part of the country. These findings differ from the findings of the study done by Kjetil S et al [5] where most of the cases were from the spring or summer season. In rainy season visibility is compromised because of rains both the pedestrians and the motorists are in a hurry to avoid the rains and the wet condition of the roads adds to the problem.

Motorcycle was the offending vehicle in highest no. of cases followed by heavy motor vehicles like bus, trucks and lorry. But studies done by Baldwin A [6] stated that the Heavy motor vehicle as the offending vehicle in highest no. of cases. This can be attributed to the fact that motorcycle/two wheelers and buses are widely used as a means of transport in this area.

Head injury was the most common fatal injury followed by the trunk as seen in table 3. These findings differed from the findings of the study done by Jean Louis Martin et al [7] which stated lower limb injuries as the most common pedestrian injury followed by the head. Whereas Longhitano et al [8] reported trunk as the most common site of pedestrian injury. This can be explained by fact that in western world countries heavy motor vehicles like mini vans, pickup trucks and SUV's are the most common mode of transport which corresponds to the level of trunk. Whereas in developing country like India two wheelers and Light motor vehicles like cars are preferred mode of transport.

The 8 hr time interval of 4:00 PM TO 12:00 AM had the highest no. of cases with an average time of 6:43 PM for this interval. Similar findings were observed by Derry D et al [9] and can be explained by the fact that twilight phase in the evening is a phase of reduced visibility. On the other hand these hours are the busiest as commuters are in a hurry to return home from their offices, colleges, schools and tuitions.

Safety/Preventive Measures:

The three E's of traffic safety – education, enforcement, and engineering are the primary tools available for pedestrian injury prevention. The Global Road Safety Partnership strongly recommends road safety education of children worldwide.

Regular eye and ear checkups have to be done for all in their fifth decade to have appropriate sensory inputs. In case of people with sensory deficits, they should always be accompanied by another healthy person when it comes to road safety.

Street lighting may prevent road traffic crashes, injuries, and fatalities for all roads [10]. It should be switched on by 6.30

pm as in our study maximum number of pedestrian related accidents occurred around 6.43 pm.

Sidewalks and refuge islands are of interest to protect pedestrians from collisions with motor vehicles. Puffin (Pedestrian User Friendly Intelligent Crossing) can be used, which will reduce casualties in case of emergencies [11].

Increase awareness of road hazards promotes injury prevention (helmet use, conspicuity aids, etc.) The Global Road Safety Partnership recommends road safety education of children worldwide to reduce road traffic accidents [12]. Increase the perception of risk related to alcohol use when cycling or walking. To potentiate prevention of alcohol consumption, strict laws as to vigilance and punishment has to be passed towards drinking and driving. According to a review, alcohol ignition interlocks, prevent drivers from starting the engine if their blood alcohol concentration/ blood alcohol level is over the legal limit, appears to be effective when the device is installed in the vehicle of potential offenders [13].

Measures to reduce speed of motorized traffic

It can be done by installing speed reducers in each vehicle determining. Sensors can be installed in the vehicle to detect pedestrians to reduce pedestrian accidents. Positioned at the front of the vehicle distance sensing and braking system will prevent injuries and save lives [14]. Research and development has to be concentrated on safer car fronts to reduce pedestrian fatalities. Exterior air bags can be installed in vehicles like car to cushion a collision with pedestrians, reducing fatalities [15].

Strategic road planning for the area based on vehicle density and population can reduce pedestrian fatalities. This initiative can be taken by the government to prevent these mishaps. Speed humps and rumble strips can be used to calm the traffic in the built up areas [9].

Finally at the time of giving licence, people can be trained in First aid so as to decrease pedestrian fatalities.

Conclusion

Pedestrians consider that crash avoidance is up to motorists only. Walking in the street is often considered a common life activity carrying no particular hazard. But pedestrian can be at fault too.

Measures should be taken to segregate motorized and non-motorized road users, especially in locations where collisions are most likely to occur.

At the time of giving license people can be given training in first aid skills so that the victims are attended immediately in the post accidental period [16].

Conflict of Interest

None Declared.

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Table 1 showing age wise distribution of cases (n=242)

Age Group	No. of cases
0-9	17
10-19	21
20-29	38
30-39	27
40-49	53
50-59	39
60-69	33
70-79	15

Table 2 showing distribution of cases according to the time of incident (Mean = 6:43 PM)

Timings	No. of cases
12:00 AM – 08:00 AM	32
08:00 AM – 04:00 PM	72
04:00 PM – 12:00 AM	131

Table 3 showing distribution of cases according to Fatal Injury (n=242)

Fatal Injury	No. of cases
Head	185
Trunk	24
Polytrauma	16
Limbs	14
Others	3

Original Research Paper

FACTORS AFFECTING ESTIMATION OF TIME SINCE DEATH BY RIGOR MORTIS

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Abstract

One of the most difficult features of rigor mortis to explain is the great variation in the interval between death and the first signs of stiffening in the muscles. This study was conducted with an objective to determine the order of appearance of rigor mortis in different body parts and its relation to age, built and season. Earliest onset of rigor mortis was seen in 1-2 hours after death with maximal onset interval being 4-5 hours. Most of the cases showed onset of rigor mortis in eyelids in 3-4 hours, jaws in 4-5 hours, neck muscles in 6-7 hours, upper limbs in 8-9 hours, lower limbs in 10-11 hours, finger &toes in 11-12 hours. Children and elderly had early onset, development and passing off of rigor mortis as compared to adults. Thin-built persons also had early onset, development and passing off of rigor mortis as compared to well-built persons. Summers showed early onset, development and passing off of rigor mortis as compared to winters.

Keywords: Rigor, Post-mortem, Death, Muscles, Stiffening, Rigidity.

Introduction

Rigor mortis (death stiffening or cadaveric rigidity) is a state of stiffening of muscles, after death, with slight shortening of the fibres.[1] After death, the muscles of the body pass through three stages: (i) Primary relaxation or flaccidity, (ii) Rigor mortis or cadaveric rigidity, (iii) Secondary flaccidity. Rigor mortis appears in involuntary muscles; the myocardium becomes rigid in an hour.[1] Classically, rigor is said to develop sequentially but this is by no means constant, symmetrical or regular. Ante-mortem exertion usually causes rigor to develop first in the muscles used in the activity. Typically, rigor is first apparent in the small muscles of the eyelids, lower jaw and neck, followed by the limbs, involving first the small distal joints of the hands and feet and then the larger proximal joints of the elbows, knees and the shoulders and hips.[2]

The rigidity of the muscle is at its maximum, when the level of ATP is reduced to 15%.[3] Simultaneously, there will be a rise in lactic acid and hydrogen ion concentration due to glycolysis. When lactic acid concentration reaches a level of 0.3%, muscles go into an irreversible state of contraction known as rigor mortis. Rigor persists until decomposition of the proteins of the muscle fibres makes them incapable of any further contraction. The muscles then soften and relax.[1]

At high atmospheric temperature rigor mortis comes early and passes off early. In cold atmospheric conditions it

comes late and stays longer. In temperate climates it may take two to three hours to develop. In India, it usually commences in one to two hours after death.[4–6]

The onset is slower, and the duration longer in those cases where the muscles have been healthy and at rest before death than in those cases where the muscles have been feeble and exhausted. Rigor mortis is said not to occur in the body of an immature foetus of less than seven months.[5] In healthy adults, it develops slowly but is well- marked and lasts longer, while in children and old people it is feeble and rapid.[1]

Material and Methods

Material for the present study comprised of 175 cases brought to Mortuary of the Rajindra Hospital, Patiala. The dead bodies comprised hospital deaths and deaths in the jurisdiction of Patiala sub-division. The study comprised recording of the particulars of the deceased age, sex, muscular state (built), atmospheric conditions, temperature. Particulars of the deceased were collected from police and relatives of the deceased, including information regarding name, age, sex, address, religion, occupational status. The cases of instantaneous death (cadaveric spasm) which were 5 and bodies kept in cold chambers which were 20 were not included in the present study. So results are calculated from data of 150 cases.

Observations

The present study was undertaken to estimate time since death by rigor mortis to note the effect of following on the onset and duration of rigor mortis. The following observations were drawn as per table 1 to 10.

Discussion

Order of appearance of Rigor Mortis in different parts of body: Our study showed that minimum duration of onset of rigor mortis in eyelids was between 1-2hours and maximum was 4-5 hours with an average of 2.73 hours. Minimum duration of onset in jaws was between 3-4 hours and maximum between 5 to 6 hours with an average of 4.49 hours. Minimum duration of onset in neck muscles was 4-5 hours and maximum 7-8 hours with an average of 6.08 hours. Minimum duration of onset in upper limbs was between 6-7 hours and maximum 9-10 hours with an average of 7.90 hours. In lower limbs, minimum duration was 8¬9hours and maximum 11-12 hours with an average of 9.96hours. In Fingers & Toes, minimum duration was 10-11hours and maximum 11-12hours with an average of 11.05 hours. This observation is in agreement with the findings of Camps.[7]

Rigor Mortis in relation to Age: As is evident from Table-2 minimum duration of onset of rigor mortis in children was between 1 to 2 hours and maximum duration was between 3 to 4 hours with an average of 2.61 hours. Minimum duration of onset in elderly was between 1 to 2 hours and maximum between 3 to 4 hours with an average of 2.5 hours. Minimum duration of onset in adults was between 2 to 3 hours and maximum between 4 to 5 hours with an average of 3.8 hours. This observation is in agreement with the findings of Mathiharan and Patnaik.[5]

As in evident from Table-3 minimum duration of complete development of rigor mortis in children was between 7 to 8 hours and maximum 11 to 12 hours with an average of 9.93 hours. Minimum duration of complete development of Rigor Mortis in elderly was between 7 to 8 hours and maximum duration was between 11 to 12 hours with an average of 9.45 hours. Minimum duration in adults was 8 to 9 hours and maximum was 11 to 12 hours with an average of 10.7 hours. This observation is in agreement with Mant.[8]

As in evident from Table-4 minimum duration of passing off of rigor mortis in children <18 yrs. was between 18-24 hours and maximum duration was between 30-36hours with an average of 28.33hours Minimum duration of passing off of rigor mortis in elderly was between 18-24hours and maximum duration was between 30-36 hours with an average of 27.81hours. Minimum duration in adults was between 18-24hours and maximum duration was between 36-42hours with an average of 31.5hours. This observation is in agreement with the findings of Reddy.[1]

Rigor Mortis in relation to build: As in evident from Table-5minimum duration of onset of rigor mortis in thin built persons was between 1-2hours and maximum duration was between 4-5 hours with an average of 2.63 hours. Minimum duration in well-built was between 1-2hours and maximum duration was between 5-6 hours with an average of 3.34hours. This observation is authenticated by many workers including Dogra and Rudra, Mathiharan and Patnaik and Reddy.[1, 5, 9]

As in evident from Table-6 minimum duration of full development of rigor mortis in thin built was between 7-8hours and maximum duration was between 11-12hours with an average of 9.52hours.Minimum duration in well-built was between 8-9hours and maximum duration was between 11-12hours with an average of 10.65hours. This observation is in agreement with Mathiharan and Patnaik.[5]

As is evident from Table-7 minimum duration of passing off of rigor mortis in thin built was between 18-24hours and maximum duration was between 30-36hours with an average of 29.42hours. Minimum duration in well-built was between 24-30hours and maximum duration was between 36-42hours with an average of 32.89hours.This observation is in agreement with Nandy.[4]

Rigor Mortis in relation to seasonal variation: As is evident from Table-8 minimum duration of onset rigor mortis in winters was between 2-3hours and maximum duration was between 5-6hours with an average of 3.47hours. Minimum duration in summers was between 1-2hours and maximum duration was between 4-5hours with an average of 2.96hours. This observation is authenticated by many workers including Mant and Nandy.[4, 8]

As is evident from Table-9 minimum duration of full development of rigor mortis in winters was between 8-9hours and maximum duration was between 11-12hours with an average of 10.85hours. Minimum duration in summers was between 8-9hours and maximum duration was between 11-12hours with an average of 9.61hours. This observation is in agreement with Mathiharan and Patnaik.[5]

As is evident from Table-10 minimum duration of passing off of rigor mortis in winters was between 24-30hours and maximum duration was between 36-42hours with an average of 32.4hours. Minimum duration in summers was between 18-24hours and maximum duration was between 30-36hours with an average of 30.6hours. This observation is in agreement with Camps.[7]

Conclusion

Rigor mortis starts various body parts in sequential order. Eyelids were involved first of all followed by jaws & neck muscles. Then upper limbs followed by lower limbs and lastly fingers & toes were involved. Development of rigor mortis occurs earlier in children up to 18years & elderly >45 years as compared to adults in age group of 18-45 years. Rigor mortis passed off completely earlier in children up to 18 years and elderly >45 years as compared to adults in age group of 18-45 years. Development of rigor mortis was earlier in thin built as compared to well built. Development of rigor mortis was earlier in summers as compared to winters. Rigor mortis passed off completely earlier in summers as compared to winters.

Conflict of Interest

None declared.

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Table 1 showing onset of Rigor Mortis in Various Body Parts in Relation to Time

Site of Rigor Mortis	Time after death (inhours)	No.of cases	Percentage
Eyelids	0-1	0	0%
	1-2	9	6%
	2-3	58	38.66%
	3-4	**76**	**50.66%**
	4-5	7	4.66%
Jaws	3-4	15	10%
	4-5	**121**	**80.66%**
	5-6	14	9.33%
Neck Muscle	4-5	6	4%
	5-6	60	40%
	6-7	**75**	**50%**
	7-8	9	6%
Upper Limbs	6-7	15	15%
	7-8	35	35%
	8-9	**45**	**45%**
	9-10	5	5%
Lower Limbs	8-9	15	10%
	9-10	60	40%
	10-11	**62**	**41.33%**
	11-12	13	8.66%
Finger & Toes	10-11	70	46.66%
	11-12	**80**	**53.33%**

Table 2 showing onset of Rigor Mortis in Relation to Age

Time ofOnset	Children <18 yrs.		Adults 18-45 yrs.		Elderly >45 yrs.	
	n	%	n	%	n	%
0-1 hours	–	–	–	–	–	–
1-2hours	3	11.11	–	–	3	10.00
2-3hours	**18**	**66.66**	9	9.68	**23**	**76.67**
3-4hours	6	22.23	**46**	**49.46**	4	13.33
4-5hours	–	–	38	40.86	–	–
5-6hours	–	–	–	–	–	–
Total	27	100	93	100	30	100

Table 3 showing complete Development of Rigor Mortis in Relation to Age

Time ofOnset	Children <18 yrs.		Adults 18-45 yrs.		Elderly >45 yrs.	
	n	%	n	n	%	N
4-5hours	–	–	–	–	–	–
5-6hours	–	–	–	–	–	–
6-7hours	–	–	–	–	–	–
7-8hours	3	11.11	–	–	2	6.06
8-9hours	5	18.52	3	3.33	7	21.21
9-10hours	**14**	**51.85**	12	13.33	**19**	**57.57**
10-11hours	3	11.11	30	33.33	3	9.09
11-12hours	2	7.41	**45**	**50.00**	2	6.06
Total	27	100	90	100	33	100

Table 4 showing passing Off Of Rigor Mortis In Relation To Age

Time of completely passed off state of Rigor Mortis	Children <18 yrs.		Adults 18-45 yrs.		Elderly >45 yrs.	
	n	%	n	%	n	%
18-24hours	3	11.11	1	1.67	3	9.09
24-30hours	**15**	**55.55**	16	26.66	**22**	**66.66**
30-36hours	9	33.33	**40**	**66.66**	8	24.24
36-42hours	–	–	3	5.00	–	–
42-48hours	–	–	–	–	–	–
Total	27	100	90	100	33	100

Table 5 showing onset of Rigor Mortis in Relation toBuild

Time of Onset	Thin built		Well built	
	n	%	n	%
0-1 hours	–	–	–	–
1-2hours	6	8.57	4	5.00
2-3hours	36	51.43	16	20.00
3-4hours	24	34.28	48	60.00
4-5hours	4	5.71	8	10.00
5-6hours	–	–	4	5.00
Total	70	100	80	100

Table 6 showing full Development of Rigor Mortis

Time of full development of Rigor Mortis	Thin built		Well built	
	n	%	n	%
4-5hours	–	–	–	–
5-6hours	–	–	–	–
6-7hours	–	–	–	–
7-8hours	3	4.41	–	–
8-9hours	8	11.76	5	6.10
9-10hours	46	67.65	14	17.07
10-11hours	7	10.29	27	32.93
11-12hours	4	5.88	36	43.90
Total	68	100	82	100

Table 7 showing passing Off Of Rigor Mortis In Relation To Built

Time of completely passed off state of Rigor Mortis	Thin built		Well built	
	n	%	n	%
18-24hours	3	4.41	-	-
24-30hours	34	50.00	18	21.95
30-36hours	31	45.59	50	60.97
36-42hours	–	-	14	17.07
42-48hours	–	–	–	–
Total	68	100	82	100

Table 8 showing onset of Rigor Mortis In Relation To Seasonal Variations

Time of Onset	Winter		Summer	
	n	%	n	%
0-1 hours	–	–	–	–
1-2hours	–	–	10	11.11
2-3hours	10	16.67	43	47.78
3-4hours	38	63.33	27	30.00
4-5hours	8	13.33	10	11.11
5-6hours	4	6.67	–	–
Total	60	100	90	100

Table 9 showing full Development of Rigor Mortis In Relation To Seasonal Variation

Time of full development of Rigor Mortis	Winter		Summer	
	n	%	n	%
4-5hours	–	–	–	–
5-6hours	–	–	–	–
6-7hours	–	–	–	–
7-8hours	_	_	–	–
8-9hours	2	5.00	20	22.22
9-10hours	4	7.50	44	48.89
10-11hours	22	35.00	18	20.00
11-12hours	32	52.50	8	8.89
Total	60	100	90	100

Table 10 showing passing Off Of Rigor Mortis In Relation To Seasonal Variation

Time of completely passed off state of Rigor Mortis	Winter		Summer	
	n	%	n	%
18-24hours	_	_	2	2.22
24-30hours	15	25.00	36	40.00
30-36hours	36	60.00	52	57.78
36-42hours	9	15.00	_	_
42-48hours	–	–	–	–
Total	60	100	90	100

Case Report

SCORPION BITE CAUSING HEMIPARESIS- A CASE REPORT

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Abstract

The scorpion bite and its consequences are an important public health problem in several parts of the world. It is particularly common in Southern and coastal parts of India. It represents a major clinical entity in these parts of India causing significant mortality and morbidity. Scorpion evenomation can lead to various clinical manifestations like cardiotoxicity, respiratory dysfunction and neurotoxicity. Cerebrovascular effects are rare but lethal as they may mimic as hemiparesis. Despite advances in the treatment, the mortality remains high in rural areas on account of access to medical facilities.

Keywords: Scorpian bite; Hemiparesis.

Introduction

Scorpion sting is an acute life-threatening medical emergency of villagers. There are 1500 species of scorpions in India. Out of these Mesobuthus tamulus and Palamneus are of medical importance [1, 2]. Cardiovascular effects are particularly common following the stings by Indian red scorpion (Mesobuthus tamulus) [3]. Cerbrovascular manifestations may be a rare presentation of scorpion sting in India. About 94% of the accidents occur during the night at homes especially in rural areas and 88% do not require any hospitalization. During the day time, scorpions take shelter under back of trees, paddy husk, beddings and cresvices of windows and doors. Other important risk factors include conditions like, dryness, drought, deserts and heat [4].

The effects of the stings depend on the delivery dose of the scorpion, age of the offender, season and the size of the victim [5]. Scorpion stings locally cause intense pain, usually without local tissue injury. Systemic effects occur in a few patients, depending on scorpion species involved and are caused by a variety of excitatory neurotoxins [6]. Male adults are most frequently stung by scorpions. However, envenomations are more severe in children in whom mortality is dramatically higher than adults [7]. The death can occur early due to cardiovascular collapse. A case of scorpion bite presenting as hemiparesis is reported here.

Case report

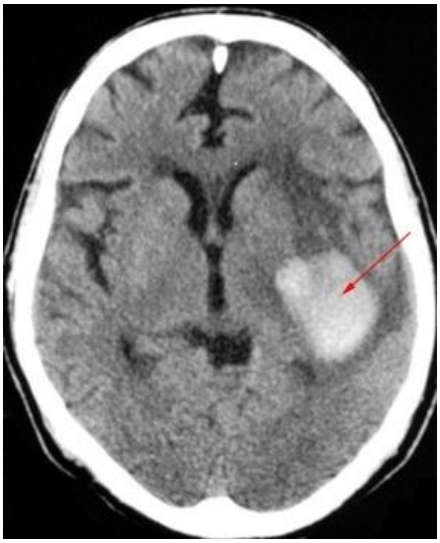
A 45 year old male patient, non-smoker, non-alcoholic, non diabetic, labourer by profession suffered a scorpion bite on the lateral aspect of the left foot in the month of June while putting on his shoes without looking at it. The killed scorpion was not available for identification. Patient initially complained of mild local pain and paresis. On examination, he was hemodynamically stable. PR was 100 per minute; BP 138/92 mmHg. CVS, Respiratory system and

abdominal examination were normal. He was treated initially with local analgesics and was kept under observation. After few hours he started having seizures. He also complained of weakness of left side of body which was of sudden onset and he was not able to move his left arm or leg, power was 0/5 in left upper and lower limbs, DTR exaggerated and planter response up, while 5/5 right upper and lower limbs,DTR normal & planter down.

Laboratory investigations revealed Hb of 8 gm%, TLC being 9600/mm^3, DLC P70L30, Platelets were 1.8 lakhs/mm^3. BT 10 minutes, CT 18 minutes, PT 30 sec(control 16 secs), BUN 32 mg%, Serum creatinine-1.0 mg%, Blood sugar-80 mg% Serum bilirubin-1.0 mg%, SGOT 34 units and SGPT 30 units. Urine examination revealed microscopic hematuria. D dimer was positive. ECG, Chest X ray and 2D echocardiography were normal. CT of brain revealed 3.2×3.4 cm size hematoma in right basal ganglia (Fig. 1).

Based on the clinical findings and investigations, patient was diagnosed to be a case of hemiparesis due to hemorrhagic stroke & was managed conservatively. Power improved over a period of a week to 4/5 from 0/5 in both right upper and lower limbs. He was discharged in satisfactory condition without and now on regular follow up.

Fig-1 showing CT brain showing intracerebral haemorrhage right basal ganglia



Discussion

In Asia epidemiological data on scorpion stings is scarce and the reported incidence is 0.6% [8]. Severity of poisoning depends on age, size of scorpion, season and time lapsed between sting and hospitalisation. Severe poisoning after a scorpion sting is characterized by serious complications, mainly cardiac. But neurological and respiratory complications also occur. CNS complications are very rare but lethal. It may present in either of two forms, like encephalopathy and stroke both of which are associated with high mortality rates. They are due to neurotoxic effects. And second presentation being stroke. Stroke [9] occurs as a result of rise in blood pressure due to autonomic storm which may rupture unprotected or diseased vessel resulting in hemorrhagic stroke, toxic myocarditis may precipitate arrhythmias and embolic stroke and associated blood coagulation changes including disseminated in travascular coagulation, vasculotoxic effect of venom causing damage to endothelial cells and vasculitis, catecholamine excess leads to severe vasoconstriction of the cerebral vessel.

Mesobuthus Tamulus may result in focal neurological manifestations like hemiparesis, hemiplegia or thrombotic stroke and the principal contributing factor for these manifestations is DIC [10]. Prevention lies in the frequent use of mosquito net & pestisides in an endemic areas of venomous sting. Vigorous shaking of clothing, bedding and shoes is recommended before putting them into use.

Conclusion

The case described above was having hemiparesis due to DIC in view of prolonged BT, CT, PT, aPTT and decreased fibrinogen level. This is probably the first case reported from this area of the country to the best of our knowledge. It is a very rare manifestation but if not managed properly becomes lethal. Early hospitalisation is recommended. The aim of reporting this case is to create awareness about this manifestation.

Conflict of interest

None declared.

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Case Report

PRECIPITATE LABOR & CONCEALED BIRTH

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Abstract

We report a case of feticide and concealed birth, which was revealed on investigation and visit to the crime scene. After examination of blood stains for blood grouping and examination of alleged mother for signs of recent delivery, it was alleged to be a case of precipitate labor. The observations signify that the actions taken at the outset of an investigation at a crime scene can play a pivotal role in the resolution of a case. We discuss the medico-social perspectives of such cases.

Keywords: Pregnancy, Precipitate, Pseudo-precipitate, Labour, Delivery, Fetus

Introduction

Precipitate or precipitous labour is rapid labour and delivery; i.e. expulsion of the foetus within less than 3 hours of commencement of regular contractions, with a reported incidence of 0.1-3% in the United States and other countries, out of which only about one-tenth are nulliparous.[1–3]

Isolated rare cases of exposure and abandonment are seen in India; as per NCRB data this crime constitutes 1.06% of total crimes against children in India.[4]Section 317 of Indian Penal Code prescribes punishment for exposure and abandonment of child under twelve years, by parent or person having care of it. Section 318of Indian Penal Code prescribes punishment for concealment of birth by secret disposal of dead body.

There are two kinds of unknown pregnancies. A denied, unknown pregnancy, graviditatasignota, is when the pregnant woman unconsciously denies the existence of the pregnancy or there is lack of awareness of being pregnant. A hidden pregnancy is a concealed pregnancy in women who know they are pregnant but actively hide their pregnancy from the family, partner, friends, teachers, colleagues, etc. Such cases are further classified as denied pregnancy or concealed pregnancy and a smaller group who attempted to forget their pregnancies. In this classification scheme, psychotic denial of pregnancy was considered a subset of denied pregnancies [5–8]. Pseudocyesis, delusion of pregnancy, false/imaginary/phantom/pseudo/spurious pregnancy is an opposite entity.

Pseudo precipitate delivery may occur in a primipara. The woman may have had labour pain but was either unaware of their significance or was not able to distinguish the sense of fullness produced by the descent of a child from the feeling of bulky evacuation. Stressors (e.g. separation from partner, interpersonal problems etc.) do play an important role as precipitating

factors for the development of an adjustment disorder with maladaptive denial of pregnancy.[9]

Case report

On the morning of 6th August 2012 the dead body of a new born male infant with umbilical cord and placenta attached was found in pool of blood in toilet in emergency department of our hospital. Blood spatter was present on the walls of the toilet. On the police request we conducted the crime scene examination along with a forensic scientist; took blood sample of child and sample of blood present on wall and around child and the blood group of all the samples found out O+ve indicating probable delivery of the child at the same place.

On police request post-mortem examination of child was performed. Rigor mortis was present over the proximal body and post mortem staining faintly visible on back. Crown-heel length was 50 cm and Crown rump length was 29 cm. Fully developed external male organs were present and both the testis descended to scrotum. Weight of infant was 2.4 kg while Umbilical cord of 35 cm length and 1cm thick not tied or clamped with placenta 15cm x 14 cm, 1.3 cm thick at centre and weighs about 500 gm. Vernix caseosa was present over joints and neck folds and Blood and was present over whole body. Scalp hairs are black and 2- 3 cm long. Lanugo is present only on the shoulders. Nails are grown over the tips of the fingers and toes. Chest was arched in shape suggesting live birth. Blood stained fluid with faecal matter was present in trachea. Position of diaphragm was at 6th rib, lungs were distended and voluminous, and pleura taut. On cut section of lungs, frothy blood oozed out and floatation test was positive. Liver, spleen and kidneys were pale. Meconium is seen at the end of large intestine.

Later, we received a query from gynaecology department that a 19 year old unmarried female having vaginal bleeding and clothes soaked in blood was admitted in surgery department at night with complaint of urinary retention and there after referred to gynaecology. Then on police request we took her blood sample also which is found out O+ve and on genital examination vaginal tear was noted and signs of recent delivery were present. On ultrasonography signs of recent pregnancy were present but the patient said that that she was neither pregnant nor had given birth to a child. Further police investigations confirmed the maternity. Paternity was attributed to an employer's son.

Figure 1 showing location of incidence with depiction of foetus (A) and blood stains (B, C)

Discussion

Many texts describe 'pseudo-precipitate labour' as a condition where a primigravida due to ignorance of the nature and character of labour pains and mistaking it to be an urge for natures' call, may deliver the newborn during defecation.[10–14]

Studies from UK, USA, Germany and Austria suggest concealment or denial might occur in about 0.04% to 0.26% of all pregnancies with almost one-third of the mothers being under 18 years of age with few having a psychiatric diagnosis.[5, 9, 15–20]

Psychotic denial of pregnancy in chronic mentally ill women may place the women and their foetuses at high risk of postpartum emotional disturbance, precipitous or unassisted delivery, foetal abuse, and neonaticide.[21]The absence of many physical symptoms of pregnancy, inexperience, general inattentiveness to bodily cues, intense psychological conflicts about the pregnancy, and external stresses

can contribute to the denial in otherwise well-adjusted women.[22] This condition poses challenging legal and ethical issues including assessment of maternal capacity, evaluation of maternal (and possibly foetal) best interests and the possibility of detention in hospital.[15]

Teen pregnancy being an unresolved social problem the reason of concealment of birth and secret disposal of body. Various socioeconomic factors like structure, types and characteristics of the family, early leaving school, incomplete education, schooling after delivery, female employment, lack of sexual education, parental and family attitudes in different periods of adolescent pregnancy, adolescent decisions on pregnancy and children and unstable partner relationship increases the early initiation of coital relations and unwanted pregnancies [23]. To terminate unwanted pregnancies commonly used methods include surgical abortion, taking misoprostol, drinking herbal and chemical preparations, and inflicting physical trauma.[24]

While the prevalence of denial of pregnancy is unclear, equally unclear are the laws that apply to a pregnant woman's personal rights. No clear legal precedents have been found to guide decision-making in the specific case of a woman with psychotic denial of pregnancy. The principles of substituted judgment and best interest may help guide clinicians in making decisions about the treatment of pregnant patients in the absence of clear legal precedent. A multidisciplinary team, comprised of social workers, nurses, obstetricians, and psychiatrists, must be involved in the evaluation and treatment of the patient.[8]

Conclusion

Precipitate and pseudo-precipitate labour is recognised entities. Denial of pregnancy is a heterogeneous condition with different meanings and different psychiatric diagnoses in different women. Teenage pregnancies have a significantly higher risk of adverse outcomes. Several studies highlight a well-established link between neonaticide and concealed pregnancy. Many authors have reported cases showing considerable unsureness or rejection of pregnancies and a significant number with little or no antenatal care. Psychosocial factors like unwanted and unplanned pregnancy, neuroticism, low education and previous history of depression have been recognised in pregnancy related depressive disorders. Psychiatric consultation must be advised for women who have denied or concealed their pregnancies. Although there is minimal evidence available, practitioners should remain alert to the pattern of concealed pregnancies.

Conflict of interest

None declared.

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Case Report

MICROSCOPIC CARDIAC CHANGES IN AN ELECTROCUTION DEATH

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Abstract

Electrical shock may result in death with variable degrees of damage to different organs. The final picture is determined by the type, voltage and intensity of the current. Except for cutaneous electrical and heat-related effects, electricity essentially leaves no trace. Though ventricular fibrillation is considered the main cause of death, no morphologic changes in heart has ever specifically been linked to electrocution. In few electrocution cases due to well moistened skin i.e. bathtub deaths, diagnosis becomes difficult as no burn marks are seen over the body. Changes in internal organs of the body can be of immense help in this regard.

We present a case of electrocution where deceased had external entry and exit wounds and changes in heart wall on histopathological examination like hypercontracted cells with square shaped nuclei and fragmentation of muscle fibres or myocardial fibre breakup (MFB) which probably is a feature of electrocution in this case.

Keywords: Electrocution, Myocardial Fibre Breakup, Joule burn, square shaped nuclei

Introduction

Electric deaths are mainly accidental and rarely suicidal or homicidal. In a study conducted in Manipal, India,the authors found that only 21 cases out of 3340 autopsies conducted over a time period from 1992-2009 were of electrocution.[1] All the cases were accidental in nature. The point of contact on the body surface may leave skin lesions called 'electrical burns', also known as Joule burn.[2] Diagnosis can be made by microscopic examination of joule burn[2], color tests[3], electrography[4], flourescent techniques[5] and observation by scanning and transmission electron microscopy.[6] Most deaths in electrocution are from cardiac arrhythmias, usually ventricular fibrillation ending in arrest. This is caused by the passage of current through the myocardium, especially in the superficial epicardial layers and possibly across the endocardium. The current has a profound effect directly upon the myocardial syncytium, the possible dislocation of the

pace making nodes and conducting systems. So the diagnosis made by the forensic pathologist is based on external skin changes, histological examination, staining techniques etc. The histological cardiac changes in electric deaths include myocardial fibre breakup [MFB].[7] But only few studies make of mention of this in literature. In the present paper, we report a case of accidental electrocution associated with histological cardiac changes and head injury.

Case Report

History:

A 48 yr old male accidentally touched the electric wire with an iron rod, which he was handling. As a result he fell down, sustained injuries and died.

Autopsy

External examination:

Multiple entry wounds with depressed centre and elevated reddish margins associated with blisters were present over the palmar surface of both hands varying in sizes from 0.7x0.5 cm to 3x1.6 cm [Fig. 1].

Multiple wounds with pale centre and reddish margins were present over the dorsum of the toes of both feet, varying in sizes from 1.5x1 cm to 3.5x2.5 cm.

Boggy swelling over an area of 8x6 cm was present over the face and head on left side.

Internal examination:

Head revealed scalp contusions over the occipital and left frontoparietal region. A fissured fracture measuring 9 cm was present over the right posterior cranial fossa. Brain showed diffuse subarachnoid haemorrhage all over the surface and contusion measuring 12x5 cm over the base of left temporal lobe. Multiple petechial haemorrhages were present over the epicardium of heart. Coronary arteries were patent.

Histopathological examination (Haemotoxylin and Eosin stain):

Wounds from the hands revealed steaming of the nuclei, vacoules in the skin layers and disaaray of the skin morphology. On staining with haemotoxylin and eosin stain, heart revealed break up of myocardial fibes, hypercontracted myocardial cells with sqaure shaped nuclei [Fig. 2].

Opinion as to the cause of death was given as complications of brain contusion, subarachnoid haemorrhage and myocardial damage caused due to electrocution.

Discussion

In this case of accidental electrocution both head injury and myocardial damage contributed to death. Electric entry wounds were present over the hands and showed typical morphological as well as histopathological findings. The wounds over both the feet appeared as entry wounds but did not show any changes of entry wound of electrocution on the histopathological examination, hence were considered as atypical exit wounds.

The electric mark sometimes reproduces the shape of the conductor, especially when it is a linear wire or a shaped metal object2. Entry wounds over hands in this case had depressed central region and elevated margins, which depicts shape of the iron rod held by the individual.

The entry mark of electrocution over skin consists of vacuolation in the epidermis and sometimes dermis, caused by the gas spaces from the heated tissue fluids splitting the cells apart. The affected tissues become more eosinophilic. The cap of epidermis may be detached and raised into a blister, with a large space beneath. The cells of the epidermis are often elongated, with the nuclei of the lower layers orientated and horizontally stretched. [2] Entry wounds in our case revealed vacoulation in epidermis and dermis, detachement of epidermis and streaming of nuclei in basal cell layer.

Fineschi Y et al processed 16 samples of myocardium in cases of fatal electrocution and head trauma; sections from each block were stained with haematoxylin and eosin. They recorded frequency, location, extent of myocellular segmentation of intercalated discs and associated changes of myocardial bundles [myofibre break-up (MFB)] [7]. The frequency of MFB in cases of electrocution and head trauma was 90% and 64% respectively. Myocardial nuclei in the hypercontracted cells had a "square" aspect rather than the ovoid morphology seen in distended myocytes. In our case too MFB was noticed along with the hypercontacted myocardial cells with square shaped nuclei. In the present case, trauma (head injury) occurred after electrocution. In such situation it becomes difficult for forensic pathologist to decide whether MFB was a result of electrocution or trauma. Review of literature does not mention of any study done, which differentiates microsopic cardiac changes due to electrocution and trauma. So there is a need of research in this direction which can be of immense help in diagnosing elctrocution deaths.

Tanaka N et al observed typical current marks with diffuse fragmentation of the myocardium with myocardial nuclei in hypercontracted cells showing a square appearance. [8] Similar results were observed in our case too.

Karger B et al observed petechial haemorrhages were present in 74% of electrocution cases and the favourite sites were the skin of the eyelids, conjunctivae, visceral pleura, and epicardium.[9] In our case multiple petechial haemorrhages were observed only over the epicardium.

Among the forensic fraternity, the cause of occurrence of the MFB is debatable. But MFB seen due to other causes like cutting by knife can be easily distinguished from MFB7. Vanderwee MA et al concluded that ischaemic myocytes contract just before actin and myosin become strongly linked to maintain the state of rigor mortis.[10] But Baroldi G et al found MFB to be absent in all cases with postmortem interval 1 to 24 hr. [11]

Guneithi B K et al in their study observed that 13 (20.96%) cases had no signs of electrocution. [12] If the external marks are absent (like in bath tub deaths), diagnosis of electrocution becomes a challenging task. In such cases internal findings can be of paramount importance in making diagnosis of electrocution. MFB, hyper contracted cardiac myocytes with square shaped nuclei as found in our case can be useful in such deaths.

Conclusion

Myocardial fibre breakup is an important internal finding in electrocution cases. But further researches are required in relation to its frequency, specificity and mechanism of occurrence in electrocution and various other types of cases like trauma.

Figure 1 showing entry wound of electrocution.

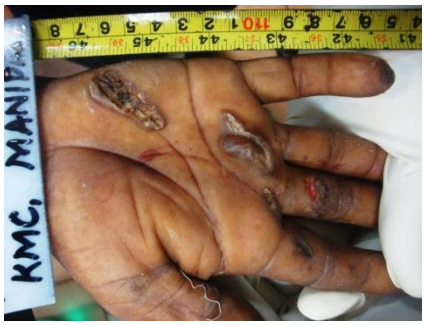
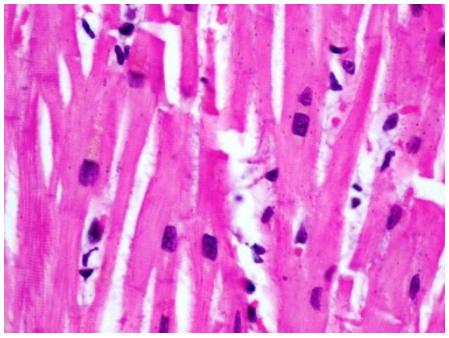


Figure 2 showing square myocardial nucleus, an expression of hyper contraction. (Haematoxylin and Eosin x 100)



Conflict of interest

None.

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Case Report

DECAPITATION IN SUICIDAL HANGING: A CASE REPORT

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Abstract

This paper reports the case of decapitation during suicidal hanging of an 82 yr old male weighing 48 kg. He reportedly jumped into a well almost 25 feet deep with a thick jute rope tied around his neck. External findings included complete transection of the neck at the level of C2-C3 vertebral body. Decapitation is an infrequent complication of hanging. The cause for such a rare event can be attributed to the inelasticity of the rope and fall of several meters.

Keywords: Hanging; Suicide; Decapitation

Introduction

Hanging is one of the most commonly resorted to methods of suicide worldwide and in India [1-3]. The cause of death in hanging is cerebral anoxia due to pressure on neck vessels along with obstruction of air passages [4]. Decapitation is an infrequent and unusual event in case of hanging. Thus, it becomes important to perform a thorough autopsy in order to differentiate it from homicidal decapitation. Here, we present a case of decapitation due to suicidal hanging of an 82 yr old man.

Case Report

The dead body of an 82 yr old man, measuring 163 cms in length and weighing 48 kg (including head) was brought to our centre for autopsy. According to acquaintances, on verbal autopsy, it was found out that the deceased committed suicide by jumping into a well, almost 25 feet deep. He tied one end of a thick jute rope around his neck, the other end being attached to the pulley of the well and then jumped into the well. The patient also had a history of joint pain since the last couple of months for which he visited the doctor often. However no medical records could be retrieved regarding the same.

The body was cold to touch and stiff at all joints. Post- mortem lividity could not be appreciated. The external injuries included complete transection of the neck at the level of C2-C3 vertebral body (Fig 1). They were cut irregularly with tissue tags. A pressure abrasion, reddish brown in colour, measuring 21X0.5 cm was present on the front and either side of the neck. On the front of the neck, it was present 8 cm above the sternal notch. The upper margin of abrasion was merged with the irregular edges of the transacted wound of the neck (Fig. 2). The pattern of the abrasion was similar to that of the rope used as ligature material.

Fig 1 showing Decapitation at cervical vertebrae (C2-C3)level

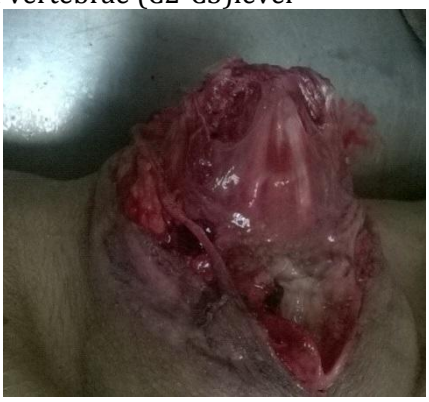
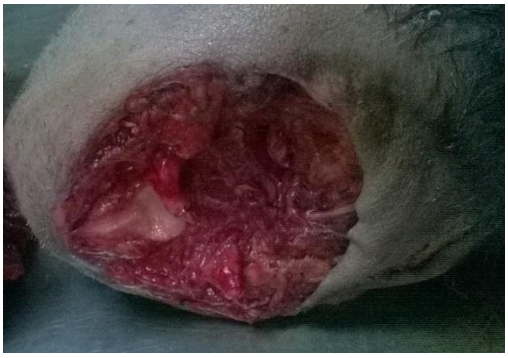


Fig 2 showing margins of decapitation wound on neck



Multiple contusions, purplish in colour were present at the tip of left shoulder and the outer aspect of left forearm in its upper one-third. No other external injuries were present.

Internal examination

Complete transection of neck at the level of C2-C3 vertebral bodies. The scalp, skull and meninges were intact and healthy. The brain was edematous. The intervertebral ligaments were stretched. The trachea and esophagus were transected at the level of C2-C3 vertebral body, corresponding to the level of transection of the neck. The coronaries and the aorta showed mild atheromatous changes. No other internal injuries were noted. Lab report was negative for poisons. The cause of death was opined to be decapitation due to hanging.

Discussion

Usually, in cases of hanging, the cause of death is ischemia and anoxia of brain due to compression of neck vessels [5]. In the present case, the victim jumped into a 25 feet deep well with a thick inelastic jute rope tied around his neck. The patterned abrasion present around the neck of the deceased matched that of the rope used for hanging. Due to the inelasticity of the rope and fall of a great height, there was axial traction and sudden tightening of the noose followed by antero-flexion of the neck. This led to complete transection of the neck at the level of C2-C3 vertebral body which is not usually seen in cases of suicidal hanging. The skin and cervical spine were also severed which is similar to the findings reported earlier [6]. It is documented that for decapitation to occur in a case of hanging, body weight, distance of fall, material of ligature, neck strength are crucial determinants.(6,7) If material is inelastic and hard, and if kinetic energy of fall is more, then the chances of decapitation are higher. In few other reported cases decapitation is documented in hanging, suggesting that complete decapitation can occur in suicidal hanging also.(7,8,9) In the present case, decapitation was a result of an amalgamation of the above stated factors. Also, history of joint pain given by the relatives raises the suspicion of an arthropathy which could have been a contributory factor.

Conclusion

Thus, it can be concluded that decapitation during suicidal hanging is a rare phenomenon and thus needs thorough investigation. Factors such as age and weight of the deceased, presence of natural disease, ligature material used and height of the fall should be borne in mind while investigating such a case. The injuries in such cases may result from traction due to sudden tightening of the ligature and pull of the body.

Conflict of Interest

None Declared

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Case Report

CHOKING WITH FOOD BOLUS LEADING TO VIOLENT ASPHYXIAL DEATH IN A HEALTHY YOUNG MALE- A CASE STUDY

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Abstract

People experience choking by accidental slipping of food bolus in their respiratory tract in their daily lives. Most of the people recover from it but a few experience obstruction of the respiratory tract that cannot be cleared and may prove fatal. Accidental choking is the commonest manner of choking. Dead body of a 26 years old male subject was brought for post-mortem examination. On autopsy examination, along with the signs of asphyxial death both externally and internally, congestion and erosion was seen in the trachea. Some traces of puffed rice was also visible along the respiratory tract by naked eyes. History furnished by the police was also corroborative. While having puffed rice with milk in his house, he suddenly became restless and turned unconscious; when taken to hospital, he was declared brought dead. This case reports about a fatal choking due to food bolus.

Keywords: Choking, Accidental, Autopsy, Asphyxial death, Fatal.

Introduction

Choking refers to blockage of the internal airways, usually between the pharynx and the bifurcation of the trachea. Death from choking can be the result of pure hypoxia from occlusion of the airway, when all the attendant signs of congestion, cyanosis and perhaps petechiae may be present, usually where the victim struggles to breathe for an appreciable period. However a large proportion of deaths occur suddenly before any possible hypoxic manifestations have time to take effect; these fatalities must be caused by neurogenic cardiac arrest, either purely neurogenic or accelerated by excess catecholamine release from the adrenaline response [1].

Case history: A 26 years old healthy male was having puffed rice with milk in his house. During this meal he started quarrelling with his wife over a petty issue. Suddenly his wife found the uneasiness within her husband. He turned restless holding his throat craving severely for air and became semiconscious. Inmates of the house rushed to the hospital and the victim was declared brought dead there.

Autopsy findings: The body was of a twenty six (26) years old male person who was of normal built, well nourishment and brown complexion. Rigor mortis was present all over the body and post-mortem staining was also appreciable on the dependent parts of the body.

External: Subconjunctival petechial haemorrhage. Face was found congested. Cyanosis of fingertips and nail bed. No external injury.

Internal: Stomach content-whitish fluid along with partially digested identifiable puffed rice (Figure 1). Some traces of puffed rice was also identifiable along the respiratory tract (Figure 2). Trachea was found to be congested and eroded

(Figure 3). All the viscera were found congested. No other significant abnormalities were found.

Cause of death:

Asphyxia as a result of accidental choking.

Discussion

Choking is almost exclusively accidental in nature. Homicidal choking is argumentatively possible with infant victims. In mental unsound persons suicidal choking can only be thought of [2]. In medical practice there are risks associated with individuals who are sedated or anaesthetized, when objects such as extracted teeth or blood from dental or ENT operations may occlude the airway without provoking the normal reflex of coughing [3]. Natural deaths are seen in individuals with acute fulminating epiglottitis, where there is obstruction of the airway by the inflamed epiglottis and adjacent soft tissue. Such individuals represent medical emergencies and can die literally in front of a physician. The individual develops a sore throat, hoarseness, respiratory difficulty, inability to speak and then suddenly collapses as the airway is completely obstructed. Inhalation of steam can cause a similar picture, with a markedly edematous, beefy-red mucosa in the larynx with obstruction [4] Different causes of death in choking are Asphyxia, Laryngeal spasm, Vagal inhibition and reflex neurogenic cardiovascular failure [5]. It is not necessary that a foreign body should be of such a size as to block the air passages completely. Even a small object blocking the lumen partially may cause death by laryngeal spasm [6]. Death from sudden reflex neurogenic cardiovascular failure may be produced by reflex parasympathetic cardiac inhibition. Sudden impaction of foreign body at the bifurcation of trachea can cause death both by asphyxia and reflex parasympathetic cardiac inhibition. [7]

In adults, choking virtually always involve food. Here it is commonly associated with acute alcoholic intoxication, bad fitting dentures, neurological injury or senility. Complications may develop after a latent interval, if the person survives. [8]

In this particular case the young man was quarrelling with his wife while having milk with puffed rice. Some part of the food went to the stomach, as was recovered from the stomach during autopsy; the same food materials were found in the trachea, within the main trunk, as well as beyond the bifurcation. Clearly, the cause of death here was asphyxia from accidental choking.

Conclusion

Usually, any object in the air passages excite violent coughing out, but if this is not successful in expelling the object out, choking results. [9] This case report is an attempt to discuss the problem of accidental choking by food bolus. As the body reached us on the day of death, there was no question of putrefaction as a reason for presence of gastric contents within trachea and moreover the food materials were identifiable and freshly swallowed. Though the person died within 24 hours of the onset of the terminal symptoms, it cannot be called as sudden death because sudden deaths are mostly natural deaths which occur immediately. [10]

Most choking deaths are accidental in manner. In 1997, as cited by DiMaio VJ and DiMaio D from a case report, there were approximately 3300 deaths ascribed to unintentional inhalation of food or other objects, resulting in obstruction of the respiratory passages. [4, 11]

Contribution of authors: We declare that this work was done by authors named in this article and all liabilities pertaining to claims relating to the content of this article will be borne by the authors.

Conflict of interest

No conflict of interest associated with this work.

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Figure 1: Presence of food material in the stomach (same as in trachea).

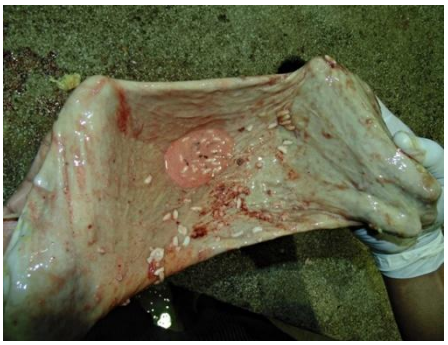


Figure 2: Food material in trachea.

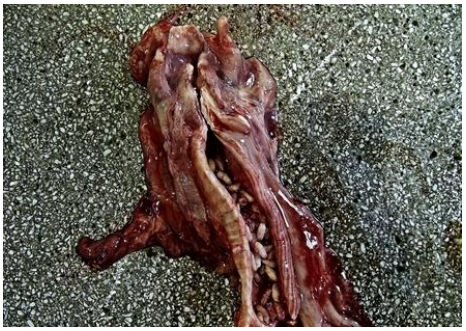
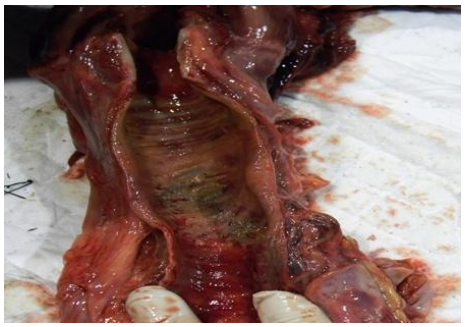


Figure 3: Congested tracheal lumen.



Case Report

SEVERE METABOLIC ACIDOSIS WITH HIGH ANION GAP: A DIAGNOSTIC CLUE FOR ETHYLENE GLYCOL POISONING

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Abstract

Ethylene glycol poisoning is very rare emergency, high clinical suspicion is required in the absence of any proper history. Rapid deterioration with severe metabolic acidosis, high anion gap, neurological complications and acute kidney injury are some of the key features to early diagnosis and treatment .

Keywords: Ethylene glycol, High anion gap, Acute kidney injury.

Introduction

Ethylene glycol is a solvent found in products ranging from antifreeze fluid and de-icing solutions to carpet and fabric cleaners. According to results from animal studies, [1] the ingested amount of ethylene glycol required to produce toxicity in animals is approximately 1.0 to 1.5 mL per kg, or 100 mL in an adult. Ethylene glycol poisoning may pose diagnostic difficulties if the history of ingestion is not available, or if the presentation is delayed. Because of its clear appearance and sweet taste its accidental poisoning is common among children [2].

The initial diagnosis of ethylene glycol is difficult and delay could be fatal so it demands rapid recognition and prompt treatment. Ethylene glycol is rapidly absorbed through the gastrointestinal tract [3]. Similar to ethanol and methanol, Ethylene glycol is metabolized by the alcohol dehydrogenase to glycolaldehyde and the action by this enzyme is the rate limiting metabolic step for Ethylene glycol [4]. Aldehyde dehydrogenase converts Ethylene glycol to glycolic acid and subsequently to oxalic acid. Severe metabolic acidosis with high anion gap is mainly caused by glycolic acid. Oxalic acid precipitates in the form of calcium salt in the renal tubules leading to acute renal failure [5].

Here we report a case of ethylene glycol poisoning, in the absence of any proper history, rapid deterioration with severe metabolic acidosis, high anion gap, neurological complications and acute kidney injury, treatment done on high clinical suspicion of poisoning and patient responded well to treatment measures.

Case Report

A 28 yrs old female brought in accident and emergency department in drowsy state, she was found in state of breathlessness and semi-consciousness in her room, without any significant history. On admission she was drowsy, responding to deep pain, pulse rate 120/min, blood pressure of 80/60 mm Hg, her respiration was deep and rapid at rate of 30/ min, pupil were mid dilated sluggish reacting to light. Immediately patient was given inotropic support. During hospitalization patient deteriorated and developed generalized tonic clonic seizure and she had to put on mechanical ventilator

support. Her biochemical investigation and blood gas showed low serum calcium, high anion gap metabolic acidosis, with high osmolar Gap (Table 1 & 2)

Table 1 showing Baseline Biochemical parameters:

	AT ADMISSION	1 DAY LATER	AT DISCHARGE
BLOOD SUGAR (mg/dL)	110	90	96
B. UREA (mg/dL)	21	90	43
S. CREATININE (mg/dL)	0.9	2.1	1.1
S. URIC ACID (mg/dL)	4.6	7.7	5.2
S. CALCIUM(mg/dL)	6.2	6.6	8.4
S.PROTEINS	7.8 mg/dL		
S,Albumin	4.1mg/dL		
S.Globulin	3.7mg/dL		

Table 2 showing Arterial Blood Gas Analysis :

AT ADMISSION	1 DAY LATER	AT DISCHARGE
Ph	7.30	7.40
Hco3	6.2	22
Sodium(meq/dL)	141	139
Potassium(meq/dL)	3.6	4.1
Chloride	106	117
Anion Gap	29	15

These finding were highly suggestive of ethylene glycol poisoning, which is commonly used as solvent. She was given sodium bicarbonate intravenously to correct metabolic acidosis. She developed acute renal failure 1 day later, this time urine microscopy showed crystals. Hemodialysis was done. She responded well to these measures, her acidosis improved, general condition improved. On further questioning she accepted to ingest some clear liquid substance kept in home as pesticide solvent most likely to be ethylene glycol.

Discussion

Ethylene glycol is a toxic alcohol that may be ingested accidentally, intentionally or consumed as ethanol substitutes. Like ethanol, this causes intoxication and is metabolized by alcohol dehydrogenase (ADH), a process that creates toxic metabolites. It is relatively innocuous until metabolized to toxic breakdown products, which explains its 4- to 12-hour latent period [6]. Ethylene glycol toxicity is divided into 3 distinct phases but can also present together, CNS depressant, patient exhibit signs of intoxication, stupor, nausea and vomiting, hallucinations and seizures. At 12 to 24 hours after ingestion the cardiorespiratory phase is heralded by the onset of hypotension, tachypnia, congestive heart failure or, occasionally, myositis. Lastly, the renal stage which appear 24 to 72 hours after ingestion is marked by flank pain and oxalate crystalluria, followed by the development of oliguric renal failure that may necessitate long-term dialysis [7].

Measurement of ethylene glycol or its by products can be helpful but they are not easily available and processing may take several days and, little correlation exists between blood levels of ethylene glycol and severity of poisoning, [1] they are not required for initiation of treatment. Indicators for a quick diagnosis of ethylene glycol poisoning include hyperventilation with neurological complication, if any, with laboratory data suggestive of an elevated anion gap metabolic acidosis and osmolar gap; the presence of hypocalcaemia; and urinary crystals [1].

The osmolar gap is calculated by subtracting the calculated serum osmolality from measured osmolality. If the serum osmolar gap is greater than 10 mOsm per kg of water, the presence of ethylene glycol poisoning is likely.

Traditional treatment of ethylene glycol poisoning consists of sodium bicarbonate, ethanol, and hemodialysis.

Fomepizole is a new agent and act as inhibitor of alcohol dehydrogenase and therefore prevent the formation of acidic ethylene glycol metabolites1

Ethanol may be administered orally or intravenously. The recommended therapeutic blood ethanol level is 100 to 150 mg per dL (22 to 33 mmol per L) [1]. Intravenous administration of ethanol should be continued until ethylene glycol levels have been reduced below 20 mg per dL and the metabolic acidosis has been corrected.

Administration of intravenous sodium bicarbonate will correct the metabolic acidosis, increase the elimination of renal glycolic acid, and inhibit the precipitation of calcium oxalate crystals, although the latter benefit has not been proved in clinical trials. Fifty to 100 mEq per L of intravenous fluid is usually sufficient, with a goal of maintaining a urine pH greater than 7.0.

As per AACT guidelines in ethylene glycol poisoning Hemodialysis is recommended if there is high anion gap metabolic acidosis metabolic acidosis regardless of drug level if there is evidence of end organ damage.

Pyridoxine (vitamin B6) and thiamine (vitamin B1) in dosages of 100 mg daily are believed to promote the conversion of intermediate by products into nontoxic metabolites but clinical data supporting their effectiveness do not exist. Therapy with 100 mg of intravenous thiamine would be appropriate if ethanol withdrawal is suspected.1 Parenteral calcium, given as gluconate or chloride salts, may be necessary for treatment of tetany and seizures caused by hypocalcemia.

Conclusion

Poisoning with ethylene glycol is not very common in this part of India, so if history is not available treatment can be delayed. Absence of a strong odor of alcohol in a patient who otherwise appears intoxicated with laboratory data suggestive of an widened anion gap, metabolic acidosis, hypocalcemia, neurological changes and acute kidney injury poisoning with ethylene glycol should be suspected and definitive treatment should be started without delay.

Conflict of Interest

None declared.

Reference

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