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Contents

Review Of Management Of Common Poisoning In India.

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ABSTRACT

Suicidal or homicidal poisoning with drugs overdosage is one of the common causes of morbidity and mortality in India. Patients generally presents with varying degree of signs and symptoms in emergency. On arrival of the patient assessment of the danger should be done followed by common procedures like gut decontamination using activated charcoal, maintenance of fluid balance and airway control. Gastric levage is an important procedure especially if done with in 60 minutes of poisoning but due consideration must be given to contraindication. In India generally two types of poisoning is seen as agriculture based and with pharmacological agents i.e. drugs. India is an agriculture-based country with over usage of pesticides. In emergency two types of pesticide poisoning is common as with organophosphates and with aluminium phosphide. The mortality rates are much lower if early treatment is started in both the case. Over dose with opioids is another important cause of mortality especially in addicts. The other poisons encountered are with paracetamol, aspirin, chloroquine, alcohol, theophylline and iron. The specific management with antidotes generally reverses the symptoms of poisoning saving the patients life.

INCIDENCE

Acute poisoning is a common problem worldwide. In the United Kingdom (UK) it accounts for an estimated 10-20% of acute medical admissions and 5-10% of the workload of Accident and Emergency (A&E) departments. Episodes of self-poisoning continue to rise, with the rates being the highest in Europe [1]. The severity of poisoning has decreased over the past decade with the introduction of safer drugs, such as the selective serotonin reuptake inhibitors. However, in India the exact incidence cannot be defined as there is under reporting of cases of poisoning. Infact, there is no check on sale of poison and any body can purchase it over the counter. This paper will address common pitfalls in the management of poisoned patients where clinical management could be improved and medico legal problems can be avoided.

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MANAGEMENT

It is generally divided in two parts as

I) General Management

A)Supportive care

It is the most important aspect of the management of poisoned patients[2]. The majority of patients who present with self-poisoning develop minimal or no clinical effects, and therefore the aim is to identify as early as possible the patients who will proceed to develop significant clinical features and complications. As a general rule, complete elimination of a drug takes approximately the duration of five half-lives of the drug and so the patient needs to be supported during this phase with monitoring for and treatment of any secondary organ dysfunction or other features that develop [2]. The initial management of the patient should be on the basis of (ABCD's) of the poisoning treatment.

☛ Airway should be cleared of vomits or obstruction with endotracheal tube if required. Even positioning in lateral decubitus position is enough to save the flaccid tongue from obstructing airway.

☛ Breathing should be assed by observation, oximetry and arterial blood gas analysis. Patient with respiratory depression should be intubated and put on ventilator.

☛ Circulation should be assed by measuring pulse rate, blood pressure, urinary output and peripheral perfusion.

☛ After establishing intravenous line blood should be drawn for estimating S. electrolytes, S. glucose and other parameters and infused with drugs.

☛ Hypoglycemia should be avoided especially in patients with altered mental consciousness and challenge with glucose should be done to avoid irreversible brain injury due to hypoglycemia. In this case Adults are given 25 gm (50 ml of 50 % Dextrose) and children 0.5 gm / kg (2ml / kg of 25 % dextrose).

B)Decontamination of body parts.

This means removing toxins from skin or GIT.
1.Skin: Remove contaminated clothes and wash the skin with soap and water.

2.GIT: There is controversy regarding efficacy of gut emptying by emesis or gastric levage after one hour of poisoning. The various methods employed are

3.Emesis- Emetic agents like ipecac syrup are sometimes used to treat childhood ingestion at home. The contraindications are corrosive poison, petroleum products and rapidly acting convulsants.

4.Gastric levage- this is done in awake or intubated patients with large sized nasogastric tube with N saline preferably.

C) Activated Charcoal

It is also called universal antidote as its large surface adsorbs many drugs. The mainstay of gut decontamination is the administration of activated charcoal within one hour of a significant ingestion of a toxin that binds to charcoal[3]. It is given in the ratio of 10:1 to charcoal: dose of toxin by weigh. However, it don't bind iron, lithium, K+ and poorly to cyanide and alcohol. Multiple-dose activated charcoal (MDAC) increases the elimination of some drugs by interrupting their enteroenteric and entero-hepatic circulation. The dose given is 50 G (1 G/kg in children) of activated charcoal every four hours[4]. Indications for MDAC are carbamazepine, dapsone, phenobarbitone, quinine and theophylline poisoning. In addition to these indications, MDAC should be considered seriously in salicylate poisoning (to prevent delayed absorption) until the salicylate level peaks. Gastric levage is much less widely used now. Although there have been descriptive case reports of removal of tablet debris using gastric levage, no clinical studies have demonstrated that this has any impact on outcome. There is also the possibility that levage may increase absorption by pushing the tablets into the small intestine[5]. It can also result in hypoxia and tacycardia. Gastric levage should therefore only be considered if it can be carried out within one hour of a life-threatening ingestion and the patient should be monitored closely during the procedure.

D) Whole bowel irrigation (WBI)[6].

WBI with Polyethylene glycol is a newer method of gut decontamination. There are published reports describing the use of WBI in poisoning with a number of substances including iron, lithium, latex packets of cocaine in body-packers, lead and sustained-release preparations including verapamil. Whole bowel irrigation is generally well tolerated, and polyethylene glycol is not absorbed and does not result in significant changes in fluid or electrolyte balance. The dosage of polyethylene glycol used for WBI is 1,500-2,000 ml/hr for adults, 1,000 ml/hr for children of six to twelve years and 500 ml/hr for children six months to six years of age; it should be continued until the rectal effluent is clear. It can be given orally or via a nasogastric tube. Indications for WBI are:

Large ingestions of agents not adsorbed to activated charcoal. e.g. lithium. Iron, large ingestions of sustained release or enteric-coated drugs e.g. calcium channel blockers. Contraindications to WBI are Bowel obstruction or ileus, significant gastrointestinal hemorrhage, hemodynamic instability.

E) Sodium bicarbonate.

This is a commonly used drug in management of acidosis due to poisoning with methanol, ethylene glycol, cyanide and salicylates and for urinary alkalinisation to enhance elimination of salicylate (and less commonly for phenobarbitone, chlorpropamide and some pesticides) or prevent renal deposition of myoglobin after severe rhabdomyolysis, cardiotoxicity caused by tricyclic antidepressants, flecanide, quinidine, chloroquine, dextropropoxyphene, procainamide, disopyramide, phenothiazines.

II) Specific Management

In clinical practice basically two types of poisoning is commonly encountered as

A) Agriculture Products Poisoning.

B) Pharmaceutical Products Poisoning (drugs).

A) Agriculture Products Poisoning (Pesticides).

India is an agriculture-based country with over usage of pesticides. In emergency two types of pesticide poisoning is common as

1. Organophosphorus Compounds (OPC).

These include DDT, parathion, malathion, diazenon and highly toxic nerve[7] gas poison like tabun, serine and soman.

Clinical Features: These can be as

2.CNS: In low concentration there can be diffuse activation of EEG and subjective altering response. In high concentration there is tremors, generalized convulsion followed by coma and respiratory arrest.

3.Eye, GIT, Respiratory and Urinary system: There can be miosis, hypotension, bradycardia, bronchospasm, voiding of urine, marked salivation and diarrhea.

Diagnosis: RBC and serum cholinesterase decrease by [3] 50% due to binding by phosphate group of pesticides. RBC cholinesterase reflect a better index than serum cholinesterase as latter is decreased in other diseases like liver diseases (hepatitis, cirrhosis, ascites, obstructive jaundice and metastatic carcinoma), congestive heart failure and congenital disorders. An experimental method[11] using frog rectus preparation has been suggested by the author and colleague. This is relatively cheap, easy to perform and convenient technique but requires technical skill.

***Treatment*[8, 9]:** Apart from general support the treatment is atropine and pralidoxime. Atropine in dosage of 2 mg IV slowly repeated at every 5 to 15 minutes

until signs of reversal (dry mouth, mydriasis) are seen. There is no theoretical limit to its dosage as more than 1 gm/ day can be given for 1 month for full control of muscarinic excess. Pralidoxime a cholinesterase reactivator is also used. It is given as infusion as 1-2 gm over 15-30 minutes. Contraindications to use are Carbamates poisoning (propoxur/baygon/carbaryl and sevin). Treatment with pralidoxime should be started early (within 24 hours) to avoid phophorylation of cholinesterase enzyme.

2. Aluminium Phosphide poisoniong[11]

Aluminium Phosphide is widely used fumigant and preservative of wheat and grains due to high potency, cheap and easy storage. Due to these factors it has become a common suicidal poison.

Mechanism of action: When taken orally it reacts with acidic media (HCL) of stomach and releases Phosphine (PH_3) gas, which is rapidly absorbed from gastro-intestinal tract by simple diffusion in body system and also lungs by inhalation. After absorption it is metabolized in liver where more of PH_3 is released accenting for prolonged action of PH_3. The absorbed PH_3 is oxidized to oxyacids and is excreted in the form of hypophosphite in the urine and a significant amount is also excreted through lungs. PH_3 is a protoplasmic poison interfering with enzymes and protein synthesis esp. that of cardiovascular and respiratory systems. The ultimate result is peripheral vascular collapse, cardiac arrest and failure, and pulmonary edema. In animal studies PH_3 has been shown to cause non-competitive inhibition of cytochrome oxidase of myocardial mitochondria.

Signs and symptoms: Symptoms are generally related to the cardiovascular and pulmonary systems. These include restlessness, irritability, drowsiness, tremors, paresthesia, vertigo, diplopia, ataxia, cough, dyspnoea, retrosternal discomfort, abdominal pain and vomiting. Signs are many like various stage of cardiovascular collapse as hypotension, reduction in cardiac output, tachycardia, oliguria, anuria, cyanosis, pulmonary edema, tacypnoea, jaundice, hepatosplenomegaly, ileus, seizures and diminished reflexes. Electrocardiographic finding includes ST elevation or depression, T wave inversion, sinus tachycardia, atrial fibrillation and infarction and atrioventricular conduction problems esp. right bundle branch block, and complete atrioventricular dissociation. The myocardial damage is reversible as ECG returns to normal in 10 to 25 days if patient survives. X ray chest reveals pulmonary edema. Lab finding reveals abnormalities in myocardial and liver enzymes. Blood urea nitrogen and creatinine levels are usually raised. Significant hypomagnesaemia and hypermagnesemia have been reported in patients with massive myocardial necrosis. Blood gas abnormalities demonstrate combined respiratory and metabolic acidosis.

Treatment: initially remove clothing and wash the skin. The next management includes oxygen, intravenous fluids, vasopressors, plasma expanders, fresh blood transfusion, bronchodilators and digoxin or calcium channel blockers depending upon clinical situation of the patients. Diuretics should be considered for pulmonary edema provided hypotension is excluded. Calcium gluconate and 25 % magnesium sulfate due to their membrane stabilizing effects have been advocated. Gastric levage with potassium permagnate solution (1:10,000 dilution) is recommended for oral ingestion as permanganate oxidizes PH3 to form phosphate esp. if given with I hour of ingestion.

B) Pharmaceutical Products Poisoning (Drugs).

Acute poisoning of drugs is very common in India and world wise.

1. Opioids.

Naloxone has been used as a specific antidote for opioids poisoning since the 1960s. A frequent error in the management of opioids poisoned patients is to administer either excessive or insufficient doses of naloxone. The goal of naloxone treatment is reversal of respiratory depression to a minimum respiratory rate of ten (with adequate respiratory depth and oxygen saturation) and reversal of central nervous system (CNS) depression aiming for a Glasgow Coma Score of [4,5]. Naloxone can precipitate acute withdrawal syndrome (AWS) in chronic opioids users.[12, 13]. The agitation, hypertension and tachycardia produced, although rarely life-threatening, may produce significant distress to both the patient and doctor. . The agitation that results from AWS makes it difficult to monitor the patient condition and patients in this state may opt to leave the A&E department against medical advice. In addition, vomiting commonly occurs in acute withdrawal and, in-patient not regaining consciousness immediately after naloxone, can result in aspiration. The bolus dose required to reverse the depressant effects of an opioids is generally between 0.4 mg and 2.0 mg IV, although this should not be given as a single large bolus dose[8]. 2 mg of naloxone is made up in a 10 ml syringe with saline and given in 100-200 mcg boluses to a maximum of 10 mg, titrated according to the effect produced. The half-life of naloxone is between 30 and 100 minutes. Because the duration of action of most opioids exceeds that of naloxone either repeated doses or an intravenous infusion of naloxone are often required[14].

2. Paracetamol.

Paracetamol remains the most common substance taken in overdose in Europe, accounting for 50 % of all self-poisoning episodes and 100-200 deaths per year. The vast majority of cases of early, lone paracetamol poisoning are asymptomatic at presentation; therefore management is best guided by blood tests[15].

***a) Risk factors for paracetamol poisoning*[16, 17].**

A number of factors may increase the risk of hepatotoxicity in patients with paracetamol poisoning and identify them as high risk. Risk factors in paracetamol overdose are: Regular ethanol consumption in excess of currently recommended limits (21 units/week in males; 14 units/week in females), Regular use of enzyme-inducing drugs (e.g. phenytoin, carbamazepine, rifampicin, phenobarbitone), Conditions causing glutathione depletion (e.g. HIV, eating disorders, cystic fibrosis, malnutrition).

b) Early paracetamol poisoning (less than 15 hours post-ingestion)

The decision to use N acetylcysteine (NAC) in patients with early paracetamol poisoning is based on the plasma paracetamol concentration plotted on the Prescott normogram [30]. Blood should be taken for a plasma paracetamol concentration on presentation (or four hours post-ingestion, whichever is later). NAC is an effective, safe antidote and if given within eight to ten hours of paracetamol ingestion provides almost 100% protection against the development of hepatic and renal toxicity[30]. Therefore, in patients who present early, if the result of the plasma paracetamol concentration is available by eight hours post-ingestion, the decision to start NAC can be based directly on the plasma paracetamol concentration. However, in patients who present at more than eight hours after ingestion of a potentially hepatotoxic dose of paracetamol (> I 50 mg/ kg or 75 mg/kg in high-risk patients), NAC should be started on presentation after blood is taken for a plasma paracetamol concentration; the NAC can be stopped if the plasma paracetamol concentration is found on analysis to be well below the relevant treatment line on the Prescott nomogram.[18].

c) Late paracetamol poisoning (more than 15 hours post-ingestion)

This group is difficult to manage. In any patient presenting within eight to twenty-four hours of ingestion of a potentially hepatotoxic dose of paracetamol, NAC should be started immediately and blood taken for International Normalized Ratio (INR), liver function tests, serum creatinine and plasma (venous) bicarbonate. The decision to continue the infusion will be determined by these blood results, the history and the clinical condition of the patient. However, in late-presenting patients, the detection limit of the plasma paracetamol assay may not be sufficient to distinguish between toxic and non-toxic amounts[19]. Therefore, the plasma paracetamol concentration in late-presenting patients needs to be considered with caution and patients may need to be given NAC on the basis of ingestion of a potentially hepatotoxic dose of paracetamol (150 mg/kg or 75 mg/ kg for 'at risk' groups) [18.The] maxim to be followed in practice is, 'If in doubt, treat.

The plasma paracetamol concentration cannot be used to assess patients presenting at more than 24 hours post ingestion. In these patients, blood should be taken for INR, liver function tests, and serum creatinine and plasma venous bicarbonate. If the patient is asymptomatic and the blood results are normal he/she may be medically discharged [20] if not, the National Poisons Information Service (NPIS) at AIIMS, New Delhi should be contacted for management advice.

d) Management of established paracetamol related hepatotoxicity

Meticulous supportive care is important in these patients and if possible liver transplant unit should be contacted for evidence of hepatotoxicity at any stage or doubt about its management, particularly if markers of severe toxicity are present[21].

Markers of severe paracetamol poisoning and indications for referral to a liver unit are: Progressive coagulopathy or INR >2 at 24 hours, INR >4 at 48 hours, INR >6 at 72 hours, Renal impairment (creatinine >200 mmol/L), Hypoglycemia, Metabolic acidosis (pH <7.3, bicarbonate <18) despite adequate rehydration, Hypotension despite fluid resuscitation, Encephalopathy.

e) Staggered paracetamol poisoning

The plasma paracetamol concentration cannot be used to guide management in those patients who have taken a number of doses of paracetamol over a prolonged period of time. A baseline INR, liver function tests, serum creatinine and plasma venous bicarbonate should be taken, but the ingested dose is the most important factor and patients who have ingested more than 150 mg/kg over a 24-hour period (75 mg/kg for high-risk patients) should be treated with NAC.

3) Chloroquine.

The mortality rate in published studies of chloroquine overdose is between 12-35%, among the highest in clinical toxicology[24]. Ingestion of more than 5 g of chloroquine is probably the most accurate predictor of a fatal outcome; death is usually due to cardiotoxicity[25]. The interval between ingestion and onset of symptoms is usually between one to three hours, with death within 12 hours. Chloroquine blood concentrations are not required for the institution of treatment. As clinical features, involving the cardiovascular and CNS are more important. Activated charcoal should be given to patients presenting within one hour of ingestion of more than 15 mg/kg chloroquine. It is essential to intubated and ventilate early in the course of chloroquine poisoning if arrhythmias, hypotension, seizures or significant CNS depression are present. Anti-arrhythmic agents should be avoided if possible as they may precipitate further arrhythmias. Intravenous sodium bicarbonate is the

treatment of choice for arrhythmias [26] and should be used in patients with widened QRS and QTc intervals (1-2 ml/kg 8,4% sodium bicarbonate repeated as necessary aiming for a pH of 7.45-7.5). Overdrive pacing is the treatment of choice for ventricular tachycardia or torsade de pointes. Inotropes may also be necessary for hypotension unresponsive to a fluid challenge. Plasma potassium should be monitored, although hypokalemia may have a protective effect and should not be aggressively corrected. In the early stages of poisoning as there is no total body deficit of potassium and attempted early correction can worsen cardiotoxicity. If hypokalemia persists beyond eight hours, potassium should be replaced cautiously. High dose diazepam has been reported to have a specific cardio protective action in severe chloroquine poisoning. It is recommended that after intubation 2 mg/kg of intravenous diazepam should be given over 30 minutes and then 1 to 2 mg/kg for two to four days. Continuous and aggressive cardiorespiratory support appears to be the most critical factor in survival.

4) Salicylate

Salicylate poisoning is much less common now a day. Delay in diagnosis is associated with a mortality of 15% compared to a much lower rate in those patients in whom early diagnosis and initiation of therapy is made[27]. Children and the elderly may suffer toxicity with lower ingested doses and at relatively lower plasma salicylate concentrations. There is no antidote to salicylate poisoning, and management is directed towards preventing further absorption and enhancing elimination of the drug. The use of MDAC is controversial in salicylate overdose[28]. The plasma salicylate concentration should be determined on admission provided that the patient is more than four hours post-ingestion [29]. As salicylates can delay gastric emptying and may form concretions in the stomach resulting in delayed absorption, the plasma salicylate concentration should be repeated every three to four hours to ensure the concentration does not continue to rise. The plasma salicylate concentration correlates very roughly with toxicity although the presence of symptoms and signs and the degree of acidosis should be considered when interpreting the plasma salicylate concentration and deciding on further management. Metabolic acidosis is a particularly important negative predictor as it increases the CNS transit of salicylate and decreases salicylate renal elimination. After ingestion of enteric-coated preparations, plasma salicylate concentrations on admission are unreliable guides to the severity of poisoning, as levels may not peak till 12-18 hours after ingestion.

***Clinical features*:** These depend on type of poisoning as

- Mild (> 150 mg/kg): Lethargy, nausea, vomiting, tinnitus, and dizziness
- Moderate (> 250 mg/kg): Mild features and tacypnoea, sweating, hyperpyrexia, dehydration, restlessness.
- Severe (>500 mg/kg): Moderate features and metabolic acidosis, hypotension, CNS features (e.g. coma, seizures), renal failure.

Treatment

Patients with salicylate poisoning are often dehydrated because of vomiting, hyperventilation and sweating; rehydration is therefore an important aspect of management. Patients with severe salicylate poisoning are also at risk of pulmonary edema, however, and it is important not to cause fluid overload; in the elderly or those with cardiac disease a central line may be necessary to guide rehydration[8].

Urinary alkalinisation is an effective method of increasing salicylate elimination and is indicated in patients with moderate salicylate poisoning[30]. In adults this is achieved by administering I L of 1.26% sodium bicarbonate over three to four hours and regularly checking the urinary pH with indicator paper aiming for a urinary pH 7.5-8.5; an increase in the infusion rate or bolus of 8-4% sodium bicarbonate may be required if an alkaline urine is not achieved as patients can have a significant base deficit. The plasma potassium should be monitored as the serum potassium can fall precipitously once adequate urinary alkalinisation is achieved and also it is very difficult to produce alkaline urine if the patient is hypokalemic. We would therefore recommend adding 20-40 mmol potassium to each liter of intravenous fluid administered. Haemodialysis reduces both the mortality and morbidity of poisoning, and, as well as being effective at increasing salicylate clearance it also corrects acid-base and fluid balance abnormalities. It should be considered if the patient has a metabolic acidosis resistant to correction with 8,4% sodium bicarbonate, especially if the pH is <7,2, the salicylate concentration is >800 mg/L in adults or 700 mg/L in children or elderly, or the patient has features of severe poisoning. It is important that alkalinisation is still achieved in those salicylate-poisoned patients undergoing haemodialysis in order to reduce plasma levels quickly, prevent acidaemia and promote elimination of as much salicylate as possible via the kidneys.

5) Uncommon Overdoses Poisoning.

The most common area of mismanagement in overdoses is failure to recognize the potential severity of poisoning and instigate early treatment.

a) Theophylline

Severe theophylline poisoning (ingestion of more than 3 g in adults or 40 mg/kg in children) is associated with a high mortality[8]. Theophylline is most commonly used in sustained release formulations leading to delayed

absorption in overdose and delayed onset of toxicity, as late as 12-24 hours post-ingestion.

Clinical features: This depends upon the severity (grades) [30] as

- Grade 1: Vomiting, abdominal pain, diarrhea, anxiety, tremor, sinus tachycardia >120 bpm, plasma potassium 2,5-3,5 mmol/L.

- Grade 2: Haematemesis, disorientation, supraventricular tachycardia, frequent ectopics, mean arterial blood pressure at least 60 mmHg but unresponsive to standard therapy, plasma potassium <2.5 mmol/L, arterial pH <7.2 or >7.6 and rhabdomyolysis.

- Grade 3: Non-repetitive seizure, sustained ventricular tachycardia, mean arterial blood pressure <60 mmHg and unresponsive to standard therapy

- Grade 4: Recurrent seizures, fibrillation, cardiac arrest.

Management is most appropriately guided by the severity on the Sessler grading scheme[31]. All patients with theophylline poisoning should receive MDAC. Vomiting, which may be profuse, occurs in overdose in more than 70% of patients[31]. It may respond to metoclopramide but is more likely to be controlled by a 5HT3-receptor antagonist such as ondansetron. All patients should have cardiac monitoring. Sinus or supraventricular arrhythmias not causing hemodynamic compromise are best left untreated. In non-asthmatic patients, symptomatic supraventricular tachycardia should be treated with propranolol (0.01-0.03 mg/kg IV) or esmolol (25-50 mg/ kg), repeated according to response. Asthmatic patients should be treated with verapamil or cautiously with esmolol (short half-life and relative beta 1 selectivity). Ventricular arrhythmias are treated with DC cardioversion or magnesium sulphate, convulsions with intravenous diazepam. The patient should be intubated and ventilated. Plasma potassium concentration should be monitored frequently (everyone to two hours in severely poisoned patients) as hypokalemia is a potentially life-threatening consequence of theophylline poisoning. The blood glucose should be checked, as hyperglycemia is also common.

b) Iron

The clinical course of iron poisoning may be divided into four stages[32]. During the initial 30 minutes to several hours after ingestion, the corrosive effects of iron result in gastrointestinal upset leading to nausea and vomiting, abdominal pain and diarrhea. In severe cases gastrointestinal hemorrhage and shock can occur. The second phase, usually lasts from six to twenty-four hours after ingestion. This phase can be deceptively asymptomatic as patient during this phase can go on to develop severe toxicity. Phase three occurs from 12-48 hours after ingestion and may include severe lethargy, coma, convulsions, gastrointestinal hemorrhage, shock, metabolic acidosis, and hepatic failure with hypoglycemia, coagulopathy, pulmonary edema and renal failure. Phase four occurs between two and five weeks if the patient survives. Scarring from the initial corrosive damage can result in small bowel strictures and pyloric stenosis. It is important when assessing the ingested dose of iron that the elemental content of different iron preparations is considered. Undissolved iron tablets are radiopaque and an abdominal X-ray (AXR) should be taken in all patients to determine the need for gut decontamination. However the absence of radiopaque material on AXR does not exclude iron ingestion. If any tablets are visible on the AXR, whole bowel irrigation should be undertaken. Blood should be taken four hours after ingestion for determination of the serum iron concentration. If desferrioxamine is to be given before four hours for severe poisoning, blood should be taken for determination of the serum iron level just prior to its administration as once desferrioxamine has been given colorimetric assay methods may underestimate the amount of free serum iron[33]. A blood level taken more than six to eight hours after ingestion may underestimate the amount of free iron because of distribution into the tissues. For sustained-release preparations an initial serum concentration should be checked at four hours and again two to four hours later. The serum iron concentration should not be interpreted in isolation but patient's clinical condition and an accurate history of the ingested dose should be considered as well. Measurement of the total iron binding capacity is of no value in the management of these patients. There have been no controlled studies looking at the use of desferrioxamine in iron poisoning. However it is recommend that it should be used in patients with hypotension, shock, severe lethargy, coma or convulsions or a serum iron level >90 mmol/L.8 Patients with a serum iron concentration of 55-90 mmol/L should be observed for 24-48 hours post-ingestion. They do not require chelation therapy unless they develop symptoms or have hematemesis / melaena. The recommended initial dose of desferrioxamine is 15 mg/kg/hour. reduced after two to four hours up to a maximum of 80 mg/kg in 24 hours. More rapid infusion rates can cause hypotension, and there is a risk of pulmonary complications such as ARDS with doses of more than 80 mg/kg/24hrs. Chelation with desferrioxamine should be continued until the urine has returned to a normal color, symptoms have abated and all radiopacities have disappeared. Haemodialysis or haemofiltration may be required to remove the iron-desferrioxamine complex in patients with renal failure.

c) Cyanide.

Although rare, acute cyanide poisoning requires immediate action as it produces its effects, which include

metabolic acidosis and CNS, cardiac and respiratory depression[19]. The immediate diagnosis of cyanide poisoning is difficult. This poses a therapeutic dilemma for the clinician who must rapidly decide whether to administer specific antidotes, some of which are themselves toxic. The blood cyanide concentration is considered the gold standard in confirmation of acute cyanide poisoning; they are, however, rarely of use in emergency management because they cannot be carried out rapidly enough to guide treatment[34]. A sample should be taken before antidote administration for cyanide quantification at a later stage. Many patients suffering from smoke inhalation or burns may also have cyanide toxicity and may present with a lactic acidosis not responding to oxygen administration. A recent study has shown that immediate and serial measurements of plasma lactate concentrations are useful in assessing the severity of cyanide poisoning in patients in whom the diagnosis is strongly suspected on a clinical basis. In burn victims without severe burns, a plasma lactate concentration of >90 mg/dL (10 mmol/L) is a sensitive and specific indicator of cyanide intoxication. Decontamination is an important aspect of management. If hydrogen cyanide gas or liquid cyanide is involved, protective clothing and breathing apparatus is necessary and if the patient is intubated a closed circuit should be used. In cyanide salt exposure, contaminated clothing should be removed and placed in sealed bags, and the skin washed with soapy water. Meticulous supportive care is important in the management of cyanide poisoning. All patients should receive high-flow oxygen, and comatose patients will require intubation. Patients who present with an established metabolic acidosis should be treated with 12 ml of 8.4% sodium bicarbonate to correct the acidosis. The other aspect of the management of cyanide poisoning is the use of antidotes. Dicobalt edetate can be associated with severe adverse effects including cardiotoxicity, facial and laryngeal edema, bronchospasm and rashes. These effects are more likely to occur if it is administered in the absence of cyanide ions or if the drug is injected too rapidly. It should therefore be used only if the diagnosis of cyanide poisoning is certain and the patient has severe clinical features[8]. If the diagnosis is uncertain or dicobalt edetate is unavailable, the patient should be treated with a combination of intravenous sodium thiosulphate and sodium nitrite[35]. Hydroxocobalamin (at a dose of 5 g for an adult) is a newer cyanide antidote and is both effective and well tolerated.

d) Methanol / Ethylene Glycol Poisoning.

This involves supportive care together with the use of competitive inhibitors of alcohol dehydrogenase (e.g. ethanol, 4-methylpyrazole) to reduce the formation of their toxic metabolites[36]. In addition, aggressive bicarbonate therapy in patients with a metabolic acidosis decreases CNS transit of both ethylene glycol and methanol, and reduces optic nerve toxicity with methanol. Ethanol remains the antidote of choice in most cases for ingestion of these substances. All patients who have ingested a significant amount of ethylene glycol or methanol should receive a loading dose of ethanol while awaiting confirmatory laboratory results. An infusion should then be commenced in patients with confirmed poisoning (usually on the basis of a history of methanol or ethylene glycol ingestion in association with a raised osmolal gap) aiming for a blood ethanol concentration of 100-150 mg/dL. Ethanol has unpredictable kinetics therefore patients who require an ethanol infusion should have hourly to two-hourly monitoring of blood ethanol concentrations until the serum ethanol concentration is 100-150 mg/dL. and then two to four-hourly monitoring once this concentration is achieved. Patients with severe methanol/ethylene glycol poisoning (e.g. severe, resistant metabolic acidosis, acute renal failure, ocular toxicity with methanol poisoning) may require haemodialysis[37]; the dose of ethanol should be increased in these patients, or ethanol put in the dialysate to maintain a serum ethanol concentration of 100-150 mg/dL. Sodium bicarbonate is frequently used in the management of poisoned patients. Dose should be titrated to clinical effect and very large doses may be required, for instance in severe tricyclic antidepressant poisoning the initial boluses of 1-2 mL 8.4% sodium bicarbonate followed by further dose titrated to a pH of 7.45-7.5. It is important that drowsy patients are ventilated to prevent the potential carbon dioxide retention that can occur and result in a paradoxical intracellular acidosis in these patients treated with sodium bicarbonate.

e) Carbon Monoxide Poisoning.

Carbon monoxide is the common cause of death by poisoning in the rural area. Two areas of particular concern are the clinical assessment of the patient and adequate oxygen therapy. Patients should have a thorough neurological examination, which includes heel-toe walking and other tests of cerebellar function. A carboxyhaemoglobin saturation level is of value in confirming the diagnosis but its level is not indicative of the severity of poisoning[22]. An electrocardiogram (ECG) is essential in any patient with severe poisoning or in those with pre-existing heart disease because arrhythmias and myocardial ischemia are a common cause of morbidity and mortality in severe carbon monoxide poisoning.64-70 Arterial blood gas analysis is also required in significant poisoning; oxygen saturation monitors are misleading as they measure both oxyhaemoglobin and carboxyhaemoglobin. All patients should receive high flow oxygen through a tightly fitting facemask and this should be continued for at least 12 hours. The use of hyperbaric oxygen is controversial and some published randomized trials disagree on its efficacy[23]. Until further, well-controlled evidence is available it is recommend that hyperbaric oxygen therapy in the groups of patients with carbon monoxide poisoning should be given. Current recommendations for hyperbaric oxygen therapy are any history of unconsciousness, carboxyhaemoglobin concentration of >40% at any time, presence of any neurological features (especially cerebellar signs), pregnancy and ECG changes.

REFERENCES

1. Hawton K and Fagg J. Deliberate self-poisoning and self injury in adolescents: a study of characteristics and trends in Oxford, 1976-89. Br. J. Psychiatry 1997;171(12):556-60.

2. Jones AL, Dargan PI. Churchill's Pocketbook of Toxicology. Edinburgh: Churchill Livingston, Harcourt; 2001.

3. Karim A, Ivatts S, Dargan PI et al. How feasible is it to conform to the European guidelines on the administration of activated charcoal within one hour of overdose? Emerg. Med. J. 2001; 18:390-2.

4. American Academy of Clinical Toxicology; European Association of Poison Control Centers and Clinical Toxicologists. Position statement and practice guidelines on the use of multi-dose activated charcoal in the treatment of acute poisoning. Clin Tox 1999; 37(6):731-51.

5. American Academy of Clinical Toxicology and European Association of. Poisons Centers. Position statement: Gastric levage. Clin Tox1997; 35(7):711-19.

6. American Academy of Clinical Toxicology and European Association of Poisons Centers. Position statement: Whole bowel irrigation. Clin Tox 1997; 35:735-62.

7. Nozaki H and Aikawa N. Serine poisoning in Tokyo subway. Lancet 1995;346:1446-47.

8. Leblanc FN, Benson BE, Gilg AD. A severe organophosphate poisoning requiring the use of atropine drip. Clin Toxicol 1986;24:69.

9. Decort NLAM, Kiestra SH and Sanster B. the use of atropine and oximes in organophosphate intoxications: A modified approach. Clin Toxicol 1988;26:199.

10. Singh N, Kumar R and Singh B. Experimental monitoring of organophosphophate compound poisoning and evaluating the prognosis in patients. Int J Of Medical Toxiocology & Legal Medicine 2002 Dec; 5(1): 29-31.

11. Sharma VK and Satpathy DK. Incidence of aluminium phosphide poisoining: its analysis amd interpretation. JFMT1999 Vol 16(2):20-25.

12. Evans LEJ, Roscoe P, Swainson CP et al. Treatment of drug overdosage with naloxone, a specific narcotic antagonist. Lancet 1973; i:452-5.

13. American Academy of Clinical Toxicology; European Association of Poison Control Centers and Clinical Toxicologists. Position statement: Single-dose activated charcoal. Clin Tox 1997;35(7):721-41.

14. Goldfrank L. Weisman RS. Errick JK et al. A dosing nomogram for continuous infusion intravenous naloxone. Ann Emerg Med 1986; 566:91-5.

15. Routledge PA, Vale JA, Bateman DN et al. Paracetamol (acetaminophen) poisoning. BMJ 1998; 317: 1609-10.

16. Prescott LF. Paracetamol, alcohol and the liver. Br J Clin Pharmacol 2000; 49:291-301.

17. Buhl R, Jaffe HA, Holroyd KA et al. Systemic glutathione deficiency in symptom-free HIV-seropositive individuals. Lancet 1989; ii: 1294-8.

18 Wallace CI, Dargan PI, Jones AL. Paracetamol overdose: an evidence-based flowchart to guide management. Emerg Med J 2002; 19:202-5.

19. Vale JA, Proudfoot AT. Paracetamol (acetaminophen) poisoning. Lancet 1995; 346:547-52.

20. Prescott LF, Critchley JA. The treatment of acetaminophen poisoning. Ann Rev Pharmacol Toxicol 1983; 23:87-101.

21. Thom SR. Keim LW. Carbon monoxide poisoning: a review of epidemiology, pathophysiology. clinical findings. and treatment options including hyperbaric oxygen therapy. Clin Toxicol 1989;27:141-56

22. Weaver LK, Hopkins RO. Chan KJ et 01. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002; 347(14): I 057-67.

23. Conso F. Death from acute poisoning in man. Vet and Hum Toxicol 1979; 21 :68-9.

24. Goldfrank LR, editor. Goldfronk's Toxicologic Emergencies. 6th ed. Stamford, CN:Appleton and Lange; 1998; 745.

25. Clemessy JL, Taboulet P, Hoffman JR et 01. Treatment of acute chloroquine poisoning: a 5-year experience. Crit Care Med 1996; 24: 1189-95.

26. Dargan PI, Wallace CI, Jones AL. An evidence-based flowchart to guide the management of acute salicylate (aspirin) overdose. Emerg Med J 2002; 19:206-9.

27 Done AK. Salicylate intoxication: significance of measurements of salicylate in blood in cases of acute intoxication. Pediatrics 1960; 26:800-7.

28 Brenner BE, Simon RR. Management of salicylate intoxication. Drugs 1982; 24:335-40.

29. Hillman RJ. Prescott LF. Treatment of salicylate poisoning with repeated oral charcoal. BMJ 1985; 291:1472.

30. Sessler CN, Glauser FL. Cooper KR. Treatment of theophylline toxicity with oral activated charcoal. Chest 1985; 87:325-9.

31. Minton NA, Henry JA. Treatment of theophylline overdose. Am J Emerg Med 1996; 14:606-12.

32. Covey TJ. Ferrous sulphate poisoning: a review, case summaries, and therapeutic regimen. J Pediatr 1964;64:218-26.

33. Helfer RE, Rodgerson DO. The effect of deferoxamine on the determination of serum iron and iron-binding capacity. J Pediatr 1996; 68:804-6.

34. Baud FJ, Boron Sw. Megarbane B et al. Value of lactic acidosis in the assessment of the severity of acute cyanide poisoning. Crit Care Med 2002; 30(9):2044-50.

35. Baud FJ, Barriot P. Toffis V et al. Elevated blood cyanide concentrations in victims of smoke inhalation. N Eng J Med 1991; 325:1761-6.

36. American Academy of Clinical Toxicology. Practice guidelines on the treatment of methanol poisoning. Clin Toxicol 2002; 40(4):415-46.

37. Brentj. Current management of ethylene glycol poisoning. Drugs 2001; 61(7):979-88.

Profile of Drowning Deaths in Mangalore, a Coastal City of Karnataka

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ABSTRACT

Background: It is estimated that 1,50,000 people die all over the world in this manner every year. Lack of reliable statistical data concerning the drowning death in costal Karnataka has motivated us to conduct this study.Objectives:To

study the incidence of drowning in costal Karnataka.To analyze and quantity of the magnitude of death from drowning and to provide epidemiological data, so that, preventive measures can be undertaken.Method: A retrospective study was done from 1999 to 2004.Results: Following observations were made as.(i)Drowning mortality rate in costal Karnataka is as high as 15.15 / lakh population. (ii)Males are at higher risk of drowning death than females.(iii)Maximum number of drowning victims were of age group 31 - 40 years(iv)Most likely locations of for drowning are river, well / pond, sea/ beach and lake respectively.(v)incidence of drowning death increased during rainy season.

CONCLUSION

Preventive measures should be taken to prevent drowning death since drowning death in costal Karnataka is much more than the average global drowning mortality rate.

Key words: Submersion , drowning death.

INTRODUCTION

The history of swimming has been described in great detail by Maiello and Pearsall(1). It seems likely that homo sapiens learnt to drown long before they learnt to swim. While archeologists have provided us with drawings of swimming 5,000 to 9000 years ago no early depictions of drowning have been unearthed. Accidental drowning is a largely preventable cause of death. Water safety organisations, the general public and legislators need adequate information about the circumstances of drowning to initiate preventive action effectively.

Death due to Submersion is of great interest from the Medico legal point of view. Drowning is not a leading cause of death in India and is not high lightened by the health authorities as a major area of concern. It is estimated that 1,50,000 persons die all over the World in this manner every year. This figure represents an average of 400 persons per day(2).

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Drowning is in third place as a cause of accidental death among children up to the age 5 (3).Increase or decrease in the incidence of this form of death over a year is closely related to climate, time of year, and geographic zone.(3-9)

The lack of reliable statistical data concerning the impact of this specific form of death in our region/country must be emphasized. Consequently there has not been much research or interest in this area. However, the reality is that each death by drowning is still one death too many. This is especially when hindsight often shows that many death from drowning are preventable. More data and knowledge about drowning can only help in better understanding of drowning and should inturn lead to more effective measures to tackle the problem. This paper aims to describe the incidence of drowning in Mangalore a coastal city of Karnataka in India ,over 6 year period

i.e. 1999 to 2004. The purpose is to analyze and to quantify the magnitude of death from drowning and to provide epidemiological data so that preventive measures can be undertaken.

MATERIAL & METHODS

The data for epidemiological analysis were obtained from Medical Record department of Dist. Wenlock Government Hospital Mangalore. All The information concerning the cases died due to drowning , that have been subjected to autopsy were recorded .Likewise inquest report, Name, Age, Sex, and other ground information and cause and manner of death were properly analysed.

Total of 3684 cases were autopsied during the six year period , 1999 to 2004.of which 350 cases ie10.52% were of drowning deaths. These drowning cases were included in the present study.

RESULTS

The results of the epidemiological study are presented in table I toV. .Table -I sets out the total no of deaths from drowning and the drowning rate per lakh population in the period under study.

Table II sets out the gender of drowning victim and the male to female ratio.

Table III sets out the age group of drowning victim.

TableIV sets out the location of drowning cases in Mangalore

TableVsets out Manner of drowning deaths from 1999 to 2004

Table I : Mangalore drowning statistics - 1999 - 2004

Year	Population	Total drowning	Rate / Lakh
1999	4,02,609	54	13.40
2000	4,09,206	62	15.15
2001	4,19,306	60	14.30
2002	4,26,779	51	11.94
2003	4,34,712	64	14.7
2004	4,42,309	59	13.31

Table II : Gender of drowning victims - 1999 - 2004

Year	Total	Male	Female	Male to Female ratio
1999	54	41	13	3.2:1
2000	62	46	16	2.9:1
2001	60	43	17	2.5:1
2002	51	34	17	2:1
2003	64	55	9	6:1
2004	59	53	6	8.9:1

Table III : Age groups of drowning victims - 1999 - 2004 (6 years)

Age (in year)	1999	2000	2001	2002	2003	2004
0-10	1	3	5	2	1	4
11-20	13	10	13	16	11	8
21-30	10	13	14	8	10	21
31-40	19	21	16	8	25	16
41-50	4	4	3	8	6	4
51-60	2	3	3	1	2	2
61-70	1	2	3	3	3	1
71-80	0	1	0	3	2	0
81-90	0	1	0			0
91-100	0	0	0			0
Unknown	4	4	3	2	4	3
Total	54	62	60	51	64	59

Table IV : Location of drowning cases - 1999 - 2004

Location	1999	2000	2001	2002	2003	2004
Sea/beach	11	13	14	15	12	12
River	19	24	20	18	27	28
Well/Pond	18	16	15	14	20	16
Lake	6	9	11	4	5	3
Swimming pool	0	0	0	0	0	0
Reservoir	0	0	0	0	0	0
Total	54	62	60	51	64	59

Table V : Manner of drowning deaths from 1999 to 2004

Year	Suicidal drowning		Accidental drowning		Homicidal drowning	
	Male	Female	Male	Female	Male	Female
1999	17	6	24	7	0	0
2000	18	8	28	8	0	0
2001	14	5	29	12	0	0
2002	13	7	21	10	0	0
2003	27	7	28	2	0	0
2004	14	4	39	1	0	1
Total &	103	37	169	40	0	1
%	(74%)	26(%)	(81%)	(19%)		(.28% out of 350

DISCUSSION

The data collected in the present study reveals several interesting facts about the drowning situation in Mangalore ,a costal city of India. Mangalore has a drowning rate per lakh population that varied from a low of 11.94 in 2002 to a high of 15.15 in 2000 in the period 1999 to2004 as shown in the table 1. This is not comparable to the drowning related mortality per lakh population of 1 to 1.3 in the high income or developed countries of Europe and America. (4,5,6) This is also more than the average global drowning mortality rate of

7.4 per 100000 population, derived from a study of data from "The 2000 Global Burden of Disease Study", probably the most comprehensive source of global data in drowning.(10).

The study data also shows that in all years under consideration, the male drowning mortality rate is much higher than the female drowning mortality rate in Mangalore. In fact, the male to female ratio ranged from a low of 2:1 in the year 2002 to high of

8.9:1 in the year 2004 as shown in the table II This correlates with the finding of RMK Tan(11) i.e. 2.7 to 11.3. This proportion is similar to 4.16 males for every female by Wintemute et al12 in USA, during the period 1974 to

1985.Other observations yield the following results 2.31 males to every females in Denmark for the period 1987-1989(9) and 5.3 males for every female in Minnesota for the period1980-1985(4)

The age group of 31-40 years had the largest number of drowning victims in our study, unlike western countries where drowning of children below the age of 15 years were generally higher. (3,10). A possible explanation in Mangalore case could be that parents here are more careful about the younger children. Further research in this area may lead to better understanding of this situation.

In this 6 years study river was the location with highest number of drowning victims as shown in the table III. This is followed by pond/well,sea and lake as the next three locations with the highest number of drowning victims respectively. In our study there was not a single death reported from bathtub and swimming pool, these may be due to less usage of bathtubs and swimming pools in Mangalore city, a developing city in a developing country. Unfortunately, very few countries report to the World Health Organisation, with the inclusion of the place of injury or related risk factors such as alcohol consumption according to the international classification of disease. Therefore the comparison with the global position of this factor could not be made. (10)

Many authors point out that the increase or decrease in the incidence of this form of death over a year is closely related to the time of year, Climatological factors, geographic zone where this death occurs .(4,5,6,7).Our study confirms that the majority of death took place during rainy season of the year, when water levels in the river, pond/lakes/well are high, in contrast to west were majority of deaths took place during the hottest months of the year, when water sports are at their most popular(4,5,6,7).

With regard to the MedicoLegal etiology, three possibilities are traditionally considered; accidental submersion, suicide and homicide although homicidal form of death is of little statistical importance.

Accidental submersion is the most common cause of drowning death. In this respect, some author as Wintemute et al(12) ,Giersten(13), Derobert,(14) Copeland (15) and gave figures that ranged between 80-90% of the total number of death due to submersion. However ,there may be variation depending on the geographic zone where the study is conducted.Our study confirms the fact that 60% ie,209 cases of all submersions were accidental, with proportion of 4 males to every female. This percentage is not similar to those given by Shephred,(3) Hedberg et al(4) O'Caroll et al,(16)) Bierens et al,(17) and) in their studies.

When suicidal submersion is analyzed 39.7% were of suicidal submersion i.e. 140 cases out of 350 cases,2.8:1 male female ratio.According to the sex of the victim clear differences is observed. In case of females drowning of this nature reach the proportion of 26% of all suicidal submersions. This percentage is very similar to the 20% obtained by Rodes et al(18) in the districts of Elda and Villena (Spain) and significantly lower than the 41.41% and 36.36% obtained by Copeland (15) in Florida and by Avis in Newfoundland(19) respectively . With regard to males, suicidal submersion reached the proportion of 74% of all submersions of this etiology as shown in the tableV. This is similar to the 80% obtained in the districts of Elda and Villena (18) and appreciably more than the 58.6% and 63.63% obtained in Miami (17) and Newfoundland (19), respectively.

On the other hand, it must be pointed out that homicidal submersion is quite uncommon, given the difficulties this form of murder entails when victim is an adult, unless the latter is first weakened by other means. Only one case of homicide was found, that of a girl aged

3years.Here the mother of the child threw the child into the well and she then committed suicide.This is a dyadic type of death. This case represented 0.29% of all deaths due to submersion, far smaller percentage compared with the 1% and

2.3% obtained by Wintemute et al. (12) and Copeland (20) in the county of Sacramento and Florida, respectively.

CONCLUSION

This study shows that Mangalore drowning rates are more than the developed countries. Males in Mangalore are at far more risk of death by drowning than females.

It should be pointed out that this study has certain limitations,that are statistics of drowning are dependent on how drowning is defined. In fact, the definition of drowning has changed over the years and differs in different jurisdictions. The age groups of a fairly significant number of drowning victims were also not known therefore the age related conclusion in this study may differ from the actual situation. Not withstanding these limitations, this study has produced some important findings and this might help to raise the awareness in this area.

Maximum number of drowning victims were of age group 31-40 years in our studies, and most likely locations for drowning in Mangalore are the river, well/pond,sea/Beach and lake respectively. Males are at higher risk of death due to drowning and majority of death due to drowning took place during rainy season.

The main measures of drowning prevention may be broadly divided into supervision. legislation, swimming lessons and aquatic safety education. The fact remains that more can be done for drowning prevention in the costal city of Mangalore. Swimming skills and water safety measures should be widely taught in the schools and other institution with special emphasis towards males, to be seriously considered by the Government. Finally more studies and research also needs to be done to provide a better understanding of the epidemiology of drowning in Mangalore and how deaths by drowning may further be reduced. In the mean time, we hope that the present study has contributed in some way towards the better understanding of this problem.

REFERENCES

1. Ian J Mackie AM - Pattern of drowning in Australia (orginal submission)http://www.mja.com.au/public/issues/iprs2/mackie/mackie.html

2. Nichter MA, Everett PB ,Profile of drowning victims in costal city.J Fla Med Assoc. 1989 Feb;76(2):253-6.

3. Shepherd S. Immersion Injury: Drowning and near drowning. Postgrad Med 1989;85:183-90.

4 Hedberg K, Gunderson P, Vargas C, Osterholm M, Mac Donlad K. Drownings in Minnesota, 1980-85; a population based study. Am J Publ Health 1990; 80: 1071-4.

5 Davis S. Smith LS. The epidemiology of drowning in Cape Town 1980-1983. S Afr Med J 1985; 68: 739-42.

6 Pearn J.,Pathophysiology of drowning. Med J Aust 1985; 142-586-8.

7 Wong LL, McNamara JJ. Salt water drowning. Hawaii Med J 1984; 43: 208-10.

8 Schuman SH, Ollison CW, Wannamaker CC. Which drowning are preventable? An in-depth study of 62 deaths in Charleston Country, South Carolina Med Assoc 1979; 74: 361-4.

9 Kringsholm B, Filskov A, Koch K. Autopsied cases of drowning in Denmark 1987-1989. Forensic Sci Int 1991; 52: 85-92.

10 Peden, MM. The epidemiology of drowning worldwide. Proceedings of the World Congress on Drowning, 26-28 June 2002, Amsterdam Stichting Foundation Drowning 2002

11 RMK Tan, The epidemiology and prevention of drowning in Singapore.Singapore Med J2004vol45(7); 324.

12 Wintemute GJ, Kraus JF, Teret SP, Wright MA. The epidemiology of drowning in adulthood: implications for prevention. Am J Prevent Med 1988; 4: 343-8.

13 Giersten JC. Drowning. In: Tedeschi CG, Eckert W, Tedeschi LG, eds. Forensic medicine, Vol. 3. Philadelphia: WB Saunders, 1977; 1317-22

14 Derobert L. Medicine Legale. Paris: Flammarion Medicine-Sciences, 1974

15 Copeland AR. Suicide by drowning. Am J Forensic Med Pathol 1987; 8: 18-22

16 O'Carroll P, Alkon E, Weiss B. Drowning mortality in Los Ageles Country, 1976 to 1984. JAMA 1988; 260: 380-3.

17 . Bierens J, Van Der Velde E, Van Berkel M, Van Zanten J. Submersion cases in the Netherlands. Am Emerg Med 1989; 18: 366-73.

18 Rodes F, Pamblanco E, Marti J, Navarro ML. El suicidio en la tercera edad. Rev Esp Med Leg 1990; 17; 7-13.

19 Avis SP. Suicidal drowning. J. Forensic Sci 1993; 38: 1422-6.

20 Copeland AR. Homicidal drowning. Forensic Sci Int 1986; 31: 247-52.

Eye is the Spy of Forensic Medicine

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ABSTRACT

Read the mind through the gesture of the *eye*. A forensic pathologist can gather a lot of medico-legal informations by examining the *eye* both in antemortem and postmortem cases. Here is a topic which describes some changes of the *eye* that undergo after death in addition to different characteristics of the *eye* during life.

Keywords: Intraoccular pressure (lOP), Vitreous, miotics, mydriatics, funduscopy

INTRODUCTION

Eye is the most important sensory organ of the body which serves not only for the sake of vision but also for the medicolegal purpose[4]. Though a lot of publications having plethora of informations in this regard already exist, a vast treasure of knowledge is still lying unearthed. Human *eye* is nevertheless, an important tool for many medicolegal information both in antemortem and postmortem cases.

MATERIALS AND METHODS

Few antemortem and mostly postmortem cases were studied in these series. About 120 postmortem cases were selected for evaluation of different information from the *eyes*.

External features, pupillary reaction to autonomic drugs, estimation of intraocular pressure with associated corneal changes, funduscopic examination of the retina and biochemical estimation of the enzymes of vitreous were incorporated in this study.

The cases having clear cornea were chosen for better visualization and objective assessment of the intraocular structures. The information so gathered were charted and compared with those of the earlier authors.

OBSERVATIONS

1. ANTEMORTEM EYE CHANGES

A. Determination of age : In our study findings in favour of the old age were:-

(a) Wrinkling of the skin of *eyelids*

(b) Xanthelasma in medial and lateral angles of the *eyelids*

(c) Long hairs of *eyebrows* and *eyelids*

(d) Arcus senilis surrounding the cornea

(e) Atherosclerotic changes in blood vessels of the *eyes* revealed by ophthalmoscopy

(f) Pale and dull condition of the fundus occuli.

B. Determination of race: For this. the colour of the iris was an important landmark in the *eyes*[3].

Its colour was brownish black in Indian or Negroid races whereas it was greyish or bluish in white races.

C. Other Informations: It was observed that the pupillar size, reflexes and its reaction to light, autonomic drugs etc. can signify trauma, poisoning and diseases during life.

2. POSTMORTEM EYE CHANGES

A. External features: According to cause of death the outward look of the *eyes* was found to vary as shown in Table 1.

Table 1: Externally visible postmortem *eye* changes

Percentage of cases	Cause of death	Externally visible *eye* changes
80%	Hanging	Eyes semiclosed or unequally opening
70%	Ligature strangulation	Eyes open with congested look
80%	Organo-phosphorus poisoning	Alcoholic *eyes*
95%	Hypovolumic shock	Eyes (Sclera) white
10%	Head injuries	Black *eyes*

3. Pupillary reaction to autonomic drugs:

Pilocarpine 4 % and tropicamide 1 % *eye* drops were used as fast acting miotic and mydriatic respectively to observe postmortem pupillary changes in the *eyes* at different time. intervals in 55 death cases as shown in Table 2.

Table 2: Papillary reaction to pilocarpine 4% and Tropicamide 1% *eye* drops at different time intervals after death

Time since death	No. of cases	Pupillary reaction to Pilocarpine 4%	Tropicamide 1%	Result
0 – 4 hrs	25	++++	+++	(WR)
4-8 hrs	20	+++	++	(MR)
8 – 12hrs	10	-	-	(NR)

WR = Well Reactive; MR = Mild Reactive and NR = Not Reactive

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In this series the postmortem pupillary reaction was found better in pilocarpine than tropicamide.

C. Intraocular tension (lOT) :

(i) IOT was tested digitally and with Schiotz Tonometer In 56 death cases. It was found to vary with the time since death as shown in tab1e 3.

Table 3: Changes In Intraocular tension (lOT) at different time Intervals after death.

Time since death	No. of cases	Changes in intraoccular tension
0 – 4 hrs	30	Tension was reduced to about half of the normal value
4 – 8 hrs	26	Tension was reduced to almost nil

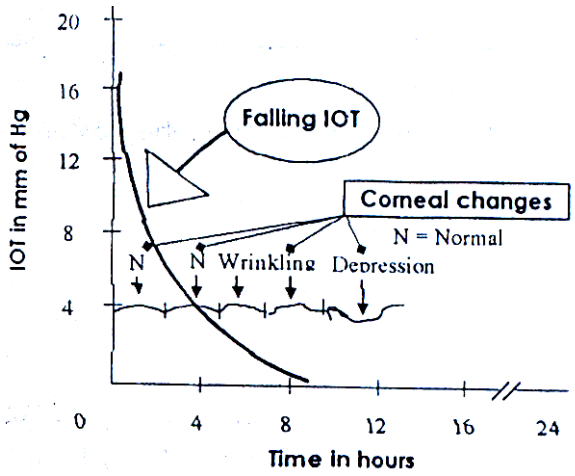
(ii) Indentation of cornea and its wrinkling was observed in 78 death cases as the IOT falls as shown in table. 4.

Table 4: Changes In cornea at different time intervals after death

Time since death	No. of cases	Changes in cornea
0 – 4 hrs	30	Corneal size was normal with smooth surface
4-8 hrs	26	Onset of wrinkling of the corneal surface
8-12hrs	12	Corneal surface was found more wrinkled
12-24hrs	10	Maximum wrinkling of corneal surface with central depression

The above changes in IOT and cornea as a function of time since death is depicted in Fig. 1.

Figure 1 .Changes in IOT and cornea as a function of time since death



D. Fundus Examination (Funduscopy):

Ophthalmoscopic examination of the fundus of the eye i.e. funduscopy was done in 8 death cases in which cornea was clear (Time since death was within 2 to 4 hours) and the findings were correlated with the cause of death as shown in Table5.

Table.5 : Postmortem funduscopic findings In cases of death due to different causes

Causes of death	No. of cases	Findings of funduscopy
Septicemia	3	Dot shaped haemorrhages.
Asphyxial death (Mostly compression to chest)	4	Blurred haemorrhages.
Sudden death (Myocardial infarction)	1	Features of diabetic Retinopathy

E. Vitreous transaminase (SGOT) estimation:

In this series. the SGOT estimation was done in 40 numbers of death cases in respect to postmortem intervals (i.e. time since death) and the findings were encouraging as shown in Table6.

Table 6 : Mean vitreous SGOT levels in death cases in respect to time since death

Time since death	No. of Postmortem	Mean SGOT level (in I.U.) in vitreous fluid
0 - 6 hrs	02	60
6 - 12 hrs	05	70
12 -18 hrs	10	85
18 - 24 hrs	13	90
24 - 48 hrs	10	105

I.U. = International Units

It may be seen from table 5 that there is a steady rise of SGOT level in the vitreous fluid as the PM interval increases. In our study, it was also observed that SGOT level was high in patients who survived some days after receiving any grave trauma before death. It was less or normal in patients who suddenly died due to accidents or homicides.

3. INJURIES

In our study subconjunctival haemorrhage, periocular haemorrhage and subcutaneous haemorrhages (i.e. black eyes) were prominent features. The incidence of black eyes in fatal head injury cases was 10% of all the head injuries. The black eyes with subconjunctival haemorrhage were associated with different types of intracranial haemorrhages. There were five cases of penetrating injuries associated with fatal head injuries. In all cases there was prolapse of internal contents like uveal tissue, lens and

vitreous. In one cases there was only one penetrating injury to eye which caused death after 24 hours. In this cases there was fracture of the orbital plate of the frontal bone with laceration of frontal cortical lobe. There was one case which had chemical burn caused by sulphuric acid which resulted in lid deformities with opacification of cornea.

DISCUSSION

For determination of age we generally take the help of ossification test in the young age group. But in the older age group. examination of eyes can be incorporated to confirm the age. For malingering cases pupillary examination can provide clues to confirm the exact damage to the vision. Postmortem pupilary reaction to pilocarpine - a miotic, was found to be better and faster than tropicamide - a miotic, however, in both instances. the resulting pupillary reaction was well reactive upto 4 hours after death in our study. As the time interval following death increases, the IOT curve falls downward abruptly. Thus the pressure gradient can be an important tool for estimating time since death. Fuduscopic examination of retina can be very useful to know the cause of death. The SGOT enzyme level continues to increases as the time since death advances. This may be due to cellular disintegration and liberation of enzymes. In addition, trauma to various organs and muscles can cause increase in the SGOT level in the vitreous of the eye. Finally black eyes is an important sign of grave intracranial injury.

CONCLUSION

From the result of various observations made in our study, many useful conclusions are drawn such as

- For estimation of age, detail examination of the eyes is helpful.
- The cause of death for most cases is reflected in the external features of the eyes.
- Postmortem time interval can be estimated from IOT, reaction to miotics and mydriatics in the early periods and SGOT level of the vitreous in the late periods after death.
- Eye injuries can predict different types of head injuries.

REFERENCES

1) Cox, Major H. M. V. - Medicolegal Court Companion.. 3rd Ed.. revised by Nagendra Kumar Bhattacharya. 1939. Eastern Law House. Calcutta. p. 84.

2) Glaister, John - Medical Jurisprudence and toxicology. 7th'Ed.. 1942. Livingstone, London and Edinberg. pp.119.635. .

3) Greva!. S. P. S. - Lyons Medicolegal Jurisprudence for India. 101h Ed., 1953, Thacker Spink, Calcutta. pp. 88. 235, 236, 139.

4) Kamath. M. A. - Medicolegal Jurisprudence. 5th Ed., 1948. Madras Law Journal Office. Madras.

5) Mont. A. Keith - Forensic Medicine.. 1960. Lloyed-Luke. London. p. 36.

6) Moor. N. J. – Modi's Textbook of Medical Jurisprudence and Toxicology, 13th ed. 1959. Tripathy. Bombay. p. 265.

7) Parikh. C. K. - A simplified textbook of Medical Jurisprudence and Toxicology. 1970. Medical Publications, Bombay p.105.

8) Shukla. K. C. - Textbook of Forensic Medicine. 1948. 1948. Dr. K.C. Shukla. 27. Old 1 aloknagar. Ahmedabad. p. 130.

9) Simpson. Keith - Taylor's Principle and Practice of Medical Jurisprudence. Vol. 1, 12th Ed.. 1965, Churchill. London, p. 243.

10) Smith. Fred J. - Taylor's Principle and Practice of Medical Jurisprudence. Vol. 1. 1905. Churchill. London. p. 510.

11) Smih. Sydney and Fidden. Frederick Smith - forensic Medicine. 10th: Ed. 1955. Churchill. London. p. 148.

12) Smith Sydney and Simpson. Keith Taylor's Principle and Practice of Medical Jurisprudence. Vol.1, 1th Ed. 1956. Churchill. London. pp. 173.315.

13) Soderman, Harry – Modern Criminal Investigator, 4th Ed., 1951, Frunk and Wagnalis, New York, P., 174.

14) Waddell. LA. - Lyons Jurisprudence for India. 8th Ed.. 1928. Thacker Spink. Bombay. Calcutta. Simla. p. 164.

Abuse of Police Power and Custodial Deaths Concerns Apex Court

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ABSTRACT

Rising human rights violation concerns every democratic society. UN charter for human rights, does not allow barbaric custodial crimes as seen many times nowadays particularly countries affected by terrorism & civil war. What was done to people in Iraq, can't be described as civil act acceptable under the UN charter for human rights. Custodial deaths are rising in India & Honorable Supreme Court of India has also expressed its displeasure as seen in recent judgement where police officials from Madhya Pradesh were convicted by trial court and High Court for killing suspect in police custody.

Key words: - Custody, human rights, constitution

INTRODUCTION

Concerned over rising human rights violations the Govt of India had set up National Human Rights Commission (NHRC), which is ordinarily being headed by retired Chief Justice of Supreme Court. It is presently headed by former Chief Justice of Apex court – Justice A.S. Anand, N.H.R.C can take suo moto action in cases where there is violation of human rights. In spite of National Human Rights Commission, human rights violation by state machinery has been going on for quite sometime Article 21 of the constitution guaranties to every citizen of India , right to life with dignity. In India more than 1,300 persons died in police or judicial custody in 2002. As per Apex court, only 40% of the cases were reported and rest were not pursued by states.

DISCUSSION

In a detailed judgement, Justice Arijit Pasayat and Justice C.K. Thakkar in cases of police officials of Madhya Pardesh where there were five accused, Apex Court confirmed conviction of one police personal while acquitted three and one died. The court has noted that abuse of police power is wide spread all over world. ***However, it observed that police is not lacking in showing scanty respect for right to life in India.*** The Apex court ruled that state can't conceal the crime. It also quoted Abraham Licolon words- If you once forfeit the confidence of our fellow citizens, you can never regain their respect and esteem. N.H.R.C. has also asked all state governments to stop encounter killings.

CONCLUSION

The present Apex court judgement has drawn attention over abuse of state machinery and human rights violation in India.

REFERENCE

Times news dated 22.11.04

A Study of Pattern and Injury Severity Score in Blunt Thoraco-abdominal Trauma cases in Manipal

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ABSTRACT

Objective: - The objective of the study was to find out the pattern of blunt thoracoabdominal trauma in Manipur as regards the sex ratio, the type of blunt trauma, survival period and cause of death of victims. It also aims at studying the injury severity scores (ISS) of these cases so as to assess what kind of injuries is incompatible with life and what are some of the frequent injury complications and was there any preventable deaths in these cases?.

Material and method: - Materials for the present study were collected from the 125 cases of blunt thoracoabdominal trauma which were brought for medicolegal autopsy at the mortuary of Forensic Medicine Department of Regional Institute of Medical Sciences, Imphal during the period from October 2001 to July 2003, and these cases were comprehensively studied.

Result: - Males outnumbered females in the ratio of 3.8 : 1. The commonest age group of the victims was 21-30 years (20.80%). Vehicular accident was the leading cause of blunt thoracoabdominal trauma (86.40%) followed by assault by blunt weapon (8%). 12.80% of the victims no associated external injuries to the thoracoabdominal region. 59 victims (47.2%) died at the spot. 15 cases (12.0%) died within 1 hour and 13 victims (10.04%) survived less than 2 hours. Only 2 victims (1.60%) survived up to more than 1 week. The commonest cause of death was haemorrhagic shock (as a result of intra thoracic and abdominal bleeding) combined with head injury in 61 (48.80%) cases followed by haemorrhagic shock alone in (44%) of the cases. Peritonitis was the cause of death in 2 (1.60%) cases. It was found that in victims with low ISS (21-30 and 31-40 ISS score ranges) survival was more as compared to the victims with high ISS (51-60, 61-70 and 71-75 ISS score ranges). The spot-death victims had a mean ISS score of 61.73 and cases who died within 1 hour showed 48.33 as the mean ISS. Mean ISS was low in those victims who survived more than 1 week i.e.27.50.

Kewords: Blunttrauma, thoracoabdominal injuries, survival period and injury severity score (ISS).

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INTRODUCTION

The road traffic accident has always been a leading cause of blunt trauma throughout the world. At the same time, blunt weapons are some of the most easily available weapons during an unanticipated fight or assault. The thoraco-abdominal region, because of its dimension and anatomical position, is a major site of impact in any form of blunt trauma viz. road traffic accidents, the fall from heights, landslides, physical assaults, etc. Subsequent to blunt trauma, the thoracic and abdominal walls may show abrasions or bruises; but the abdominal wall usually escapes gross injury by transmitting the force of violence to more resistant organs inside the abdominal cavity, which get injured[1]. Contusions or lacerations of the lungs and the heart may be produced by blows from a blunt weapon or by compression of the chest even without fracturing any bone of the thorax or showing marks of external injury[2]. So, there is always a possibility of fatal thoracoabdominal injuries to be unnoticed leading to their late detection and fatal outcome. Moreover, injuries to the chest and abdomen are commonly associated with injuries to other parts of the body, namely the head, spine, limbs, etc. Hence, the presence of intra-thoracic and intra-abdominal injuries may be overlooked or discovered later. Early detection of the injury and prompt treatment are necessary in saving the lives of many of these victims.

The Injury Severity Score (ISS), which was first formulated by Baker et al.[3] from the Abbreviated Injury Scale (AIS), is an anatomical scoring system that gives an overall score for cases with multiple injuries. Thus, a postmortem study of injury severity score in blunt thoracoabdominal trauma was carried out as the autopsy of injured persons dead on the spot can point out what kind of injuries is incompatible with life as well as with their severity. The autopsy of injured persons who survived trauma can also point to the most frequent injury complications, clinical diagnosis and preventable deaths[4].

MATERIAL AND METHODS

Materials for the present study were collected from the 125 cases of blunt thoracoabdominal trauma which were brought for medicolegal autopsy at the mortuary of Forensic Medicine Department of Regional Institute of Medical Sciences, Imphal during the period from October 2001 to July 2003, and these cases were comprehensively studied.

The criteria used for selection of cases for this study were as follows: A) All the cases showing fatal thoracic injuries with or without external injuries were considered for this study. B) All the cases showing fatal thoracic and/ or abdominal injuries with any associated body injuries, both of which having jointly contributed towards the death were also included. C) All those cases of thoracic and/ or abdominal injuries with or without any associated body injuries, which were hospitalized following trauma and subsequently succumbed to their injuries, were also included in the study. Decomposed bodies and those cases where the nature of sustenance of injury was not known were not included in the study.

For the establishment of injury severity score, the injuries are ranked on a scale of 1 to 6 as follows as per AIS (Abbreviated Injury Scale).

AIS Score	Injury
1	Minor
2	Moderate
3	Serious
4	Severe
5	Critical
6	Unsurvivable

Each injury is assigned an Abbreviated Injury Scale (AIS) score and is allocated to one of six body regions i.e.

1. Head or Neck injuries - include any injury of the cervical spine, cervical spinal cord, skull, brain and ears.

2. Face injuries - include mouth, eye, nose and facial bone injuries.

3. Chest injuries - include injuries to all of the internal chest cavity organs, the diaphragm, thoracic spine and rib cage.

4. Abdominal injuries- include injuries to all the internal abdominal organs, pelvis and the lumbar spine.

5. Extremities injuries- include all sprains, fractures, amputations and dislocations.

6. External injuries- include all contusions, abrasions, and lacerations independent of their location.

For the calculation of the ISS, only the highest AIS score in each body region is used. The 3 most severely injured body regions have their score squared and added together to produce the ISS score which ranges from 1 to 75. If a victim has any injury with an AIS value of 6, the ISS is assigned a value of 75.

RESULTS

Out of the 125 cases of blunt thoracoabdominal trauma studied, males comprised 79.20% of cases; and the male: female ratio was 3.8 : 1. Male victims in the age group of 21-30 years (20.80%) followed by the age group of 31-40 years (18.40%) were the commonest victims (Table-1).

As shown in Table-2, it was observed that vehicular accident was the commonest cause accounting for 86.40% of the cases. Other causes of blunt trauma included assault by blunt weapon in 10 cases (8.00%) and 3 (2.40%) victims who were hit by fall of heavy objects viz. boulder.

In the present study, 87.20% of the cases showed associated external injuries on the thoracoabdominal region while the remaining 12.80% did not show any external injuries (Table-3). But all the victims showed some amount of injuries in the form of abrasions, bruises and lacerations on the face and limbs.

59 victims (47.20%) of blunt thoracoabdominal organ trauma died at the spot. 15 cases died within 1 hour. 6 of out these 15 cases (12.00%) were declared brought dead when they reached the hospital since they died on the way. It took almost 45 to 50 minutes for them to reach the hospital from the site of the incident and they were declared brought dead when they reached the hospital. 10.04% of the cases survived less than 2 hours. Only 2 victims (1.60%) survived up to more than 1 week as shown in Table–4.

The commonest cause of death observed was haemorrhagic shock (as a result of intra thoracic and abdominal bleeding) combined with head injury in 61 (48.80%) cases followed by haemorrhagic shock alone in (44%) of the cases. Cardiac tamponade was the cause of death in 3 (2.40%) cases and peritonitis combined with head injury in 4 (3.20%) cases. Peritonitis alone was the cause of death in 2 (1.60%) of the cases with intestinal lacerations (Table - 5).

It was observed that in victims with low ISS (21-30 and 31-40 ISS score ranges) survival was more as compared to the victims with high ISS (51-60, 61-70 and 71-75 ISS score ranges). The spot-death victims had a mean ISS score of 61.73 and cases who died within 1 hour showed 48.33 as the mean ISS. Mean ISS was low in those victims who survived more than 1 week i.e.27.50 (Table- 6).

DISCUSSION

Males outnumbered females in a ratio of 3.8 : 1, which is in concurrence with the findings observed by several workers[5,6,7,8]. This male dominance is explainable by the fact that males are more exposed to hazards of roads, industry and violence as they are the working and earning members in majority of families.

In the present study of thoracoabdominal trauma victims, it was observed that the majority of the cases were in the age group of 21-30 years (23.20%) followed by the age group of 31-40 years (22.40%). Similar findings were reported by workers like Chandra J. and Dogra T.D.[5], Chandulal R.[9], Sinha S.N et al.[10]. The large number of cases in this age group can be explained by the fact that this age group is the most active period in life, and young persons in this age group are at the peak of their creativity and have the tendency to take unwarranted risk, thereby subjecting themselves to the danger of accidents and injuries.

In the present series, majority of blunt thoracoabdominal injuries were due to vehicular accidents (86.40%). This finding is also in agreement with those observed by numerous workers[10,11,12,13]. Ameh E.Z. *et* al.[14] also observed that the commonest cause of blunt abdominal trauma was vehicular accident (57%). This could be explained by poor maintenance of roads as well automobiles and gross indiscipline by drivers, unlicensed drivers, over-speeding, reckless driving as well as reckless movement on the roads by pedestrians.

Mason J.K.[15] observed that minor abrasions and bruises to ragged lacerations and degloving injuries in all the pedestrians with face, arms and legs being injured on every occasion. In the present study too, since the majority of the victims (86.40%) were victims of traffic accidents, the external injuries were seen on the face, limbs and thoracoabdominal region even though 12.80% of the victims did not show any associated injury of the region.

On the spot emergency medical care and rapid transportation from the incident site to the hospital is emphasized by the fact that 6 of out these 15 cases died within hour were declared brought dead since it took almost 45 to 50 minutes for them to reach the hospital from the site of the incident. However, Daly K.E. and Thomas P.R.[16] observed that majority of deaths due to multiple injuries (70%) occurred before arrival at a hospital.

The finding of Haemorrhagic shock combined with head injury as the commonest cause of death (48.80%) in the present study is in agreement with the findings observed by Brainard B.J. *et* al.[17]and Segers P. *et* al.[8]. This could be explained by the fact that thoracoabdominal trauma is often accompanied with head injuries.

Cases of peritonitis combined with trivial head injury died within 1 to 7 days of hospitalization while they were being treated for head injuries. But the presence of intestinal and stomach injuries were overlooked as it was associated with the head injury. Moreover, the possibility of any intraabdominal injury was sidelined since there were no associated external injuries of the region in 2 of these 4 cases. Similar observation was made by Daffner R.H. *et* al.[21].

Only 2 (22.22%) cases died of peritonitis following intestinal injuries. In these two cases, no surgical intervention was made and they could have been saved by a prompt treatment. Sahdev P. *et* al.[19] also opined that out of the 177 autopsies of road traffic accidents, 23% of the deaths were preventable. Similar observations were made by Nikolic S. *et* al.[4].

Anderson S. *et* al.[22] analysed 390 trauma cases (95% Blunt) with ISS (Injury Severity Score) greater than 12 and identified 61 missed injuries (Abbreviated Injury Scale, AIS>1) in 54 victims (13.8%) of which three were abdominal injuries. Further Brainard B.J. *et* al.[17] observed an average injury severity score (ISS) of 20.5 among all the cases. It was observed in the present study that the spot death victims had a mean ISS of 61.73. The victims who survived for less than 1 hour had a mean ISS of 48.33, while mean ISS for those victims who survived for more than 1 week was 27.50, thereby showing that victims with low ISS survived longer than victims with higher ISS values. This is in agreement with the findings observed by Nikolic S. *et* al.[4]. These variations in survival period with ISS can be of value in the management of thoracoabdominal trauma cases.

CONCLUSION

Thoracoabdominal organ injuries may occur without any external injury in the region, so any victim with a history of forceful impact on the area without any visible external injury should be promptly and thoroughly examined to find out any serious damage in the internal organs. A timely diagnosis and surgical treatment would help in diminishing the morbidity and mortality rates in these cases. All the victims of head injury with coma and developing shock soon after must be considered as having intrathoracic or abdominal injury until confirmed otherwise.

The relationship between ISS and survival period can be of immense value in the management of thoracoabdominal trauma cases. Simultaneously, from the medicolegal point of view, it can also find out whether death was due to trauma; whether it was the consequence or complication of injury; what could be the cause and nature of death; whether it was preventable; whether there were possible malpractice or negligence, *etc*[4].

REFERENCE

1. Mukherjee J.B: Injuries, Forensic Med. and Toxicology; Gulab Vazirani for Arnold Assoc, Delhi, 2nd Edn.1; 1994: 397-409

2. Modi J.P.: Regional injuries, Modi's Medical Jurisprudence and Toxicology; C.A. Franklin, N.M. Tripathi Pvt. Ltd., Bombay, 21st Edn; 1989: 319-327

3. Baker S.P., O'Neill B., Haddon W. Jr. and Long W.B.: The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care.J Trauma. Mar 1974;14(3):187-96,.

4. Nikolic S, Micic J, and Mihailovic Z.: Correlation between survival time and severity of injuries in fatal injuries in traffic accidents, Srp Arh Celok Lek, Nov-Dec 2001;129(11-12):291-5

5. Chandra J. and Dogra T.D.: Pattern of injuries in various road users involved with different vehicles in fatal accidents, J. Police Research and Development, April-June 1978; 26-28

6. Ghosh P.K.: Epidemiological study of the victims of vehicular accidents in Delhi, J-Indian-Med-Assoc., Dec.1992; 90 (12): 309-312

7. Luby S., Hassan I., Jahangir N., Rizvi N., Farooqi M., Ubaid S., Sadruddin S.: Road traffic injuries in Karachi: the disproportionate role of buses and trucks, Southeast Asian J Trop Med Public Health, Jun1997; 28(2):395-8

8. Segers P., Van Schil P., Jorens P., Van Den Brande F.: Thoracic trauma: an analysis of 187 patients, Acta Chir Belg., Nov-Dec 2001;101(6):277-82

9. Chandulal R: Fatal road accidents, J.Police Research and Development, July-Sep1971;17-19

10. Sinha S.N., Sengupta S.K.and Purohit R.C.: A five year review of deaths following trauma, P N G Med J, Dec 1981; 24(4):222-8

11. Chandra J., Dogra T.D. and Dikshit P.C.: Pattern of Cranio-intracranial injuries in fatal vehicular accidents in Delhi (1966-76), Med-Sci-Law,1979;19 (3):186-194

12. Sharma O.P.: Traumatic diaphragmatic rupture-not an uncommon entity - personal experience with collective review of the 1980s, J-Trauma, May 1989; 29 (5): 678-682

13. Banerjee K.K., Aggarwal B.B.L. and Kohli A.: Study of thoraco-abdominal injuries in fatal road traffic accidents in North east Delhi, Jour.For.Med.Tox., Jan-Jun 1997; 14(1): 40-43

14. Ameh E.A., Chirdan L.B. and Nmadu P.T.: Blunt abdominal trauma in children: epidemiology, management, and management problems in a developing country, Pediatr Surg Int, 2000;16(7):505-9

15. Mason J.K.: Injuries and death in road traffic accidents and Pedestrian injuries and death, The pathology of trauma; Edward Arnold, Hodder & Stoughton Ltd., London, 2nd Edn, 1993; 1-29.

16. Daly K.E. and Thomas P.R.: Trauma deaths in the South West Thames region, Injury, 1992; 23 (6): 393-396

17. Brainard B.J., Slauterbeck J., Benjamin J.B., Hagaman R.M. and Higie S.: Injury profiles in pedestrian motor vehicle trauma, Ann-Emerg-Med., Aug 1989; 18 (8): 881-883

18. Barashkov G.A. and Gubar L.N.: Characteristics and structure of automobile injuries, Vestn-Khir., May 1978; 120 (5): 73-78

19. Sahdev P., Lacqua M.T., Singh B. and Dogra T.D.: Road traffic fatalities, Acci-Anal-Prev., Jun 1994; 26 (3) : 377-384

20. Brathwaite C.E, Rodriguez A., Turney S.Z., Dunham C.M., and Cowley R.: Blunt traumatic cardiac rupture-A 5-year experience, Ann Surg.,Dec1990; 212(6):701-4

21. Daffner R.H., Debb Z.L., Lupetin A.R. and Rothfus W.E.: Patterns of high speed impact injuries in motor vehicle occupants, J-Trauma,Apr1988; 28 (4): 498-501

22. Anderson S., William M.C. and Lane P.: Unidentified injuries in multiple trauma -a second look, J-Trauma, Jul 1995; 39(1): 161

TABLE - 1. SHOWING AGE AND SEX WISE DISTRIBUTION OF CASES

Sl.No.	Age Group in Years	Males		Females		Total	P.C (%)
		No.	P.C(%)	No.	P.C(%)		
1.	0-10	2	1.60	4	3.20	6	4.80
2.	11-20	13	10.40	4	3.20	17	13.60
3.	21-30	26	20.80	3	2.40	29	23.20
4.	31-40	23	18.40	5	4.00	28	22.40
5.	41-50	15	12.00	6	4.80	21	16.80
6.	51-60	13	10.40	3	2.40	16	12.80
7.	61-70	6	4.80	0	0	6	4.80
8.	71-80	1	0.80	1	0.80	2	1.60
	Total	99	79.20	26	20.80	125	100.00

TABLE - 2. SHOWING THE TYPES OF BLUNT THORACOABDOMINAL TRAUMA

Sl. No.	Type Of Trauma	No.	P.C (%)
1.	Vehicular Accident	108	86.40
2.	Assault by Blunt Weapon	10	8.00
3.	Hit by fall of heavy objects (boulders)	3	2.40
4.	Fall from Height	3	2.40
5.	Kick by Horse	1	0.80
	Total	125	100.00

TABLE-3. SHOWING THE RELATIONSHIP BETWEEN BLUNT THORACOABDOMINAL ORGAN INJURIES AND ASSOCIATED EXTERNAL INJURIES IN THE REGION

Sl.No.	Thoracoabdominal organ injuries	No.	P.C (%)
1.	With associated external injuries	109	87.20
2.	Without associated external injuries	16	12.80
	Total	125	100.00

TABLE - 4. SHOWING SURVIVAL PERIOD OF VICTIMS

		SURVIVAL PERIOD							
	Spot	< 1 Hr	>1-2 Hrs	>2- 6 Hrs	> 6-12 Hrs	>12-24 Hrs	>1- 7 Days	>1- 2 Wks	Total
No. of cases	59	15	13	19	5	3	9	2	125
P.C.	47.20	12.00	10.04	15.20	4.00	2.40	7.20	1.60	100.00

TABLE – 5 . SHOWING THE CAUSES OF DEATH IN 125 CASES OF BLUNT THORACOABDOMINAL TRAUMA

Sl. No.	Causes of death	No.	P.C
1	Haemorrhagic sock + Head Injury	61	48.80
2	Haemorrhagic shock	55	44.00
3	Cardiac tamponade	3	2.40
4	Peritonitis + Head injury	4	3.20
5	Peritonitis	2	1.60

TABLE-6. SHOWING SURVIVAL PERIOD IN RELATION TO INJURY SEVERITY SCORE

Sl. No.		ISS SURVIVAL PERIOD							
		Spot	< 1 Hr	>1-2 Hrs	>2-6 Hrs	>6-12 Hrs	>12-24 Hrs	>1-7 Days	>1-2 Wks
1.	0-10	0	0	0	0	0	0	1	0
2.	11-20	1	0	1	1	0	0	1	0
3.	21-30	3	2	1	4	1	1	4	2
4.	31-40	7	1	2	8	4	2	0	0
5.	41-50	4	7	8	5	0	0	3	0
6.	51-60	8	2	1	1	0	0	0	0
7.	61-70	4	0	0	0	0	0	0	0
8.	71-80	32	3	0	0	0	0	0	0
	Total	59	15	13	19	5	3	9	2
	Mean ISS	61.73	48.33	39.38	35.42	33.60	32.33	30.22	27.50

Traumatic Dislocation of Implanted Joint

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ABSTRACT

In the section 320 IPC, it is clearly mentioned that dislocation of the joint is a grievous injury. Nowhere anything is mentioned about the dislocation of the implanted joint. Probably when the IPC was framed, joints were not implanted. Nobody had the forethought that joint will be implanted and a section will be needed to describe the displacements of implanted joints. Dislocation of such a joint, during a quarrel is a rare occurrence.

KEY WORDS: Grievous injury, implanted joint

INTRODUCTION

Due to accidents or during quarrels a displacement of joints is not an uncommon occurrence. The surgeries for joint replacements are increasing day by day. In the population particularly middle aged and old persons, we are now having people with implanted joints. Such people are as prone to meet accidents and assaults as any member of the society is.

CASE REPORT

A case reported in the emergency of the Rajindra Hospital with injuries by pointed weapons and a blunt weapon. He was a male aged 50 years. He was coming back from the court after giving the evidence when his opponents attacked him on the way back to his home. In addition to three other injuries, patient had pain in the right hip joint. He was not able to move the right lower limb. He never gave the history of the implanted hip joint. When radiological examination of the hip joint was carried out, to know the cause of pain and inability to move the right lower limb after assault, displacement of the right implanted hip joint was detected.

The injury was declared as grievous in nature, keeping in mind the clause-7 of section 320 IPC as dislocation of joint cannot be declared as simple in nature[1]. Police registered a case under section 325 IPC. Still this case has not undergone the scrutiny of the court. This case is being presented, as being a rare and unusual case, where IPC is silent.

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Professor & Head
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DISCUSSION

With the changing trends of life support, new problems are bound to arise in the treatment and care of the persons. As other branches of medicine need updating of the facilities and knowledge, so is the case with forensic medicine.

New problems do crop up, where nothing is so clear-cut. Courts usually clarify such things, when these cases are taken up. When a rare case like this comes up first, it has to be handled by forensic medicine experts. Police will always insist for an opinion, even if there is no clear-cut opinion anywhere in the literature. Keeping in mind, that culprit is responsible for the injuries to the victim as he is and holding him responsible for the damage to the victim, this opinion was given .

It was a peculiar case and literature reviewed to find out the nature of injury in any similar previous case, did not reveal any such medicolegal case. However, according to Victorian WorkCover Authority's 'The Nature of Injury / Disease Classification System for Victoria' damage to artificial aids like broken dentures, damaged artificial limbs, damaged prosthesis, etc. are included in classification of injuries that qualify for workers' compensation claim[2].

REFERENCE

1. Indian Penal Code (Act 45 of 1860). Criminal Manual. 15th Ed. Lucknow: Eastern Book Company. 2003. p.74

Victorian WorkCover Authority. The Nature of Injury / Disease Classification System for Victoria. Version 1.1 Australia. November 2003.

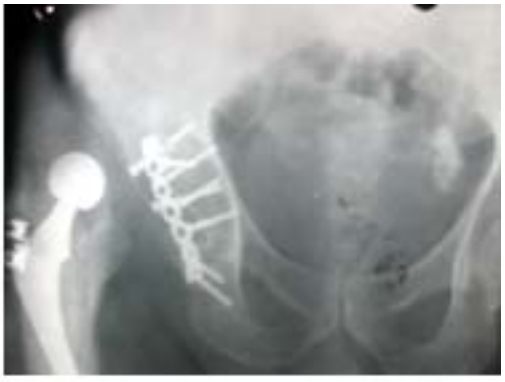

Figure 1: Photograph of X-Ray showing the dislocation of implanted hip joint

Ambiguity in Juvenile Act Cleared by Apex Court

Bhardwaj DN, Additional Professor, Forensic Medicine, AIIMS, New Delhi

Key word:- Juvenile, Supreme Court, Offence

SUMMARY

Prior to year 2000, children below 16 yrs were tried in Juvenile court for any criminal offence. However, after passing the Juvenile Justice Act 2000, the age was raised to 18 yrs. Hence, children below 18 yrs are being tried nowadays in Juvenile Courts renamed as Juvenile Justice Boards. It has one Principal Magistrate of rank equitant to metropolitan magistrate and two members from Non Govt. Organization (NGO). However, still there was doubts over definition of Juvenile. Apex Court has ruled ***that the date of Committing on offence would determine whether an accused was entitled to Juvenile Justice Act benefit or otherwise.***

An accused below 18 yrs of age is tried in Juvenile Justice Board and not sent in ordinary jail but reformatory homes.

Juvenile is also entitled to leniency of adjudged guilty and sentenced for a crime. ***The court further ruled that all those between 16-18 yrs age at the time of commission of crime prior to 2000 would not be tried along with normal criminals but in Juvenile Justice Boards.***

Conclusion

The Judgement has given relief to many alleged youth criminals who were treated at par with other criminals including hardened criminals and facing charges prior to 2000. Many of then were in jails also because of lack of proper guidelines

REFERENCE

Time news network dated . 3.02.2005.

Bhopal is still Drinking Poison even After 20 Years

Key words: Toxic, poison, tragedy

Summary

More than 4000 people had died due to leakage of MIC (Methyl Iso Cynate) in Union Carbide factory in Bhopal in 1984. There were lot of disabilities also. However, thousands of tones of toxic waste are still lying stored inadequately thus affecting water supply of town.

The team of BBC took a sample of drinking water from near the site and found pesticide level 500 times higher than the maximum limits recommended by WHO as claimed in report. This is a matter of concern to health of people. There is need for safe practices for storage of pesticides.

REFERENCE

Time news dated : 15.11.2004

Life sentence likely to become a life time affair

ABSTRACT :

The Union Government is closely studying a proposal to make life imprisonment last a full life time instead of 14 years as is the practice now, The idea to change this punishment to make it stringent in various crimes like rape, murder and other organised crimes.

The suggestion has come from Delhi Chief Minister Sheila Dikshit and being supported by many other higher rate of conviction. The idea of proposal is to chief ministers. There is also proposal to provide protection to the witness as many times they are threatened to turn hostile in court of law thus causing embarrassment to the state.

REFERENCE

Times News Network dated 23-05-2005.

Spectrum of Organophoshorous Poisoning in Manipal

***Palimar Vikram** MD, *****Arun M** MD, DNB,
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ABSTRACT

Organophosphorous compounds are extensively used in agriculture as insecticides. 153 cases of organophosphorous poisoning presented to Kasturba Hospital Manipal, Karnataka between January 2001 to December 2002 formed the material for the study. Most common reason for poisoning was suicide (98.7%). 21-30years was the commonest age group(36.6%) and 75.1% were males. In 65.4% poison was consumed during day time. In 31.4% of the cases poison was consumed in the summer months. Methyl Parathion was the most common compound consumed. Respiratory failure was the most common complication (43.1%). Mortality was 26.2% and 30% of the victims died with in 24 hours.

Key Words: Insecticides, organophosphorous, poisoning, suicide

INTRODUCTION

Organophosphorous insecticides are used extensively in horticulture and agriculture. Because of its easy availability these are a significant cause of morbidity and mortality in developing countries including India. As reported by the World Health Organisation, 3 million people consume these compounds resulting in 40,000 deaths annually.[1] Because of the easy availability and rapidity of their lethal action even in smaller doses, they are one of the popular suicidal poisons. [2]

In India these compounds have been imported since 1951, but few people knew the nature of these compounds as a virulent poison till the occurrence of Kerala food poisoning tragedy in 1958, which took a toll of hundred odd lives due to inadvertent stocking of food stuff and parathion packages in the same hold where the parathion containers leaked and contaminated the gunny bags containing the food stuff.[3]

Developed countries have well established poison information centres for prevention and control of the poisoning but in the developing countries like India, the picture is totally different where there is inadequacy of infrastructure.

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An attempt has been made in the present study to find the pattern of organophosphorous poisoning in this part of the world and thereby to suggest preventive measures which will considerably reduce the morbidity and mortality associated with these compounds.

MATERIAL AND METHODS

During a two year period between 1st January 2001 and 31st December 2002, one hundred and fifty three cases of organophosphorous poisoning were admitted at Kasturba Hospital, Manipal, which is a tertiary care teaching hospital located at coastal belt of Karnataka State of South India. The information regarding manner of poisoning, age, sex, religion, marital status, occupation, diurnal and seasonal variation, nature of the compound consumed, complications, outcome and the duration of survival were noted from the hospital files obtained from Medical Records Department of Kasturba Hospital, Manipal. The data obtained were tabulated and analysed.

RESULTS

Out of the one hundred and fifty three cases of organophosphorous poisoning the manner of death was suicidal in 151 cases (98.7%) and accidental in 2 cases (1.3%). Fifty-six victims were aged between 21-30 years (Fig.1). Males accounted for 115 cases (75.1%) and females formed 38 cases (24.9%). Majority of the victims were Hindus (94.1%) followed by Christians and Muslims (3.3% and 2.6% respectively). Ninety four victims (61.4%) were married and 59 victims (38.6%) were unmarried. Agriculture was the most common occupation of the victims (Table-1). Consumption of the poison was more during day time (6am- 6pm) (Table-2). In 31.4% of the cases poison was consumed during the summer months (Table-3). Methyl parathion was the most common compound consumed (Table-4). Respiratory failure was seen in 43.1% (Table-5). 113 victims (73.8%) survived and 40 patients (26.2%) expired. One third of the victims survived for a period of less than a day after consuming poison (Table 6).

DISCUSSION

In the present study, it was observed that in majority of the cases the poisoning was suicidal in nature which is consistent with observations made by other workers[4-9]. The studies from Australia[10] and Almeria[11] observed accidental poisoning to be more common. In Costa Rica[12] it was observed that occupational poisoning was common in men, attempted suicide was common in women and accidental poisoning was more common in children. In our series 2 cases were accidental and no homicidal cases were seen.

Of the 2 accidental cases, one was due to consumption of the poison, confusing it with some medication and in the other case the poisoning occurred while the person was spraying the field with organophosphates. Suicidal poisoning is very common since these compounds are easily available and whenever there is an impulse to commit suicide they are ready in hand. 21-30 years was the commonest age group affected which is similar to other works[4,5,9] . In Krakow[8] 30-39 years was the commonest age group to be affected. This age group in all probability is more vulnerable to the various emotional conflicts which occur during this phase of life. Males were the common victims in the present study. This finding is in concurrence to the findings of other workers.[5,9,11] However in Nepal[4] the victims were predominately females. Deliberate self ingestion is more common in men. This could be attributed to the fact that they are exposed to more stresses of life than women and perhaps they are less efficient in managing the same when compared to women. Majority of the victims were Hindus. This is because in this part of the world, the major chunck of the population follow Hinduism as their religion and hence the occurrence of the poisoning is high. A large number of the victims were married which is in contrast to the work in Nepal.[4] Married people probably experience comparatively more stress and strains of life than their unmarried counterparts. Agriculturists were the common people to be affected by poisoning with these compounds. In Nepal[4], the students were commonly involved where as in Almeria[11] greenhouse workers predominated. Self poisoning is common in agriculturists due to failure of crops coupled with easy availability of the poison in their household and whenever there is a precipitating factor one might be tempted to end one's own life. In this series, the majority of the subjects consumed poison during day time. It is likely the person would be exposed to more stress during the day time and the stress will be in its peak during the day. In the present study, the poisoning commonly occurred during the summer months. This is mostly because during the summer months the agricultural income would be nil and hence there would be an enhanced risk in committing suicide in those months as he is unable to meet all the needs of his dependents. Methyl parathion was the commonest compound of organophosphate consumed in 37.9% of the cases and the compound consumed was unknown in 39.8% of the cases. This is because the exact history regarding the compound could not be ascertained either from the relatives or from the victim himself.

Methyl Parathion was the commonest compound in Nepal[4]. Dimethoate was the commonest compound in other study.[9] Respiratory failure was the most common complication observed which is consistent with the findings of other worker[5]. It results from a combination of respiratory muscle weakness, central respiratory depression, increased bronchial secretions, bronchospasm and pulmonary oedema[5].

Pancreatitis a rare complication was observed in one case. Three cases of pancreatitis was observed by other worker[11]. The mechanism of pancreatitis is yet to be ascertained. Present study observed a mortality of 26.2%. The literature mentions the mortality to be between 2.9% to 32.6%. [4,7-10,12-16] One third of the patients survived for a period of less than 24 hours after consuming the poison. In Srilanka,[9] 18.7% of the patients died within 3 hours of admission. This emphasizes the fact that the initial 24 hours is the crucial period for the outcome and stresses the need for decontamination of the poison at the primary care level before being referred to a tertiary care hospital for further management.

Preventive measures should be considered at all levels starting from manufacturing of these compounds to their safe disposal. During transport and storage, strict guidelines should be followed in preventing contamination of food, clothings and body surfaces of the handlers. Health education is essential in relation to the toxic effects of these compounds. Effective legislation regulating the sale of these insecticides is the need of the hour in minimizing the occurrence of poisoning.

The personnel involved in primary health care delivery systems should be adequately trained in decontamination of the poison and other first aid measures. The goal of safe and effective use of insecticides is best achieved by an integrated and interdisciplinary application of the skills as well as the knowledge of farming industry, chemistry and medicine.

REFERENCES

1. Sqn Ldr Piplani S, Sqn Ldr Attanda, Sqn Ldr Aggarwal R, Sqn Ldr Gupta BK, Wg Cdr Mishra SC, Lt Col Ray P, Maj Gen Gupta IDG. Organophosphorous poisoning with intermediate syndrome. Med J Armed Forces Inst 2002; 58: 81-3.

2. Davies JE. Changing profile of pesticide poisoning. New Eng J Med 1987; 316: 807-8.

3. Subrahmanyam BV. In. Modi's Medical Jurisprudence and Toxicology, 22nd edn. Butterworths India: New Delhi, 1999: 85 (Toxicology section).

4. Karki P, Hansdak SG, Bhandari S, Shukla A, Koirala S. A clinicoepidemiological study of organophosphorous poisoning at a rural based teaching hospital in East Nepal. Trop Doct 2001; 31(1): 32-4.

5. Goel A, Joseph S, Dutta TK. Organophosphate poisoning: Predicting the need for ventillatory support. J Assoc Physcians Ind 1998; 46(9): 786-90.

6. Karalliedde L, Senanayake N. Organophosphorous insecticide poisoning. Br J Anaesth 1989; 63: 736-50.

7. Abebe M. Organophosphate pesticide poisoning in 50 Ethopian patients. Ethiop Med J 1991; 29(3): 109-18.

8. Kamenczak A, Jasinska-Kolawak, Targosz D, Szkolnicka B, Sancewicz-Pach K. Acute pesticides poisoning in the Krakow. Department of Clinical Toxicology in 1986-1995. Przegl Lek 1997; 54(10): 671-6.

9. Karalliedde L, Senanayake N. Acute organopshophorous poisoning in Srilanka. Forens Sci Inter 1988; 36: 97-100.

10. Emerson GM, Gray NM, Jelinek GA, Mountain D, Mead HJ. Organophosphate poisoning in Perth, Western Australia, 1987-1996. J Emerg Med 1999; 17(2): 273-7.

11. Martin Rubi JC, Yelamos Rodriguez F, Laynez Bretones F, Cordoba Escamez J, Diez Garcia F, Lardelli Claret A, Blanco Coronado JL, Vincente Rull JR. Poisoning caused by organophosphate insecticides. Study of 506 cases. Rev Clin Esp 1996; 196(3): 145-9 (Article in Spanish).

12. Ingianna J, Herrero R, Albertazzi C. Comparative study of cases of poisoning by organophosphorous insecticides in various regions of Costa Rica. Rev Biol Trop 1983; 31(1): 139-44 (Article in Spanish).

13. Singh S, Wig N, Chaudhary D, Sood NK, Sharma BK. Changing pattern of acute poisoning in adults: Experience of a large North-West Indian Hospital (1970-1989). J Assoc Physic Ind 1997; 45(3): 194-7.

14. Sinha US, Kapoor AK, Agnihotri AK, Srivastava PC. A profile of poisoning cases admitted in SRN hospital, Allahabad with special reference to aluminium phosphide poisoning. J Forens Med Toxicol 1999; 16(1): 40-3.

15. Vaswani V, Patil VD. Spectrum of childhood poisoning: A Belgaum experience. J Forens Med Toxicol 1998; 15(1): 50-2.

16. Aggarwal P, Handa R, Wali JP. Common poisonings in India. J Foren Med Toxicol; 1998; 15(1): 73-9.

Table-1: Occupation of the Victim.

Occupation	No of Cases (n=153)	Percentage
Farmer	45	29.4
Manual Labourer	22	14.4
Housewife	22	14.4
Student	13	8.5
Miscellaneous#	12	7.8
Unknown	39	25.5

= Hotel worker, typist, clerk, auto driver.

Table-2. Diurnal Variation of Consumption

Time Of Poisoning	No of Cases (n=153)	Percentage
Day (6AM-6PM)	100	65.4
Night (6PM-6AM)	41	26.8
Unknown	12	7.8

Table-3. Seasonal variation of consumption.

Seasonal Variation	No of cases (n=153)	Percentage
Summer (Mar-May)	48	31.4
Rainy (Jun-Aug)	34	22.2
Spring (Sep-Nov)	32	20.9
Winter (Dec-Feb)	39	25.5

Table-4. Type of organophosphorous compound consumed.

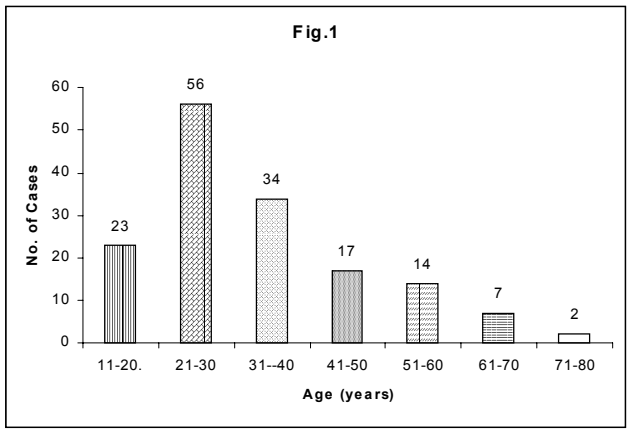
Compound Consumed	No of cases (n=153)	Percentage
Methyl parathion	58	37.9
Malathion	2	1.3
Quinalphos	7	4.6
Monocrotophos	11	7.3
Chlorpyriphos	2	1.3
Dimethoate	7	4.6
Ethion	2	1.3
Phosphamidon	1	0.7
Ediphenphos	2	1.3
Unknown	61	39.8

Table 5: Complications Observed

Complications	No. of Cases	Percentage
Respiratory Failure	66	43.1
Cardiac Arrest	46	30.1
Pneumonia	25	16.3
Septicaemia	4	2.6
ARDS	3	2
Acute Renal Failure	2	1.3
Pancreatitis	1	0.7

Table-6. Survival period in fatal cases.

Duration of Survival	No of Cases (n=40)	Percentage
< 24 hrs	12	30
1 – 3 days	10	25
3 – 7 days	10	25
> 7 days	8	20



Legend:

Fig1: Age profile of the Victims.

LEGAL ASPECTS OF PATIENT CARE

"Legal Aspects of Patient Care" is written with the aim of educating **medical practitioners** and para-medical staff about **patient's rights, current laws** relating to **medical practice** and how to **face menace of malpractice suits**. The book has several chapters relating to **doctor-patient relationship, consent, medical ethics, role of medical council, medical records,** and most importantly, **medical negligence.** The emphasis has been laid on defining **medico-legal aspects of injury in general, head injury** and **regional injuries.** The common **medico-legal problems/ mishaps** that occur in major medical practices like **anesthesia, gynecology** and **obstetrics practice, surgical practice** and **practice by physicians** have also been dealt with. A chapter on **professional indemnity policies** offered by the insurance complains also included. A major emphasis of this book is how to face **common medico-legal problems** like refusal of treatment, arrival of dead body and non-payment of fee etc. Guidelines on how to **avoid medical malpractice suit/medical negligence** are prescribed. It is hoped that this book will help the persons working in medical practice to discharge safe medical practice.

ABOUT THE AUTHOR

Dr. R.K. Sharma completed his MBBS and MD from the prestigious All India Institute of Medical Sciences, New Delhi. After completing his studies, he joined as a faculty member in the Department of Forensic Medicine at All India Institute of Medical Sciences, New Delhi and is presently working as an Additional Professor there. He has published a large number of scientific papers in national and International journals. He has also supervised many research projects of national importance. He has toured extensively all over India and abroad to gain vast medico-legal experience. He is an examiner for many universities in India. He is an office bearer of many national and international organizations. His main zeal is education of medical practitioners about consumer rights and legal aspects of patient care so that they can discharge safe medical practice. He is a strong advocate of patient's rights and seeks improvement in patient care in India. He has delivered many talks regarding this at the Indian Medical Association, Punjab Health System Corporation, Central Health Scheme and various hospitals and nursing homes. He is the Editor-in-Chief of the international journal **"Medico-Legal Update"**.

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CONFERENCE CALENDER

2005, August 10-12,
The Florida Association of Medical Examiners 2005 Conference- 21st Century CSI Technology and Death Investigation in Sonesta Beach Resort, Key Biscayne, FL, USA
Contact: E-mail: housekm@pathology.ufl.edu
Website: http://maples-center.ufl.edu

2005 August 15-19,
International Congress on Medical law in Korea Convention & Exhibition Center, Seoul, Korea
Contact: E-mail:jcpark@sharptravel.co.kr
Website: www.icml2005.com

August 21-26, 2005
17th Meeting International Association Forensic Sciences in Hong Kong
Contact: E-mail: iafs2005@govtlab.gov.hk,
Web: www.iafs2005.com

2005. August 29-September 2,
The International Association of Forensic Toxicologists (TIAFT) 43rd International meeting in Lotte Hotel, School, Seoul, Korea
Contact: E-*mail: hschung7@nisi.go.kr*
Website: www.tiaft2005.org

2005, September 13-17,
21st Congress of the International Society for Forensic Genetics
Contact: E-mail: Isfg2005@ipatimup.pt

2005, September 14-16
National Organisation for the Treatment of Abusers. 15th annual Conference 2005 in Dublin
Contact: www.nota.co.uk

2005 September 15-16,
MINNESOTA Division of the International Association for Identification 2005 Fall Educational Conference
Contact: E-mail :peowally@charter.net, jlg6975@comcast.net; website: www.minnesotadivisioniai.org

2005, September 19-23,
Bloodstain Evidence Institute
Contact: E-mail: forensiclab@stny.rr.com.

September 19-24, 2005, Hamburg, Germany
6th International symposium on Advances in legal Medicine (6th ISALM)
Contact: E-mail: 6.isalm@rechtsmed-hh.de
Web: www.isalm2005.rechtsmed-hh.de

2005, September 26-29,
6th International Symposium on Human Identification
Contact: E-mail: carol.bingham@promega.com
Website: www.promega.com./geneticsympl6

2005, October 3-7,
MIDWESTERN Association of Forensic Scientists (MAFS) Annual Fall Meeting
Contact: E-mail: bhampton@saintcharlescounty.org;
Website: http://mafs.net

2005, October 4-7,
The Association of Forensic Quality Assurance managers 2005 Meeting
Contact: E-mail: lschultz@indygov.org
Website: www.afqam.org

2005, October 5-7,
International Association of Bloodstain Pattern Analysts Annual Training Conference
Contact: Website; www.iabpa.org

2005, October 11-15,
California Association of Criminalists Fall 2005 Seminar
Contact: E-mail: v9273@lapd.lacity.org
Website: www.cacnews.org

2005, October 18-22,
52nd Annual meeting of the Canadian Society of forensic Science
Contact: 780-451-7404: Fax: + 780-495-6961

2005, NOVEMBER 4-6,
Sexual Offences
Contact:
E-mail; Tracey@forensic-science-society.org.uk

2005, November 10-12,
Justice for All: A national Symposium on the Most Significant Criminal justice Legislation of Our Times
Contact: E-mail: justiceforall@duq.edu.

November 15-19, 2005, Toronto, Canada
American Society of Criminology Meeting
Contact: Web: www.asc41.com

February 11-17, 2006
95th Annual Meeting United /states and Canadian Academy of Pathology at Orleans, USA
Contact: E-mail: iap@uscap.org
Web: www.uscap.org

February 28 – March 4, 2006
43rd Annual Meeting Academy of Criminal Justice Sciences at Baltimore, USA
Contact: E-mail: lmonaco@acjs.org
Web: www.acjs.org

2006, March 2-5,
ACLM 2006 45th Annual Conference at Harrah's Las Vegas Hotel & Casino, Las Vegas, Nevada

2006, April 2-7,
18th International Symposium on the Forensic Sciences Classroom to Courtroom at Esplanade Hotel, Freemantle, Western Australia
Contact: Dr. Clive Cooke, Chair, Organising Committee.

2006, May 15-16,
International Symposium on Craniofacial Reconstruction
Contact: E-mail: guy.willems@med.kuleuven.ac.be
Web: www.mfo.be

2006, May 17-19,
Annual conference of the Association of forensic physicians
Contact: E-mail: admin@afpweb.orq.uk
oragdott@doctors.org.uk

2006, May 17-20,
International Symposium on Forensic Odontology
Contact: E-mail: guy.willems@med.kuleuven.ac.be
Web: www.mfo.be

2006, june 14,
EAFS 2006 4th European Academy of Forensic Science meeting
Contact: E-mail: erkki.sippola@krp.poliisi.fi

2006, August 23-26,
20th Congress of International Academy of Lkegal medicine in budapest
Contact: Web: www.motesz.hu

2006, October 16-18,
The 20th World Congress of the International Traffic medicine Association (ITMA)
Contact; E-mail: MorrisO@vifm.org;
Web:www.trafficmedicine.org

November 1-4, 2006
American society of Criminology Meeting at Los Angeles, USA
Contact: Web: www.asc41.com

2007, May 16-20,
9th Cross Channel Conference 2007
Contact; E-mail: ianwall@doctors.org.uk
www.palacetorquary.co.uk

Erratum

Article entitled **'Geriatric Poisoning Fatalities- A Manipal Perspective'** published in the **Medico-Legal Update**, Jan-March 2005, Vol.5, No.1, Pg: 7 & 9.

1. In page no. 7, the address of author Dr. Nagesh K.R, Assistant Professor, Forensic Medicine Kasturba Medical College, Manipal should be read as **Dr. Nagesh K.R, Assistant Professor, Forensic Medicine, Kasturba Medical College, Mangalore.**

2. In page no. 9, **the references are correlated and should be read continuously as 1 to 20** instead of references No.1-9, 1-7 and 1-4.

Editor-in-Chief

Marvelous Tools of Identification–Bite Marks

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**** Professor, Department of Forensic Science, Punjabi University, Patiala
***** Professor & Head, Department of Prosthetics, Govt. Dental College, Patiala

ABSTRACT

Forensic Odontology is a newer developing branch of Forensic Medicine in which knowledge of dental expertise is utilized for solving many forensic problems. Of course the study of bite marks, still in infancy, is now getting due importance which was long overdue. Bite marks of one person never matches with that of another person. In this study, proper dental casts of volunteers were prepared and were matched with their bite marks. In this study, identification of an individual was done through bite mark, by comparison techniques using transparent overlays from study dental cast models and life size photograph of bite marks in different materials like skin, cheese, fruits or clay. Interesting conclusions of this study are presented in this paper.

KEY WORDS: Bite Marks, Identification, Odontology

INTRODUCTION

Interestingly the oldest investigation based on Forensic Sciences was Bite marks. They were the oldest evidence used, even by God when He came to know about the 'first sin' by looking at the bite marks on the apple[1].

The second half of the last century saw a great deal of work on bite marks all over the world, a positive step. The forensic community used bite marks as an evidence to punish the guilty in the court of law. Bite marks have come a long way, and like fingerprint, it has been recognized in the court of law.

In 1971, DeVore used ink models to place marks on living volunteers and cadavers. Photographs of the marks were taken in several body positions. Skin from the cadavers bearing the ink was excised. He concluded that there is a large margin of error in using bite marks photographs and unsecured excised skin[2].

Incidentally, in 1906 a burglar was convicted at the Cumberland assizes from the marks of his teeth in cheese at the scene of crime[3].

Biting is used by human being as both offence and defense. The bite of a suckling baby whose tooth has erupted causes considerable excoriation and pain. Bites of five year olds are definitely painful[4]. All the places that can be kissed are also the places that can be bitten, except upper lip, the interior of mouth and the eyes.

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There are the words from KamaSutra. These bites are found in adolescent and emotionally active persons[4]. Another category of bite marks is caused by habitual or nervous habit of chewing for example a Pencil, Key, Pen or Pipe[5]. No bite marks can be made with full dentures[6].

A forensic odontologist collects, preserves, evaluates and interprets the bite mark evidence. In an attempt to make, bite mark evidence more acceptable, the American Board of Forensic Odontology (ABFO) is currently trying to use standards, though not been universally accepted.

Criminals often go scot-free, due to the lack of knowledge and awareness on the part of investigating authorities on bite marks. In cases where bite marks are present, namely sexual assaults, fights, child abuse, they form the basis and cornerstone for investigations provided the investigation agency utilize its full potential. Evaluation and interpretation of evidence is most difficult aspect as there are only few experts with the training and experience to carry out these complex procedures[5].

Mac Donald defined a bite mark as a mark made by the teeth either alone or in combination with other mouth parts[7]. Bite marks are found on human body or food or on materials such as wooden cabinet, bottle cap, cigar and cigarette holders, pipes and musical instrument mouth pieces[4,5]. Usually bites are the result of sexual or physical assault by an adult on a child, rape or attempted rape, quarrels and fights among men[5]. Not all human bites are associated with homicides, sexual assaults or child abuse cases. They may be produced due to self-defense when defending against aggressive animal or self inflicted as tongue bite in epileptic fits or fall from heights[8].

Mac Donald's etiological classification[5] of bite marks is as follows:

a) Tooth pressure marks caused by incisial edge of anterior teeth

b) Tongue pressure marks seen as impression of the palatal surface

c) Tooth scrapes mark may be scratches and abrasions that can indicate irregularities in the teeth such as incisial fractures, restorations or attrition

d) Complex marks are a combination of all the above, occasionally complicated by multiple bites.

Gustafson's clinical classification[9] of bite marks is as follows:

a) Sadistic or sexual bite is usually made slowly and is therefore well defined.

b) Aggressive bite is made quickly with force and is caused by scribing across the tissue.

c) Most aggressive bite results in tissue being bitten off usually and involves ears, nose, and nipples.

Injuries of bite marks may be abrasions, bruises or lacerations. Teeth crush the superficial epithelium and leave its imprint, which can be photographed appropriately. It is imperative to take an early photograph as a bite bruise usually disappears in twenty minutes. However, infrared photographs can show deep-seated bleeding[10]. Lacerations having ragged edges are mostly found in cases of animal bite.

Human bite mark is almost circular or oval, whereas animal bite mark is narrowing U-shaped[9]. Not all teeth are involved in a bite and usually consist of three or four upper teeth and one or more lower opposing teeth[11].

Bite marks investigation starts with the examination of wound and if identified as bite mark, to identify the teeth involved, notify any peculiarity of size, shape, position to detail the individual tooth features seen in marks and those seen in tooth of the accused, followed by photography[12].

Further analysis of bite marks is done by superimposition of marked transparency of inked edges of the plaster model of teeth over the bite mark[13,14,15,16]. Presently, scanning electron microscope and computerized axial tomography (CAT) technique is also used to develop precise registration of incisial edge for comparison of bite mark[17].

The principle of identification by bite mark is 'Comparison' of the bite mark with the cast model of teeth and computer has become a very handy instrument to do the same with great accuracy and speed; however, it is the man behind the machine, which actually matters. In the present study, computers and digital cameras are used extensively; however, the actual comparison was done manually.

Almost every developed country has a Forensic Odontology Association where experiments are carried out and the subject is taught.

MATERIAL AND METHODS

100 volunteers were taken, median age being 29 yrs. Age, sex and address of volunteer was noted and a serial number was allotted to each volunteer. Bite marks were taken on three different groups of items:

a) Skin which is a very poor impression material[4]

b) Perishable substances like cheese, fruits and chewing gum which may be usually found at crime scene[5]

c) Non-perishable substances like clay wax which is flexible synthetic material, which reproduces marks well and is dimensionally stable. Some other non-perishable objects that have been bitten include bullet, pipe, pencils and soap[18].

The volunteers were divided into 4 groups of 25 each and each group took bites on skin, clay-wax, fruits or cheese. For identification, standard method of comparison technique by transparent overlays from study dental cast model and life size photography of bite marks was used. This is the most commonly used method until date, because of its simplicity and authenticity. This study was performed in two parts:

a) Collection of bite marks of volunteers in different material by photograph

b) Comparison of bite marks with dental cast of the same volunteer biter by comparison method.

The process involved production of bite marks by volunteers by biting one of the four objects, then photography of bite marks was taken and dental model cast from biters teeth was made. Further tracing of overlays from dental study cast was made. Lastly, comparison was done (both direct & indirect).

A digital camera was used for photography and photos were transferred to a computer for printing and analysis. A five-rupee coin of diameter of 2.20 cm was kept to size the images to life size. Photography was done vertically, so that the chances of distortion of photograph were reduced to minimum. A scale was also kept, as it served duel purpose, firstly obtaining relative size and secondly allowed accurate enlargement. A scale with certified accuracy should be used and the same scale should be used through out the whole photographic process[5].

Impressions of volunteers' teeth were taken in irreversible Hydrocolloid impression material (Alginate) in a dental tray, which gave negative replica of teeth, which sets in 45 seconds. Then positive replica, a model was obtained with dental stone powder, which sets 10 minutes.

The model casts were evaluated for dentition and impression. The size of arch, abnormal position of teeth, rotation, wear and tear, and missing tooth were looked for. A sheet of transparency film and fine tipped felt pen was used to mark the perimeter of the biting surface of each teeth, by hand tracing[19,20].

Indirect Comparison

In this method, transparent overlay was placed directly over the bite marks on photographs and the match was noted[21].

Direct Comparison

In this method model was placed directly over photograph and the concordant points demonstrated i.e. the fit of the incisial edge. Hence, the model was moved on the photograph[22]. The results so obtained were tabulated.

Overall analysis

To identify the characteristics of the bite marks and comparison of dental cast with photograph and to give expert comments, if the match was positive or else.

Match Positive

If there were no doubts about the bite marks when compared with dental cast it was termed as match positive.

Match Non-contributory

In case the dental cast did not at all match with photograph of bite marks or there was some degree of doubt due to technical fault in making of cast or photograph, then the comment of expert was reserved as match 'non-contributory' rather than match negative

OBSERVATIONS

Table 1: Distribution of Volunteers According To Age

Age group (years)	No. of cases
0 – 10	0
11 – 20	7
21 – 30	59
31 – 40	19
41 – 50	12
51 – 60	3
Total	**100**

Table 2: Factors of Non-Contributory Match

Cause	Proper	Non-contributory	Total
Model/Casts	95	5	100
Photographs	92	8	100
Others	83	4	87
Total	83	17	100

CONCLUSIONS

In case of bite marks in skin, the match was positive in 60% cases, while it was positive in 100% cases in clay wax, in fruit, the match was positive in 84% cases and in cheese, the match was positive in 88% cases.

The match was non-contributory due to defective model cast in 5% cases and due to defective photography in 8% cases.

In comparing direct and indirect method of match, it was found that when the comparison by indirect comparison method remains inconclusive, the direct comparison method tends to match.

More study is required to find out if direct method is more sensitive than indirect comparison method.

REFERENCE

1. Father D' Coto. Bible Old Testament. Sunshine Publisher. 2nd Edition reprint. 1942: 51.

2. DeVore D T. Bite marks for Identification. Med Sci Law 1971; 11 (3): 144-5.

3. Numble B H. Identification by means of teeth. Brit Dental J. 1933; 54: 529.

4. Harvey W. Bites and Bite marks: Dental Identification and Forensic Odontology. Henry Kempton publisher. 4th Ed. 1976: 1, 88, 90,129.

5. Clark D H. Bite marks in tissue and in inanimate objects: analysis and comparison. Practical Forensic Odontology. Wright. 3rd Ed. 1980: 107, 128, 129, 149, 152, 153, 159.

6. Levine L. Forensic Odontology. Int Microform J Deg Med. Card 5B4-13, 1971: 3-6

7. MacDonald D G. Bite Marks, Recognition and Interpretation. J Foren Sci 1979; 14: 229.

8. Onmura T. Examination of bite wounds in reference to a rape and murder case of a young girl. Acta Crim Med Deg Jap 1968; 34: 51

9. Gustafson G. Forensic Odontology. Staples Press, London. 1966

10. Parikh C K. Mechanical Injuries; Parikh's Textbook of Medical Jurisprudence and Toxicology CBS Publisher. 5th Ed. 2000: 79,144,235

11. Smith S and Fiddes F S. Forensic Medicine: Bite marks. Churchill, London. 10th Ed. 1955: 93

12. Whittaker D K and MacDonald D G. Bite marks and flesh: A Colour Atlas of Forensic Dentistry. Wolfe Medical. 4th Ed. 1989: 108

13. Sorup A. Odonotoskopie ein Beitrag Sun gerichtlichen Medizin Vjschn Zahnheilk. 1924; 30:385

14. Humble B E. Identification by means of teeth. British Dent J 1933; 54: 528

15. Simpson K. The health care. Police J 1947; 2: 266.

16. Furuhata T and Yamamoto K. Forensic Odontology. Thomas, Springfield. 1967

17. Thomas J. J Forens Sci 1986: 31.

18. Jonason C O, Frykholm K O and Frykholm A. Three-dimensional measurement of tooth impression of criminological investigation of teeth in soap. Br Dent J 1984; 157: 270-1

19. Luntz L and Luntz P. Hand book for dental identification. Lippincott, Philadelphia. 1973: 154

20. Sweet D and Bowers M. Accuracy of bite marks overlays. Paper presented at the meeting of American Academy of Forensic Sciences, Odontology section, New York. 21 Feb 1997.

21. Morgan H. Zat Frage der Bisspureh. Rdsch 1943; 52: 791-6

22. Farness J. A new method for the identification of teeth marks in cases of assault and Homicide. Br Dent J 1968; 124: 2161-6

Table 3: Bite Marks Matches

Bite marks on	No. of cases	Positive	Direct	Indirect	Non-contributory
Skin	25	15	15	15	10 (40%)
Clay	25	25	25	25	0
Fruit	25	21	21	21	4 (16%)
Cheese	25	22	22	22	3 (12%)
Total	**100**	**83**	**83**	**83**	**17**

Book- Review

By Dr. R.K. Sharma
Editor In Chief Medico-legal Update

1. **Modern Medical Toxicology by V.V. Pillay 3rd Edition, Published by Jaypee Brothers, Medical Publishers New Delhi ISBN 81-8061-519-7 pp 499 Price Rs. 250/-**

This is a short concise book ob medical toxicology quite useful for undergraduate and post-graduate students.

Indian perspective has been widely discussed. Latest concepts of diagnosis and treatment have been corporated. Forensic and medico-legal aspects of poisons have been described in great detail. Recent references have also been added. Case histories is a salient feature of this book. The text is simple to follow especially for under-graduate students. Tables have been provided at appropriate places with illustrations. Mnemonics have been added for quick memory for students. The book is readable and worth its price.

2. Lyon's Medical jurisprudence and Toxicology Revised by T.D. Dogra and a Rudra 11th Edition 2005, Published by Delhi law House, Delhi ISBN 18-86976-46-9 pp 1550 Price Rs 1295

Lyon's Medical Jurisprudence and Toxicology dates book to 1856 when first manual addition come since then, the book has travelled to eleventh edition in 2005. This is quit exhaustive book where most of the contributors have been postgraduate students or who have just completed their post-graduate training. Inspite of where inexperience, they have done splendid job under the guidance of main editor. The book is quit useful for under-graduate and post-graduate students. The latest case laws have been incorporated. The new concepts have been described in detail. The text of the book is simple and easy to follow. The book would also be quit useful for lawyers and Judges to understand medical jurisprudence. In short, a good book and good effort.

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