Unintentional household poisoning due to rodenticide in a child

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ABSTRACT

Introduction: The household poisoning is a major problem in India, and the awareness of these poisons among the people is meager and sometimes they ignorant and careless regarding these poisons. The various types of rat killer or rodenticides used in household, and their improper usage and safety may pose serious health issues. The rat killer poisoning with phosphide, warfarin, and superwarfarin are increasing day by day.

Case report: A three years old boy unintentionally consumed rat killer paste, when his mother was out of the house. He had complaints of nausea and vomiting, and he admitted in a primary health care center. Then he was referred to tertiary care center and during the treatment he developed the features of acute renal failure and hepatotoxicity due to the rat killer poison and succumbed on a second day of admission. The features of rat killer poison that is zinc phosphide were evident on thorough autopsy examination, clinical findings, and ancillary examinations.

Discussion: There is a need for proper preventive and treatment strategies to reduce the number of exposures, morbidity and mortality due to household poisons including rat killer poisons. In medical practice identifying the rat killer poisoning cases, their proper management, and its medicolegal aspects are very important.

Key words: Rat killer poison, zinc phosphide, unintentional poisoning, household poison.

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INTRODUCTION

Household poisoning is a serious problem in developed and developing countries. It is a major problem in India also. The extensive use of pesticides like rat killer poisons or rodenticide is also common in our country. The awareness, safety practices of these poisons among the people is meager and sometimes they ignorant and careless regarding these poisons.

Poisoning in variety of forms is an important cause of morbidity and mortality. It occurs in all societies, races, and socioeconomic groups. According to World Health Organization annual report of 2004, about 3,45,814 people of all ages died worldwide; as a result of accidental poisoning. Even though a majority of these occurred among adults, 13% happened among children and young people under the age of 20 years.1 The poisoning material in a particular area depends largely upon the predominant occupation of the area, agricultural settings and availability of various substances under the existing law.2

According to National Crime Records Bureau, in 2012, accidental poisoning constitutes 7.8% of all accidental deaths in India.3 An epidemiological study of poisoning cases reported to the National Poisons Information Centre found that most common causes are household poisoning (44.1%) followed by drugs (18.8%).4 The study on childhood poisoning revealed more than one-third of cases occurred in children less than five years. The most common route of poisoning was ingestion (84%) and most occurred inside the house (80%), and survival rate of 95% of the cases.5 Unintentional poisoning is mainly due to household chemicals, medications and plants in developed countries. In developing countries, it is much more serious due to ingestion of kerosene, caustic acids in toilet cleaners and herbal remedies, etc. Poisoning in infants and young children are almost always unintentional because of their explorative behavior and tendency to place any objects in their mouth.6

Rodenticides are used mainly for the storage of grains in large quantity and to kill the domestic rodents. The classification of rodenticide:7,8
(a) Inorganic preparation: barium carbonate, zinc or aluminium phosphide.
(b) Organic preparation: fluoroacetate
(c) Convulsant: strychnine
(d) Anticoagulants: first generation (warfarin) and second generation (superwarfarin).

Superwarfarin compounds again divided into three subgroups; hydroxycoumarin derivatives with a 4 bromo (1,1 biphenyl) side chain, coumatetryls, and indanediones. Among the acute rodenticides, zinc phosphide is most widely used in South Asia and is a basis for 80-90% of rodent control operations. First generation anticoagulants have not gained much publicity in India due to their multiple dose requirement and development of resistance. Second generation anticoagulant, bromadiolone is available in India from 1988 onwards and using till now as only small single dosage is required to kill rodents and less development of resistance.9 Bromadiolone is an anticoagulant, acts by inhibiting the carboxylation of vitamin K- dependent clotting factors (II,VII,IX & X).10,11

Zinc phosphide (Zn3P2) is being used as a vertebrate pest control agent for many decades. It is preferred in the USA and Australia for the field control of rodents because of its reasonably low risk of secondary poisoning and absence of environmental persistence.12 Zinc phosphide is a gray crystalline compound with a garlicky odor. It reacts with water or acid in the stomach to form phosphine gas. This phosphine gas is very toxic and irritant to respiratory tract and whole systems of the body. It inhibits cytochrome C oxidase and oxidative respiration. In addition to this energy failure of cell, free radical generation also increased leading to lipid peroxidation. The reactions that phosphine causes at the cellular level give rise to various disorders and failures in organs and systems.13,14,15 It produces widespread organ damage due to cellular hypoxia and directly damages the vessels and red blood cells membrane. Lethal dose of zinc phosphide for human is 20- 40 mg/kg.16 Unfortunately there is no specific treatment for zinc phosphide poisoning.10,17 Because of the low cost and easy availability of metal phosphides, and it is more common in suicidal and accidental poisoning.18

CASE REPORT

A three-year-old developmentally normal, a male child had alleged history of accidental consumption of rat killer paste at home. His mother was out in the paddy field for work, and no one was present at the home to look after the child at the time of incidence. The incident was noticed by a neighbor as he was eating the paste, and the quantity not known. He had 4-5 episodes vomiting containing food material mixed with frothy secretion after 4 hours of ingestion. He given first aid at nearby primary health center and then referred to our hospital for further treatment. On enquiry, we revealed he had unintentionally consumed kerosene one year back, and he admitted for four days.

He was conscious, oriented, his vital were normal, per abdomen examination showed epigastric tenderness at the emergency department of our hospital. The laboratory investigation showed normal blood counts, elevated blood urea (50 mg/dl) and serum creatinine (2.1 mg/dl). Elevation of aspartate aminotransferase (AST-88 IU/L), alanine aminotransferase (ALT – 67 IU/L) and alkaline phosphatase (ALP-213 IU/L) also noted. Prothrombin time (PT-22.2 sec) and international normalized ratio (INR-1.7) mildly elevated. He closely observed in pediatric intensive care unit and started on intravenous fluids, antacids, and other supportive measures. On the second day, he developed features of shock like hypotension, tachycardia and prolonged capillary refill time (5 sec). Laboratory investigations revealed features of hepatotoxicity i.e. total bilirubin (4.6 mg/dl), AST (685 IU/L), ALT (112 IU/L) and raised PT & INR (3.37). Then he was in a state of altered sensorium and arterial blood gas analysis showed features of metabolic acidosis with respiratory alkalosis. Subsequently he developed ventricular tachycardia, dysrhythmia and succumbed on the second day. His body sent for autopsy at the postmortem center of our hospital.

Autopsy examination shows averagely nourished and thin built body of length (92 cm) and weight (10kg). Congestion of face, yellowish discoloration of sclera, and no evidence of any external injuries noted over the body. On internal examination, brain shows patchy

subarachnoid hemorrhages over bilateral parietal lobes, both lungs congested and edematous. Peritoneal cavities contain 200 ml of yellow colored fluid. Stomach shows 30 ml of reddish colored fluid without any peculiar smell and mucosa hemorrhagic. Liver enlarged with yellowish discoloration and other organs are congested.

Histopathology examination of lung showed pulmonary hemorrhage and congestion. The section from heart detected focal vacuolar degeneration of cardiac myocytes with myophagocytosis. Liver showed focal lobular hepatocellular necrosis with microvesicular steatosis with a mild increase in portal lymphocytes. Interstitial congestion, hemorrhage, and focal acute tubular necrosis noted in the kidney. Chemical analysis revealed zinc phosphide by gas chromatography at the regional forensic science laboratory. Our final opinion for the cause of death is pulmonary hemorrhage, liver necrosis and acute tubular necrosis due to zinc phosphide poisoning.

Figures and Figure legends:

Figure 1: Section from lung showing pulmonary hemorrhage on H&E staining (100X).

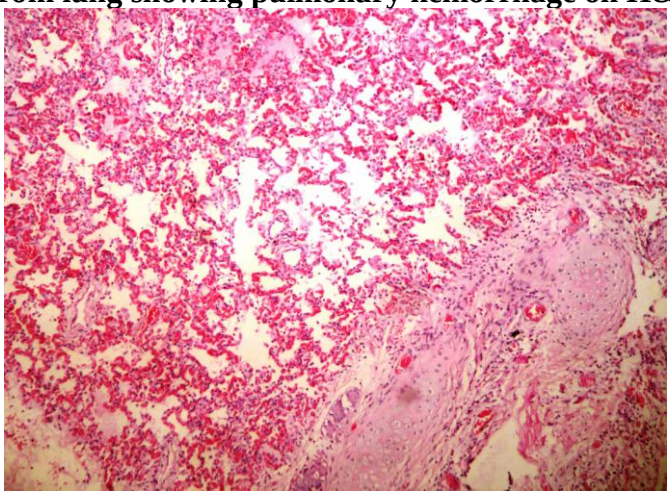


Figure 2: Section of liver showing micro-vesicular steatosis on H&E staining (400X).

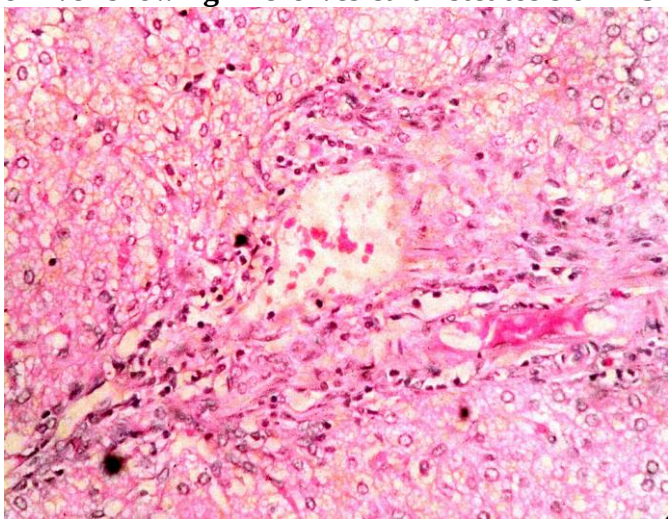
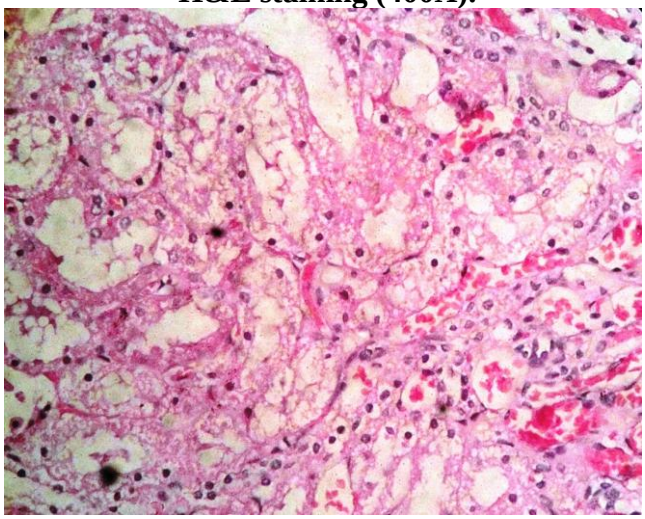


Figure 3: Section from kidney showing patchy acute tubular necrosis on H&E staining (400X).



DISCUSSION

The problem of poisoning either intentional or unintentional is getting more complicated day by day as newer drugs and chemicals are producing in large numbers. Rehman et al. observed that nausea, vomiting, and abdominal pain are the most common presenting symptoms of phosphide poisoning.13 We also observed the same clinical findings in our case. Mehmet TG et al. revealed that the most common metabolic complication of zinc phosphide was metabolic acidosis with respiratory alkalosis and elevated liver function tests.15 As per Bumbrah et al. the time progresses; there will be tachypnea, ventricular tachycardia and dysrhythmia leading to cardiogenic shock, which found to be most common cause of death.17 The focal myocardial necrosis that probably results in trans-membrane exchange of ions (Na+, K+, Mg+, Ca+) causing arrhythmias and rapid death.19 The classical signs and symptoms were correlating with other studies.13,15,17,19 It is very difficult to manage a case of zinc phosphide ingestion, which is supportive and symptomatic. Lavage with potassium permanganate recommended previously, but there is no sufficient evidence for its efficacy.20 A review of phosphide poisoning reported that autopsy examination will show bluish discoloration of face and froth around the mouth. Internally all organs will be congested, and lung shows petechial hemorrhages.17 Saleki S et al. noticed that, common histopathological feature of liver in zinc phosphide poisoning is necrosis and microvesicular steatosis.21 As per the study by Mehrpour et al. lung histopathology revealed congestion, edema and hemorrhage in majority of the cases. 22 According to Bumbrah et al Focal myocardial infiltration with necrosis also seen.17 Similar features observed in this case also.

Abbas SK et al observed that, unintentional poisoning is most common in the age group of 1 – 5 years with a male preponderance. Pharmaceutical products as a whole are the most common cause followed by kerosene oil. Most common route is ingestion and mostly occurs at home.23 Ghosh VB et al studied that most common cause of childhood poisoning is

kerosene oil followed by hydrochloric acid, and drugs used by adults.24 The use of preventive programs and measures resulted in reduction of unintentional poisoning cases among children in developed countries. The exposure frequency reflects the product availability, packaging, and accessibility to the child. Child resistant packaging is defined as "difficult for the child below the age of 5 years to open, but not difficult for a normal adult to handle". The usage of child resistant packaging is shown to be effective in reducing such mortalities and morbidities.6 In the study of Schmertmann et al. observed that the usage of more positive control by mothers, storing of poisons at accessible location, low parenting stress with high psychological stress, and less supervision of child are the major risk factors for unintentional pediatric poisoning.25

Due to low socioeconomic background and both parents are working in the rural areas on daily wage basis, no one is there to take care of the children properly. One year back, he had accidentally consumed kerosene in the house. So this repeated exposure may be due to the negligence of caretaker like keeping these materials within the reach of children, storage in soft drink bottle or food packets or not instructing the child properly. The rat killer paste packaging is very similar to toothpaste and children may easily attract; if not stored properly. We have differentiated zinc phosphide poisoning from bromadiolone by clinically, autopsy and histopathology features; chemical analysis confirmed our opinion.

CONCLUSION

Here we discussed a case of unintentional zinc phosphide poisoning in a child with previous kerosene poisoning and observed how carelessly the people are handling all these poisonous materials at home. Zinc phosphide affects all the systems of the body leading to multi-organ failure and very difficult to manage also. Improper storage facilities, lack of care by parents, keeping at the reach of children, low level of knowledge regarding phosphide and household poisons may cause such incidences. Parental education, awareness regarding the storage, and proper supervision of children will be useful for prevention of such calamities. The child resistant packaging should be used for zinc phosphide and other household poisons. Child attractive packaging should be avoided and shouldn't resemble nontoxic household products. Highlighting the caution over the covers and labels in major languages should be enforced. Properly dispose of the zinc phosphide poison and their containers after the use. Preventive strategies and guidelines should be formulated to prevent such type of poisoning in the community.

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