Case Report

Zinc Phosphide Poisoning: Case Report and Brief Review

Prateek Rastogi

ABSTRACT

Zinc phosphide, a commonly used rodenticide is emerging as one of the popular self-poisoning agents in adults due to its low cost, easy availability and highly toxic nature. Zinc phosphide acts by liberating toxic phosphine gas on coming in contact with gastric acid, and fatal period varies from a few hours to 24 hrs.

In this paper, two cases of zinc phosphide poisoning autopsied at the mortuary of Kasturba Medical College, Mangalore are presented. In both cases, the deceased were young adults who consumed the poison in the vicinity of their own house and died after a few days of hospitalization. No external injuries were present on the body.

Chemical analysis report was positive for the presence of zinc ions in the preserved viscera and blood. Histopathology of internal organs was non-specific.

In a developing country like India, where agriculture is the main source of livelihood of the majority of the population, loss of crops due to insects cannot be ignored and thereby the use of rodenticides cannot be abolished. Hence, measures for the safe usage and disposal of rodenticides should be adopted. In addition, it is advisable to make available expert psychiatric services to the community which could help in identifying high-risk individuals who are likely to commit deliberate self-harm.

Key Words: Zinc phosphide; Suicide; Rodenticide

Introduction

Zinc phosphide is a highly effective rodenticide, and is used widely to protect grain in granaries, and during its transportation. Acute poisoning with this compound during its approved use may be direct due to ingestion of the salts, or indirect from accidental inhalation of phosphine gas generated on zinc phosphide coming in contact with gastric acid, with a fatal period varying from a few to 24 hrs.[1,2] Zinc phosphide acts by inhibiting cytochrome oxidase, and sometimes also catalase and peroxidase. In addition, phosphine can interact with hydrogen peroxide to form the highly reactive hydroxyl radical; both mechanisms result in hydroxyl radical associated damage such as lipid peroxidation.[3]

There is usually only a short interval between ingestion of phosphides and the appearance of systemic toxicity. Phosphine-induced impairment of myocardial contractility and fluid loss leads to circulatory failure, and critically, pulmonary oedema supervenes. Metabolic acidosis, or mixed metabolic acidosis and respiratory alkalosis, and acute renal failure are frequent. Other features include disseminated intravascular coagulation, hepatic necrosis and renal failure.[2,4,5]

In this paper, two cases of zinc phosphide poisoning autopsied at the mortuary of Kasturba Medical College, Mangalore are presented.

Case Reports

Case 1: A 24 year-old female was brought to hospital with a history of consumption of rat poison. She died

Dept of Forensic Medicine & Toxicology, Kasturba Medical College, Mangalore, Karnataka.
E-mail: rastogiprateek@rediffmail.com

after 8 days of hospitalization and an autopsy was performed. Hospital records showed that the patient was unconscious throughout the hospital stay, and was brought with a history of abdominal pain and vomiting. Hepatic encephalopathy ensued with jaundice, pallor, and serum bilirubin level of 6 mg%. On examination, no external injuries were present on the body, but blood tinged fluid was oozing from the mouth and nostrils. Brain was oedematous, lungs were congested, and pleura and peritoneum contained straw-coloured fluid. Stomach contained 300 ml of reddish black liquid without any unusual odour, while the gastric mucosa was eroded and haemorrhagic at places. A few haemorrhagic spots were present on the surface of the liver. The usual viscera (stomach, part of the small intestine, liver, kidneys) and body fluids (blood, urine) were preserved for chemical analysis, and was subsequently reported to be positive for the presence of zinc ions (consistent with zinc phosphide). Histopathological examination of liver showed chronic venous congestion. Cause of death was opined as hepatic encephalopathy due to consumption of zinc compound.

Case 2: A 26 year-old male was brought to hospital with a history of consumption of some unknown poison. He died after 3 days of hospitalization. Hospital records showed that the patient was brought with history of abdominal pain and vomiting. No positive diagnosis was made. On autopsy examination, no external injuries were present on the body. Brain was oedematous, spleen was congested, and stomach contained 200 ml of reddish black liquid with a peculiar odour. Gastric mucosa was eroded and haemorrhagic at places. A few haemorrhagic spots were present on the surface of the liver. Routine viscera and body fluids (outlined above) were preserved for chemical analysis, and was reported positive for the presence of zinc ions. Histopathological examination of liver showed chronic venous congestion. Cause of death was opined as hepatic encephalopathy due to consumption of zinc compound.

Discussion

Zinc phosphide is a steel-grey coloured crystalline powder, insoluble in water and alcohol with garlicky odour.[2] It is available in formulations such as baits, pellets, granules and dust, under various trade names as Ratol, Agrophos and Sudarshan.[6] Zinc phosphide is a popular rodenticide, first used in 1947 by the US department of agriculture.[7] Zinc phosphide liberates phosphine on reaction with moisture, although the poison is most effective on ingestion, but toxicity is seen even upon inhalation of fumes, with dermal and eye contact. Hydrolysis requires acidic pH, and is completed in a duration of 12–18 hrs at gastric pH. Phosphine as well as zinc ions are absorbed through GIT and are responsible for toxic effects.[6-8]

A victim of acute toxicity clinically presents with metallic taste, nausea, vomiting, vertigo, diarrhoea, garlicky (fishy) odour, intense thirst, cough, cold and clammy skin, and burning epigastric pain. In severe cases, there may be hypotension, tachycardia/bradycardia, heart block, respiratory distress, laboured breathing and cyanosis. Effects on the central nervous system include ataxia, tremors, diplopia, headache, dizziness, convulsions and coma. In chronic toxicity, weakness, anaemia, toothache, jaw necrosis, weight loss and spontaneous fractures are seen. In complicated cases, impairment of myocardial contractility resulting in circulatory failure and pulmonary oedema, metabolic acidosis, respiratory alkalosis, hypokalaemia, hypomagnesaemia, acute renal failure, DIC, tender hepatomegaly and hepatic necrosis may be encountered.[1,3,6,8]

Zinc phosphide is emerging as one of the increasingly popular self-poisoning agent in adults in India, probably due to its low cost, easy availability and highly toxic nature. Its fatal dose is 2–4gms, while toxicity of phosphine is 400–600ppm in 30 minutes. Diagnosis is mainly based on history, clinical features, ECG changes (sinus tachycardia, ST-T wave changes, bradycardia with heart block). Urine shows occult blood, bilirubin, glucose and albumin. Prothrombin time, blood urea and serum creatinine are elevated with altered liver function tests.[6,7,9]

There is no specific antidote for phosphine or metal phosphide poisoning, and many patients die despite intensive care. Supportive measures are all that can be offered and should be implemented as required.[4,5] The following are recommended: IV fluids (4–6 L over 6 hrs) and dopamine IV (4–6mg/kg/min) to treat shock, 100% oxygen, intubation and assisted ventilation to treat respiratory distress, sodium bicarbonate IV (50mEq/15min) for metabolic acidosis, ranitidine IV (50mg 8 hrly) for gastric irritation, diazepam IV (5–10mg over 2–3 min) and phenytoin IV (10–15mg/kg) for convulsions, magnesium sulfate IV (3gm bolus, 6gm infusion over 24 hrs for 5–7 days) for arrhythmias, and furesemide (contraindicated in shock) IV (20–40 mg) for pulmonary oedema. Vitamin K and whole blood transfusion are also recommended

if a product containing a combination of zinc phosphide with an anticoagulant rodenticide such as brodifacoum has been consumed, with consequent bleeding disorders.[8-10]

In the case of death of the patient, autopsy often shows widespread hypoxic damage resulting in congestion and petechiae, garlicky odour with haemorragic mucosa and mucosal shedding in the stomach, microscopic necrotic changes in the liver and kidneys, pulmonary oedema, and toxic myocarditis with fibrillar necrosis. Laboratory analysis of viscera may reveal presence of zinc ions/phosphorus/phosphine as well as zinc phosphide.[6,9]

In our cases, both the patients were shifted to hospital within a few hours of ingestion of the poison. Although all possible treatment was administed, the patients expired. Zinc phosphide has a tendency to adhere firmly to the crypts in mucous membranes, and even if a small quantity remains in the stomach after stomach wash, it is sufficient to cause death by absorption.[2] This is probably what happened in these cases, where-in even after gastric lavage and treatment the patients died and still the viscera tested positive for the poison after days of hospitalization. This emphasizes the toxicity of zinc phosphide. Also, some medicolegal issues like alleged negligence on the part of the attending physician may be encountered, as patients sometimes show delayed collapse after an initial recovery.

In a developing country such as India, where agriculture is the main source of livelihood of the majority of the population, loss of crops due to insects cannot be ignored, and thus, these compounds will always remain in use. Hence, measures for safe usage and disposal of rodenticides should be adopted. In addition, extending psychiatric services to the community may help in identifying high-risk individuals who are likely to commit deliberate self-harm. Also, the setting up of poison information centers for providing effective treatment guidance in poisoning cases is the need of the hour.

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