A CASE REPORT ON ZINC PHOSPHIDE POISONING AND ITS RARE EFFECTS

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ABSTRACT

BACKGROUND

Zinc phosphide is widely in use as a rodenticide. After ingestion, it gets converted to phosphine gas, which is subsequently absorbed into the bloodstream through the stomach and the intestines and gets captured by the liver and the lungs. The toxic effects of zinc phosphide poisoning is through the phosphine gas that produces various metabolic and non-metabolic intermediate compounds. Patients develop features of shock, myocarditis, pericarditis, acute pulmonary oedema and congestive heart failure. In this case report, we present a common complication of the poison that manifested earlier than it is depicted in the current literature.

KEYWORDS

Zinc Phosphide, Liver Dysfunction, Side Effects.

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BACKGROUND

Zinc phosphide is a dark grey, crystalline compound. It is used as a rodenticide and pests such as mice, rats, field mice and squirrels.

It is possible, though rare, to be exposed to zinc phosphide poisoning by accident, but majority of the inflicted patients do it in an attempt of suicide. As it is ingested, it gets converted to dangerous phosphine gas, which is the main culprit for various toxic intermediate compounds and also has the potency to be a lethal toxin by itself. There are no antidotes currently known. The mortality rate of zinc phosphide poisoning is around 37-100%.

Consumption of zinc phosphide has seen a recent upsurge as far as demographics are concerned as it continues to be one of the leading causes of morbidity and mortality of socially backward and economically down trodden agriculturists in developing world and otherwise.

CASE REPORT

A 23-old-year male, an agriculturist by occupation, a nondiabetic and non-hypertensive came with alleged history of consumption of approximately 2 teaspoonsful of pellets of zinc phosphide mixed with water and later buttermilk in an attempt to commit suicide. There were three episodes of vomiting, which were non-projectile, non-blood tinged and non-bilious. The patient underwent gastric flushing with activated charcoal treatment at the local government hospital and was admitted for few hours for resuscitation. The patient was dispatched from there to the higher centre in view of impending ventilator support. He was received in the casualty of the tertiary centre and gastric lavage with activated charcoal was done and was treated in the emergency ward as he was relatively stable. He was conscious with a blood pressure of 160/100 mmHg, pulse of 82 per minute and blood sugar of 364 mg/dL.

As the patient was admitted in the emergency ward and subjected to the initial intervention, next few hours saw a steep decline in his sensorium owing to which the patient was taken to the intensive care unit. His blood pressures started declining. Activated charcoal application at 2 mg/kg was continued and supportive therapy was initiated in the form of inotropes titrated accordingly. In order to improve the low urine output of less than 0.5 mL/kg, intravenous furosemide was started after proper rehydration and blood pressure monitoring. The patient was administered oxygen at a rate of 5 L/min. through an orofacial mask. Complete blood count, biochemistry, coagulation parameters and Arterial Blood Gas (ABG) readings of the patient were normal. His urine output was 0.5 mL/kg/hour and the patient was haemodynamically stable for next 4 days.

After receiving intensive care for 5 days, patient developed typical GTCS seizures twice with 1 hour gap between each episode with uprolling of eye balls and involuntary micturition. He was started on intravenous phenytoin loading dose of 20 mg/kg stat followed by 100 mg thrice daily. Serum electrolyte reports were showing hyponatraemia of 121 mg/dL. So, 3% hypertonic saline was started with 6th hourly monitoring of electrolyte levels. Renal

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function tests showed urea to be 80 mg/dL and creatinine as 1.6 mg/dL. Prothrombin time was 86.8, INR was 7 and activated partial thromboplastin time was 57.7. So, injection vitamin K1 ampule IV once daily dose was started and 4 pints of fresh frozen plasma were given. His liver function tests read as total bilirubin 4.8 mg/dL, direct bilirubin 3, aspartate transaminase was 7150 IU, alanine transaminase was 3981 IU and alkaline phosphatase was 229 IU. A detoxifying syrup with glutathione was added along with ursodeoxycholic acid 150 mg twice daily dose. Patient was clinically stable. The following day morning serum electrolyte readings were read as sodium 126 mmol/L and potassium 2.8 mmol/L. Potassium correction was done with addition of 2 ampules of KCl to normal saline and repeat coagulation profile was PT being 26, APTT being 52.4 and INR being 3.7. The antibiotics coverage included piperacillin and tazobactam along with metronidazole given thrice daily dose.

Next day morning, the patient was irritable and developed hypotension that was unresponsive to fluid replacement. As arterial blood gas analysis was normal, the patient was started on a 5 mcg/kg/minute dopamine infusion. Due to low sensorium and increased respiratory distress, the patient was intubated and mechanically ventilated. After being on ventilator for more than 10 hours, he developed metabolic acidosis and was treated with hundred milliequivalents of soda bicarbonate loading dose. According to the blood gas values, the metabolic acidosis did not show any signs of improvement. Blood pressures were further declined and noradrenaline and dopamine were started subsequently and titrated according to the blood pressure.

Even after aggressive resuscitation as per the standard protocols, the patient could not be revived and was declared dead.

DISCUSSION

Phosphide is being widely employed as means of committing suicide. In a study of G. S. Bumbrah, K. Krishan et al, "phosphide poisoning- a review of literature," Forensic Science International, the average age of patients who attempted suicide was 27 years.[1] The characteristics of our case and the age of our patient match the patient profile in that literature.

Zinc phosphide's mechanism of action upon oral ingestion is unclear.[2] Most possible explanation would be that of the phosphine gas forming in the stomach after oral intake of zinc phosphide.[3] Phosphine is rapidly absorbed and inhibits of cytochrome oxidase as a result of which oxidative respiration is impaired at a cellular level. Patients die early due to irreversible damage caused to the vital organs.

Phosphine gas is known to cause various metabolic and non-metabolic toxic effects. Clinically, patients land in refractory hypotension due to myocarditis, pericarditis, acute pulmonary oedema and congestive heart failure. It will affect gastrointestinal system resulting in symptoms like nausea, vomiting and diarrhoea. Later hepatomegaly, severe metabolic acidosis and acute kidney failure are observed in some patients. Retrosternal pain, shortness of breath and cyanosis are not infrequent symptoms. Studies show that severe hypoglycaemia, delirium, tonic-clonic seizures with acute severe metabolic acidosis (distal renal tubular acidosis) and thrombocytopenia could be seen in cases of zinc phosphide poisoning.

In our patient, liver cell damage in terms of biochemical markers like aspartate transaminase and alanine transaminase were more than 7000 and 3000, respectively and occurred lately during the course as against to the established and accepted notion. Later zinc phosphide poisoning-related circulatory collapse and lung damage developed. At the same time, the very severe hypotension and resistant metabolic acidosis that did not respond to bicarbonate treatment have been found to be in line with the literature.[4] In cases of phosphide poisoning hypotension is a common occurrence, may develop quickly and maybe resistant to treatment.

In patients poisoned with rodenticide as per Karanth and Nayyar study, decompensated liver failure was reported after phosphide ingestion with increased Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST) values.

Pulmonary oedema is commonly observed, 4-48 hours following oral ingestion of zinc phosphide without an increase in the pulmonary artery pressure.[5] Both ARDS-related pulmonary oedema and nonspecific pulmonary oedema are observed. Oedema fluid maybe protein-rich with or without basal exudates and mostly haemorrhagic. Generally, after undergoing intensive care for approximately 9 to 12 hours, patients develop an acute pulmonary oedema.[6]

CONCLUSION

Zinc phosphide is a substance that causes life-threatening complications and most often than not ends in mortality. There is neither an effective antidote nor specific treatment for it. Despite a quick and aggressive supportive therapy, heart or lung damage due to zinc phosphide poisoning is bound to happen.[7] Liver failure is usually noted after fifth day and the regular monitoring of liver function tests are needed in cases of rodenticide poisoning. Treating physician must be cautious regarding the late and severe toxicity and allied manifestations of hepatobiliary system as it is in our case and should explain grave prognosis to the attenders throughout the course of treatment.

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