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A Case Report of Rodenticide Poisoning with Acute Kidney Injury

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ABSTRACT

Rodenticide is widely used around the word. It contains various toxic components such as zinc or aluminum phosphide, yellow phosphorus, arsenic, thallium, sodium monofluroacetate. Rodenticide is easily available. Toxic effects may be produced by inhalation or ingestion. It may be accidental or intentional.

The rodenticide in this case contains 38 percent zinc phosphide. Zinc phosphide produces phosphine gas when hydrolyzed by gastric acid. Phosphine gas is highly toxic. It produces toxicity within 30 minutes. It may produce symptoms such as nausea, vomiting, abdominal pain, hematemesis, arrhythmias, acute respiratory syndrome, respiratory failure, cardiac failure, liver failure and renal failure. Phosphine gas is highly toxic to respiratory system it inhibits cytochrome C oxidase.

Poisoning from metal phosphide is prevalent, particularly in tropical nations. Through the phosphine gas-mediated effects on cellular respiration, aluminum and zinc phosphide poisoning have harmful effects. Majority of deaths are due to acute pulmonary edema, metabolic acidosis or distal renal tubular acidosis.

Key words: Zinc phosphide, Rodenticide, Poisoning.

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INTRODUCTION

This case of rodenticide poisoning contains major quantity of zinc phosphide (38 percent). It is easily available all over the world and is very cheap. Hence it can be easily used as poisoning agent and is also consumed accidentally. It may also enter the body through inhalational or through the skin. It is important to start treatment early, to prevent complications. Most of the deaths reported are due to hypovolemia and acute kidney injury with fulminant liver failure [1].

CASE PRESENTATION

A 29 year male patient presented with consumption of 2.3 g of rodenticide powder which was grey in colour, mixed with water 5 hours back. The commercial packet of rodenticide has zinc phosphide as the major content. 5 minutes after consumption he had 2 bouts of vomiting and had complaints of gastric and throat irritation, giddiness and abdominal pain. Patient was taken to an outside clinic where stomach wash was done and then patient was referred to us. On presentation his BP was 110/70mmHg, pulse rate was 110 bpm, saturation was normal in room air.

Examination showed that he was icteric and pale [2, 3].

His complete blood count showed low platelet count(80,000), Hemoglobin - 12.8, total count-6480, Liver function test showed AST - 200U/L, ALT- 1247 U/L Alkaline phosphate - 420U/L, Urea - 40, creatinine - 1.7.electrolytes were within normal limits. Amylase -245(22-80), APTT was 31(30-36), INR was 0.90. Urinalysis showed albumin 2+, RBCs 8-9. ABG showed metabolic acidosis [4].

ESR was 28mm in the first hour. CRP was 8.0. Chest X-ray, USG abdomen was normal. ECG showed normal sinus rhythm [5].

Patient was admitted in ICU. On second day of admission Patient's urine output was decreased,

RFT showed urea- 48, creatinine - 2.4. Intake was maintained at less than 1000ml/day [6].

On third day urea was 60 and creatinine was 3.1. The patient was started on hemodialysis. After the first cycle, serum creatinine started to decrease. Three cycles of hemodialysis was done. On day 6, patient was shifted toward [7].

DISCUSSION

Poisoning with zinc phosphide is acknowledged as a substantial source of morbidity and mortality in age groups with poor socioeconomic status but active economies, particularly in developing nations. Acute toxicity of zinc phosphide caused human mortality at doses of 4 to 5 g (55–70 mg/kg).Following consumption of zinc phosphide; our patient had acute kidney injury, increased hepatic enzymes with hyperbilirubinemia, acute pancreatitis, and dynamic ECG abnormalities likely caused by an element of myocarditis.

Following inhalation, abdominal pain, cough, giddiness, headache, and sore throat are common symptoms of zinc phosphide poisoning. Ingestion-related symptoms include nausea, vomiting, pain in abdomen, tightness in the chest, agitation, cyanosis, loss of consciousness, and convulsions.

The severity of the symptoms varies between purposeful and accidental phosphide poisoning, and patients who appear with intentional phosphide poisoning require cautious observation. Thrombocytopenia, metabolic acidosis, distal rental tubular acidosis, hypomagnesaemia, severe hypocalcaemia, hypokalemia, disseminated intravascular coagulation, adult respiratory distress syndrome, oliguric acute kidney injury (in about 50% of patients), fulminant hepatic failure and seizures. ECG changes may be seen such as sinus tachycardia, bradycardia, supraventricular ectopic, ventricular ectopic, atrial fibrillation, ventricular fibrillation, wide QRS complex, A-V conduction defects, bundle branch block, complete heart block, and ST-T changes like ST depression, ST elevation. It also results in myocarditis and pericarditis.

Glycemic derangement is a known side effect of phosphide poisoning. Because hepatic glycogenolysis and gluconeogenesis are less frequent, severe hypoglycemia is more frequent. Rarely occurring temporary hyperglycemia may be caused by pancreatic involvement. The literature comes to the conclusion that treating hyperglycemia lowers cellular oxygen consumption by allowing glucose to enter the intracellular compartment once more. One should search for hyperglycemia that necessitates prompt correction because delayed presentation and having a hyperglycemia that could infrequently occur due to unknown reasons predict a worse prognosis. The conclusion of an analytical investigation on aluminum phosphide poisoning provided more support for this point, showing that the patients who had not survived proportionally significantly had been higher glucose readings than the survivors.

CONCLUSION

Zinc phosphide poisoning can cause symptoms such as nausea, vomiting, stomach pain, tightness in the chest, agitation, cyanosis, loss of consciousness, and convulsions. Refractory hypotension and arrhythmias have been found to be the primary causes of the majority of deaths, which occurred in the first 12 to 24 hours. ARDS, liver failure and renal failure were the most common causes of the late deaths (those that occurred after 24 hours). With delayed presentation, coagulopathy development, hyperglycemia, and multi-organ failure with high liver enzymes, the result is worse.

Zinc phosphide is cheap and widely accessible in Asian nations. So, it is a substance that is frequently used in suicides. As there is no known treatment for phosphides, management is only symptomatic and supportive. Physicians are therefore faced with a significant difficulty due to the lack of specific antidotes. Delay in treatment this also due to non-availability of renal replacement at primary health centres and peripheral hospitals. Actions should be conducted to update the medical staff's understanding of the prompt management of life-threatening end organ damage at peripheral hospitals and to educate the rural public about the lethal implications of such poisoning. In order to reduce deaths from major organ failure, local hospitals' resuscitation programmers and the way patients are transferred to tertiary care facilities need to be improved.

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