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Rodenticide induced acute fulminant hepatitis and acute pancreatitis: A case report

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ABSTRACT

Rodenticides are one of the most toxic substances found in home. Yellow phosphorous component, if ingested leads to severe liver consequences. We report a case of 25 year old female, who was brought to hospital for consumption of rodenticide, developed the symptoms of hepatic failure. She was managed conservatively and was discharged after 16 days.

Keywords: Rodenticide, Fulminant Hepatitis, Pancreatitis

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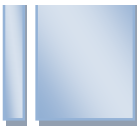
INTRODUCTION

Rodenticides are commonly used suicidal poison in India. The varieties of rodenticides used over the years are heavy metals (arsenic, thallium), red squill, Alpha naphthyl thiourea, Strychnine, Cholecalciferol-containing rodenticides, warfarin-type anticoagulants, phosphorous. In most of the cases, the outcome is good. Elemental phosphorus exists in two forms—red and yellow. Phosphides used as rodenticides include: aluminium phosphide, calcium phosphide, magnesium phosphide, zinc phosphide. The acid in the digestive system of the rodent reacts with the phosphide to generate the toxic phosphine gas. Red phosphorus is nonvolatile, insoluble, and unabsorbable, and therefore nontoxic when ingested. Yellow phosphorus (also referred to as white phosphorus), on the other hand, is a severe local and systemic toxin causing damage to gastrointestinal, hepatic, cardiovascular, and renal systems. White phosphorus is used as rodenticides and in fireworks. In India, suicidal or accidental poisoning with rodenticides containing metal yellow phosphorous (Ratol® which has Zinc Phosphide in paste form) is a more frequent cause of drug-induced ALF. We describe here a case of rodenticide poisoning leading to acute fulminant liver failure and acute pancreatitis, who eventually recovered with conservative management.

CASE REPORT

A 25 year-old female was brought to the casuality with an alleged history of consumption of an unknown quantity of rodenticide paste (Ratol, containing 3% yellow phosphorus) and complaints of pain abdomen and 2 episodes of vomiting. There was no history of breathlessness, hemoptysis and hematemesis. On examination; pulse 80/min, respiratory rate - 18/min, blood pressure - 120/76 mmHg, and systemic examinations were normal. She was provided symptomatic treatment in the form of stomach wash (gastric lavage) and intravenous saline, proton pump inhibitor and antiemetic. Blood investigations done at the time of admission were normal. She became symptomatically better 24 hours later and she was shifted to ward for observation. On the third day, she



complained of abdominal pain and 3 episodes of vomiting, altered level of consciousness, irritability.

On examination, patient was drowsy, icteric, temperature of 101.5°F, pulse rate 144/min, respiratory rate of 32/min and blood pressure of 100/70 mmHg in the supine position. Glasgow coma scale score was 10/15. Liver palpable 2 cm below the right costal margin, with liver span of 17 cm in right mid-clavicular line. No splenomegaly, no ascitis, pupils were sluggishly reacting to light and plantar reflex was bilaterally flexor. Her condition deteriorated further with Glasgow coma scale of 6/15 and eventually the patient went into deep coma. Meanwhile her blood investigations on day 3 revealed Hemoglobin 9.9 Gm%, TLC 4500/cumm, platelet 1.9 lakhs/mm^3, RBS – 90mg/dl, total bilirubin 7.8 mg/dl, direct – 5.2mg/dl, indirect- 2.6 mg/dl, SGOT – 822 U/L, SGPT – 936 U/L, Alkaline phosphatase – 310 U/L , total protein 7.4 mg/dl , albumin- 4.2 mg/dl, globulin 3.2 mg/dl , serum lipase – 1746 u/l , serum amylase 922 u/l, Prothrombin time was 46 seconds and INR was 2.34, sodium- 142, potassium -3.7, urea- 35 mg/dl, creatinine- 0.73 mg/dl, blood smear for malarial parasites was negative. HIV, Hepatitis B surface antigen, Hepatitis C, Hepatitis A IgM and Hepatitis E IgM were also negative. ABG revealed metabolic acidosis. Sonography of abdomen revealed fatty liver and mild ascitis. Chest x ray was normal. Furthur CECT Abdomen was done which revealed diffuse low attenuation of hepatic parenchyma with sparing of quadrant lobe. Patient was treated with intravenous fluids, fresh frozen plasma and whole blood, vitamin K, cefotaxime, omeprazole, ondensetron, l-ornithine-l-aspartate and lactulose enema. Her vitals were checked at regular intervals. After about 4 days she showed clinical improvement. Blood investigations were repeated daily which showed improvement gradually, liver functions normalized on 10th day. Amylase and lipase levels came to normal level thereafter. With all parameters being normal, she was discharged on 14th day.

DISCUSSION

Phosphide is one of the most common mode of suicidal poisoning in developing countries which is readily available as rodenticides, military ammunition, fire crackers, and fertilizers. The acid in the digestive system of the rodent reacts with the phosphide to generate the toxic phosphine gas. It is a protoplasmic poison which inhibits various enzymes and protein synthesis leading to multi organ failure. It usually goes into three phases. First phase within 24 hours which is asymptomatic period and the patient may be discharged prematurely.[1] Second phase is asymptomatic elevation of liver enzymes. Third phase of advanced liver disease and resolution after few days of poisoning.[1] Rodenticide poisoning usually presents with gastrointestinal effects[2] which may proceed to cause acute hepatic failure, coagulopathy, and deranged liver function,[3,4] and it was witnessed in our patient. Early elevations in transaminase, alkaline phosphatise, derangement in prothrombin time, metabolic acidosis associated were significantly associated with mortality.[5] In addition, our patient had pancreatitis. It may also cause neurological manifestations, cardiac toxicity and renal failure. As there is no specific antidote for yellow phosphorous, supportive therapy, gastric lavage with potassium permanganate are the usual mode of treatment given. Further management aimed at regular monitoring of liver and renal functions and liver transplantation[4] if necessary. Poor prognostic factors are shock, altered mental status, high APACHE II score, acute kidney injury, low prothrombin rate, hyperleucocytosis, requirement of mechanical ventilation, lack of vomiting after ingestion, hyperglycemia and time lapsed after exposure.[6] Emphasis should be on prevention by restricting access to the poison, indiscriminate use of yellow phosphorus in the manufacture of fireworks should be eliminated. High degree of suspicion by the treating physician, expecting the effects and complications of phosphorous, avoiding premature discharge and followup of the patient for 5 to 7 days after poisoning with biochemical marker evaluation is of paramount importance.

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