[Downloaded free from http://www.toxicologyinternational.com on Tuesday, April 26, 2016, IP: 14.139.224.243]

Successful Management of Rodenticide Induced Acute Liver Failure in a Patient

Sir,

A20-year-old female presented to us with alleged history of consumption of 10 g of rat poison (zinc phosphide) around 10.30 am and was received in casualty at 11.00 am, with complaint of nausea and three episodes of vomiting.

There was no history of breathlessness, hemoptysis, hematemesis, abdominal pain, bleeding, dyspnea, and seizures. On examination; afebrile, pulse - 96/min, respiratory rate - 20 cycles/min, blood pressure - 110/70 mmHg, and other systemic examinations were normal.

Potassium permanganate gastric lavage was given to the patient. She was admitted and observed. Investigations: Routine hemogram and liver and kidney function tests were normal. Coagulation profile was normal.

On the 2nd day, patient became drowsy.

General examination: Pulse- 44/min, BP 148/98 mmHg, and icterus present. Per abdomen: Liver span -5 cm in right midclavicular line. Neurological examination: Glasgow coma score- 7/15(E 2 M 3 V 2), brainstem signs: Dolls eye present, pupils were semi dialated sluggish lyre acting to light, and plantars were bilateral extensor. Over next 4 h she developed deep coma.

Investigations revealed: Blood glucose - 68 mg%; international normalized ratio (INR)- 5.72; prothrombin time (PT)- 2 min 44 s; total bilirubin 5.89 mg%; unconjugated bilirubin- 1.88 mg%; conjugated bilirubin- 4.01mg%; aspartate transaminase (AST)- 677 U/L; alanine transaminase (ALT)- 755 U/L; serum ammonia- 46.8 mg/dl; kidney function test (KFT) serum urea - 43 mg%, creatinine- 1.41 mg%, sodium - 134 meq/L, and potassium - 4.4 meq/L; arterial blood gas (ABG) normal; fundus examination showed- early papilledema; computed tomography (CT) scan of brain- normal; acute viral markers for hepatitis A virus (HAV), HBV, HDV, and HEV negative; and malaria and leptospira infection ruled out.

In view of coma - coagulopathy and altered liver enzymes -acute liver failure was suspected.

She was treated with dextrose, endotracheal intubation and mechanical hyperventilation, mannitol, lactulose, high bowel washes, fresh frozen plasma, proton pump inhibitors, and intravenous (IV) prophylactic antibiotics.

The patient gradually showed signs of recovery over next 4 days and completely recovered in next 1 week.

DISCUSSION

Zinc phosphide is a highly potent rodenticide that was first registered in 1947. It has a wide range of uses commercially and residentially, including the protection of food crops and grasses. It may also be used as an insecticide.[1] Zinc phosphide targets household rodent pests, such as mice and rats, in addition to field rodents including voles, ground squirrels, pocket gophers, prairie dogs, and jack rabbits.

Zinc phosphide is converted into phosphine via ingestion, which is the compound which causes its high toxicity. It is a protoplasmic poison which inhibits various enzymes and protein synthesis. 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 pulmonary edema supervenes, though whether this is a cardiogenic or noncardiogenic is not always clear.[2,3]

Zinc phosphide which is used as rat poison contains inorganic phosphorous which is hepatotoxic. Inorganic phosphorous can be lethal in small doses. Usually, rodenticide poisoning presents with gastrointestinal and cardiovascular side effects.[2] Acute liver failure though rare is one of the most dangerous side effects. That's why it should always be kept in mind while treating such cases. Interestingly, liver failure signs and symptoms and biochemical parameters for liver failure may become abnormal even after 3–4 days of consumption of toxin, so meticulous clinical examination for signs of liver failure and repeated liver function test, coagulation profile should be done.[4,5] Early arrival to the hospital, potassium permanganate gastric lavage and absence of other bad prognostic indicators like cardiovascular instability, arrhythmias, adult respiratory distress syndrome (ARDS), anoxia, gastrointestinal bleeding, and seizures might have also contributed to recovery in our case.[6] We treated the liver failure in the general lines of management of fulminant hepatic failure.

[Downloaded free from http://www.toxicologyinternational.com on Tuesday, April 26, 2016, IP: 14.139.224.243]

Sourya Acharya, Samarth Shukla[1], Nitin Raisinghani

Departments of Medicine, and [1]Pathology, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Science Sawangi (Meghe) University, Wardha, Maharashtra, India

Address for correspondence:
Dr. Sourya Acharya,
Department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Science Sawangi (Meghe) University, Wardha - 442 004, Maharashtra, India.
E-mail: souryaacharya@yahoo.co.in

REFERENCES

1. Bumbrah GS, Krishan K, Kanchan T, Sharma M, Sodhi GS. Phosphide poisoning: A review of literature. Forensic Sci Int 2012;214:1-6.
2. Sogut O, Baysal Z, Ozdemir B. Acute pulmonary edema and cardiac failure due to zinc phosphide ingestion. J Emerg Med 2011;40:e117-8.
3. Proudfoot AT. Aluminium and zinc phosphide poisoning. Clin Toxicol 2009;47:89-100.
4. Chugh SN, Aggarwal HK, Mahajan SK. Zinc phosphide intoxication symptoms: Analysis of 20 cases. Int J Clin Pharmacol Ther 1998;36:406-7.
5. Karanth S, Nayyar V. Rodenticide-induced hepatotoxicity. J Assoc Physicians India 2003;51:816-7.
6. Mostafazadeh B, Farzaneh E. A novel protocol for gastric lavage in patients with aluminum phosphide poisoning: A double-blind study. Acta Med Iran 2012;50:530-4.

Access this article online

Quick Response Code:

Website: www.toxicologyinternational.com

DOI: 10.4103/0971-6580.155394