Rodenticide-induced Hepatotoxicity

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Abstract
Hepatotoxicity following acute poisoning with rodenticides has been infrequently reported in literature. To emphasize the fact that this form of clinical presentation is not unusual we are reporting two cases of rodenticide poisoning masquerading as severe hepatic dysfunction.

INTRODUCTION

Acute poisoning with rodenticides is an uncommon problem but an important cause of death among patients admitted to the intensive care unit (ICU) with attempted suicide. Most of the reports in Indian literature on rodenticides highlight the toxicity of zinc and aluminium phosphide that presents as cardiovascular and multisystem failure. Medical literature from the West on the other hand, describes rodenticide poisoning presenting as a hemorrhagic syndrome as warfarin-like compounds is the main form of commercially available rodenticides in those countries. Hepatotoxicity following an acute poisoning with rodenticides has been infrequently reported in literature. A review of recent Indian literature revealed only one case series that reported three cases of hepatocellular dysfunction amongst six patients of zinc phosphide poisoning with suicidal intent.

The following report describes two patients with rodenticide poisoning who presented to us with severe liver dysfunction. One of the patients (Case 1) died due to acute hepatocellular necrosis and fulminant hepatic failure.

CASE REPORT

Case 1

A 21 years housewife consumed a phosphorus-based rodenticide six days prior to admission into our ICU. Following the ingestion no immediate first-aid was administered. Twenty-four hours later she developed vomiting and abdominal pain for which she was taken to a peripheral hospital where she received primary care in the form of a stomach wash and some other symptomatic therapy before being discharged. Forty-eight hours after the alleged ingestion she again developed intractable vomiting and thus was shifted back to the same hospital where she was admitted this time. On the sixth day after the incident in view of worsening of her sensorium she was referred to our institution. She reached the ICU in shock (with a blood pressure of 80 systolic, pulse rate of 160/minute and cold peripheries), febrile (temperature 100.6°F.) and \( O_2 \) on saturation 95% on room air. Systemic examination was normal except for the presence of a decreased Glasgow Coma Scale (GCS) of 7/15 (Eye opening 1, Verbal response 1, Motor response 5). Investigations on admission revealed an acute hepatitis-like picture with a total bilirubin 6.1mg%, ALT 826U/L, AST 389U/L and prolonged prothrombin time of greater than two minutes. Serum ammonia was also elevated to 46.2 mcg/dl. Arterial blood gases revealed well compensated metabolic acidosis. Sonographic imaging revealed fatty liver with minimal ascites. Thus a diagnosis of acute fulminant hepatic failure secondary to toxic hepatitis following a rodenticide poisoning was made. Accordingly she was started on antihepatic precoma measures, low-dose dopamine and other symptomatic measures. Infective causes of acute hepatitis including malaria, leptospira and other viral markers were negative. Through the next forty-eight hours the patient showed gradual worsening of her sensorium which dropped to a GCS of 4/15 by day three of admission (ninth day following the alleged ingestion of poison). She continued to be in this state till the fifth day following admission (eleventh day after the alleged ingestion), when further worsening of her sensorium was noted. By now she had also developed hemodynamic instability requiring increasing levels of inotropic support. On the following day she had an episode of bradycardia followed by a ventricular fibrillation. She was resuscitated and placed on mechanical ventilation. However through the course of the day, she worsened and expired at 6 PM that day.

A post-mortem liver biopsy was done which showed collapsed reticulin framework with fibrosis between the hepatocytes showing a bubbly and vacuolated cytoplasm suggestive of an acute fulminant hepatitis (Figs. 1 and 2).

Case 2

A 25 years housewife presented with alleged history of ingestion of an unknown quantity of a rodenticide, Ratol (containing 3% yellow phosphorus) mixed with alcohol about
five days prior to admission. There were no symptoms immediately following the ingestion. However she confessed to having taken the poison four days after the incident. Following primary care in a nearby hospital she was referred to our institution. Examination revealed her to be icteric, with normal vitals and presence of a soft, non-tender hepatomegaly. Investigations at the time of admission revealed a picture of acute hepatitis with total bilirubin of 7.8mg%, direct bilirubin of 3 mg%, ALT 1055U/L, AST 824U/L, alkaline phosphatase 13 U/L and a prolonged PT of over two minutes. An abdominal ultrasonography revealed hepatomegaly with diffuse increase in echotexture of liver with areas of sparing associated with ascites.

Through her stay in the hospital initially there was increasing icterus that was noticed which was also supported by a biochemical evidence showing increasing total bilirubin till about the seventh day of admission, following which there was a gradual decline in the serum bilirubin levels. However all through this period there was a steady decrease in, serum ALT and AST levels associated with an improvement in the prothrombin time suggesting improving hepatic function. smear for MP was repeatedly negative. Hepatotropic viral markers were also negative. Patient was administered vitamin-K for three days along with other symptomatic measures. She was admitted in the hospital for a period of two weeks and later discharged. At the time of discharge her liver function tests read as follows: Total bilirubin 3.5mg%, ALT 245U/L, AST 136U/L and prothrombin time 22.3 seconds.

**DISCUSSION**

Hepatotoxicity due to ingested poisons can occur due to a wide variety of chemical compounds. Inorganic phosphorus is a potent hepatotoxin that is still widely available in our country as a household rodenticide. Our report of two cases highlights the dangers of inorganic phosphorus as a toxin that is known to be lethal even in miniscule quantities. The first patient in this case report died with fulminant hepatic failure following ingestion of a phosphorus-based rodenticide. Liver biopsy in this patient showed classical signs of acute hepatocellular necrosis as described above. The second patient also had biochemical evidence of acute hepatitis along with features of hepatocellular failure but survived.

Our case report emphasizes the fact that presentation with hepatocellular dysfunction following rodenticide ingestion is not unusual. With increasing focus on aluminium and zinc phosphide, the significance of inorganic phosphorus as a rat poison is often disregarded. Features of hepatotoxicity with inorganic phosphorus often develop 72 hours after ingestion of the poison. During this time, the patient has only minor gastrointestinal symptoms or no symptoms at all. Unless looked for specifically clinical evidence of icterus or an abnormality in liver function tests can be missed and elevation of prothrombin time can be wrongly attributed to a warfarin containing rodenticide.

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**References**