Annals of Health & Health Sciences
Vol. 1, No. 1 January-June 2014, 68-70
DOI: 10.5958/j.2322-0422.1.1.014

Case Report

Yellow Phosphorus Poisoning

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ABSTRACT

Acute liver failure from yellow phosphorus poisoning is uncommon and has been rarely reported. Firecrackers or rodenticide pastes containing yellow phosphorus may be ingested accidentally or with suicidal intent. Acute poisoning can manifest with features of gastrointestinal tract, hepatic, cardiovascular, central nervous system and renal involvement, which can rapidly progress to multiorgan failure and death. The management is mainly supportive but liver transplantation has been done successfully to salvage the patients of acute liver failure. We report a case of suicidal ingestion of rat killer paste with acute liver failure who succumbed to poisoning.

Keywords: Yellow phosphorus, Poisoning, Acute liver failure, Rodenticide, Warfarin

INTRODUCTION

Yellow phosphorus (also referred as white phosphorus) has been used in rodenticides, firecrackers, fertilizer industry and military ammunition. Yellow phosphorus has received less attention as a constituent of rat poisons compared to others like zinc phosphide, aluminum phosphide and warfarin. In developing countries, poisoning commonly results from ingestion of rodenticide pastes with suicidal intent or firecrackers accidentally. Case fatality, as high as 20–50%, has been reported in severe cases.[1] Complications involve acute toxic hepatitis with liver failure, renal failure, encephalopathy, cardiac arrhythmias, profound cardiogenic shock, coagulopathy and bleeding.[2] Extensive skin burns and systemic toxicity may result from yellow phosphorus explosions.[3] We report a case of suicidal ingestion of rat killer paste with acute liver failure who succumbed to poisoning.

CASE REPORT

A 24-year-old married lady was brought with alleged ingestion of unknown quantity of rat killer paste (containing 3% white phosphorus) 2 days prior to the hospital admission. She presented with nausea, vomiting and abdominal pain. There was no bleeding gums or petechial haemorrhages. On day 2 of hospital admission, she developed deepening of jaundice, irritability and altered behaviour (fourth day post ingestion). On admission (day 3 post ingestion), she was afebrile, haemodynamically stable, icteric and was in grade 2 hepatic encephalopathy. Her chest, cardiovascular and abdominal examinations were unremarkable. Laboratory evaluation revealed conjugated hyperbilirubinemia, elevated liver enzymes, coagulopathy, renal dysfunction, normal serum calcium and phosphate levels and thrombocytopenia (Table1). The markers of acute viral hepatitis (HBsAg, anti-HAV IgM and anti-HCV antibodies) were negative. Ultrasonographic scan of the abdomen with colour Doppler evaluation of hepatic venous outflow was normal. Urinary pregnancy test was negative.

She was admitted to intensive care unit for continuous haemodynamic monitoring. She was treated intravenous fluids, lactulose, metronidazole, vitamin K and mannitol. Her encephalopathy worsened to grade 4 on day 2 of hospital admission. She was intubated and put on

mechanical ventilation. On day 2 of hospital admission, she developed progressive hypotension and had to be started on vasopressors (noradrenaline and dopamine). The arterial blood gas analysis revealed severe metabolic acidosis. She developed refractory shock, anuria and later succumbed on day 2 of hospital admission (fourth day post ingestion) despite all the measures.

Table 1: Laboratory investigations

Lab parameter	Day1*	Day2*
Hb (g/dl)	11	11.7
WBC (per µl)	4,000	6,000
Platelets (per µl)	56,000	40,000
T.bilirubin (direct), mg/dl	4.8(2.6)	7.2(3)
ALT (U/l)	440	680
AST (U/l)	400	450
ALP (U/l)	100	120
Serum albumin (g/dl)	3.8	3.6
Serum creatinine (mg/dl)	1.8	2.5
Prothrombin time (s)	92	86
Serum calcium (mg/dl)	8.0	8.4
Serum phosphate (mg/dl)	5.2	4.8

Abbreviations: ALT - alanine aminotranferease; AST - aspartate aminotransferase; Hb - haemoglobin; WBC -white blood count: ALP - alkaline phosphatase; *Day of hospital admission.

DISCUSSION

White phosphorus is a highly toxic inorganic substance that readily catches fire at room temperature. This accounts for the classical description of 'smoking' and luminescence of vomitus and stool.[4] It is readily absorbed from gastrointestinal tract, respiratory tract, skin and mucus membranes. The mechanism of toxicity may involve abnormalities of ribosomal function and protein synthesis, defective regulation of glucose metabolism,[5] abnormalities of lipoprotein synthesis and secretion of triglycerides leading to intracellular lipid accumulation with fatty degeneration of organs like liver, kidney, brain and heart.[6]

The clinical course of acute poisoning following ingestion may involve three stages.[2] In stage 1 (up to 24 h), nausea, vomiting and abdominal pain predominate. In stage 2 (latent stage, between 24 and 72 h), laboratory evaluation and liver biopsy may show early toxic hepatitis changes but patient may be asymptomatic. In stage 3 (after 72 h), clinically overt hepatitis (with or without acute liver failure), acute renal failure and, in severe cases, multi-organ failure can ensue. Liver histology at this stage may show steatohepatitis and necrosis.[7] Renal biopsy may show acute tubular necrosis. Some patients depending on the dose consumed may recover completely and some may have a variant course with more rapid progression.[1]

Among patients who ingested fireworks, 27% developed fulminant hepatic failure and succumbed in a study.[7] More than 10 times elevation of alanine transaminase, severely prolonged prothrombin time, hypoglycemia and metabolic acidosis predicted adverse outcomes. Cardiovascular complications like arrhythmias, hypotension and refractory cardiogenic shock have been described.[5] Acute bone marrow suppression with cytopenias has also been reported.[8] There is no specific diagnostic test and serum phosphate levels are not useful for the diagnosis.[2]

Treatment is mainly supportive and is directed at removal of the poison, as there is no specific antidote for yellow phosphorus. Gastric lavage with potassium permanganate is recommended to convert the phosphorus to relatively harmless oxides. The yellow phosphorus as the hazard of chemical skin burns and direct contact with vomitus and stools should be avoided while managing the patients. The hepatic and renal function should be monitored carefully. *N*-Acetyl cysteine did not significantly alter outcomes of hepatotoxicity in a study.[7] Where facility is available, suitable patients of liver failure should be offered liver transplantation as there are reports of successful rescue of patients with transplantation.[6] Silver nitrate solution can be used for treatment of cutaneous burns.

CONCLUSION

To conclude, acute yellow phosphorus poisoning can result in fatal acute liver failure with multi-organ dysfunction that can lead to death in severe cases. Management is mainly supportive and liver transplantation can be used to rescue patients with acute liver failure.

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How to cite this article: Praveen Kumar AS, Praveen Kumar BA, Anupama MP. Yellow Phosphorus Poisoning. Ann Health & Health Sci 2014; 1(1): 68-70.