Fulminant Hepatic Failure with White Phosphorus: A Case Report

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ABSTRACT

A 3-year-old female child was brought with alleged history of consumption of rat poison (white phosphorus) 6 days prior to admission. She was in stage I hepatic encephalopathy with coagulopathy. She was managed symptomatically but did not respond and worsened to stage III hepatic encephalopathy. In view of progressive deterioration, a liver transplantation was planned. However, before embarking on that, a trial of N-acetylcysteine was given, following which patient improved dramatically.

Key words: White phosphorus, hepatic failure, N-acetylcysteine

Introduction

Acute poisoning with white (or yellow) phosphorus is relatively uncommon in Kerala. White phosphorus is used as a rodenticide, cockroach poison, and in the manufacture of incendiaries and fireworks. Lethal hepatotoxicity following acute poisoning with white phosphorus is well known, but has been infrequently reported in literature.

The following report describes a child with white phosphorus poisoning who presented to us with severe liver dysfunction and responded to a trial of N-acetylcysteine, suggesting the need for additional studies to determine its efficacy in phosphorus induced liver failure.

The Case

A 3 year old girl who was apparently well with normal growth and development was brought with complaint of jaundice. There was a history of alleged accidental consumption of rat poison 6 days ago. At the time of the incident, the child had vomited, and after that she had been taken to a local hospital where her symptoms subsided, and she was discharged after 2 days. However, on day 4, she developed jaundice, and subsequently just prior to being brought to our hospital, she became drowsy and lethargic. There was no history of bleeding tendency.

Physical examination revealed a drowsy child who was arousable by verbal commands; HR of 90/min, RR of 30/min, and BP was 94/83 mmHg. She displayed deep jaundice. Liver was just palpable with span of 7.5cm, and was nontender. CNS examination did not reveal any focal deficits. A provisional diagnosis of Stage I hepatic encephalopathy was made. Laboratory investigations revealed the following: Total bilirubin - 9.2 mg/dl, Direct bilirubin - 4.9 mg/dl, AST - 988 IU/L, ALT - 1905 IU/L, S. Ammonia – 76 umol/L, PT - 178 s (control: 12.8s), INR - 32, APTT - 74s (control: 32s), T. Protein - 6.3g/dl, S.Albumin - 3.9 g/L, and RBS - 85mg/dl; USG Abdomen was normal.

The child was immediately placed on standard hepatic encephalopathy treatment regimen including FFP for the coagulopathy. She however did not respond and worsened to stage III hepatic

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encephalopathy, and also developed profound hypoglycemia. LFT and other parameters worsened. Toxicological analysis failed to reveal phosphorus in urine and blood. Other possible etiologies for hepatic failure including viral and leptospiral causes were ruled out by relevant investigations. The manufacturer of the rat poison was contacted and presence of white phosphorus as an ingredient was confirmed. In view of progressive deterioration, a liver transplantation was planned as advised by the transplant surgeon. However, a trial of N-acetylcysteine was given as a last resort, following which surprisingly she gradually improved, both clinically and biochemically. At the time of discharge she was active and playful even though she still displayed jaundice. At 4 weeks follow up, she was totally anicteric with normal LFT.

Discussion

Systemic white phosphorus poisoning may occur following oral, inhalational, and dermal exposure. Phosphorus is a protoplasmic poison and is a potent hepatotoxin. Toxicity is enhanced when phosphorus is mixed with alcohols, fats, or oils. The fatal dose is said to be roughly 1 mg/kg or 60 mg. Clinical effects following acute poisoning have classically been divided into 3 stages, with an initial gastrointestinal stage characterized by vomiting, diarrhea and abdominal pain[1]. Vomitus and stools may be luminescent, and may have a garlicky odor. Faint fumes may emanate from the stools (*smoky stool syndrome*).[2,3] The second stage which may last for several days, is an essentially symptom free period, while the third stage could terminate in acute liver and renal failure with metabolic derangements. This stage is due to the systemic effects of phosphorus after it has been absorbed. Early hypoglycemia carries a grave prognosis.

Management of acute poisoning includes the use of activated charcoal, and gastric lavage with potassium permanganate (1:5000), which oxidizes phosphorus to relatively less toxic phosphoric acid and phosphates. Alternatively, a 0.2% solution of copper sulphate may be used for stomach wash, which converts phosphorus to non-toxic copper phosphide. It is preferable to avoid milk or any oily or fatty foods since this will enhance the absorption of phosphorus. Supportive measures include the management of hypotension, hypoglycemia, seizures, coagulopathy, and arrhythmias, including torse de pointes.

Available data indicate that phosphorus induced liver damage may be a free radical mediated process. Administration of antioxidants could therefore play a role in mitigating the resultant tissue injury.[4] Reports on N-acetylcysteine in phosphorus poisoning point to its efficacy in preventing progression of liver damage during stage I of phosphorus poisoning. A dose regimen of 150 mg/kg in 200 ml of D5W for 15 min, then 50 mg/kg in 50 ml of D5W for 4 hrs, followed by 100 mg/kg in 1000 ml D5W for 16 hrs has been suggested, and was followed in this case.[5]

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