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Acute hepatic failure due to yellow phosphorus ingestion

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Abstract

We report a case of three year old girl, who was brought to hospital for accidental consumption of rat-poison (3% phosphorus). The patient was asymptomatic for first 48 hours. Later on she developed the symptoms of hepatic failure. She was managed conservatively and was discharged after 14 days.

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Full Text

Introduction

Elemental phosphorus exists in two forms--red and yellow. 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. The most readily available source of yellow phosphorus today is rodenticides. Rodenticides are available as powders or pastes containing 2 to 5% of yellow phosphorus. [1] We describe here a case of acute yellow phosphorus poisoning that led to fulminant hepatic failure and eventually, the patient recovered with conservative management.

Case Report

A 3-year-old girl was brought to the hospital with an alleged history of accidental consumption of an unknown quantity of rodenticide paste (Ratol, containing 3% yellow phosphorus). The child had mistaken it to be tooth-paste. She was made to rinse her mouth with salt water and remained asymptomatic until 8 hours when she started vomiting. She was then brought to the hospital 14 hours after poisoning due to persistent vomiting. There was no history of lethargy, altered sensorium, or convulsions. There was no history of jaundice, abdominal pain,

breathlessness, or oliguria.

On admission, the child was conscious and afebrile with a heart rate of 122/minute and respiratory rate of 32/minute. Blood pressure was 90/70 mm Hg in right upper arm in the supine position. Mild pallor was present, but no icterus or edema was seen. The liver was palpable 2 cm below the costal margin, soft, and with rounded margins. The liver span was 7 cm. Other systems were essentially normal. The child was admitted and gastric lavage done. There was no garlicky odor.

Results of the investigations done on the day of admission and subsequently are shown in [Table 1]. Blood smear for malarial parasites was negative. Hepatitis B surface antigen and hepatitis A IgM were also negative. Abdominal sonography done on day 2 showed mild hepatomegaly.{Table 1}

On the second hospital day, the child developed fever and was noticed to be sleeping excessively and not taking feeds. A diagnosis of stage 1 hepatic encephalopathy was made and the patient was given intravenous (IV) fluids with 10% dextrose, IV cefotaxime vitamin K, injection ranitidine, and oral ampicillin. She was also started on oral lactulose. Intake and output was strictly monitored and blood glucose was measured six-hourly.

Despite initiation of antihepatic failure therapy, the child progressed to stage 3 encephalopathy on day 3 and had malena for which she was administered fresh frozen plasma. Packed red cells were also transfused. After a plateau period of 4 days, the sensorium started improving on the 8 th hospital day, with a simultaneous improvement in her laboratory parameters [Table 1]. Thereafter, the child made a complete recovery and was discharged on day 19.

Discussion

Yellow phosphorus is an inorganic substance used in military ammunition, fire crackers, fertilizers, and as rodenticide. It emits smoke and has very strong garlicky odor. It can get absorbed through skin, mucus membrane, respiratory and gastrointestinal epithelium. Intoxication occurs with suicidal or accidental ingestion. After absorption, it is distributed to all tissues, particularly the liver, and the peak level is reached after 2 to 3 hours of toxic oral ingestion. Bile salts are important for absorption of phosphorus. Because of water content and low oxygen tension, phosphorus remains stable in gut for longer period. [1]

Phosphorus is a general protoplasmic poison causing cardiac, hepatic, renal, and multiorgan failure. The patient with white phosphorus intoxication passes through three stages. The first stage occurs during the first 24 hours in which patient is either asymptomatic or has signs and symptoms of local gastrointestinal irritation. The second stage occurs between 24 to 72 hours after ingestion. It is an asymptomatic period and the patient may be discharged prematurely. There may be mild elevation of liver enzymes and bilirubin in this stage. The third stage (advanced) occurs after 72 hours until the resolution of symptoms or death. [1]

Patients may present with acute hepatic failure, coagulopathy, and deranged liver function, as was witnessed in our patient. [2] Some patients may develop acute tubular necrosis and present with acute renal failure. Central nervous system effects include changes in mental status like confusion, psychosis, hallucinations, and coma. Cardiac toxicity includes hypotension, tachycardia, arrhythmias, and cardiogenic shock. [3]

There is no specific antidote for yellow phosphorus. Treatment is directed at removal of the poison and supportive therapy. Gastric lavage with potassium permanganate is recommended to convert the phosphorus to relatively harmless oxides. Careful monitoring of hepatic and renal function and management of their failure is required. Liver transplantation has been done in suitable candidates for acute hepatic failure. [4]

Fernandez and Canizares in a series of 15 patients have reported a mortality of 27%, confirming that yellow phosphorus is extremely lethal when ingested. [2]

The safest way to deal with such a lethal substance would be prevention. The indiscriminate use of yellow phosphorus in the manufacture of fireworks should be eliminated. Since rodents are developing resistance to rodenticides containing warfarin, rat poisons containing yellow phosphorus are making a big come-back. The yellow phosphorus rodenticides pose a special problem in that the product directions suggest that the paste be applied to bread to enable ingestion by rodents, thus making it appealing to children as well. [5] Physicians should therefore be aware of the toxicity and its management.

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