Case Report

'Para-suicide Pact' by Yellow Phosphorus Ingestion

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ABSTRACT

Suicide by yellow phosphorus poisoning is not very common in some parts of Karnataka state of India, such as Mysore. We report two cases of deliberate suicide attempt by yellow phosphorus poisoning (Ratol – yellow phosphorus 3%) involving a 21-year -old man and a 17-year-old woman. Both were asymptomatic at the time of admission and they developed symptoms of poisoning only on the third day. Both patients recovered completely with conservative management supplemented by N-acetyl-cysteine (NAC) and discharged on the ninth day.

Key Words: Phosphorus; Rat poison; Rodenticide; N-acetylcysteine; NAC; Para-suicide pact

INTRODUCTION

Acute poisoning with rodenticides is not an uncommon problem in India, and mortality among patients admitted to the intensive care unit (ICU) due to attempted suicide with these compounds is quite high. Yellow phosphorus is used in some rodenticides, besides being also used in fertilizers, water treatment, and in fireworks. Rodenticides containing phosphorus are available as powders or pastes containing 2–5% of yellow phosphorus.[1] There is no specific antidote for yellow phosphorus. The use of NAC as an adjuvant in the management of yellow phosphorus poisoning improves survival when presented early.[2] We present two cases of acute yellow phosphorus poisoning by consumption of rat paste (Ratol) as part of a 'para-suicide pact' admitted to Cauvery Hospital, a tertiary care hospital in Mysore, Karnataka.

Case 1: A 21-year-old male was brought to the hospital with a history of 'para-suicide pact' by consumption of half tube of rodenticide paste (Ratol, containing 3% yellow phosphorus). He was in love with a girl for 3 years which came to the notice of both their parents who refused this alliance. Upset with this, both of them consumed yellow phosphorus poison. He was treated with gastric lavage at a local hospital and brought to our centre five hours after consumption.

On admission, patient was conscious, alert and obeying commands. He was afebrile and his vital signs were normal. No icterus or oedema was seen. Systemic examination was unremarkable. Baseline haematological and biochemical investigations were normal except for a mildly elevated prothrombin time (PT). His liver function tests and renal function tests were normal on admission. Results of relevant investigations from the day of admission to the time of discharge are depicted in **Table 1**. Ultrasound abdomen also revealed normal findings.

He was admitted to the ICU following gastric decontamination, and subjected to supportive measures, including intravenous fluids, dextrose injections, and proton pump inhibitors, along with antiemetics. He complained of mild gastrointestinal symptoms such as nausea and abdominal discomfort, but remained largely asymptomatic.

In view of deranged PT, he was started on vitamin K 10 mg IV. He was initiated on N-acetyl cysteine IV at a dose of 150 mg/kg (loading dose) in 250 mL of 5%

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Table 1 Results of Investigations from Day of Admission to Discharge – Case 1

Day	1	2	3	4	5	6	7	8
Prothrombin time (sec) test / control		19.7 /15	19.3/14	21.6/14	23.6/15	27.4/15	19.4/ 15	15/15
INR		1.35	1.3	1.5	1.5	1.8	1.3	1
SGOT	29 U/L				41 U/L		63 U/L	
SGPT	36 U/L				45 U/L		70 U/L	

dextrose over one hour, followed by a maintenance dose of 50 mg/kg in 500 mL of 5% dextrose over 4 hrs. Subsequent maintenance doses were given as an infusion of 10 mg/kg for 7 days. He was closely monitored for coagulation abnormalities and signs of liver failure. On day 3, his GI symptoms increased with severe nausea and vomiting, and he could not tolerate even small sips of fluids. He also developed lip ulcerations. The coagulation profile deteriorated further. He was managed conservatively with IV fluids, proton pump inhibitors, and antiemetics. On day 5, he developed mild icterus in addition to his GI symptoms and also had deranged liver functions, which worsened further on day 7. After that however, he made a steady recovery, the GI symptoms resolved, and he started taking oral feeds. Liver function improved and jaundice gradually disappeared. He was discharged on day 9 after psychiatric counseling.

Case 2: A 17-year-old girl was brought to the hospital with an alleged history of 'para-suicide pact' by consumption of half a tube of rodenticide paste (Ratol, containing 3% yellow phosphorus). On admission, she was conscious, alert, and obeying commands. She was afebrile, her vital signs were normal, and general examination was unremarkable. All systems were normal. She was admitted to the ICU, after thorough gastric decontamination in the emergency room (ER).

Her baseline haematological and biochemical investigations were normal. Renal parameters and liver function tests were also normal, except for mildly increased prothrombin time (PT). She was monitored daily for signs of liver failure. Liver enzymes and PT, INR, were checked daily, the results of which are tabulated in **Table 2**. She was treated with intravenous fluids, dextrose, proton pump inhibitors and antiemetics. She was also given vitamin K 10 mg IV OD, in view of raised PT, and for the same reason was also started on N-acetylcysteine which was continued for 3 days in the same dose, and later was given orally for the next 3 days at 600 mg thrice a day. On day 3, she developed mild vomiting 3–4 times, which settled the next day. She remained asymptomatic showing no signs of liver failure whatsoever, and was discharged on day 7 after psychiatric counseling.

DISCUSSION

Yellow phosphorus is commonly used in rodenticides, fertilizers and in fire crackers. It is easily available in the market as 'Ratol' (3% yellow phosphorus - powder or paste containing 2–5% of the chemical). It is a protoplasmic poison and has very strong garlicky odour. It can get absorbed through skin, mucous membranes, and respiratory and gastrointestinal epithelium. After absorption, it is distributed to all tissues, particularly the liver, and the peak level is reached after 2–3 hrs of ingestion.[1]

Table 2 Results of Investigations from Day of Admission to Discharge – Case 2

Day	1	2	3	4	5	6	7	8
Prothrombin time (sec) test / control		21/15	21.1/15	22.5/15	19.9/15	19.6/15	16.6/15	15.5/15
INR	1.5	1.4	1.5	1.3	1.3	1.1	1.04	1
SGOT	23 U/L		26 U/L					
SGPT	28 U/L		33 U/L					

Poisoning occurs in substantial ingestions (suicidal or accidental) which may end up with cardiac, hepatic, renal, and multiorgan failure. The lethal dose is about 1 mg/kg or 60 mg.[3]

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 hrs in which the patient is either asymptomatic or has signs and symptoms of local gastrointestinal irritation.

The second stage occurs between 24–72 hrs 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.[2,4]

The third stage (advanced) occurs after 72 hrs until the resolution of symptoms or death. Patients may present with acute hepatic failure, coagulopathy or with deranged liver function. Liver transplantation may be the only resort when patients develop fulminant hepatic liver failure, and a few have underwent this successfully.[2]

Some patients may develop acute tubular necrosis and present with acute renal failure. Central nervous system effects include changes in mental status such as confusion, psychosis, hallucinations, and coma. Cardiac toxicity includes hypotension, tachycardia, arrhythmias, and cardiogenic shock.[4]

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. Results with treatment of N-acetylcysteine is conflicting in literature.[2,5] We went ahead and initiated these two patients for whatever benefit they may get and the relatively safe profile of the drug. 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.[5]

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 abolished. 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.[6] Physicians should therefore be aware of the toxicity and its management.

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