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Case Study

# A RARE CASE OF YELLOW PHOSPHOROUS POISONING WITH ACUTE CHOLESTATIC HEPATITIS, BICYTOPENIA, AND IMPENDING HEPATIC FAILURE

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**ABSTRACT**

Yellow phosphorus is a protoplasmic poison (a potent hepatotoxin). Fulminant poisoning results from ingestion of more than 1 to 2 g. An 18 y old adult male presented with ingestion of rodenticide poison containing 3 percent phosphorous in paste form. He was asymptomatic during the first day of hospitalization. He developed jaundice on the second day. On evaluation, his total leukocyte count was 1.800 ($10^9$/l) on the day of admission, which improved to 4.400 ($10^9$/l) on the 7th day. His platelet count was 1.53 ($10^9$/l) on the day of admission, which decreased daily till 0.80 ($10^9$/l) on the 7th day of hospitalization. His prothrombin time started deranging from the second day of admission with a crest of 38 s and INR 2.98 noted on the 5th day. He was given N-acetyl cysteine on the 5th day following which PT/INR began to improve and reached to a baseline normal. The regimen of N-acetyl cysteine used was a loading dose of 150 mg/kg IV over 1 h, followed by 50 mg/kg IV over 4 h and 100 mg/kg IV over 16 h. He was also treated with fresh frozen plasma and intravenous vitamin k. The man made a dramatic recovery and became stable.

**Keywords:** India, Kerala, Kottakkal, Phosphorous, Poisoning, N-acetyl cysteine, Hepatitis, Hepatic failure



## INTRODUCTION

Yellow or white phosphorus is a waxy, yellowish, crystalline solid with a garlicky odor. Yellow phosphorus is used in the military as an ingredient of smoke screens, tracer bullets, air-sea rescue flares and incendiary bombs. It is also used in the manufacture of matches, fireworks, rodenticide and fertilizers. Yellow phosphorus is a protoplasmic poison (a potent hepatotoxin). It causes direct hepatotoxicity typically resulting in periportal injury. There are several pastes and powders available in India that contain phosphorous. The usual fatal dose is approximately 1 mg/kg of body weight. Fulminant poisoning results from ingestion of more than 1 to 2 g. severe hypotension can occur as one of the major clinical features. Death usually pursues in 12 to 24 h. [1, 2].

The clinical manifestations in acute poisoning occur in three stages. The first stage occurs within 3 d characterized by abdominal pain, vomiting, and diarrhea. The second stage usually occurs several days after the first stage and is usually a symptom-free period. During the third stage manifestations of liver injury are prominent. Jaundice, tender hepatomegaly and finally hepatic encephalopathy with bleeding manifestations may occur [1, 2]. Management of yellow phosphorous poisoning is supportive. There is no antidote for yellow phosphorous poisoning. Liver transplantation may be lifesaving in candidates with acute hepatic failure. In acute poisoning, intravenous vitamin K is used to combat hypoprothrombinemia and fresh frozen plasma to correct coagulation defects.

## CASE REPORT

An 18 y old man presented with ingestion of rodenticide poison containing 3 percent phosphorous in the form of a paste. There were no vomiting, abdominal pain, bleeding manifestations and jaundice on presentation. He was asymptomatic for one day. He developed one episode of vomiting with abdominal pain the next day.

On evaluation, the patient had jaundice on the second day of admission. There was no evidence of hepatic encephalopathy. He had no pruritis. His total leukocyte count was 1.800 ($10^9$/l) on the day of admission, which improved to 4.400 ($10^9$/l) on the 7th day. He developed a spike of fever on the 5th day of hospitalization. His platelet counts were 1.53 ($10^9$/l) on the day of admission, which decreased daily from 1.28 ($10^9$/l) on the second day to 0.80 ($10^9$/l) on the 7th day of admission. Platelet counts reached normal levels on the 11th day. His prothrombin time started deranging from the second day of admission with a crest of 38 s and INR 2.98 noted on the 5th day. His total bilirubin levels mounted up to 10.5 mg/dl on the 9th day of admission with liver enzymes SGPT and SGOT peaking to 1722 U/l and 2954 U/l respectively. His Liver span varied from 9.5 cm on the day of admission to 9 cm on 4th day and 10.5 cm on the 7th day of hospitalization. The results of the investigations from the day of admission till recovery are shown in table 1. Serology for HbsAg, HCV, Dengue, Leptospira and hepatitis A were negative. Ultrasound scanning of the abdomen showed no significant findings.

**Table 1: Investigations are showing hepatitis and hematological involvement in yellow phosphorous poisoning.**

| Day | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|
| Total leukocyte Count ($10^9$/l) | 1.80 | 1.97 | 1.92 | 2.07 | - | - | 4.40 | 4.90 | 7.00 | 8.30 |
| Platelet Count ($10^9$/l) | 1.53 | 1.28 | 0.92 | 1.04 | - | - | 0.80 | 1.0 | 2.1 | 2.4 |
| Neutrophil ( %) | 58 | 41 | 41 | 48 | - | - | - | - | - | - |
| Prothrombin Time (s) | 15 | 22 | 31 | 29 | 38 | 22 | 14 | 14 | 15 | 14 |
| Hemoglobin (g/dl) | 17 | 18.2 | 17.3 | 16.7 | - | - | - | 13.7 | - | - |
| INR | 1.0 | 1.56 | 2.38 | 2.14 | 2.98 | 1.56 | 1.0 | 1.0 | 1.0 | 1.0 |
| Total Bilirubin (mg/dl) | 0.8 | 2.7 | 4.2 | 6.3 | 8.1 | 9.1 | 10.2 | 9.8 | 3.9 | 3.4 |
| Direct Bilirubin (mg/dl) | 0.2 | 1.0 | 1.4 | 3.0 | 4.8 | 4.5 | 5.2 | 4.0 | 1.3 | 1.5 |
| Indirect Bilirubin (mg/dl) | 0.6 | 1.7 | 2.8 | 3.3 | 3.3 | 4.6 | 5.0 | 5.0 | 2.6 | 1.9 |
| SGPT (U/l) | 18 | 36 | 44 | 937 | 1722 | 867 | 630 | 487 | 173 | - |
| SGOT (U/l) | 50 | 54 | 59 | 1617 | 2954 | 572 | 392 | 269 | 64 | - |
| ALP (U/l) | 418 | 440 | 408 | 625 | 832 | 713 | 640 | 512 | 504 | - |
| Liver Span (cm) | 9.5 | 9.5 | 9.5 | 9 | 9.5 | 10 | 10.5 | 10.5 | 9.5 | - |

He was treated with intravenous vitamin K, IV fluids, IV antibiotics and Fresh frozen plasma. In view of impending fulminant hepatic failure, patient's relatives were made cognizant of the need for liver transplantation and shifting the patient to a specialist center for liver transplantation if such a state of affairs took place. He was given N-acetyl cysteine, a loading dose of 150 mg/kg IV over 1 h, followed by 50 mg/kg IV over 4 h and 100 mg/kg IV over 16 h. Patient's PT/INR became stabilized. Thereafter, the man made a recovery and became stable. He was discharged from the hospital on day 15. On follow-up after 2 w INR remained 1.11 with SGPT 29 (U/l), SGOT 27 (U/l) and total bilirubin 1.8 mg/dl.

## DISCUSSION

Acute yellow phosphorous poisoning has been divided into 3 stages. The first stage is characterized by nausea, abdominal pain, and vomiting. This usually occurs within 24 h. All investigations may be normal. During the second stage, patient may be asymptomatic but with deranged liver function test. In the third stage, Acute liver failure occurs which may require liver transplantation [1-2].

AJ Tafur *et al.* reported a case where leukopenia with neutropenia occurred after 24 h of hospital admission with spontaneous resolution noted during the next 48 h [3]. In humans, yellow phosphorous poisoning present as a temporary proliferation of red blood cells and a decrease in the leukocyte count [4]. In our case, we noted a significant decrease in both leukocytes and platelet counts, which spontaneously resolved after 6$^{th}$ and 11$^{th}$ day respectively.

Management of yellow phosphorous poisoning is supportive. There is no antidote for yellow phosphorous poisoning. Liver transplantation may be lifesaving in candidates with acute hepatic failure [5].

Studies were done by Marin GA *et al.* have shown no clear benefit from N-acetyl cysteine, corticosteroids or exchange transfusion [6]. But in this case, our patient improved within 3 d after giving N-acetyl cysteine. His INR improved from 2.98 to 1.0 within 3 d and stabilized thereafter. The regimen of N-acetyl cysteine used was a loading dose of 150 mg/kg IV over 1 h, followed by 50 mg/kg IV over 4 h and 100 mg/kg IV over 16 h.

Fernandez *et al.* reported a mortality of 27 % in their case series, confirming that yellow phosphorus is highly toxic when ingested [7].

Mc Carron *et al.* reported mortality rates of 23 % for patients who had early symptoms of vomiting or abdominal pain; 73 % for those where the first manifestations of intoxication were restlessness, irritability, drowsiness, stupor or coma; and 47 % for patients who had a combination of both GI and CNS symptoms. Garlic odor, mucosal burns, and phosphorescent vomitus or feces occurred in only a small percentage of cases [8].

## CONCLUSION

An 18 y old male who ingested the paste form of 3 percent phosphorous went into cholestatic hepatitis with impending hepatic failure. He also had leucopenia and thrombocytopenia. He recovered within 3 d after giving N-acetyl cysteine (20 h regimen) which has shown clinical benefit in the presenting case.

## CONFLICT OF INTERESTS

Declare none

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