Rare Hit of Yellow Phosphorus Poisoning

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Abstract

Yellow phosphorus, also known as white phosphorus, on ingestion causes multi-organ dysfunction and has a high mortality rate. Hematological manifestations are rare. Here we present a 26-year-old man with selective myeloid series suppression and severe hepatic involvement following yellow phosphorus poisoning.

Introduction

Phosphorus exists as red, black, and yellow phosphorus. Red phosphorus is insoluble and unabsorbable therefore nontoxic. Yellow phosphorus is an ingredient in rodenticides, fertilizer, and fireworks. It is a protoplasmic toxin, major of its toxic effects affecting the gastrointestinal, hepatic, cardiovascular, and renal systems.

Keywords: Yellow phosphorus, hematological, neutropenia.

Case report

The twenty-six-year-old young man was brought with a history of intentional ingestion of a packet of ratol, containing 3g of rodenticide poison (yellow phosphorus) on 26-4-18 at around 8 pm. He had multiple episodes of vomiting, melena. Initially, he was admitted to an outside hospital and was managed conservatively with gastric lavage and N-acetyl cysteine. Initial investigations were normal.

Jaundice was noticed on day two, and serial LFT showed a worsening trend. Referred for further management to our hospital, on examination, the patient was conscious, oriented, and hemodynamically stable. He was icteric. Systemic examination showed right hypochondrial tenderness, and other systemic examinations were within normal limits. There were no features of hepatic encephalopathy.

Patient investigation revealed that leucopoenia with myeloid series suppression (TC of 1.9 and ANC of 665) showed improvement in 48 hours (TC of 7.9 with ANC of 1896 with a shift to the left). The patient also had thrombocytopenia which also improved with conservative treatment. Deranged liver parameters, namely SGOT, and SGPT peaked at 4134 and 1494, respectively, and elevated PT of 60.2 and INR of 5.65 on day four after toxins ingestion. Treated with FFP for coagulopathy, and liver supportive was continued. Following which serial LFT and coagulation parameters showed improvement. Initially patient had coagulopathy; hence bone marrow study was not done; during the later half of the hospitalization, blood counts were improving; hence bone marrow study was deferred.

The patient was monitored in ICU. He had no cardiac dysrhythmias, Input/Output was strictly monitored, and blood glucose was measured every 4th hour, which was normal. The patient was clinically stable without any symptoms, and laboratory parameters showed improvement with Haemoglobin of 13.9, TC of 7.9, and platelets of 86,000 (improving trend) LFT – SGOT – 109, SGPT – 255, PT of 10.8, and INR of 0.8, discharged on day 9. On follow-up after two weeks, repeat parameters were normal.

Discussion

Yellow phosphorus is used as rodenticide because of the development of resistance to warfarin derivatives which was previously used as a rodenticide (1). It has a garlic odor. Absorption can occur through

skin, mucous membrane, respiratory and gastrointestinal epithelium. The rate of absorption is rapid and is distributed to all tissues and concentrated in the liver; approximately 69 – 73% of the total ingested dose gets accumulated in the liver (2)(3).

The clinical feature of toxicity is mostly related to the GI system(1). Renal and CNS involvement is rare, whereas hematological involvement is rare, and to date, we have very few countable case reports.

In the body, by means of an exothermic reaction, it produces phosphoric acid that causes direct tissue damage by free radicals against organic molecules bringing changes in ribosomal function and protein synthesis and fatty degeneration of multiple organs and also decreases the production of ATP and inhibit fatty acid oxidation by its toxic effect on mitochondria which altogether leading to multi-organ failure(4) is the mechanism of toxicity. It is also directly hepatotoxic, causing periportal injury with ballooning degeneration and vacuolization. (5)(2)

Reports of hematological disturbance are rare. Yellow phosphorus intoxication produces a decrease in granulocyte count. Our patient had a decrease in granulocyte count and platelet count. In vitro studies showed a reduction in cellular replication rate. (3)

There is selective suppression of myeloid series in the bone marrow, which may be due to metaphase arrest or immune-mediated destruction, or pharmacogenetic factors determining its toxicity profile. Spontaneous recovery is seen (1).

Those who had bone marrow involvement had less hepatic involvement, as noted in previous studies (3)(1). But our patient had marrow involvement along with severe hepatic involvement.

N acetylcysteine treatment in yellow phosphorus studies has not yielded consistent results. The survival benefit was noted if the patient is treated with NAC early in the course of disease vs. late treatment. (6)

There is no antidote; liver transplantation is the life-saving measure in hepatic failure. (5) Poor outcome indicators

Elevation of liver transaminases and ALP, more than tenfold increase in alanine aminotransferase, deranged PT, metabolic acidosis, hypoglycemia. (6)

MELD score has been described as a prognostic indicator in rodenticide poisoning. the average score for those who died was 40.5 as compared to 11.7 for survivors. (7) Patient's meld was 31 points (estimated three months

Clinical Stages following Yellow phosphorus poisoning (1)

Stage	Time duration	Common Manifestation	Rare Manifestation	Complications
1	First 24 hours	Gastrointestinal manifestations: nausea, vomiting, diarrhea, abdominal pain		Cardiac arrhythmia, dyselectrolytemia.
2	1 – 4 days	Asymptomatic period: liver enzymes increase because of toxic hepatitis	Hematological involvement: low hemoglobin level, agranulocytosis, and thrombocytopenia.	
3	4 – 7 days	Multi-organ failure: acute liver failure, acute kidney injury	The cholestatic pattern of liver involvement.	Metabolic derangement, encephalopathy, coagulopathy, cardiogenic shock, psychosis, coma.

mortality of 52.6%). Even though the MELD score was high, fortunately, our patient made it up.

Death ranges from 23% among those presenting GI symptoms to 73% among those with CNS symptoms. (3) The lethal dose to the liver is 1mg/kg (Fernandezou, Canizares, 1995). The LD 50 dose in yellow phosphorus poisoning is 10mg/kg, rarely as low as 100mg, resulting in death. (6) Mortality is due to hepatic or cardiovascular collapse but not due to hematological involvement. (3)

Conclusion

Even though rare complete blood count, Hematological involvement to be monitored during the first week helps to detect the rare toxicity profile of yellow phosphorus on bone marrow. Previous case reports showed that patients with hematological involvement had less hepatic involvement were as our patient had severe hepatic dysfunction along with the hematological participation. Further studies are needed to identify the mechanism of selective myelosuppression and genetics behind the varied clinical presentations.

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Legend

Rare manifestations of yellow phosphorus poisoning picked up by serial monitoring of complete blood count. Thrombocytopenia and selective myeloid series suppression showed spontaneous improvement.