Case Study

A Case of Ratol Poisoning and Review of Literature

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Abstract

We report a case of a 17 year old lady who presented with severe abdominal pain for 4 days. Patient developed fulminant hepatitis and disseminated intravascular coagulation during her hospital stay. History of Ratol (3% yellow phosphorus) ingestion was revealed on the second day of admission. She was managed with NAC infusion for 3 days along with supportive treatment and was discharged on the 10th day of admission. Accidental ingestion of Ratol is common in rural India. Early diagnosis and management of yellow phosphorus can improve the outcome of the patient. It is important to identify predictors of outcome of patients with toxin-induced liver injury.

Keywords: Yellow phosphorus poisoning, Acute liver failure, Disseminated intravascular coagulation, N-acetylcysteine, Toxin Induced Liver Injury

Introduction

Ratol (3% yellow phosphorus) is commonly used as a rodenticide in the agricultural industry in India. Yellow phosphorus is readily absorbed by mucous membranes of the gastrointestinal tract and metabolized by the liver. It can cause direct toxicity to the liver[1][2].

Current scientific literature suggests that a dose of >1mg/kg of yellow phosphorus can be lethal. Yellow phosphorus acts as a protoplasmic poison. Liver injury and neurologic manifestations are commonly reported, with the latter being associated with a poorer prognosis. Bone marrow suppression and ventricular arrhythmias have been reported[2,3,4]. One autopsied case also reported pancreatic damage due to Ratol poisoning. There is no antidote available and treatment is mainly supportive. While some patients have been treated with N-acetylcysteine infusion, the results have been inconclusive. Some patients may also require plasmapheresis. Accidental ingestion of the paste is not uncommon.

Few cases report complete recovery after exposure. We report a case of a 17 year old lady who presented to us with severe liver injury and pancytopenia after Ratol ingestion. Informed consent was taken to share the patient's case from her parents for academic purposes.

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Case Study

A 17-year old lady presented chief complaints of severe epigastric pain and three to four episodes of vomiting and diarrhea, which were non-bloody, non-bilious and lacking any specific odor for four days.

She initially presented to another hospital("Hospital A") where an abdominal ultrasonography(USG) suggested changes of subacute pancreatitis and mild ascites. A CECT abdomen was suggestive of an ileo-ileal intussusception. Blood amylase and lipase levels were elevated, along with thrombocytopenia, leucopenia and transaminitis(see Table 1 below). Dengue IgM and IgG were negative. She was treated with broad spectrum intravenous antibiotics and supportive treatment for four days before she presented to our hospital("Hospital B").

Fig 1: CECT abdomen suggestive of an ileo-ileal intussusceptions

Table 1: Complete blood count, LDH, Ferritin, Troponin levels

Parameters	Day 0 [26/12/23] Hospital A	Day 4 [29/12/23] Hospital B	Day 7 [1/1/24]	Day 13 [7/1/24] DISCHARGE Hospital B	Reference values
Hemoglobin (g/dL)	12.7	11.9	10.4	8.3	12-14 g/dL
RBC count (x106/mm3)	5.01	4.8	3.88	3.04	4-5 million/ mm3
Hematocrit		37.5	30	24.6	Male 41%-53% Female 36%-46%
Reticulocyte count		2			0.5%-1.5%
WBC count (x103/mm3)	12.32	1.7	2.46	11.08	4500-11,000/ mm3
Platelet count (/mm3)	217,000	49,000	23,000	206,000	150,000-400,000/mm3
LDH		1352		351	45-200 U/L
Ferritin		>1650			Male 20-250 ng/mL Female 10-120 ng/mL
CRP	7.1	1.6		0.6	<0.9 mg/dL

Table 2 Liver Function Tests, Renal function tests, Coagulation profile & electrolytes

Liver Function Tests	**Day 0 [26/12/23] Hospital A**	**Day 4 [29/12/23] Hospital B**	**Day 13 [7/1/24] DISCHARGE Hospital B**	**Reference values**
Total Bilirubin (mg/dL)		3.43	2.86	0.1-1.0 mg/dL
Direct (mg/dL)		2.5	1.92	0.0-0.3 mg/dL
Indirect (mg/dL)		0.93	0.94	<0.7 mg/dL
SGPT or ALT (U/L)	147	423	121	10-40 U/L
SGOT or AST (U/L)		328	76	12-38 U/L
ALP (U/L)		91	216	25-100 U/L
Total Protein (g/dL)		4.95	5.66	6.0-7.8 g/dL
Serum Albumin (g/dL)		3.13	3.27	3.5-5.5 g/dL
S. Globulin (g/dL)		1.82	2.39	2.3-3.5 g/dL
Renal function				
Creatinine (mg/dL)		0.72	0.33	0.6-1.2 mg/dL
Urea nitrogen		44.5	9.8	7-18 mg/dL
Coagulation profile				
PT/INR		91.9/7.4	16.4/1.11	11-15 seconds /0.8-1.1
APTT		60.3	33.1	25-40 seconds
D-Dimer		3.05	2.09	≤250 ng/mL
Electrolytes				
Serum Na+	123	136	139	136-146 mEq/L
Serum K+	3.26	2.34	3.5	3.5-5.0 mEq/L
Serum Mg2+		1.16	1.7	1.5-2.0 mEq/L
Serum Ca2+		6.9	9.1	8.4-10.2 mg/dL

On presentation to our hospital, she was afebrile, hypotensive (blood pressure was 90/60mmHg) and had tachycardia(Heart rate 100/min, regular). She weighed 60 kgs. The abdomen was non-tender without any guarding and there was no hepatosplenomegaly. She had mild pallor and icterus. She had presented on the sixth day of her menstrual cycle with persistent heavy bleeding with clots and a change of three pads/day. A gynecology opinion was sought and she was treated with tranexamic acid and injectable vitamin K.

Repeat abdominal ultrasound at our hospital ("Hospital B") was normal. A repeat CECT abdomen showed mild hepatomegaly and minimal free fluid in pelvis and peripancreatic area. There was no evidence of active pancreatitis. Blood analysis revealed leucopenia, thrombocytopenia, transaminitis, hypokalemia (2.67 mEq/L), hypomagnesemia (1.16 mEq/L) and hypocalcemia (6.5 mEq/L), altered coagulation profile and elevated serum amylase and lipase. Serum LDH and Ferritin levels were elevated[Tables 1,2]. The electrocardiogram was suggestive of sinus tachycardia. 2D ECHO showed preserved ejection fraction with no regional wall abnormalities.

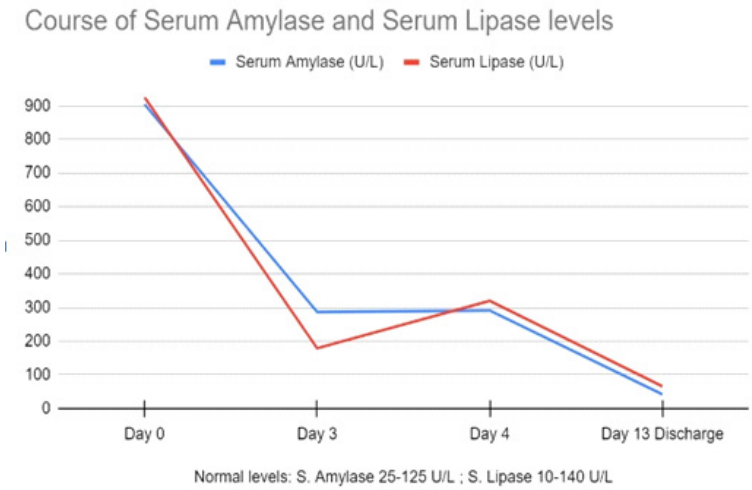

Figure 2: Graphical representation of S. amylase and lipase levels

On the second day of admission to our hospital, she revealed a history of ingestion of Ratol (3% yellow phosphorus), five days prior. The amount of ingestion of the substance could not be retrieved.

Eight Units of Fresh Frozen Plasma(FFP) and four units of Platelet-Rich Concentrates (PRCs) were administered. Menstrual bleeding subsided on the fourth day of admission. All culture reports were negative. A gastroenterology opinion was sought and a diagnosis of toxin induced pancreatitis and hepatitis was made.

A final diagnosis of toxin induced disseminated intravascular coagulation (DIC) (6 Points according to ISTH Criteria- s/o overt DIC), subacute pancreatitis with hypokalemia and pancytopenia was made. She was started on N-Acetylcysteine(NAC) infusion; supportive fluids and broad spectrum antibiotics were continued. She was continued on NAC infusion for three days and then switched to oral form. Intravenous Folinic acid was given for four days in view of pancytopenia.

Her general condition, counts and electrolytes started improving on the third day of admission. A psychiatric evaluation was sought and she was counseled for suicidal attempt prior to discharge. She was discharged on the tenth day of admission at hospital B.

Discussion

Yellow phosphorus is a common ingredient used in fireworks and rodenticides. Accidental or intentional ingestion of yellow phosphorus is quite common, especially in developing countries[1,4]. It is a protoplasmic poison and direct toxicity to the liver has been described. It undergoes exothermic reactions to produce phosphoric acid which causes tissue damage and also forms phosphorus pentoxide which further reacts with organic molecules[5]. Calcium binds preferentially to phosphorus in serum which may result in hypocalcemia[6].

The clinical sequelae of acute phosphorus poisoning are commonly divided into three phases: In the first phase, the patient usually experiences gastrointestinal symptoms like nausea, vomiting, diarrhea and burning pain in mouth, throat and retrosternal chest pain. This phase generally lasts for the first 24 hours. This phase is followed by an asymptomatic phase which may last for the next 48 hours. The third phase (>72 hours) is characterized by systemic toxicity where gastrointestinal symptoms may reappear. Patients may develop pancreatitis, acute liver failure, acute renal failure, pulmonary edema, severe metabolic acidosis, shock and cardiotoxic manifestations like arrhythmias, ischemia or even arrest [1,2,4,7]. Previous studies show that patients presenting with neurological manifestations like hallucinations, confusion, headache, tinnitus, delirium, psychosis and coma, have generally poorer prognosis. Leukopenia and thrombocytopenia have also been described [3,7].

Our patient also followed the classical course of illness. The patient presented to us on the fourth day of Ratol ingestion,in the third phase of toxicity. Initial radiological investigations at the previous hospital were contradictory- suggesting that the patient had developed subacute pancreatitis or intussusception. A case report by Prabhat et al described a 23 year old lady who developed pancreatitis four days after ingestion of yellow phosphorus[8]. Autopsy findings in a case report by Jai Prakash Soni et al showed focal fat necrosis along with necrosis of large areas of pancreatic parenchyma [9].

Ultrasonography and CT abdomen at our hospital revealed no active changes in the pancreas and serum amylase and lipase levels were also in a reducing trend, suggesting that the inflammation was resolving [see Chart 1]. This may be attributed to the treatment that patient had received prior to presentation at our hospital. However, blood investigations were suggestive of severe leukopenia, thrombocytopenia and electrolyte abnormalities-hypokalemia, hypocalcemia, hypomagnesemia, along with transaminitis and features of disseminated intravascular coagulation (DIC) (elevated Fibrin degradation products(FDPs), reduced fibrinogen levels, raised D-dimer) (6 points for overt DIC according to ISTH Criteria).

Decontamination and supportive therapy is given to patients as there is no antidote for yellow phosphorus. Since our patient revealed a history of ingestion on the fourth day since onset of symptoms, gastric lavage wasn't done.

Reports on clinical improvement after timely initiation of NAC are conflicting. In our case, treatment with NAC showed a good outcome compared to the report of Nanditha et al [1]. NAC acts as an antioxidant thus is used for treatment of yellow phosphorus poisoning.

Patients might need plasma exchange, if supportive measures fail, or even a liver transplant if severe organ damage has occurred. Delayed resuscitation, jaundice, hepatic encephalopathy, the elevation of AST and ALT to >1000 IU/L, metabolic acidosis, and refractory shock are reliable predictors of a bad outcome [10]. Ravi Mohanka et al suggested that patients with lower dose ingestion (<17.5 g), absence of cardiotoxicity, <grade 3 HE, lactate < 5.8, SOFA score < 14.5, and increase in SOFA score by < 5.5 were more likely to survive. Apart from hepatic encephalopathy, markers of severe acute liver injury such as PT-INR > 6.0, MELD score > 37, persistently elevated serum lactate despite resuscitation and with plasmapheresis PT-INR > 2.5; at least 12 h after second cycle have been proposed for indication for liver transplantation [2]. Our patient presented with jaundice, transaminitis and pancytopenia. The dose of toxin ingested by our patient could not be determined. Our patient also had a markedly raised PT/INR and SOFA score of 5 points (<33% mortality) on admission and a MELD score of 34 points (52.6% estimated 3-month mortality). Our patient also had an R-factor of 13.9 suggestive of hepatocellular injury- indicating toxin induced liver injury. These were suggestive of poor prognosis for the patient.

As reported in literature, victims of yellow phosphorus poisoning may be initially asymptomatic; however, recovery is observed after 2-3 days, and later on, signs of acute hepatic failure develop [2]. In our case, the patient had an acute progression of complications. Hence, patients with acute yellow phosphorus poisoning warrant close monitoring for at least a week. Psychiatric evaluation and counselling to prevent recurrent suicide attempts should be performed.

There is a statistically significant association between the patient's age, amount of poison consumed, time taken between poison consumption and seeking medical help and the outcome of discharge or death. The study done by McCarron et al. has shown varying mortality rates - 23% for patients with GI symptoms and 73% for those with CNS manifestations [11]. Patients in the younger age group and having a lower dose of ingestion were more likely to recover.

Conclusion

Diagnosing yellow phosphorus poisoning in a patient presenting in emergency is imperative and if treated in a timely manner, patients may show full recovery. Liver damage is found to be the most common complication of yellow phosphorus poisoning. Accidental ingestion of rodenticide should be suspected in cases of severe liver injury, especially in developing countries.

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