Case Report: Yellow Phosphorus Induced Acute Fulminant Hepatitis and Acute Kidney Injury in a Male

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Abstract

Yellow Phosphorus (YP) is an active component present in rodenticides, pesticides and fireworks. It is a potential hepatotoxic agent which can cause Acute fulminant hepatitis. Amongst the other organs involved GIT and kidneys are also vulnerable. We present a case acute yellow phosphorus poisoning leading to fulminant hepatic failure and acute kidney injury in a young male.

Keywords—*Yellow phosphorus, fulminant hepatitis, toxin, acute kidney injury.*

I. INTRODUCTION

Elemental phosphorus exists in two forms, red and yellow. Red is non-volatile, insoluble and un-absorbable and therefore nontoxic when ingested. Yellow phosphorus which is also known as white phosphorus has severe local and systemic toxins that cause damage to hepatic, renal, cardiovascular systems. [1,2] Yellow phosphorus is used in rodenticides and fireworks out of which rodenticides are the most readily available source of yellow phosphorus today.[3,4]Yellow phosphorus in the form of rodenticide is available as powder or paste containing 2 to 5 percent yellow phosphorus.

II. CASE REPORT

A 48 year old male,was referred from a private clinic with history of consumption approximately 4 grams of 3% of rat kill poison (Ratol) 5 days ago at his home following which patient complained of pain in abdomen, and vomiting .Patient was taken to local clinic where primary treatment in form gastric lavage was given and on day 3 repeat blood investigations showed derangement of liver function and renal function for which he was referred to this hospital for further management. There was no history of convulsions, haematemesis, diarrhoea, blood in stools, chest pain or breathlessness.

On examination patient was afebrile with heart rate of 98 beats per minutes, respiratory rate-26 cycles/min, blood presure-140/90 mmHg in right upper supine position, icterus and oedema feet were seen. Liver was palpable 2cm below costal margin which was soft and tender with round margins. Liver span was 13cm. CNS examination -patient was disoriented but responding to verbal commands, judgment and abstract reasoning were impaired ,acalculia and construction apraxia was present. He was moving all four limbs and all the reflexes were preserved, asterixis were present, cranial nerves were normal. Cardiovascular and respiratory system examination was normal.

On investigations haemogram was normal ,serum biluribin was 6 mg/dl with direct fraction of 4mg /dl, serum ALT was 1800 and AST was 1400 IU/L ,Alkaline phosphatise-220 IU/L, Prothrombin time was 28 seconds, INR-3 times of control ,serum urea-118 mg/dl, serum creatinine-3.5 mg/dl. ABG-pH-7.296 Po2-98 mmHg Pco2-36 so2-98.6%,HCO3-14 mmol/L suggesting severe metabolic acidosis, serum potassium 6.8 mEq/L ,ECG showed tall T waves, serum CPK-MB,Trop I were normal. Other investigations like HbsAg,Anti-HCV,IgM Anti-HAV, Serum Ceruloplasmin and ferritin level was negative and USG abdomen was normal. With the above report a diagnosis of Yellow phosphorus [YP] induced acute fulminant hepatic failure with acute kidney injury was entertained.

The patient was treated with intravenous N-acetyl cysteine [NAC] 150 mg over one hour as loading dose followed by 50 mg/kg/hr for next 4 hrs and 100 mg/kg/hr for next 15 hours, injection vitamin K 10 mg was given s.c. for 3 days, 10 unit of fresh frozen plasma [FFP] was transfused to make INR less than 2, and achieve normal coagulation profile. Encephalopathy was treated with high bowel wash 8 hourly, syp lactulose 30 ml tds through NG tube. In view of metabolic acidosis and persistent hyperkalaemia not responding to calcium gluconate and GI drip patient was taken for haemodialysis daily till the metabolic profile and uremic profile were normal.

Over the next 5 days his liver enzymes level decreased and encephalopathy, uremic complications improved and patient was discharged on the tenth day,on one week follow up the liver enzymes were mildly raised (ALT-220 IU/L, AST-182 IU/L), INR - 1.4, and all other reports were essentially normal and patient was asymptomatic, patient is awaiting second follow up after 2 weeks.

III. DISCUSSION

"RATOL" is a trade name of rodenticide which contains 3 percent yellow phosphorus. Yellow phosphorus is a potential hepatotoxic agent. It also cause GIT toxicity in form of nausea, vomiting ,abdominal pain, renal toxicity in form of acute tubular necrosis, haematological complications like bone marrow failure, cardiovascular effects like cardiac arrhythmias ,haemodynamic collapse and central nervous system complications in form of altered sensorium ,seizure and coma. [3,4,5,6,7].

Yellow Phosphorus poisoning present in three phases ,first phase usually presents as GIT side effects and last for 24 hrs .Second stage 24-72 hours, is usually asymptomatic but lab investigations may start revealing features of hepatotoxicity in form of transaminitis. The second stage may progress to a third stage ,acute fulminant hepatic failure with full blown hepatic encephalopathy and coagulopathy, which may require liver transplant. This stage of liver failure can occur within any time within seven days of ingestion of poison[8].

MELD score in our patient was 37, a study showed that MELD score more than 40 usually indicated grave prognosis in yellow phosphorus ingestion[9].Outcome of patients of yellow phosphorus poisoning was variable in different studies. One study found that 87 percent had hepatic involvement out of which 27 percent died of fulminant hepatic failure[9].The American association of study of liver disease (AASLD) recommends treatment with vitamin K,FFP transfusions for the coagulopathy associated with YP poisoning. [9] N-acetyl cysteine should be administered because it is hepatoprotective and cost effective and is safe.[10,11] Though some studies suggest the role of NAC is controversial. [12] The final treatment of fulminant hepatic failure is liver transplant.

IV. CONCLUSION

To conclude cases of YP poisoning should be treated conservatively as there is no antidote available. The initial stage of GIT effect may gradually progress to fatal fulminant hepatic failure anytime within seven days of ingestion. So, all patient of yellow phosphorus should closely observed by daily assessment of hepatic profile at least for a week

REFERENCES

[1] PHOSPHORUS, ELEMENTAL (2017) National Library of Medicine HSDB Database [Internet].

[2] Mauskar A, Mehta K, Nagotkar L, Shanbag P (2011) Acute hepatic failure due to yellow phosphorus ingestion. Indian J Pharmacol 43(3): 355-356.

[3] Mohideen SK, Kumar KS (2015) Should ratol paste be banned? Indian J Crit Care Med 19(2): 128-129.

[4] Lakshmi CP, Goel A, Basu D (2014) Cholestatic presentation of yellow phosphorus poisoning. J Pharmacol Pharmacother 5(1): 67-69.

[5] Saoji AA, Lavekar AS, Salkar HR, Pawde GB, Tripathi SS (2014) A Case on Suicidal Poisoning Associated with Ratol and a Perspective on Yellow Phosphorus Poisoning. Int J Re Sci& Techi 10: 223-225.

[6] McCarron MM, Gaddis GP, Trotter AT (1981) Acute yellow phosphorus poisoning from pesticide pastes. Clin Toxicol 18(6): 693-711.

[7] Tafur AJ, Zapatier JA, Idrovo LA, Oliveros JW, Garces JC (2004) Bone marrow toxicity after yellow phosphorus ingestion. Emerg Med J 21(2): 259-260.

[8] Pahadia M, Registrar S, Bhushan B, Professor A, Saluja M, et al. (2013) Impact on Morbidity & Mortality by Rodenticidal Poisoning. Indian Medical Gazette.

[9] Nalabothu M, Monigari N, Acharya R. Clinical Profile and Outcomes of Rodenticide Poisoning in Tertiary Care Hospital. International Journal of Scientific and Research Publications.2015; 5(8): 1-12.

[10] Fernandez OU, Canizares LL. Acute hepatotoxicity from ingestion of yellow phosphorus-containing fireworks. J Clin Gastroenterol. 1995; 21(2): 139-142.

[11] Bassem W G, Timothy S L, Gajanan B P, Reshma D S, Priyank J. "Successful Treatment of Acute Liver Failure due to Yellow Phosphorus Ingestion in a Rural, Low Resource Setting". Biomed J Sci &Tech Res. 2018; 4(4).

[12] Akman SA, Cakir M, Baran M, Arikan C, Yuksekkaya HA, Tumgor G, et al. Liver transplantation for acute liver failure due to toxic agent ingestion in children. Pediatr Transplant 2009;13:1034-40.