Case Report

EARLY MANAGEMENT OF YELLOW PHOSPHORUS TOXICITY DECIDES THE PROGNOSIS: A FATAL CASE REPORT

Journal Homepage :www.jist.org.in; Email: article@jist.org.com

Pankaj Verma,[1] Pradeep Kumar Mishra,[1#] Rashmi Kulkarni[1]

ABSTRACT

Yellow phosphorus is used as an insecticide, rodenticide, in ammunition and fireworks manufacturing. More or less it affects all organs of the body but target organs of this poison are gastro-intestinal tract, liver, kidneys and brain. It is absorbed by the body very quickly and accumulates in various organs producing fatal effects thereof. Early detection and institution of treatment without delay is paramount importance in managing such cases. We report a fatal case of yellow phosphorus poisoning due to delayed institution of treatment. A 26 years old adult female came to emergency department at Sri Aurobindo Medical College, Indore with complaints of nausea, generalized swelling, anuria and shortness of breath with abnormal vitals. Three days back she had consumed a tube-full of rat poison paste. The preserved gastric lavage fluid, vomited material, blood and urine samples were sent to the Forensic laboratory for Toxicological analysis. Qualitative screening of samples using Ammonium Molybdate and Mitscherlich methods detected presence of yellow phosphorus derivatives. The patient was succumbed to the complications of hepatic failure a day after admission to the hospital.

Keywords: yellow phosphorus; insecticide; fireworks; rat poison; gastric lavage; analytical toxicology; hepatic failure

INTRODUCTION

Phosphorus is an inorganic irritant poison and found in two forms i.e. White/Yellow and Red phosphorus. Yellow phosphorus emits white fumes and ignites at low temperature. Yellow phosphorous is widely used in preparations of vermin pastes containing 1 to 4% yellow phosphorous and in rodenticides in 2-5% concentration. Absorption of yellow phosphorus occurs through skin, gastrointestinal epithelium, mucus membrane and respiratory epithelium. For its absorption, bile salts play very important role. Intoxication is mostly accidental and suicidal in nature. [1]It acts as hepatotoxic protoplasmic poison and disturbs normal cell metabolism by affecting cellular oxidation. If ingested, it produces widespread fatty infiltration with degeneration of cells of different organs, especially of liver and the cells of cerebral cortex.[2]It is highly toxic with a fatal dose of 60-120 mg and fatal period of 2- 8 days and there is no antidote. [3]

There are three stages in acute poisoning. In first stage gastrointestinal effects are more common, followed by 2-3 days of non-symptomatic second stage. In third stage, as a result of systemic toxicity there is rapid decline in the condition of the patient due to increased gastro-intestinal irritation and adverse effects over liver, kidneys, heart and brain. Central nervous system includes confusion, hallucination and coma.[4] [5] Other findings are persistent left ventricular dysfunction in heart and acute pulmonary oedema in lungs as a result of yellow phosphorus intoxication. [6] [7] In chronic poisoning there is necrosis of lower jaw in the region of decayed tooth, also known as Phossy Jaw. [3]

CASE REPORT

A 26 years old average built adult female came with complaints of nausea, generalized swelling, anuria

[#]**Correspondence Author**: Department of Forensic Medicine & Toxicology. Sri Aurobindo Medical College & Post Graduate Institute, Indore, Madhya Pradesh, E-mail: pradeep_sus1074@yahoo.com.

[1]Department of Forensic Medicine & Toxicology. Sri Aurobindo Medical College & Post Graduate Institute, Indore, Madhya Pradesh.

and shortness of breath to Emergency Department at Sri Aurobindo Medical College, Indore with history of ingestion of one complete tube of Rat poison(Ratol® paste) in form of paste, three days back. Yellow phosphorus is a major constituent of Ratol®. On examination, vitals were abnormal (blood pressure 90/60mmHg in supine position, respiratory rate 30/min, tachycardia with pulse 140/min), with mild pallor, icterus, oliguria and altered consciousness. Investigations were suggestive of acute hepatitis with 6.7mg% total bilirubin, ALT- 986 U/L and AST- 438 U/L and prolonged prothrombin time of more than 2 minutes. Her kidney function showed Urea 106 mg/dl and serum creatinine 5.77 mg/dl. Electrolytes were normal. Hemoglobin was 9.6 gm/dl, total WBC count 20900/cmm. Hepatitis B surface antigen was negative. Arterial blood gas revealed pH7.20, PCO_2 32.0, PO_2 81.9, HCO_3 12.5 suggesting metabolic acidosis. Hepatomegaly was noticed in ultra-sonography. All viral markers were negative. Vomit material, blood and urine were sent to our Departmental Analytical Toxicology Laboratory and it came out to be positive for yellow phosphorus by Phosphate test using Ammonium Molybdate method and Mitscherlich method. She was treated with intravenous (IV) Vitamin K, IV fluids, IV antibiotics, and fresh frozen plasma. Intake and output was strictly monitored and blood glucose was measured six hourly. Metabolic acidosis was corrected by sodium bicarbonate infusion. Central line insertion and endo-tracheal intubation was done. The patient was put on ventilatory support. Her falling blood pressure was managed with intravenous fluids, vasopressors and inotropes. Despite of all available measures taken, the patient died within a day of admission and sent for post mortem examination. On external examination, there was no evidence of any injury. Blood mixed fluid was coming out of mouth and both the nostrils. Rigor mortis was present all over the body. Post mortem hypostasis was present over back and fixed. On internal examination, duramater was showing yellowish discoloration and brain was congested. Petechial hemorrhages were present over both the lungs all over at places. Both lungs were edematous and congested. Stomach was containing about 100 ml of yellowish liquid material and mucosa was congested all over with patchy hemorrhagic areas at places. Mucosa was also hemorrhagic in duodenal part of small intestine. Hemorrhages were present within cortico-medullary junction of both kidneys. Liver was showing fatty changes. Viscera sent for chemical examination and part of liver and kidneys were sent for histopathological examination.

Histopathology Findings

Vacuolar appearance of hepatocytes and diffuse fat globules in parenchyma of liver was present. Both micro-vesicular as well as macro-vesicular lipid droplets accumulations were found in hepatocytes. Vacuolar degeneration of proximal tubular epithelium of kidneys noticed.

DISCUSSION

Yellow phosphorus emits smoke and has very strong garlicky odor. Absorption process is very fast with accumulation of about 70% in liver within 2-3 hours. [8] Yellow phosphorus is most commonly used poison of all rodenticide poisons available in market. [9] Systems affected as a result of yellow phosphorus poisoning were gastrointestinal tract (100%), followed by liver (66.70%), cardiovascular system, nervous system along with metabolic abnormalities (66.70%).[10] In the present case, gastro-intestinal irritation, CNS depression, respiratory as well as circulatory system was affected. The symptoms manifested by the patient in this case report are consistent with the known pharmacological effects of yellow phosphorus. The clinical manifestation of intoxication in our casewas nausea, generalized swelling, anuria, shortness of breath, irritability, drowsiness, jaundice, hypotension, tachycardia, along with primary GI symptoms which are signs of renal, hepatic and cardiovascular system compromise. Another important aspect in yellow phosphorus poisoning is dose and time interval between ingestion and treatment, which play a significant role in the survival of patient. A study done by McCarron MM et al [11], observed that mortality increases by involvement central nervous system along with gastrointestinal system. In post mortem examination, gross changes were noticed in liver and kidneys while only congestion was noticed in brain and heart. And this shows the toxicity of yellow phosphorus over liver and kidneys. If proper history is not available, then it is quite difficult for the doctors to diagnose yellow phosphorus poisoning in first 72 hours, as there are very less or no symptoms of GIT irritation during this period. Hepatotoxicity often develops after 72 hours of consumption of poison. [12] Present case came with history of ingestion of yellow phosphorus 3 days back developed hepatic and renal failure and died even after treatment. In a study, those cases who got medical attention after 24 hours of ingestion of yellow phosphorus with dose >1mg/kg developed fulminant hepatic failure

Figure 1: Multiple petechial hemorrhages present over the lungs.

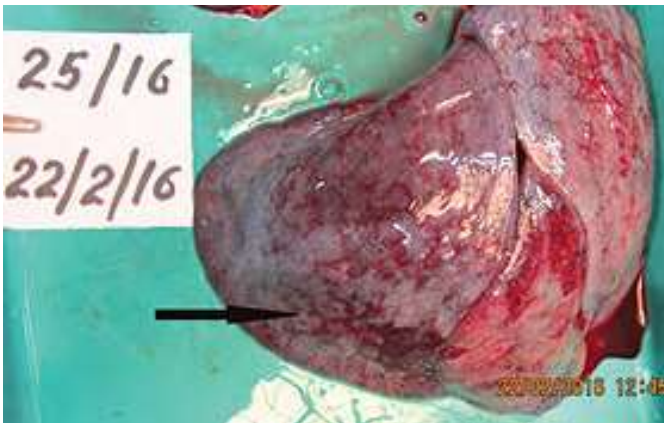


Figure 2: Marked congestion and hemorrhage found in kidneys.

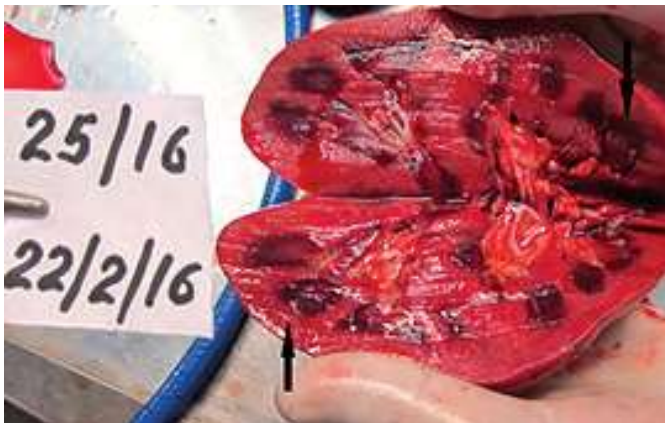


Figure 3: Showing the dissected liver showing features of fattyde generation.

Figure 4: Cut opened gastric cavity showing confluent areas of sub-mucosal hemorrhages due to ingestion of yellow phosphorus.

Figure 5 : Photomicrograph of liver showing features of fatty changes as complication of yellow phosphorous toxicity (H&E, 10X)

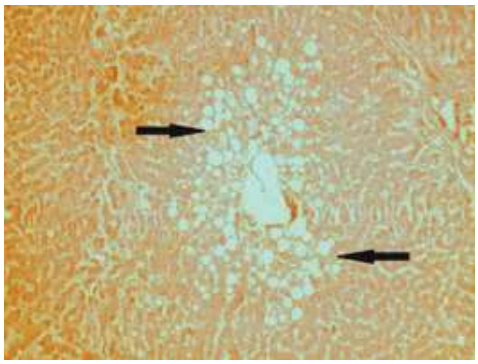
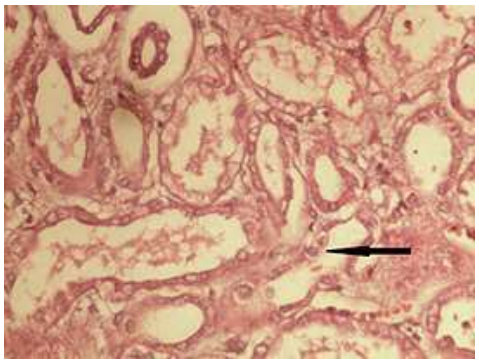


Figure 6: Photomicrograph of kidneys showing diffuse vacuolar degeneration of tubules and interstitium subsequent to the yellow phosphorous toxicity of kidney. (H&E, 40X)



and 100% mortality.[13] In another study on 15 cases of yellow phosphorus poisoning, the mortality rate was 27% and confirming it extremely lethal when ingested. [14] Fatty degeneration of liver and kidneys are the most common pathological findings in death due to yellow phosphorus poisoning. [8]

CONCLUSION

The interval of consumption of yellow phosphorus and the onset of treatment usually decides the outcome of the prognosis of the cases. Nevertheless, there is no specific antidote available for yellow phosphorus poisoning. Patients must be kept under observation for 3-4 days to see all the effects till the time period of completion of third stage. Keep such chemicals away from food items to avoid contamination. In households, it should be kept in the places which are not accessible by the children. Government should take strict steps in sale of such poisons in open market that are easily available and teach people how to dispose these poisons.

Conflicts of interest: declared none.

REFERENCES

1. Brent J, Wallace KL, Burkhart KK, Phillips SD, Donovan JW. Phosphorus in: Critical care toxicology- Diagnosis and management of the critically poisoned patient. Philadelphia, PA, USA. Elsevier Mosby; 2005. p. 851–61.
2. Mukherjee JB. Forensic medicine & toxicology. 3rd ed. Kolkata: Academic Publishers; 2007. p. 956-7.
3. Reddy KSN, Murty OP. The essentials of forensic medicine and toxicology. 33rded. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2014. p. 550-1.
4. Mauskar A, Mehta K, Nagotkar L, Shanbag P. Acute hepatic failure due to yellow phosphorus ingestion. Indian J Pharmacol 2011; 43:355-6.
5. Saoji AA, Lavekar AS, Salkar HR, Pawde GB, Tripathi SS. A case on suicidal poisoning associated with ratol and a perspective on yellow phosphorus poisoning. Int J Recent Trends Sci Technol 2014; 10: 223-5.
6. Pooja PS, Padmakumar AV, Suresh CG. Persistent left ventricular dysfunction in a teenager with yellow phosphorus poisoning. JCR 2015; 5: 137- 40.
7. George P. An unusual case of pulmonary oedema and its successful management: a case of phosphorus poisoning. J Clin Diag Res 2010; 4: 3554- 7.
8. Hiran S. Ventricular arrhythmia due to yellow phosphorus poisoning. J Case Rep Stud 2017; 5(3): 303.
9. Nalabothu M, Monigari N, Acharya R. Clinical profile and outcomes of rodenticide poisoning in tertiary care hospital. IJSRP 2015; 5: 1-12.
10. Chikkaveeraiah SK, Marijayanth M, Reddy PK, Kaluvakuri S. Clinical profile and outcome of rodenticide poisoning in patients admitted at tertiary care teaching hospital in Mysore, Karnataka, India. Int J Res Med Sci 2016; 4: 5023-7.
11. McCarron MM, Gaddis GP, Trotter AT. Acute yellow phosphorus poisoning from pesticide pastes. Clin Toxicol 1981; 18(6):693- 711.
12. Haddad, Winchester. Clinical management of poisoning and drug overdose: W. B. Saunders Company 1990; 2: 1118- 9.
13. KafeelSI , Chandrasekaran VP , Eswaran V. Role of N-acetyl cystine in outcome of patients with yellow phosphorus poisoning- An observational study. National J Emerg Med2012; 1:1.
14. Fernandez OU, Canizares LL. Acute hepatotoxicity from ingestion of yellow phosphorus-containing fireworks. J Clin Gastroenterol 1995; 21:139- 42.