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ORIGINAL ARTICLE

The toxicological profile of acute phosphorus poisoning

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Abstract

Phosphorus is a very common nonmetallic mechanical irritant and protoplasmic poison which affects the cellular oxidation. Yellow phosphorus is highly toxic and is widely used in fertilizers, fireworks, pesticides and rodenticide pastes. Cases of poisoning by consumption are very common in developing countries. Clinical manifestation ranges from acute hepatitis to fulminant hepatic failure. Even cardiotoxicity and multi organ dysfunction are also seen in this poisoning leading to significant mortality and morbidity. The purpose of this study is to evaluate the clinical outcome, progression of a case of acute phosphorus poisoning and to compare the autopsy and histopathological findings with respect to the duration of survival. It is an observational study conducted on patients admitted to Mcgann Hospital, Shimoga and autopsies conducted in the mortuary. As per inclusion and exclusion criteria cases are included and excluded, and the collected data was entered in a prestructured proforma. Cases were mainly distributed in young adult age group. Few cases were also noted in children which were found to be accidental in nature. There was no such gender variation regarding consumption of this poison. High mortality rate found in this poisoning with hepatotoxicity, cardiotoxicity and cardiogenic shock. Most of the cases with yellow phosphorus poisoning were stable on first and second day, subsequently worsened on third or fourth day with multiple organ dysfunction due to action of the absorbed poison. In most of the autopsies hepatic necrosis was observed as a sign of fulminant hepatic failure. So, the chemical content of poison is very important for the prognosis, intensive monitoring and early interventions.

Keywords

Hepatotoxicity; Cardiotoxicity; Rodenticides; Phosphorus; Rodenticide

Introduction

Elemental phosphorus exists in the environment in form of four allotropes—red, white, violet and black. Among these four, red phosphorus is odourless, tasteless, nonvolatile, insoluble, and unabsorbable, and therefore nontoxic when ingested. It is formed by heating white phosphorus to 250 degrees centigrade in vacuum. Pyrophoric, translucent, waxy white phosphorus (with impurities, it becomes yellow), on the other hand, is a severe local and systemic toxin causing damage to gastrointestinal, hepatic, cardiovascular, and renal systems. Yellow phosphorus (YP) is used in fireworks, fertilizer and rat baits.[1] Rodenticides are available as powders or pastes containing 2%–5% of YP.[2] Being an uncoupler of oxidative phosphorylation in hepatocytes, phosphorus decreases ATP production which leads to decrease transformation of triglyceride to beta lipoprotein and thereby rapid rise of hepatic triglyceride. Massive hepatic steatosis and zone 1 hepatic necrosis is the hallmark of YP toxicity. The estimated dose of YP that is lethal to the liver is 1 mg/kg, and the ingestion of that amount results in death due to acute liver failure and cardiovascular collapse.[3] In developing countries, accidental poisoning by oral intake in children, is common. The only definitive treatment for acute liver failure due to the ingestion of YP is liver transplantation because no Antidote or medical treatment is available to reverse the toxic effects on the liver.[4,7] This study was conducted to evaluate the clinical outcome, progression of the cases of acute phosphorus poisoning and to compare the autopsy and histopathological findings with respect to the duration of survival.

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Materials and Methods

It is an observational study conducted on patients admitted to Hospital and autopsies conducted in the mortuary of Shimoga institute of medical science, Karnataka. As reported cases of phosphorus poisoning is quite rare in this Malnad region, all the cases of phosphorus poisoning reviewed between January 2016 and January 2017 were include in this study. Total 11 cases (6 females and 5 males) of yellow phosphorus poisoning cases were noted in this time duration. Here we have included all those clinically diagnosed cases of phosphorus poisoning admitted in the hospital and the autopsies conducted in the mortuary with alleged history of phosphorus poisoning which later on confirmed by chemical analysis report. Details regarding age, sex, marital status, occupation, type of poison, route of exposure, outcome of

poisoning, laboratory parameters and organ specific findings were collected from the hospital records and postmortem reports. The data so collected were entered in a pre-designed proforma, compiled, tabulated and were analyzed by descriptive statistical methods and representing the data in the form of appropriate tables and graphs, to draw the correlation between the different factors and outcome.

Autopsy cases in which the manner of death concluded as natural after autopsy and unidentified and decomposed bodies due to lack of proper history and proper findings were not included in the study. Hospital admitted cases which were referred to other centers before necessary investigation and confirmation were also excluded.

Results

In the present study, 11 cases of yellow phosphorus poisoning were reviewed. The majority of the cases were in the age group of 21-40 years. It was also found that the incidence of poisoning decreased with increasing age. In all the cases, the route of exposure was oral. Males (five, 45.45%) and females (six, 54.54%) and 7 cases (63.63%) were married. Occupation wise, poisoning was more commonly found among housewives (five, 45.45%). Most of the cases were suicidal (nine, 81.81%). Two cases were accidental in nature. Six patients (54.54%) had subclinical hepatic injury and two patients (18.18%) had no clinical or biochemical evidence of hepatic damage. Liver function test (LFT) and renal function test (RFT) derangements were seen mostly with yellow phosphorus after 2-3 days of consumption of poison in our study (Table 1). All of the patients recovered with supportive therapy except for two cases of hepatocellular necrosis and 1 case of acute fulminant hepatic failure who succumb to death after day 5. In autopsy yellow greasy soft fatty enlarged liver along with associated jaundice was noted. Along with that tongue, esophagus and stomach were congested (Figure 1). Gastric erosion and hemorrhage were present (Figure 2). Sub-endocardial and epicardial hemorrhage was also noted in few cases (Figure 3). Kidneys were pale and swollen.

On histopathological examination, sinusoidal dilation, micro and macroscopic steatosis & signs of hepatic necrosis were observed (Figure no 4 & 5). Organs were sent for chemical analysis which confirmed the presence of phosphorus. Mortality rate of our study was 27%, confirming that Phosphorus is very lethal when ingested. Out of these three deceased, one case was a pregnant female. After admission within 48 hours, fetus was aborted at 20 weeks of gestation. Then because of minimum health issues, the family members taken her to home but unfortunately brought her again to the emergency after deterioration on day four. She died on day six and brought for autopsy. On autopsy signs of recent abortion along with suggestive features of hepatotoxicity were present.

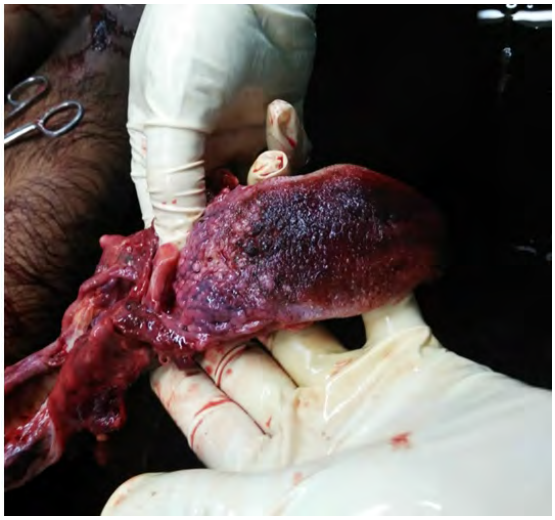

Figure 1: Congested tongue and esophagus

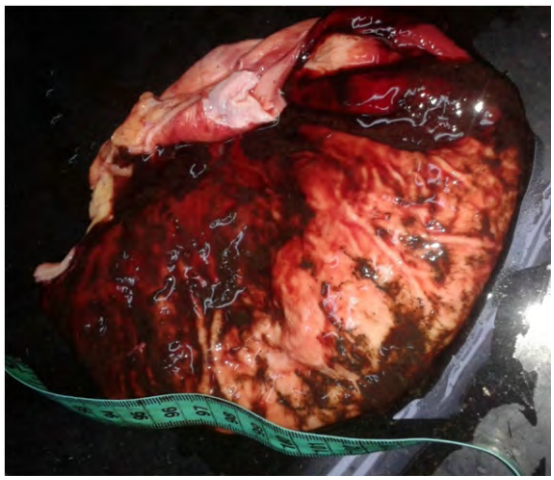

Figure 2: Gastric erosion and hemorrhage

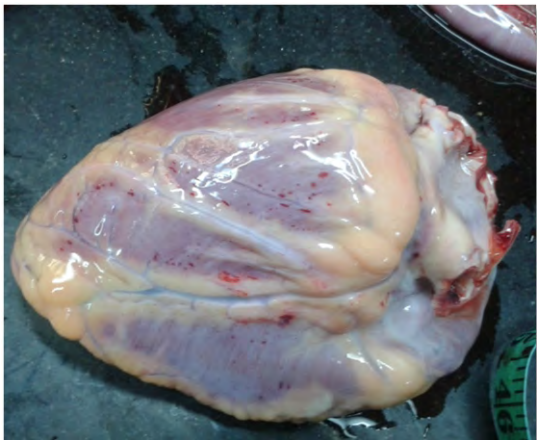

Figure 3: Epicardial hemorrhage

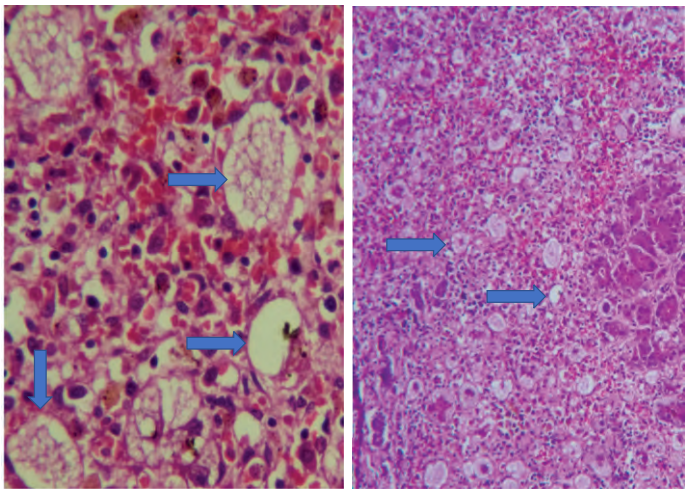

Figure 4: Histopathological picture of massive hepatic steatosis

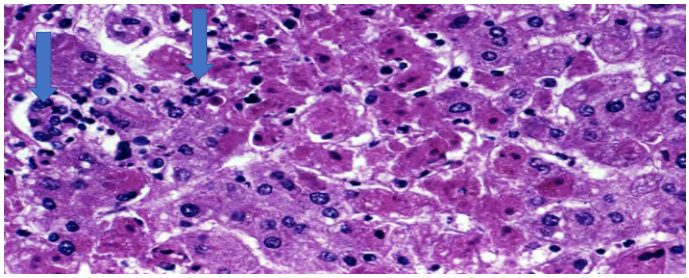

Figure 5: Histopathological picture of hepatic necrosis with inflammatory cell infiltration

Table 1: Liver and renal function test reports of hospital admitted cases

Parameter	Day 1	Day 3	Day 5	Day 6	Day 7-8	Day 10
TOTAL Bilirubin (mg)	1.4	2.7	7.1	---	3.5	1.2
SGOT (IU/L)	---	1436	---	1180	945	40
SGPT (IU/L)	---	358	---	193	175	35
Alk P. ase (IU/L)	---	48	---	51	58	---
PT	17	28	---	---	15	---
S. Creatinine (mg/dl)	0.7	1.4	1.2	1		0.8
B. Urea (mg/dl)	29	43	41	47		38

Discussion

In the present study, 11 cases of yellow phosphorus poisoning were reviewed between January 2016 and January 2017. In all the cases, the route of exposure was oral. Males (five, 45.45%) and females (six, 54.54%) and 7 cases (63.63%) were married. Our study correlates to the findings of others authors regarding this parameter. Ates et al. in their study, presented the outcome of 10 children who ingested fireworks containing yellow phosphorus, among them 6 boys and 4 girls were there. [7] Fernandez and Canizares reviewed 15 cases of yellow phosphorus fireworks poisoning where he studied its hepatotoxic effects on these patients.[2]

The majority of the cases were in the age group of 21-40 years. It was also found that the incidence of poisoning decreased with increasing age. Age related distribution of the cases was consistent to the findings of Fernandez and Canizares, Talley et al. Santos et al. Mauskaret al., nd McCarron et al. [2-6] This pattern of distribution can be explained by the fact that this age group people are mainly suffering from stress of the modern lifestyles, failure in love, family problems. Occupation wise, poisoning was commonly found among housewives (five, 45.45%). Factors, such as dowry, cruelty by the in-laws, family quarrels, maladjustment in married life, and dependence of women on husband, are responsible for the higher incidence of poisoning among homemakers. This finding was consistent with the findings of Srivastava and Arora, Geeta et al., Singh et al., and Statistics of NCBI 2010. [8-11]

Most of the cases were suicidal in nature (nine, 81.81%). Two cases were accidental in nature. The yellow phosphorus rodenticides pose a special problem in that the product directions suggest that the paste be applied to bread to enable ingestion by rodents, thus making it more appealing to the children thereby resulting in accidental deaths in this age group. Because of the luminescence and strong garlicky odour, it is rarely used for homicidal purpose.

Six patients (54.54%) had subclinical hepatic injury and 2 patients (18.18%) had no clinical or biochemical evidence of hepatic damage. All of these patients recovered with supportive therapy except for 3 cases of severe hepatic damage who succumb to death after day 5 (Table no 1). Generally, intoxication due to phosphorus passes through three stages. The first stage occurs during the first 24 h in which patient is either asymptomatic or has signs and symptoms of local gastrointestinal irritation. There may be the mild elevation of liver enzymes and bilirubin in the second stage. The third stage (advanced) occurs after 72 h until the resolution of symptoms or death. In this stage we can see rapid elevation of liver enzymes. In our study LFT and RFT derangements were seen mostly with yellow phosphorus after 2-3 days of consumption which shows gradual regression latter on (Table no 2). Similar to the findings of Fernandez and Canizares[2], patients with YP poisoning mainly present with acute hepatic failure, coagulopathy, and deranged liver function, early elevations in transaminase, alkaline phosphatase, derangement in PT, and associated metabolic acidosis and was witnessed in our study

group also.

As histopthological finding sinusoidal dilation, micro and macroscopic steatosis, hepatic necrosis was notes in the slides (Figure no 4 & 5). As phosphorus prevents oxidative phosphorylation in hepatocytes, ATP production will be decreased automatically. This phenomenon leads to decrease conversion of triglyceride to beta lipoprotein and thereby leads to accumulation of hepatic triglyceride in the hepatocytes. Being a protoplasmic poison, which affects the cellular oxidation leads to hepatic necrosis. That's why massive hepatic steatosis and hepatic necrosis with infiltration of inflammatory cell in the cytoplasm of hepatocytes (zone 1) are the hallmark of YP toxicity. This kind of histopathological findings corresponds to the findings observed by Fernandez and Canizares, and Santos et al.[2,4] Out of these three deceased, one case was a pregnant female. Because of minimum health issues, the family members taken her to home but unfortunately brought her again to the emergency after deterioration on day four. She died on day six and brought for autopsy. It can be explained by the fact that second stage which occurs between 24 and 72 h after ingestion, is apparently an asymptomatic period, and the patient may be wrongly discharged prematurely.

Conclusion

Yellow phosphorus consumption associated with the late manifestation of liver cell injury and MODS. So should be observed for one week without early discharge of the patient. As there are no antidotes for these compounds' active symptomatic management with early stomach wash may be benefitted. Public should get alerted with the high mortality rate of rat killer poison consumption and should prevent it.

Ethical clearance: A prior approval was obtained from the Institutional Ethics Committee

Conflict of interest: None to declare

Source of funding: None to declare

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