A fatal case of multi-organ failure due to acute yellow phosphorus poisoning

Abstract

Yellow ~~P~~phosphorus is a non-metallic irritant used in various industrie~~sector~~s such as the rodenticide, firecracker, match, and fertili~~s~~zer industries. Yellow ~~P~~phosphorus poisoning is ~~responsible for deaths among~~fatal in both children and adults. Accidental yellow phosphorus poisoning is frequently reported in children, whereas suicidal consumption is not uncommon among adults. Herein, we present the case of a 30-year-old ~~female patient~~woman who ingested Ratol paste containing yellow phosphorus in an attempt to commit suicide. Her initial chief complaints were nausea, vomiting, and diarrhoea during ~~hospitalization~~hospitalisation, followed by a symptomless phase with stable ~~vitals~~ vital parameters on the second day of hospitalisation. She~~; she~~ was managed ~~conservatively~~conservatively and discharged ~~. She took discharge~~ against medical advice on the second day. ~~She~~ However, she was readmitted to the same hospital on the fourth day after Ratol ingestion ~~after two days~~ with complaints ~~ing~~ of ~~generalized~~ generalised weakness, body pain, drowsiness, loss of appetite, and breathing difficulties. She developed severe complications owing~~due~~ to the intoxication and died. An autopsy was performed, and ~~. Histopathological~~ histopathological and ~~the~~ toxicological examination~~s~~ ~~were carried out. We found~~revealed characteristic features of yellow phosphorus toxicity in her organs. We concluded that the cause of death was hepatic encephalopathy and multi-organ dysfunction syndrome caused by ~~the~~ yellow phosphorus poisoning.

Keywords: Phosphorus, Hepatic encephalopathy, Multi-~~ple~~ organ failure

Introduction

In India, ~~around~~ approximately 70% of rural households still depend primarily on agriculture for their livelihood [1].[1] ~~The~~ An unchecked growth of rodents ~~like~~ such as rats around houses and sometimes in farms and fields can be problematic because ~~they~~ rodents can potentially spread diseases that would hamper ~~the~~ food supply and consequently lead to economic loss. Hence, rodenticides are widely marketed in India, and various preparations are available, such as yellow phosphorus in the form of Ratol paste and powder. ~~This preparation is cheap and easily available in the open market and on online e-commerce sites in India, which contributes to the frequently reported cases of suicides due to Ratol ingestion. Accidental consumption of Ratol paste at home is not uncommon among children.~~ Available r~~R~~odenticides contain~~ing~~ 3-5% yellow phosphorus, which is highly toxic; no antidote is available for these rodenticides. ~~are currently available, and Ratol paste contains 3% yellow phosphorus.[2] It is highly toxic and does not have any antidote.~~ Ratol paste contains 3% yellow phosphorus and is cheap and readily available in the open market and on online e-commerce sites in India. Accidental home consumption of Ratol paste is not uncommon among children because~~Accidental poisoning with Ratol paste is very common since~~ it is ~~very~~ similar to toothpaste [3–5].[3-5] Easy availability contributes to the frequently reported cases of suicide due to Ratol paste ingestion. Yellow phosphorus is also used in fireworks and matches, which leads to chronic poisoning in workers [6].[6]

Yellow phosphorus is a kind of non-metallic protoplasmic poison. It is rapidly absorbed from the digestive tract and is primarily ~~metabolized~~ metabolised by the liver [7].[7] The ~~smallest~~ minimum fatal dose of yellow phosphorus is 8 mg;~~,~~ ~~but~~ however, the usual fatal dose is 1 mg/kg [8].[8] The v~~V~~omitus after phosphorus ingestion is luminescent and has a characteristic garlic odour. If the patient

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survives the initial gastrointestinal irritation phase, hepatic toxicity ensues secondary to systemic poisoning. Herein, we report a ~~fatal~~ case of suicidal yellow phosphorus poisoning.

CASE REPORT

A 30-year-old ~~female patient~~woman was admitted to the Department of Trauma ~~&~~ and Emergency Medicine in a tertiary care hospital with complaints ~~complaining~~ of a burning sensation in the mouth, nausea, and vomiting. The vomitus had a garlic odour. Her husband stated that she had ingested 10-12 g Ratol paste approximately ~~about~~ 2 h~~ours~~ before. The patient was conscious with a blood pressure of 140/80 mmHg, pulse rate of 84 beats per minute, and respiratory rate of 20 breaths per minute. Results of different tests performed at the time of admission are shown in Table 1. Gastric lavage ~~was done~~ with 1:5000 $KMnO_4$ and activated charcoal was performed. ~~. The patient was conscious with a blood pressure of 140/80 mmHg, pulse rate of 84 beats per minute, and respiratory rate of 20 breaths per minute. Results of different tests at the time of admission are shown in Table 1.~~

Bedside abdominal ultrasonography ~~(USG) of the abdomen~~ was unremarkable. Symptomatic management was achieved~~done~~ with N-acetylcysteine (NAC). On the second day of admission, the~~r~~ patient's clinical status improved, and she was asymptomatic. The patient, ~~and~~ supported by her relative, ~~took~~ was discharged against medical advice~~discharge despite insistent medical advice to remain hospitalized~~. On the fourth day after Ratol ingestion, the patient returned to the hospital with ~~generalized~~ generalised weakness, body pain, drowsiness, and breathing difficulty. She ~~was drowsy~~presented with hypotension (blood pressure of 88/58 mmHg), tachycardia (110 b~~pm~~eats per minute), and tachypnoea.

On general examination, icterus was present. Laboratory workup results from~~on~~ the fourth day are shown in Table 1. ~~A bedside~~Bedside abdominal ultrasonography~~USG~~ showed hepatomegaly and fatty changes. Hence, a provisional diagnosis of yellow phosphorus poisoning with hepatic encephalopathy and multi-organ failure was made. On the fifth day, the patient developed sudden-onset bradycardia and hypotension, which rapidly ~~led~~ progressed to ~~the patient's~~ death with no opportunity to perform liver transplantation. An autopsy was ~~conducted~~performed.

AUTOPSY FINDINGS

On post-mortem examination, scleral icterus was present. Both pleural cavities were filled with approximately 350 ~~ml~~ mL of haemorrhagic fluid ~~each~~. Interlobular fissures of both lungs showed petechial haemorrhages. The right and left lungs weighed 410 g and 455 g, respectively (RV: 450 g and 375 g, respectively). The cut section showed that the lungs were congested. Pinpoint haemorrhages were present over the heart surface. The ~~weight of the~~ heart weighed ~~was about~~ approximately 270 g (RV: 243 g). The peritoneal cavity was filled with 750 ~~ml~~ mL of haemorrhagic effusion. The stomach mucosa was haemorrhagic. Pinpoint to pinhead-sized petechial haemorrhages were present over the mesenteries, liver, and kidneys (Figure 1A, 1B). ~~Yellowish~~ A yellowish discoloration ~~of the~~was present on the liver, kidney~~s~~, and brain surfaces ~~was present~~. The weight of the liver was about 1100 g (RV: 1100 g); the weights of the right and left kidneys ~~was~~ were 114 g and 129 g, respectively (RV: for both 288 g); and the weight of the brain was 1177 g (RV: 1233 g). ~~The cut~~Cut sections of the liver (Figure 1C) and kidneys (Figure 1D) showed pinpoint haemorrhages.

Figure 1. Gross examination of the~~:~~ (A) ~~–~~ liver showing pinhead-sized haemorrhages over the surface and yellowish discoloration; (B) ~~–~~ kidneys showing pinpoint haemorrhages over the surface and

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yellowish discoloration; (C)— cut surface of the liver depicting pinpoint haemorrhages within the hepatic parenchyma; (D)— cut section of the kidney depicting pinpoint haemorrhages and yellowish discoloration of the renal parenchyma.

Histopathological examination ~~of the lung~~ showed acute interstitial inflammatory cell infiltrates along with multiple focal alveolar haemorrhages in both lungs (Figure 2A, 2B). The heart showed focal necrotic fibres with focal acute myocarditis (Figure 2C).

Figure 2.

Photomicrographs of the (A and B) lung ~~(A and B)~~ and (C) ~~the~~ heart ~~(C)~~. (A and B)— Inflammatory cell infiltrates ~~along with~~and focal alveolar haemorrhage (H&E, 4X and 10X, respectively); (C)— Focal necrotic fibres with neutrophilic infiltrates, suggestive of focal acute myocarditis~~.~~ (H&E, 10X).

The liver showed non-zonal necrosis, karyorrhexis, ~~vacuolization~~vacuolisation, intracellular bile pigment deposition, mild periportal inflammation, and piecemeal necrosis (Figure 3A, 3B). The kidneys showed ~~vacuolization~~ vacuolisation of proximal tubular cells and multifocal necrosis of the ~~lining~~ epithelial cell linings with sparing of the glomeruli (Figure 3C). The pancreas showed focal fat necrosis along with necrosis of large areas of the pancreatic parenchyma (Figure 3D). Histological analysis of the bone marrow was not ~~carried out~~performed. All features were suggestive of multi-organ failure comprising submassive hepatic necrosis, focal acute myocarditis, acute renal tubular necrosis, and pancreatic necrosis typical of phosphorus poisoning, which was confirmed after a qualitative chemical analysis report showed the presence of phosphorus in tissues sampled during ~~the~~ autopsy.

Figure 3.

Photomicrographs of the (A, B) liver ~~(A, B)~~, (C) kidney ~~(C)~~, and (D) pancreas ~~(D)~~. (A)— Non-zonal necrosis, karyorrhexis, ~~vacuolization~~vacuolisation, and piecemeal necrosis (H&E, 4X);~~,~~ (B)— ~~Vacuolization~~ Vacuolisation in liver tissue (H&E, 10X); (C)— ~~Vacuolization~~ Vacuolisation of the proximal tubular cells and multifocal necrosis of the lining epithelial cells with sparing of the glomeruli (H&E, 10X); (D)— Parenchymal necrosis of the pancreas (H&E, 10X).

DISCUSSION

In this report, we present the case of a 30-year-old woman who died of yellow phosphorus poisoning. Yellow phosphorus is a toxic substance that is used in matches, fireworks, and rodenticides [9].[9] Several cases ~~Developing and underdeveloped countries have reported~~ of intoxication with yellow phosphorus in developing and underdeveloped countries have been reported;~~,~~ ~~but~~ however, this is rarely reported in developed countries. In developing countries, ~~the~~ intoxication generally results from accidental oral ingestion, although suicidal ingestion is also not uncommon [10,11].~~10,11~~

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Ratol paste contains 3% yellow phosphorus, a far more toxic substance than red phosphorus [12,13].[12,13] Yellow phosphorus is ~~categorized~~ categorised as a highly lethal rodenticide when ingested in doses exceeding 1 mg/kg. In our case, the deceased ingested approximately 10-12 g of yellow phosphorus rodenticide. ~~As reported in the literature, victims of~~Patients with yellow phosphorus poisoning may be initially symptomatic; however, ~~; however,~~ recovery is observed after 2-3 days of ingestion.~~,~~ ~~but later~~Nevertheless, symptoms of acute liver failure subsequently develop [12].[12] In our case, the patient had a similar progression of complications. Hence, we surmise that patients with acute yellow phosphorus poisoning should be monitored closely for ~~a~~ 1 week since mortality is not recorded ~~-~~after 8 days [14].[14]

Yellow phosphorus not only affects the liver~~,~~ but ~~its toxic effects are~~ also ~~observed in~~ the central nervous system~~,~~ and may cause ~~including~~ restlessness, irritability, drowsiness, lethargy, stupor, and coma owing~~due~~ to liver ~~disfunction~~dysfunction [15,16].~~;~~[15,16] Moreover, cardiovascular toxicity with arrhythmias and hemodynamic instability [12,13],[12,13] acute tubular necrosis, and bone marrow toxicity such as thrombocytopenia are ~~also~~ observed [17].[17] Fernandez and Canizares [18][18] reviewed 15 cases of yellow phosphorus poisoning and found that 87% patients had some hepatic derangement after yellow phosphorus poisoning, and 27% developed fulminant hepatic failure and died. Histological analysis of the liver shows steatohepatitis and necrosis. Santos et al. [19][19] described three cases of white phosphorus intoxication with acute liver failure secondary to the consumption of firecrackers. In one case, liver injury improved with supportive care; in the second, the patient required liver transplantation; and the third ~~case had a fatal outcome~~patient died. Similarly, Nalabothu et al. [4][4] found a 28% mortality rate ~~in his study~~ that was associated with the Model for End-stage Liver Disease (MELD) score. A MELD score greater than 40 was related to death, ~~while~~ whereas survivors presented a score lower than 12. Our patient's highest MELD score was 36. McCarron [16][16] observed a mortality rate of 23-73% associated with yellow phosphorus toxicity, depending on clinical manifestations; ~~those~~ patients with early central nervous system manifestations had a poorer prognosis. There is no antidote for phosphorus poisoning, [12,15][12,15] and the only treatment is early decontamination followed by monitoring of liver function and supportive care [12,13].[12,13] Some researchers recommend gastric lavage with 1:5000 $KMnO_4$ followed by activated charcoal and using mineral oil as a cathartic [16,17].[16,17] Fernandez et al. [18][18] found ~~that~~ no benefit from NAC ~~shows~~ use~~no benefit~~, whereas Nalabothu et al. [4][4] suggested that the early administration ~~use~~ of NAC improves outcomes for all patients with rodenticide poisoning~~s~~ ~~with~~ having liver failure, and survival rates vary with the timing of NAC administration. In their study, the survival rates ~~was~~ were 76%, 40%, and 23% if NAC was administered on Day 1, 2, and 3, respectively, following ~~40% if administered on Day 2, and 23% if administered on or after Day 3 of~~ rodenticide ingestion [4].[4] However, this outcome ~~is~~ was confounded by an early gastric lavage in patients who were ~~hospitalized~~ hospitalised immediately after rodenticide ingestion. In our case, NAC was administered on Day 1 after yellow phosphorus poisoning was confirmed; however, the outcome was unfavourable. Yellow phosphorus is rapidly absorbed through the gastrointestinal mucosa, and approximately 70% accumulates in the liver within 2 to 3 h~~ours~~ of ingestion. It also accumulates to a lesser extent in the heart (12%), kidneys (4%), pancreas (0.4%), and brain (0.39%), and leads to damage in the~~e~~ose organs [16].[16] Histopathological changes, in our case, were mainly detected in the lungs, heart, liver, kidneys, and pancreas. No significant pathological change, other than congestion, was observed in the brain. The characteristic histopathological findings are fat infiltration, vacuolization, and necrosis in different organs, mainly the liver and kidney, along with focal myocarditis. Multi-organ failure, with fulminant hepatic failure, acute tubular necrosis, and toxic myocarditis, is responsible for a fatal outcome. The toxic effect of yellow phosphorus occurs in the endoplasmic reticulum and ~~the mitochondria~~mitochondria, leading to (i) decreased synthesis of

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the apolipoprotein portion of very low-density lipoproteins ~~(VLDL)~~, (ii) decreased production of adenosine triphosphate, and (iii) inhibition of fatty acid oxidation. This combined effect leads to fat deposition and cellular damage in different organs [20].~~39~~

CONCLUSION

Yellow phosphorus is a cheap and effective rodenticide. However, the number of cases of accidental poisoning~~s~~ and deaths among children and adults cannot be neglected. ~~We agree with several other authors that the use of yellow phosphorus in rodenticides and fireworks in its currently packaged and lethal form should be banned.~~ We have ~~also~~ discussed the diagnosis, ~~and~~ management, and prognosis of liver failure due to yellow phosphorus ~~and its prognosis~~poisoning and suggest that the use of yellow phosphorus in rodenticides and fireworks in its currently packaged and lethal form should be banned. Early and effective supportive care is ~~the~~ key ~~factor in~~ to reducing the morbidity and mortality associated with yellow phosphorus poisoning.

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