International Journal of Biological and Pharmaceutical Sciences Archive

ISSN: 0799-6616 (Online)

Journal homepage: https://ijbpsa.com/

(CASE REPORT)

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A case report on Ratol paste poisoning in addition with oleander buds

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International Journal of Biological and Pharmaceutical Sciences Archive, 2024, 08(02), 063-066

Publication history: Received on 08 November 2024; revised on 18 December 2024; accepted on 21 December 2024

Article DOI: https://doi.org/10.53771/ijbpsa.2024.8.2.0092

Abstract

Rodenticide poisoning, particularly with yellow phosphorus-based formulations like Ratol paste, poses a significant public health challenge in rural India due to its widespread availability and high toxicity. This report describes a fatal case of suicidal poisoning involving a 20-year-old male who ingested a lethal dose of Ratol paste alongside oleander buds. Despite prompt medical interventions, including gastric lavage, intravenous N-acetylcysteine (NAC), vitamin K, plasmapheresis, and supportive care, the patient succumbed to progressive hepatic encephalopathy, coagulopathy, and septic shock.

Yellow phosphorus, present in Ratol paste, is a potent toxin that rapidly affects multiple organ systems, primarily targeting the liver. Its mechanism includes disruption of fatty acid oxidation and ATP production, leading to severe hepatic and systemic toxicity. Oleander ingestion exacerbated the condition due to its additional cardiotoxic properties. The case underscores the critical need for stringent regulation of hazardous substances like Ratol paste and enhanced public awareness to mitigate accidental and intentional poisoning incidents.

Keywords: Rodenticide poisoning; Coagulopathy; Oleander poisoning; Plasmapheresis; Toxic hepatitis

1. Introduction

In rural India, where approximately 70% of households rely on agriculture as their primary source of livelihood [1], the unchecked proliferation of rodents, particularly rats, poses a significant challenge. These pests not only damage crops but also have the potential to spread diseases, further exacerbating the economic hardships faced by farmers. To mitigate these risks, rodenticides are commonly used, and among the various products available, yellow phosphorus-based formulations, such as Ratol paste and powder, are widely marketed. These rodenticides are inexpensive and easily accessible through both local markets and online platforms, making them a popular choice.

However, the widespread availability of Ratol paste, which contains 3% yellow phosphorus, has led to an alarming increase in cases of accidental poisoning, especially among children. [2] The paste's similarity to toothpaste makes it particularly dangerous in households with young children, who may ingest it unintentionally. [3,4,5] Yellow phosphorus, a highly toxic substance, has no known antidote and poses a severe threat to health even in small amounts. The smallest fatal dose is reported to be as low as 8 mg, with the typical lethal dose being around 1 mg/kg of body weight. [6]

Upon ingestion, yellow phosphorus is rapidly absorbed by the gastrointestinal tract and is primarily metabolized in the liver. The toxicity manifests in two stages: initial gastrointestinal distress, followed by hepatic toxicity if the patient survives the early phase. [7] A characteristic sign of poisoning is the luminescent vomit, which often has a distinct garlic odor. Given the high toxicity of yellow phosphorus, it is critical to address the public health risks associated with the

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availability of such substances. This report discusses a fatal case of suicidal poisoning due to the ingestion of Ratol paste, highlighting the need for greater regulation and public awareness regarding the dangers of these rodenticides.

2. Case report

A 20-year-old male patient was brought to the hospital with a reported history of ingesting Ratol paste (1 tube = 15 g) and oleander buds (4) at approximately 7:30 pm. Upon initial evaluation, gastric lavage was performed at a multispecialty hospital, and the patient was subsequently referred to a tertiary care facility. At the time of admission, the patient did not exhibit any signs of seizures, altered sensorium, vomiting, diarrhea, dyspnea, or chest/abdominal pain. Vital signs were stable, and the patient was afebrile, well-hydrated, and fully alert and oriented.

The management plan began with intravenous fluids, including Ringer's lactate (75 ml/hr), Normal Saline, and Dextrose Normal Saline, in addition to intravenous N-acetylcysteine (NAC) (30 mg/kg for 4 hours, escalated to 150 mg/kg for 12 hours, then 1.2 g IV BD). Vitamin K (10 mg IV OD) and Pantoprazole (40 mg IV) were also administered. On the second day, Ursodeoxycholic Acid (UDCA) 300 mg (1-1-1) was added to the treatment regimen. Abdominal and pelvic ultrasounds were performed, which showed no significant abnormalities.

After 12 hours of nil per os (NPO), the patient was transitioned to a liquid diet. Given concerns for potential toxin-induced shock or bicytopenia on day 3, intravenous Cefotaxime (1 g) was initiated, with continued administration of the other prescribed medications. Hourly blood pressure monitoring remained stable throughout the day. By day 4, the clinical diagnosis was confirmed as poisoning from rat killer paste and also with oleander buds, complicated by hepatic encephalopathy grade 1. The patient was also noted to have recovered from initial coagulopathy and shock, though bicytopenia was suspected. Due to increasing drowsiness, Rifaximin (550 mg) was added to the treatment regimen.

Laboratory testing for HIV, hepatitis B surface antigen (HBsAg), and hepatitis C (anti-HCV ELISA) were all negative. A nephrology consultation led to the placement of a double-lumen plasmapheresis (PLEX) catheter using the modified Seldinger technique, with 1 liter of plasma removed. Following the procedure, 10 units of fresh frozen plasma (FFP) and 500 ml of normal saline were administered. The patient's blood pressure remained stable at 130/80 mm Hg.

On day 5, hepatic encephalopathy worsened to grade 2, accompanied by electrolyte imbalances, including hyponatremia, hypokalemia, and hypocalcemia, which were corrected with intravenous calcium gluconate and potassium chloride. Two cycles of plasmapheresis were performed, and the patient's clinical status remained stable. However, by day 6, the patient developed grade 3 hepatic encephalopathy and coagulopathy, with progressive deterioration of liver function. Laboratory results indicated rising levels of prothrombin time (PT), alkaline phosphatase (ALP), alanine aminotransferase (SGPT), aspartate aminotransferase (SGOT), and total protein, suggestive of advancing hepatic failure. Renal function tests also showed increasing abnormalities, and the patient's INR rose to 9.9 with a PT of 50.8 seconds.

Table 1 Progression of SGOT and SGPT levels over consecutive days.

DAYS	SGOT	SGPT
DAY 1	11	5
DAY 2	18	10
DAY 3	25	22
DAY 4	346	319
DAY 5	911	853
DAY 6	1075	1891

On day 7, the patient was suspected of having grade 4 hepatic encephalopathy, toxic hepatitis, coagulopathy, and septic shock. Despite efforts to monitor blood pressure and SpO2, the patient's respiratory rate was 36 breaths/min, and pulse rate was 100 beats/min. The patient was stuporous with minimal response to pain. Intravenous noradrenaline was started at 0.5 mcg/kg/min and titrated to 1 mcg/kg/min to support circulation. Unfortunately, the patient's condition continued to deteriorate, and he became unconscious with no detectable respiratory or cardiovascular activity.

Following a cardiac arrest, cardiopulmonary resuscitation (CPR) was initiated, and intravenous adrenaline (1 cc) was administered. Despite intensive efforts, the patient could not be resuscitated and was declared dead.

3. Discussion

Yellow phosphorus is a highly toxic substance used in various industries, including match manufacturing, fireworks production, and as a rodenticide. While cases of yellow phosphorus poisoning are rare in developed countries, they are more frequently reported in developing and underdeveloped regions, where intoxication typically results from accidental ingestion, although suicidal ingestion is also common. [8,9,10]

Ratol paste, a common rodenticide in many developing countries, contains 3% yellow phosphorus, a substance that is significantly more toxic than red phosphorus. [11,12] When ingested at doses exceeding 1 mg/kg, yellow phosphorus is considered a highly lethal rodenticide. In the present case, the deceased consumed approximately 15 grams of yellow phosphorus, a lethal dose that led to acute toxicity. Victims of yellow phosphorus poisoning often present initially without symptoms, but they may later develop features of acute liver failure. [11]

In a study by Fernandez et al. [13] the use of N-acetylcysteine (NAC) showed no significant benefit in improving outcomes, whereas Nalabothu et al. [3] suggested that early administration of NAC can improve survival rates in rodenticide poisoning, particularly when liver failure is involved. In our case, NAC administration did not appear to have a positive effect, which may be attributed to the large dose of poison ingested in conjunction with the additional consumption of oleander buds.

Oleander poisoning, even in small amounts, can be fatal. Osterloh et al. [14] calculated that a lethal dose of oleander leaves in their patient was approximately 4 grams. Fernandez and Canizares [13] reviewed 15 cases of yellow phosphorus poisoning and found that 87% of patients experienced some form of hepatic derangement, and 27% developed fulminant hepatic failure, which proved fatal. Similar to these cases, the patient in this study exhibited gradual liver function deterioration, eventually progressing to liver failure.

Yellow phosphorus is rapidly absorbed through the gastrointestinal mucosa, with approximately 70% of the substance accumulating in the liver within 2 to 3 hours. It also accumulates in smaller quantities in other organs, including the heart (12%), kidneys (4%), pancreas (0.4%), and brain (0.39%), causing damage to these tissues as well. [15] The toxic effects of yellow phosphorus primarily target the endoplasmic reticulum and mitochondria within cells. These effects include (i) decreased synthesis of the apolipoprotein component of very low-density lipoproteins (VLDL), (ii) reduced production of adenosine triphosphate (ATP), and (iii) inhibition of fatty acid oxidation. The combined impact of these disruptions leads to fat deposition and cellular damage in multiple organs. [16]

4. Conclusion

This case emphasizes the severe toxicity of yellow phosphorus-based rodenticides like Ratol paste and the fatal outcomes associated with their misuse. It highlights the need for strict regulations, increased public awareness about the dangers of these substances, and enhanced medical strategies for managing poisoning cases to prevent such tragedies.

Compliance with ethical standards

Acknowledgments

We are greatly indebted to our highly respected and beloved madam, Dr. C N Nalini, M Pharm, Ph.D., Principal, C L Baid Metha College of Pharmacy for her benevolent and ever-helping arms which provided us with all the essential facilities in bringing out this paper. We would like to thank our beloved parents for trusting and supporting us. Above all, we would like to thank the Almighty God for their grace and blessings throughout the entire work.

Disclosure of conflict of interest

The authors declare that there is no conflict of interest.

Statement of informed consent

Oral informed consent was obtained from the study participants.

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