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CASE SERIES OF ZINC PHOSPHIDE POISONING PRESENTING TO TERTIARY CARE HOSPITAL

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ABSTRACT

Background: This case series analyzes 10 patients with ZP poisoning to assess clinical presentation, management strategies, and outcomes. **Methods:** A retrospective review of 10 patients admitted with ZP poisoning was conducted. Data on demographic details, clinical symptoms, quantity and form of ZP ingested, time to presentation, laboratory findings, treatment interventions, and outcomes were analyzed. **Results:** The most common symptoms were vomiting, abdominal pain, and central nervous system involvement such as drowsiness and disorientation. Higher doses (≥30 g) and yellow phosphorus formulations were associated with increased mortality. Three patients underwent plasmapheresis, with one surviving. Gastric lavage was controversial due to the risk of phosphine gas release and mucosal injury. The overall mortality rate was 50%, with delayed presentation, severe acidosis, and high-dose ingestion identified as poor prognostic factors. **Conclusions**: Zinc phosphide poisoning remains a critical medical emergency requiring early recognition and aggressive supportive care. Antioxidant therapy and plasmapheresis in select cases may improve outcomes. Avoiding gastric lavage while focusing on early decontamination, electrolyte correction, and organ support is essential.

KEYWORDS : Zinc phosphide , Metabolic acidosis, Plasmapheresis.

INTRODUCTION:

Zinc phosphide (ZP) is a widely used rodenticide in agricultural settings, posing a significant public health threat due to its high toxicity. Upon ingestion, ZP reacts with stomach acid to produce phosphine gas, a potent systemic poison that can lead to multi-organ dysfunction and death.(1) This case series aims to comprehensively analyze the clinical presentation, management challenges, and outcomes of patients with ZP poisoning admitted to our institution. By examining these cases, we aim to enhance our understanding of this potentially fatal condition, improve diagnostic and therapeutic approaches, and ultimately contribute to better patient outcomes. Here we present case series of 10 patients with alleged history of consumption of zinc phosphide in various form.

Case Series:

Case 1- (IPD:22540) 42 years old male, factory worker, resident of Vadgaon Budruk, Pune, presented in a drowsy, gasping state to casualty with alleged history of consumption of Rat Kill 30 gm cake form at his factory on 23/5/23 at 12:00 pm.

Case 2 - (IPD: 36069) 25 years old, male, student, resident of Kondhwa, Pune, presented to casualty in a drowsy arousable state with history of severe vomiting and breathlessness with alleged history of consumption of Rat Kill 6 gm in a powder form, on the terrace of his house on 17/10/23 at 12: 30pm

Case 3- (IPD: 42893)16 years old male, student, resident of Narhe, Pune, came to casualty with irritation of throat with alleged history of consumption of Rat Kill around 5 gm in a powder form at his house on 1/1/24 at 4:30pm

Case 4- (IPD: 44981) 28 years old, male, factory worker, resident of Narhe, Pune, presented to casualty with vomiting, headache, abdominal pain in a drowsy state with alleged history of consumption of Rat Kill 40 ml in a liquid form, in his house on 20/2/24 at 5: 30pm

Case 5- (IPD: 53453)20 years old, female, student resident of Katraj, Pune, came to casualty with c/o vomiting followed by sweating and irritability with alleged history of consumption of Rat Kill 10 gm in a powder form, in her house on 21/5/24 at 7: 30pm

Case 6- (IPD: 57460) 21 years old, female, student, resident of Katraj, Pune, came to casualty with c/o 10-12 episodes of vomiting and abdominal pain with alleged history of consumption of Rat Kill 5gm in a powder form at her house on 15/9/24 at 10: 30pm

Case 7- (IPD: 58634)38 years old male, IT professional, resident of Katraj, Pune, referred to casualty in intubated state from outside hospital with alleged history of consumption of Rat Kill 25 gm in a cake form at his home on 17/09/2024 at 10:00pm

Case 8- (IPD: 59664)16 years old, female, student, resident of Vadgaon Budruk, Pune, presented to casualty in drowsy irritable state with multiple episodes of vomiting with alleged history of consumption of Rat Kill 50 gm in a powder form at home on 30/09/2024 at 6pm

Case 9- (IPD: 59987) 26 years old, female, housewife resident of Narhe, Pune, came to casualty with c/o 5 episodes of vomiting, giddiness and headache with alleged history of consumption of Rat Kill 10gm in a powder form at her house on 15/11/24 at 4: 30pm

Case 10- (IPD: 64674) 21 years old, female, student, resident of Narhe, Pune, presented with severe vomiting, giddiness, abdominal pain with alleged history of consumption of Rat Kill 60 gm in a powder form, at her house on 26/12/24 at 7: 30pm

Table 1 : Summary Of All 10 Cases Presentation , Laboratory Findings And Their Outcome.

CASES : UHID	CLINICAL PRESENTATION	CONTENT OF SUBSTANCE ABUSE	TIME LAPSE BETWEEN CONSUMPTION AND PRESENTATION	LABORATORY FINDINGS	TREATMENT	OUTCOME

CASE 1: 22540 42/M	Drowsy, disoriented and gasping state, intubated i/v/o low GCS.	30 gm zinc phosphide cake. (2- 5 % yellow phosphorus)	8 HOURS ICU STAY : 4 HOURS	Severe metabolic acidosis with acute hepatitis.	Mechanical ventilation INJ. SODABICARBONATE INJ. N-ACETYLCYSTEINE	SUCCUMBED
CASE 2: 36069 25/M	Drowsy, arousable state with multiple episodes of vomiting.	6 gm zinc phosphide powder. (5-10% red phosphorus)	3 HOURS ICU STAY : 48 HOURS	Metabolic alkalosis with hypokalemia.	IV Fluids with bsl monitoring during NBM status. KESOL correction. ACITVATED CHARCOAL INJ. MGSO4	DISCHARGED.
CASE 3: 42893 16/M	Throat irritation with nausea and vomiting.	5 gm zinc phosphide powder. (5-10% red phosphorus)	2 HOURS ICU STAY : 48 HOURS	Within normal limits.	IV Fluids with bsl monitoring during NBM status.	DISCHARGED
CASE 4: 44981 28/M	Irritable, drowsy not responding to verbal commands	40 ml zinc phosphide liquid. (1-3% yellow phosphorus)	7 HOURS ICU STAY : 6 HOURS	Severe metabolic acidosis	IV Fluids with bsl monitoring during NBM hours. Mechanical ventilation. ACITVATED CHARCOAL INJ. SODABICARBONATE INJ. N-ACETYLCYSTEINE	SUCCUMBED
CASE 5: 53453 20/F	Vomiting, sweating and irritability.	10 gm zinc phosphide powder. (5-10% red phosphorus)	2 HOURS ICU STAY : 48 HOURS	Within normal limits.	IV Fluids with bsl monitoring during NBM hours ACITVATED CHARCOAL	DISCHARGED
CASE 6: 57460 21/F	10-12 episodes of vomiting with abdominal pain.	5 gm zinc phosphide powder. (5-10% red phosphorus)	8 HOURS ICU STAY : 48 HOURS	Hypokalemia with hypochloremia	IV Fluids with bsl monitoring during NBM hours KESOL correction.	DISCHARGED
CASE 7: 58634 38/M	Referred in intubated state with no response to DPS . GCS : E1VTM1	25 gm zinc phosphide cake. (2- 5 % yellow phosphorus)	2 HOURS ICU STAY : 12 DAYS	Sever metabolic acidosis with compensated respiratory alkalosis with acute fulminant hepatitis with transaminitis.	INJ. N-ACETYLCYSTEINE INJ. GLUTATHIONE ACITVATED CHARCOAL Mechanical ventilation. INJ. MANNITOL Inj MGSO4 3 cycles of PLASMAPHARESIS.	DISCHARGED
CASE 8: 59664 16/F	Drowsy irritable state with multiple episodes of vomiting	50 gm zinc phosphide powder. (5-10% red phosphorus)	2 HOURS ICU STAY : 16 HOURS	Sever metabolic acidosis with dilated cardiomyopathy (EF 25%)	IV Fluids with bsl monitoring during NBM hours. Mechanical ventilation. ACITVATED CHARCOAL INJ. SODABICARBONATE INJ. N-ACETYLCYSTEINE INJ. GLUTATHIONE	SUCCUMBED
CASE 9: 59987 26/F	5 episodes of vomiting with giddiness and headache.	10 gm zinc phosphide powder. (5-10% red phosphorus)	4 HOURS ICU STAY : 48 HOURS	Within normal limits.	IV Fluids with bsl monitoring during NBM hours ACITVATED CHARCOAL	DISCHARGED
CASE 10: 64674 21/F	Multiple episodes of vomiting, giddiness, abdominal pain and irritability.	60 gm zinc phosphide powder. (5-10% red phosphorus)	6 HOURS ICU STAY : 12 HOURS	Sever metabolic acidosis with transaminitis	IV Fluids with bsl monitoring during NBM hours. ACITVATED CHARCOAL INJ. SODABICARBONATE INJ. N-ACETYLCYSTEINE INJ. GLUTATHIONE Mechanical ventilation. PLASMAPHARESIS.	SUCCUMBED

DISCUSSION:

Zinc phosphide poisoning continues to be a severe toxicological emergency due to its high fatality rate and lack of a specific antidote. It poses a severe toxicological challenge, with diverse clinical presentations and significant mortality risk, as highlighted in this case series of 10 patients.

For many years, it was believed that clinical presentation of zinc phosphide poisoning was due to liberation of phosphine (PH 3) on contact with acidic content of the stomach. However, relatively long hiatus between ingestion of Zn2P3 and presentation of its systemic toxicity, and progression of acute liver failure could not be account by the current opinion.

Hence, other innovative theory signified that phosphonium, an intermediate product will create and pass through the stomach, which then will reduce to produce PH 3 in the luminal tract (1)

Common clinical presentation include gastrointestinal symptoms like nausea, vomiting (sometimes severe and persistent), abdominal pain, and throat irritation, which were typically among the earliest signs of toxicity. Central nervous system involvement was significant, with patients presenting with drowsiness, irritability, disorientation, reduced responsiveness and in severe cases involved respiratory failure requiring mechanical ventilation, metabolic acidosis, and organ dysfunction, including transaminitis and dilated cardiomyopathy. (1)

A dose of 5 g of zinc phosphide is toxic and can cause death. In about 30 minutes of ingestion phosphides produce toxicity rapidly and within 6 hours of ingestion death may occur (1). Patients who ingested higher doses (30–60 g) presented with profound systemic toxicity, while those with smaller doses (5–10 g) had milder symptoms or normal laboratory parameters. Patients who ingested formulations with higher red phosphorus content (5–10%) in powder form with high doses generally exhibited metabolic acidosis and systemic toxicity, often leading to poor outcomes, including fatalities. Conversely, formulations with yellow phosphorus (1–3%) in liquid and cake form were associated with severe systemic toxicity and fatalities even at smaller doses, reflecting the higher bioavailability of yellow phosphorus formulations. (2, 3)

Role of gastric lavage in zinc phosphide poisoning is controversial and defined in various literatures. To decrease the absorption of the zinc phosphide in the exposed patient, gastric lavage and activated charcoal are described as effective measures. These should not be done with water because it would favor the formation of phosphine; therefore, the literature reports performing gastric lavage with coconut oil, potassium permanganate (1:10,000) or liquid paraffin, considering that stomach acid stimulates the conversion to phosphine gas (4). However gastric lavage is not recommended for several reasons such zinc phosphide causing severe inflammation and necrosis of the gastrointestinal tract, increasing the risk of perforation during gastric lavage.(4) Gastric lavage may not effectively remove the toxin, as zinc phosphide is rapidly absorbed into the bloodstream which can delay the administration of more effective treatments, such as activated charcoal and supportive care (5)

Treatment with antioxidant therapy and magnesium electrolyte supplementation is indicated from the beginning. Nacetylcysteine is used to prevent myocardial oxidative damage and restore intracellular glutathione levels. Similarly, to prevent liver necrosis, consider that phosphine induces oxidative stress, causing lipid peroxidation, and reduces glutathione, leading to magnesium and magnesium(7)dependent glutathione depletion, and electrolyte supplementation with magnesium sulfate is initiated. Plasmapheresis is used in selected cases to resolve metabolic problems and oxidative stress. In one case in this study, a patient with severe metabolic acidosis and functional impairment underwent early and multiple plasma exchange cycles. Patients showed good results with prompt administration of antioxidant therapy, including plasma exchange and osmotic diuretics.(8)

CONCLUSION :

Despite these efforts, mortality remains at 50% and occurs mostly in patients with delayed presentation, severe acidosis, or high intake. Survivors benefit from rapid recovery, improved metabolism, and close monitoring.(9) This study highlights the importance of early diagnosis, intensive supportive care, and the need for further research into treatments to improve the outcome of zinc phosphide poisoning. Phosphide poisoning is fatal and its symptoms are numerous. Gastric lavage should be avoided because it is ineffective and potentially dangerous. Lethality and severe toxicity depending on zinc phosphide content and toxin ingestion. Early diagnosis and intensive support are critical to improving patient outcomes.(11)

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