




CASE REPORT

Suicide attempt using zinc phosphide rodenticide: A case report and literature review

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Abstract

The patients with a history of intentional or unintentional consumption of rodenticide compounds, especially ZnP, it is necessary to assess ABG and abdominal radiography.

KEYWORDS

hepatic encephalopathy, rodenticide, suicide, zinc phosphide

1 | INTRODUCTION

Zinc phosphide (ZnP) is a gray-to-black inorganic chemical compound, which is used as an effective rodenticide and is abused for suicide as well. A 20-year-old man was referred with gastrointestinal symptoms following intentional ingestion of ZnP powder. Despite intensive therapeutic procedures, hepatic encephalopathy occurred and he died.

Zinc phosphide [Zn_3P_2] (ZnP) is an effective rodenticide, and it protects grain against different species of mice. It is a gray-to-black inorganic chemical compound that is available in different forms, such as paste, bait, granules, dust, and tracking powder formulations.¹⁻³ ZnP is also

potentially toxic for mammals in acute exposure, even for humans, accidentally or intentionally through suicidal or homicidal uses.^{1,2,4} No existing specific antidote for ZnP poisoning leads to a high mortality rate of 37–100%, and only supportive care is considered.^{5,6} ZnP is converted into phosphine (PH₃) gas by hydrolysis in the acidic circumference of the stomach. PH₃ gas is an active metabolite that affects different parts of the body through the bloodstream.^{1,2,7}

As it was stated in many papers, there are several mechanisms involved in PH₃ poisoning, substantially inhibition of cytochrome C oxidase and oxidative respiration.^{4,7} The following mechanisms have also been described for ZnP toxicity: anticholinesterase activity leading to

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deterioration of oxyhemoglobin molecules, inhibition of catalase and peroxidase activity leading to lipid peroxidation (LPO), DNA damage, and alteration in mitochondrial morphology and oxidative respiration by inducing hypoxia in the liver.^{1,8}

PH₃ exerts both metabolic and non-metabolic toxic effects. PH₃ can affect the cardiovascular, respiratory, hepatobiliary, and hematologic systems, and also the gastrointestinal (GI) tract. Also, electrolyte and metabolic abnormalities, nephrological and neurological defects, and ultimately multi-organ failure have all been recognized with phosphide poisoning.^{2,4,7,8}

Herein, we present a 20-year-old patient who was referred to the emergency department after 12 days with acute liver failure due to ZnP poisoning.

2 | CASE PRESENTATION

A 20-year-old man was referred to the emergency ward on March 31, 2019, following the intentional consumption of a box of ZnP powder 1 week ago. He was admitted to a local hospital and received conservative treatment and gastric lavage. After 2 days, he was transferred to the general hospital for further evaluation.

The vital signs were as follows: blood pressure (BP): 80/30 mmHg, pulse rate (PR):122 beats/minute, temperature: 36.5°C, respiratory rate (RR): 23/minute, and O₂ saturation: 98%. The patient was awake and complained of nausea and vomiting (N/V). There were no abnormal findings on physical examination and no history of other medical disorders. The electrocardiogram (ECG) showed sinus tachycardia. The results of laboratory tests on admission are shown in Table 1.

He received an intravenous (IV) infusion of N/S serum 3 Lit/24 h, stat IV pantoprazole 40 mg, and IV metoclopramide 10 mg. On the second day of hospitalization, the patient had mild tenderness in the right upper quadrant of the abdomen and no defecation. Charcoal and sorbitol were also given as a stat, which was then repeated every 4 h. The abdominal X-ray showed some pieces of radiopaque materials in the stomach and duodenum (Figure 1).

After 2 days, due to no clinical response, loss of consciousness (still responding to painful stimulations), PR of 140 beats/min; RR >30/min; and BP of 86/40 mmHg, the patient was transferred to the ICU and intubated under synchronized intermittent mandatory ventilation (SIMV) mode. The rise in liver enzymes (ALP = 363, SGOT = 2034, SGPT = 206, LDH = 1446), prothrombin time (PT: 27s), bilirubin (direct: 6.4 mg/dL, total: 15.4 mg/dL), and icteric appearance revealed ALF. He still had no defecation and developed oliguria following a progressive

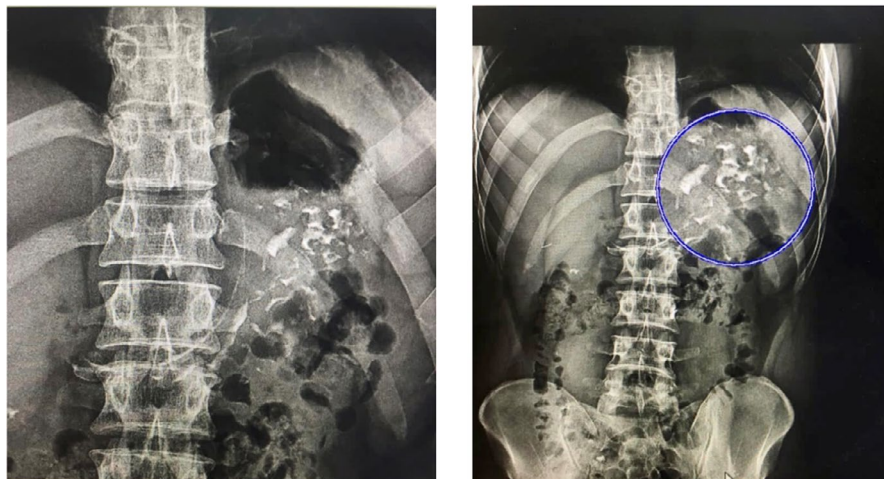
TABLE 1 Patient's on-arrival laboratory tests results

Laboratory tests	Reference values	On admission
HG: gr/dL	12–16	15
RBC: ×10 ⁶ mm ³	4.7–6.1	5.04
WBC: ×10 ³ mm ³	4.5–11	8.6
Lymph: %	20–50	44
Poly: %	37–72	51
HCT: %	35–47	42
Plt: ×10 ³ mm ³	130–400	147
BG-Rh -	–	AB(+)
PT: seconds	11–13.5	27
PTT: seconds	25–35	51
INR:	0.8–1.1	4.1
pH:	7.35–7.45	7.25
HCO ₃ : mmol/L	19–27	7.8
PCO ₂ : mmHg	35–45	18
SO ₂ c: %	95–98	98
ALP: IU/L	20–140	363
SGOT: IU	10–40	2034
SGPT: IU	10–40	206
CPK: U/L	39–308	6895
Bil T: mg/dL	0.1–1.2	15.4
Bil D: mg/dL	Less than 0.3	6.4
Na: mEq/L	135–145	143
K: mEq/L	3.5–5	4.3
Mg: mg/dL	1.7–2.2	3.1
Ca: mmol/L	1.0–1.3	8.3
Iron: micg/dL	40–165	268
Cr: mg/dL	0.5–1	0.8
Urea: mg/dL	7–20	35
BS: mg/dL	Less than 100	91

increase in serum creatinine level. A nasogastric tube was placed, and the following medications were prescribed: N/S 250 mL plus the volume equivalent to the urine output every 6 h, bisacodyl suppository, stat intramuscular (IM) dimercaprol 200 mg, then every 6 h for 2 days, IM vitamin K 10 mg twice a day, and 4 sachets of polyethylene glycol (PEG) (every sachet in 1 L of water, gavage per hour). He had defecation within 24 h.

On the next day, the patient became unresponsive to painful stimulation. His icteric sclera and skin, metabolic acidosis, and reactive bilateral pupils revealed the hepatic encephalopathy phase. He received 2 units of fresh frozen plasma, and also 2 more sachets of PEG to complete the elimination of radiopaque materials. In addition, liver transplantation was considered for him, but he died because of cardiac arrest before any preparations were made

FIGURE 1 Patients' abdominal X-ray showing radiopaque materials in stomach and duodenum



on April 5, 2019. Written informed consent was obtained from the patient's parents for publication of this report. This study was conducted according to the declaration of Helsinki principles. Also, CARE guidelines and methodology were followed in this study.

3 | DISCUSSION

Zinc phosphide is a low-cost substance that people with poor socioeconomic status in Asia have easy access to, and Iran is no exception.^{4,8–10} The mortality rate of phosphide toxicity due to suicidal intentions is estimated at about 2.6% in Iran.¹¹ Moreover, the mortality rate of ZnP poisoning is much higher.² The average age of patients who misused ZnP intentionally for suicidal purposes is 27 years,¹ and the increased rate of suicide among younger has become a recent concern in Iran.^{9,11} It is reported that consumption of 5 grams of ZnP (55–70 mg/kg) results in death. It takes 30 min to manifest acute toxicity, and death could occur within the first 6 h after ingestion.^{2–4}

Most patients with PH₃ intoxication are referred to hospital with common signs and symptoms, including N/V, retrosternal burning, metabolic acidosis, hypotension, hypoglycemia, delirium, and tonic-clonic seizures, which can lead to severe clinical findings such as circulatory collapse, dysrhythmias, congestive heart failure, pulmonary edema, acute liver, and renal failure that can cause early death.^{8,11} Our patient was presented to the hospital with N/V, hypotension, and sinus tachycardia on ECG. It was interesting that our patient was referred to the hospital after 1 week of ingestion, while in the AA Mashali et al. study, the peak of clinical symptoms and time to elevate liver and cardiac enzymes were reported as 12 and 24 h after hospitalization, respectively. Also, the amount of ingested ZnP and time till hospitalization were considered as the possible predictive factors for liver and heart

toxicity in that study, which both were positive in our case and obviously affected his prognosis.¹²

ALF is one of the most severe adverse effects of ZnP. Hepatic encephalopathy as a poor prognosis manifestation can occur at any time, and the only treatment is liver transplantation.¹³ In spite of considering treatment, our patient's general condition became worse. Following the elevation of liver enzymes, prolonged PT, hyperbilirubinemia, and icteric appearance with the loss of consciousness, hepatic encephalopathy was suspected and the patient was placed on the liver transplantation list.

It was found that patients with positive X-ray for ZnP poisoning are more likely to show severe intoxication even with no serious symptoms on admission,¹⁴ which was also seen in a fatal zinc phosphide poisoning case with unusual abdominal radiologic findings due to zinc's radiopaque property.⁹ A serial abdominal X-ray is a useful diagnostic tool for poor prognosis patients to confirm complete excretion of radiopaque materials, which are responsible for toxicity.¹¹ So, PEG was administered for GI decontamination in this patient.

Although PH₃ can cause ventricular tachycardia, ventricular fibrillation, ST-segment elevation/depression, and a wide QRS complex, cardiovascular damage due to ZnP toxicity can lead to early death without any changes in ECG,⁸ as seen in our patient (CPK = 6895 U/L).

4 | CONCLUSION

In hospitalized patients with a history of intentional or unintentional consumption of rodenticide compounds, especially phosphides (ZnP, CaP, and MgP), it is necessary to assess arterial blood gas (ABG) and abdominal radiography. In the presence of radiopaque materials, GI decontamination with PEG, in addition to a serial abdominal X-ray, should be initiated. After removing of

radiopaque substance from the GI, patient discharge can be considered.

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Declared none.

CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

AUTHOR CONTRIBUTIONS

ZZ involved in the interpretation and collecting of data and editing of the manuscript. ZN, MM, and ESB involved in drafting the first version of the manuscript and editing it. KR and AH involved in writing, editing, and preparing the final version of the manuscript. MF is involved in critically revising the whole manuscript. RT is responsible for collecting data and submitting the manuscript. All authors reviewed the paper and approved the final version of the manuscript.

CONSENT

Informed consent for publication of this case report was obtained from the patient's parents.

DATA AVAILABILITY STATEMENT

The data are available with the correspondence author and can be achieved on request.

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