Case Report

Successful management of zinc phosphide poisoning

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Abstract

Zinc phosphide (Zn_2P_3) rodenticide, is generally misused intentionally for suicidal purpose in Iran. For many years, scientists believe that liberation of phosphine (PH_3) on contact with acidic content of the stomach is responsible for clinical presentations. However, relatively long time interval between ingestion of Zn_2P_3 and presentation of its systemic toxicity, and progression of acute liver failure could not be explained by the current opinion. Hence, an innovative theory intended that phosphonium, as an intermediate product will create and pass through the stomach, which then will reduce to produce PH_3 in the luminal tract. Here, we present a case of massive Zn_2P_3 poisoning. In our case, we used repeated doses of castor oil to induce bowel movement with an aim of removing unabsorbed toxin, which was proved by radiography. Interestingly, the patient presents only mild symptoms of toxicity such as transient metabolic acidosis and hepatic dysfunction.

Keywords: Management, poisoning, zinc phosphide

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Introduction

Zinc phosphide (Zn_2P_3) has been used in rodenticide biats. Commercial products are often available in dark grey powder or pellets. It is generally misused intentionally for suicidal purpose and accounts for about 2.6% of the mortality due to poisonings in an Iranian report.[1-3] It is proposed that after ingestion of metal phosphides, phosphine (PH_3), the active ingredient, will release on contact with moisture or hydrochloric acid in the stomach.[4] However, some doubts made recently because of the unique presentations after Zn_2P_3 poisoning, and a novel idea of liberation and absorption of PH_3 via luminal tract was formed.[5,6]

Case Report

An 18-year-old male presented to emergency department approximately 5 h after consumption of about 20 g of Zn_2P_3. On arrival, he was conscious but irritable, with pulse rate of 85/min, blood pressure (BP) of 120/75 mmHg, respiratory rate of 14/min, and temperature of 37.2°C. There was slight flushing of the face, but he was otherwise asymptomatic. The first venous blood gas (VBG) analysis was normal. Per abdominal examination revealed normal. An intravenous access was established and monitoring with electrocardiogram (ECG), BP, and pulse oximetry. An abdominal X-ray showed radiopaque material throughout the luminal tract. The patient was given 60 cc of castor oil to induce bowel movement. The first episode of defecation occurred after an hour. Despite three-time of defecation, control abdominal X-ray 6 h later showed a considerable amount of the radiopaque material at the end of the transverse colon at the splenic flexure. Hence, 60 cc of castor oil was prescribed for the patient twice within the next 6 h. Afterward, a

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repeat abdominal X-ray was clear. During this period, pulse oximetry, BP, and ECG monitoring were normal. Serial VBG obtained every 4 h over the first 24 h of admission. Although the first and second VBGs were normal, the third VBG components revealed a slight metabolic acidosis (pH = 7.34, PCO_2 = 30.1 mm Hg, HCO_3 = 16.2 mEq/L), which was recovered to normal levels after the decontamination of luminal tract, without administration of alkalization agents. Afterward, the pH and bicarbonate levels remained stable, indicating that the patient did not develop severe systemic toxicity. In the 2nd day of admission, the patient developed mild coagulopathy (prothrombin time: 18 s, partial thromboplastin time: 32 s, and international normalized ratio of 2.15), as well as marked aminotransferase elevations (aspartate aminotransferase (AST): 68 IU/L, alanine aminotransferase (ALT): 37 IU/L). After 2 days his coagulation tests were normal, however, his liver function tests (LFTs) components changed to AST: 69 IU/L and ALT: 92 IU/L. Hence, he discharged with Silymarin 70 mg three times a day for 2 weeks and psychology outpatient follow-up. A follow-up LFT was normal 2 weeks after discharge from hospital.

Discussion

However, authors generally believe that liberation of PH_3 gas in the stomach is responsible for causing symptoms, the clinical manifestation of aluminum and Zn_2P_3 poisonings are not usually similar. The most important dissimilarities are the relatively long time interval between ingestion of Zn_2P_3 and presentation of its systemic toxicity as well as the progression of acute liver failure during the 1st week, exclusively in the case of Zn_2P_3 poisoning.[6,7] Hence, an innovative report disproved the current belief of PH_3 liberation after contact of Zn_2P_3 and gastric contents. It intended that phosphonium (PH_4^+), as an intermediate product will create and pass through the stomach, which then will reduce to produce PH_3 in the luminal tract.[5] However, the PH_4^+ is not radiopaque, zinc, the potentially radiopaque ingredient is visible on abdominal radiography. Recently, Hassanian-Moghaddam *et al*. suggested performing abdominal X-ray in the case of suspected Zn_2P_3 poisoning, even if the patient is asymptomatic. They indicated that a positive abdominal radiography could be considered as poor prognostic factor.[8] This fact can confirm the idea of PH_3 absorption through luminal mucosa. This novel theory suggests novel practical approaches. We start castor oil to induce bowel movement. Despite several episodes of defecation, a control abdominal X-ray 6 h later revealed residual radiopaque material in colon. However, the patient remained asymptomatic during the first 12 h of admission; the third VBG components revealed a slight metabolic acidosis; which was recovered to normal levels after the decontamination of luminal tract. As the determination of metabolic acidosis may reflect the severity of PH_3 toxicity,[9,10] we can argue forceful intestinal decontamination in this case could prevent more toxin absorption. However, Hassanian-Moghaddam *et al*.[8] in their report administered polyethylene glycol to help the patient move toxin through his bowel, since it is a water-soluble product; we use castor oil to prevent reduction of PH_4^+ in the luminal tract. This approach was first proposed to prevent more PH_3 liberation after ingestion of metal phosphides in Shiraz, Iran.[11]

Hepatotoxicity due to Zn_2P_3 has been occasionally reported from India.[12] Saraf *et al*. reported that Zn_2P_3 poisoning is the most common cause of drug/toxin-induced acute liver failure in India.[7]

However, we still do not know what the exact mechanism is, but in our case, development of coagulopathy and elevation of aminotransferase levels in the 2nd day could be explained by direct toxic damage of PH_3 to the liver, after being transported by the portal vein. In fact, based on the theory of PH_4^+ reduction and PH_3 absorption from the luminal tract, it just directly reaches to hepatic cells.[6] Hence, compared to other metal phosphides, acute liver dysfunction might be an exclusive presentation of Zn_2P_3 poisoning. Moreover, Saraf *et al*. reported that higher doses may result in earlier onset of hepatotoxicity and acute liver failure.[7] By the way, in our case, despite ingestion of large amounts of toxin, after forceful intestinal decontamination, this complication was provisional, and could not take the patient to a fatal outcome.

However, there are many reports of fatal outcome due to cardiovascular collapse or hepatic failure; it seems that we have more time to use decontamination techniques in the case of Zn_2P_3 poisoning compared to other metal phosphides. In our case, we used castor oil to induce bowel movement with an aim of removing unabsorbed intermediate toxic product, the PH_4^+. However, the theory of production of PH_4^+ is not unanimously accepted; it could explain every fact in the case of Zn_2P_3 poisoning. According to an article published in 2014, more aggressive gastrointestinal decontamination is necessary in patients with positive radiography. We also strongly suggest following removal of the radiopaque material by repeated abdominal radiography to ensure complete decontamination.

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Conflicts of interest

There are no conflicts of interest.

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