

Successful outcome in managing of aluminum phosphide poisoning

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Abstract

Aluminum phosphide (ALP) is a potent lethal substance, that use for agriculture purpose, as a pesticide. this substance may use for suicide, and it will kill the patient rapidly. we want to report a patient who use ALP for suicide purpose and was managed quickly in the emergency department and he became alive.

Keywords: Aluminum phosphide, Suicide, Outcome

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Introduction

Aluminum phosphide (ALP) is a highly toxic pesticide that is used as a rodenticide and insecticide. Its toxic effects are due to liberation of phosphine gas upon exposure to moisture (1-4). This gas can easily be absorbed from gastrointestinal and pulmonary tracts or on dermal absorption (1,2). Phosphine as an oxidant can cause hypoxic damage at the cellular level due to binding cytochrome oxidase and inhibiting oxidative phosphorylation (4). A wide range of symptoms and signs of ALP toxicity are reported including vomiting, diarrhea, cyanosis, pulmonary edema, respiratory failure, renal failure, metabolic acidosis, hepatic failure, cardiovascular collapse, dizziness and death (4,5). Some authors describe hemolysis due to ALP toxicity (2). Mortality rate of ALP poisoning is reported between 50% to 60% in literature.

We report a survived 22-year-old male patient who was admitted to the emergency department (ED) after a suicidal ingestion of ALP.

Case presentation

A 22-year-old man was brought to the ED after a suicidal ingestion of two tablets of ALP (6 g) and 100 tablets of paracetamol. He was fully conscious, mildly anxious, and suffered from abdominal pain. He had a blood pressure of 80/60 mm Hg, a heart rate of 100 beats/min, a respiratory rate of 42, and a body temperature of 36.7°C. Heart and lungs were normal in auscultation and initial electrocar-

diogram (ECG) taken in ED had no abnormal changes. Abdomen was soft without tenderness. Nasogastric tube was placed immediately and gastric lavage was performed with sunflower oil. The initial arterial blood gas (ABG) analysis showed a metabolic acidosis (pH=7.33) and bicarbonate 19 mmol/dl. He was treated with sodium bicarbonate, 0.9% saline, calcium gluconate, and magnesium sulfate. [Glucose, Insulin, Katrium (Potassium)] GIK protocol was commenced for patient with Glucose, 60 units of regular insulin and 40 mEq of potassium in normal saline. He was paralyzed, intubated and connected to mechanical ventilator because of acidosis and tachypnea. Due to progressive hypotension infusion of 10 µg/min epinephrine was added to treatment. N-acetyl cysteine (NAC) was commenced.

Patient was admitted in intensive care unit (ICU). Treatment continued with magnesium sulfate (1 g/6h), calcium gluconate (1 g/6h), sodium bicarbonate (50 mmol/h), regular insulin (6 unit/2h), hypertonic glucose, and potassium chloride (20 mEq in one liter of 0.9% saline). After achieving normal blood pressure, epinephrine infusion was discontinued. NAC infusion continued with 70 mg/kg/4h. In the second day of admission, potassium chloride (KCL) infusion decreased to 10 mEq in each liter of crystalloid and infusion of glucose, insulin, magnesium sulfate, and calcium gluconate was discontinued. Hydrocortisone (200 mg/12h) started. In third day, NAC infusion changed to oral administration and continued with



600 mg/4h. Patient was extubated and hydrocortisone decreased to 100 mg/12h. Because of patient's abdominal pain, an upper gastrointestinal endoscopy was performed which was reported normal.

In the fifth day, the patient was admitted to psychiatry ward with no morbidity due to ALP toxicity.

Discussion

ALP is a highly toxic pesticide that is used widely in agriculture since 1940s (1,4). It is cheap and easily achievable in countries, such as Iran and India (4). ALP poisoning is common either as an accidental intoxication or for suicidal reasons and is a serious public health problem in developing countries, mainly in Northern India where this intoxication is the main reason of poisoning (3). Because of high toxicity and no existence of effective antidote, ALP poisoning has a high mortality rate (2,5).

After exposure to moisture, ALP releases phosphine gas which is the active toxic component (1,4,5). Each three grams tablet can release one gram phosphine (4). Phosphine gas could be absorbed from the respiratory system, gastrointestinal tract or on dermal absorption (1,5). By inhibiting mitochondrial cytochrome oxidase, phosphine can inhibit the oxidative respiration by 70% and can also make oxidative damage by producing oxidant free radicals (1,2,4). Clinical manifestations include various signs and symptoms. Nausea, vomiting, abdominal pain, diarrhea, palpitation, dizziness and altered consciousness are common symptoms (4). Hypotension, tachycardia, severe cardiac arrhythmias, tachypnea, severe metabolic acidosis, acute renal failure, tubular necrosis, hepatic failure, and hemorrhage are some of common complications of ALP poisoning (4). Death, in most times, is due to arrhythmia, cardiopulmonary collapse or metabolic acidosis (2). The lethal dose of phosphine for a 70 kg person is 150-500 mg (2,4) and survival is unlikely after ingestion of 1500 mg of ALP (1,3).

Due to its high toxicity, ALP poisoning should be treated as soon as possible (5). There is no antidote and treatment is conservative (2,5). The first and the most important step for successful treatment is an early resuscitation of shock (4). Central vein pressure (CVP) should be kept at around 12-14 cm of water (4). Hydrocortisone 200 mg each 2-4 hours could be helpful to combat shock and prevent the patient from acute respiratory distress syndrome (ARDS) (4).

Potassium permanganate oxidizes phosphine to non-toxic phosphate and gastric lavage with potassium permanganate (1:10,000) can reduce phosphine absorption (4,5). Although administration of activated charcoal is contro-

versial (5,6) but the use of approximately 100 g of charcoal within the first hour could be useful to reduce absorption (4,5). In situations like our case that potassium permanganate is not achievable immediately; gastric lavage with coconut oil or any other vegetable oil can reduce phosphine release and absorption.

Intravenous sodium bicarbonate for aggressive correction of metabolic acidosis could improve patient outcome (5). Serum bicarbonate level less than 15 mEq/L requires sodium bicarbonate intravenously administration (4). Hypomagnesaemia is common (3,4). Many studies described the potential role of magnesium sulfate in reducing the fatal outcome in acute ALP poisoning, but at present, the routine use of magnesium intravenous administration is controversial since some literature showed hypermagnesemia in some cases (5). The NAC as an effective treatment in ALP poisoning, can prevent hepatic complications and delays mortality latency time (4).

In our case early arrival, aggressive resuscitation and early prevention of phosphine absorption by using vegetable oil were important factors for a successful outcome.

Ethical issues

we got inform consent from patient to report.

Authors' contributions

Samad Shams Vahdati, idea and case management; Reza Shahab Moghadam, data gathering and managing; Zahra Vanderajabpour and Shahrard Tajoddini, article review; Pouya Paknejad, writing case and critic.

References

1. Bogle RG, Theron P, Brooks P, Dargan PI, Redhead J. Aluminium phosphide poisoning. *Emerg Med J*. 2006; 23(1): e3.
2. Sood AK, Mahajan A, Dua A. Intravascular haemolysis after aluminium phosphide ingestion. *J R Soc Med* 1997; 90(1): 47-8.
3. Louriz M, Dendane T, Abidi K, Madani N, Abouqal R, Zeggwagh AA. Prognostic factors of acute aluminum phosphide poisoning. *Indian J Med Sci* 2009; 63(6): 227-34.
4. Moghadamnia AA. An update on toxicology of aluminum phosphide. *DARU* 2012; 20(1): 25.
5. Gurjar M, Baronia AK, Azim A, Sharma K. Managing aluminum phosphide poisoning. *J Emerg Trauma Shock* 2011; 4(3): 378-84.
6. Mehrpour O. Comment on "An update on toxicology of aluminum phosphide". *DARU* 2012; 20(1): 50.