Updates in
Emergency Medicine

Case Report

Successful Treatment of Acute Aluminum Phosphide Poisoning: Possible Benefit of Olive Oil - A Case Report

Mohammad Hossien Kamaloddini [1], Parvin Zohoorian[2], Mahdi Foroughian [2], Sara Hemmati Awli[2], Ali Teimouri*[3]

1 Toxicology Research Center. Mashhad University of Medical Sciences, Mashhad, Iran.
2 Department of Emergency Medicine, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran.
3 Student Research Committee, Mashhad University of Medical Sciences, Mashhad, Iran.

Correspondence: Ali Teimouri, Student Research Committee, Mashhad University of Medical Sciences, Mashhad, Iran.

Email: ali.teimouri.1994@gmail.com.

Abstract:

Aluminum phosphide (ALP) is a highly efficient pesticide that is widely applied in agriculture, but it is also one of the most commonly used suicide poisons. The management of ALP poisoning is limited to primarily supportive care in the absence of a particular antidote. Treatment options include gastric lavage with different solutions as well as the potassium permanganate, activated charcoal and sorbitol, coconut or almond oil, administration of N-acetyl cysteine, Magnesium sulfate, and methylene blue supplementation. While still there is no reliable protocol available and ALP poisoning is associated with a high mortality rate. Here we report a new method of gastric decontamination with olive oil and supportive medical therapy with vitamin E, N-acetyl cysteine, and magnesium sulfate. A 33-year-old man was admitted to the Emergency Department because of the intentional ingestion of three ALP tablets. The patient had decreased consciousness, sweating, paleness, and weak peripheral pulses. On bedside echocardiography, ejection fraction (EF) was 30%, showing cardiac toxicity of ALP. The patient was also suffering from metabolic acidosis (pH=7.37). Gastric decontamination was started with Olive Oil in Emergency department and medical therapy with vitamin E, N-acetyl cysteine, and magnesium sulfate was performed. During the hospitalization period, EF and metabolic acidosis were improved and the patient was discharged 8 days later.

Keywords: Aluminum phosphide, Phosphine, Olive Oil, Gastric lavage, Emergency department

Citation:
Kamaloddini MH., Zohoorian P., Foroughian M., Hemmati Awli S., Teimouri A., (2021). Successful Treatment of Acute Aluminum Phosphide Poisoning: Possible Benefit of Olive Oil - A Case Report. 1*(1): 47-51.*

Received: June 15, 2021
Reviewed: June 17, 2021
Accepted: July 30, 2021
Published: August 14, 2021

Introduction

Aluminum Phosphide (ALP), also known as rice pellet in Iran, is used as a pesticide in agriculture *(1)*. ALP poisoning can happen accidentally or intentionally. According to studies conducted in Iran, 95% of APL poisonings have been suicidal *(2)*. The approximate weight of ALP is 3 grams, when this tablet is exposed to moisture, about 1 gram of phosphine gas gets released *(3)*. Phosphine gas smells like rotten fish or garlic. Phosphine causes a non-competitive inhibition of the enzyme cytochrome c oxidase, which inhibits cellular respiration. This gas damages several organs such as the liver, heart, gastrointestinal tract, kidneys, and respiratory tract *(3)*. Symptoms of acute ALP poisoning include respiratory symptoms (dyspnea, tachypnea, and acute respiratory distress syndrome), gastrointestinal symptoms (nausea, vomiting,

epigastric pain), neurological symptoms (lightheadedness, restlessness), and cardiac symptoms (chest pain). Heart failure and cardiac dysrhythmia are not unexpected *(3)*. Abnormalities in laboratory tests can be observed in patients with rice ALP poisoning. Levels of serum electrolytes may get altered and increased aspartate aminotransferase and alanine aminotransferase may be observed. Also, metabolic acidosis with or without respiratory alkalosis might happen *(3)*. Since there is no specific antidote for ALP, the treatment of these patients is supportive and includes correction of electrolyte disorders, gastric lavage, treatment of hypotension, use Methylene blue for the treatment of methemoglobinemia, correction of acidosis, use of magnesium sulfate, N-acetylcysteine, and various other methods as well as the hemodialysis *(1)*. In this study, a patient who was intoxicated with aluminum phosphide and underwent supportive treatment, application of vitamin E, n-acetylcysteine, and olive oil, was discussed.

Case presentation:

The patient was a 33-year-old man who was sent to the Toxicology ward of Imam Reza Hospital in Mashhad 6 hours after an intentional ingestion of 3 rice tablets. At the admission, the patient had decreased level of consciousness (Glasgow Coma Scale (GCS):10), sweating, paleness, weak peripheral pulses, systolic blood pressure (SBP) of 80 mm Hg, and diastolic blood pressure (DBP) of 60 mm Hg, and heart rate of 130 beats per minute, Respiratory rate of the 17 breaths per minute with a blood O2 saturation of 97%. Also, in bedside echocardiography (MyLab™X8, made in Italy), the patient had an EF of 30%; while electrocardiogram (ECG) was normal at arrival.

Blood tests showed hemoglobin (Hb) of 10.7 g/dL (Normal range: 12.3-15.3 g/dL); White blood cells (WBCs) of 3.77 ×106/µL (Normal range: 4.5-5.9 /µL); Platelet count of 379 ×103/µL (Normal range: 150-450×106/µL); Urea of 26 mg/dL (Normal range: 17-45 mg/dL); Creatinine of 0.9 mg/dL (Normal range: 0.7-1.4 mg/dL); Calcium of 9.4 mg/dL (Normal range: 8.5-10.5 mg/dL); Magnesium of 1.88 mg/dL (Normal range: 1.7-2.7 mg/dL); Sodium of 140 mEq/L (Normal range: 135-145 mEq/L);; and Potassium of 4.1 mEq/L (Normal range: 3.5-5.3 mEq/L).

For gastric decontamination, 50 cc of olive oil was administered every 2 hours through the nasogastric (NG) tube. The patient was also treated with 400 units of Vitamin E Pearl every 12 hours, Intravenous (IV) N-acetylcysteine (NAC) with a total dose of 18 grams, and magnesium sulfate 200 mg/kg intravenously. Then the patient was transferred to the intensive care unit (ICU). Finally, after 3 days of hospitalization in the intensive care unit, the patient was transferred to the ward in good general condition. After improved clinical tests and correction of acidosis, the patient was discharged on the 8th day (Figure 1).

Discussion:

The main fatal consequence of aluminum phosphide is usually a cardiovascular dysfunction through the direct effect of phosphine gas on the myocardium and consequent arrhythmia *(4)*. Our case showed an acute decreased EF to 30%. ECG changes have been seen from prolonged PR and QRS intervals to changes in the ST segment and complete heart block *(5)*. Other common and fatal complications of include acute renal failure and shock *(6)*.

Cardiovascular failure, severe hypotension and acidosis, and acute renal failure are the most common complications of ALP poisoning and our case showed all these signs except acute renal failure. Acute lung injury is a result of the direct effect of phosphine gas on alveoli and their destruction that could be followed by an acute

respiratory distress syndrome *(7)*; while our case did not have respiratory problems.

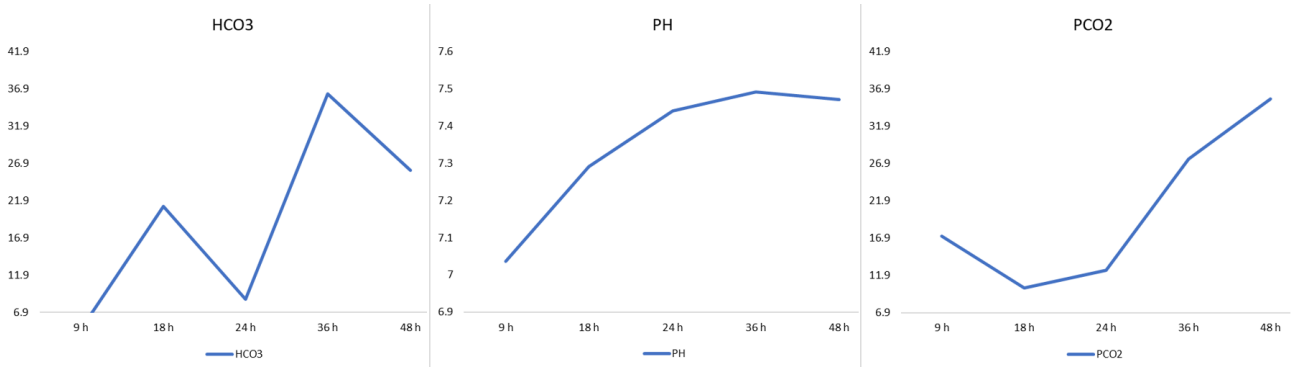

Figure 1. Patient arterial blood gases (ABG) changes during hospitalization

Symptoms of ALP often appear very quickly, within 10 to 15 minutes. Vomiting is the first symptom of the effects of aluminum phosphide on the gastrointestinal tract and has been reported in some studies to be the body's help to excrete aluminum phosphide and reduce phosphine gas release *(8)*. Vomiting may be severe and prolonged *(9)*. The presence of classic signs and symptoms of ALP, such as upper abdominal pain (epigastrium), nausea, and shock reflecting severe hypotension, are reactions that have been reported in many studies *(10)*. Most deaths occur within the first 12-24 hours of intoxication and are often due to arrhythmias and cardiac arrest. Mortality after 24 hours is more due to liver failure *(9,10)*.

Symptoms begin after the pill comes in contact with stomach Joice, which releases phosphine gas within minutes of swallowing. Toxicity symptoms include vomiting, abdominal pain, restlessness, tachycardia, tachypnea, acidosis, hypotension. The course of ALP poisoning is from nausea and vomiting to multiple organ failure and death, which mostly occurs during the first 24-48 hours after poisoning *(9,10)*.

The exact mechanism of the effects of aluminum phosphide in humans is not clear and it seems that severe cell damage is the main consequence of ALP which does not involve a single organ and this causes several organs failure *(11)*. Studies in rats have shown that phosphine gas released from metal phosphides has the ability to inhibit the enzyme cytochrome oxidase C, thereby disrupting the cellular respiration cycle *(12)*. It causes non-competitive inhibition of cytochrome C oxidase enzyme in mitochondria and electron transfer system and oxidative phosphorylation *(13)*. This inhibition results in the process of energy production in the electron transfer chain and eventually the cessation of cellular respiration. Following these changes, free radicals are produced that can damage organs *(13)*. Given the ability of phosphine to generate free radicals in many tissues, some studies have concluded that organs that need more oxygen, such as the heart, brain, kidneys, lungs, and liver, are more sensitive to phosphine gas damage. This is consistent with the occurrence of histopathological changes in such organs *(11)*. Phosphine gas also inhibits protein synthesis and enzymatic activity, especially in the mitochondria of heart and lung cells *(11)*.

As phosphine gas starts with ALP contact with water, various researchers have studied methods of gastric decontamination to reduce phosphine gas release. According to Pajumand's study *(14)*, gastric lavage with potassium permanganate 1:1000, charcoal, and coconut oil can be used as the

first step in an emergency room. According to Bajwa study *(15)*, gastric lavage with sodium bicarbonate and coconut oil can be used. But there is not enough evidence about the olive oil. In one study, zinc phosphide poisoning was successfully managed by olive oil gastric wash *(16)*.

Conclusion:

Although there is no specific antidote for the treatment of ALP poisoning, according to our study, the use of olive oil for gastric lavage can be helpful, maybe due to the decrease in the absorption of phosphine gas.

Consent For Publication:

Patient consent for publication of study if his personal data stays confidential.

Availability of Data and Materials:

All data are available in article.

Competing interests:

None.

Funding:

None.

Authors' contributions:

MHK, PZ, MF, and SH contributed to the management of the patient. AT, MHK, and PZ have written the draft manuscript. All authors confirmed the final manuscript.

Acknowledgments:

None.

REFERENCES:

1. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. Arh Hig Rada Toksikol 2012;63:61-73.

2. Hosseinian A, Pakravan N, Rafiei A, Feyzbakhsh SM. Aluminum phosphide poisoning known as rice tablet: A common toxicity in North Iran. Indian journal of medical sciences. 2011; 65*(4)*:143-50.

3. Moghadamnia AA. An update on toxicology of aluminum phosphide. DARU journal of Pharmaceutical Sciences. 2012 Dec;20*(1)*:1-8.

4. Akkaoui M, Achour S, Abidi K, Himdi B, Madani A, Zeggwagh AA, Abouqal R. Reversible myocardial injury associated with aluminum phosphide poisoning. Clinical toxicology. 2007 Jan 1;45*(6)*:728-31.

5. Soltaninejad K, Beyranvand MR, Momenzadeh SA, Shadnia S. Electrocardiographic findings and cardiac manifestations in acute aluminum phosphide poisoning. Journal of forensic and legal medicine. 2012 Jul 1;19*(5)*:291-3.

6. Mohan B, Singh B, Gupta V, Ralhan S, Gupta D, Puri S, Goyal A, Aslam N, Tandon R, Wander GS. Outcome of patients supported by extracorporeal membrane oxygenation for aluminum phosphide poisoning: An observational study. Indian heart journal. 2016 May 1;68*(3)*:295-301.

7. Gurjar M, Baronia AK, Azim A, Sharma K. Managing aluminum phosphide poisonings. Journal of emergencies, trauma and shock. 2011 Jul;4*(3)*:378.

8. Gupta S, Ahlawat SK. Aluminum phosphide poisoning—a review. Journal of Toxicology: Clinical Toxicology. 1995 Jan 1;33*(1)*:19-24.

9. Nosrati A, Karami M, Esmaeilnia M. Aluminum phosphide poisoning: A case series in north Iran. Asia Pacific Journal of Medical Toxicology. 2013 Sep 1;2*(3)*:111-3.

10. Montazer H, Laali A, Khosravi N, Amini Ahidashti H, Rahiminezhad M, Mohamadzadeh A. Epidemiological, clinical and laboratory features in patients poisoned with aluminum phosphide. Journal of Mazandaran University of Medical Sciences. 2016 Jun 10;26(137):188-95.

11. Hosseini SF, Forouzesh M, Maleknia M, Valiyari S, Maniati M, Samimi A. The molecular mechanism of aluminum phosphide poisoning in cardiovascular disease: Pathophysiology and diagnostic approach. Cardiovascular Toxicology. 2020 Oct;20*(5)*:454-61.

12. Anand R, Kumari P, Kaushal A, Bal A, Wani WY, Sunkaria A, Dua R, Singh S, Bhalla A, Gill KD. Effect of acute aluminum phosphide exposure on rats—A biochemical and histological correlation. Toxicology letters. 2012 Nov 23;215*(1)*:62-9.

13. Singh S, Bhalla A, Verma SK, Kaur A, Gill K. Cytochrome-c oxidase inhibition in 26 aluminum phosphide poisoned patients. Clinical Toxicology. 2006 Jan 1;44*(2)*:155-8.

14. Pajoumand A, Jalali N, Abdollah M, Shadnia S. Survival Following Severe Aluminium Phosphide Poisoning. Journal of Pharmacy Practice and Research. 2002; 32*(4)*:297-9.

15. Agrawal VK, Bansal A, Singh RK, Kumawat BL, Mahajan P. Aluminum phosphide poisoning: Possible role of supportive measures in the absence of specific antidote. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine. 2015; 19*(2)*:109-12.

16. Altintop I, Tatli M. Successful management of zinc phosphide poisoning: possible benefit of virgin olive oil. Medicine Science International Medical Journal. 2017;6.