Incidental Aluminum Phosphide Poisoning: Case Report and Current Management

Özlem Çakın¹, Gokhan Tazegul¹, Ayça Gümüş², Melike Cengiz², Atilla Ramazanoğlu²

¹ Department of Internal Medicine, Akdeniz University School of Medicine, Antalya, Turkey
² Department of Anesthesiology and Reanimation, Akdeniz University School of Medicine, Antalya, Turkey

Correspondence: Gokhan Tazegul, Akdeniz Üniversitesi Tıp Fakültesi Dekanlık, 07070 Kampus, Antalya, Turkey
E-mail: drgtazegul@gmail.com
Tel: + 90 531 661 6518

Received: 01 Oct 2017
Accepted: 29 Nov 2017
Published Online: 02 Jan 2018
Published: 28 Sept 2018

Key words: aluminum phosphide, poisoning, inhalation, phosphine

Citation: Çakın Ö, Tazegul G, Gümüş A, Cengiz M, Ramazanoğlu A. Incidental aluminum phosphide poisoning: case report and current management. Folia Med (Plovdiv) 2018;60(3): 464-7.
doi: 10.2478/folmed-2018-0001

Aluminum phosphide (AlP) is a commonly used cheap rodenticide, insecticide, and fumigant. Most intoxications in the literature are suicidal ingestions, however, AlP may cause incidental inhalational toxicities as well. After ingestion or inhalation, nausea, vomiting, dyspnea and abdominal pain develops within minutes. Hallmark of toxicity is refractory hypotension, cardiac failure and severe metabolic acidosis developing within a matter of hours are the major cause of mortality. In Turkey, AlP tablets are widely accessible and are sold without any restrictions. However, there are few local case reports in the literature. Additionally, incidental AlP intoxications are rarely reported.

Herein, we present a 25-year-old male patient incidentally poisoned with AlP. He was found unconscious in a grain storage unit protected by aluminum phosphide tablets. He had hypotension and tachycardia. Arterial blood gas analysis did not reveal metabolic acidosis. He was quickly intubated and admitted to Intensive Care Unit (ICU). Supportive care crystalloid solution, n-acetyl cysteine and norepinephrine infusion was administered. After 36 hours, he was extubated and discharged without any complications.

In Turkey, AlP tablets are widely accessible and are sold without any restrictions. However, there are few local case reports in the literature. Additionally, incidental AlP intoxications are rarely reported. Herein, we present a case of a 25-year-old male patient incidentally poisoned with AlP, our treatment approach and current literature regarding pathogenesis and treatment.

INTRODUCTION

Aluminum phosphide (AIP) is a commonly used cheap rodenticide, insecticide, and fumigant. 3-gram AIP tablets are widely used in grain storage. AIP is a common cause of acute chemical toxicity, particularly in India and Iran. Intoxication has high mortality and a dose between 150 and 500 mg is lethal for a 70-kg person. Most intoxications in the literature are of suicidal ingestions; however, AIP may cause incidental inhalational toxicities as well.

There is no specific antidote or treatment for AIP toxicity. Literature is controversial regarding treatment approach. Inhalational toxicity may occur under extreme conditions, as presented in this case report. Preventive strategies should be considered to reduce incidents. Clinicians should also be aware that AIP is a widely available and highly toxic compound that has no specific antidote and toxicity needs to be urgently treated with best supportive care.

CASE REPORT

On a rainy day, a 25-year-old male private security guard was brought in, by emergency response medical team. He was working for an agriculture company and was found unconscious without a face mask, 30 minutes after he disappeared in a grain storage unit, reportedly protected by aluminum phosphate tablets. Emergency response medical team reported the roof of the storage unit was leaking...
Incidental Aluminum Phosphide Poisoning

rain water and they have seen several dozen wet tablets on the ground. On physical examination, he was unresponsive and Glasgow coma scale score was 3. Blood pressure was 83/51 mm Hg with heart rate (HR) 121 beats per minute. Temperature and saturation were within normal limits. Extremities were cold and moist. Electrocardiography (ECG) showed sinus tachycardia. He was quickly intubated and admitted to Intensive Care Unit (ICU). Arterial blood gas analysis did not reveal metabolic or lactic acidosis. Blood count and biochemistry were within normal limits. Gastric lavage was performed with potassium permanganate and sodium bicarbonate. Patient was treated with 30 mL/kg/hour crystalloid solution, n-acetyl cysteine 300 milligrams twice daily, and 0.1 mcg/kg/hour norepinephrine infusion. During follow up, he did not develop metabolic acidosis, hence sodium bicarbonate infusion was not started. After 36 hours of ICU admission, norepinephrine support was gradually ceased and he was extubated. Blood pressure was 124/71 mm Hg with HR 91 beats per minute after extubation. Body temperature and oxygen saturation were normal as well. Arterial blood gas analysis, blood cell count, and biochemistry tests were within normal limits. ECG revealed sinus rhythm. Patient was discharged without any complications.

DISCUSSION

AlP is a widely available and highly toxic pesticide. When the compound comes in contact with water, it quickly dissolves, emitting phosphine (PH3) gas, which is colorless, odorless, flammable, and highly toxic. After suicidal or accidental ingestion or inhalation, PH3 gas is absorbed rapidly through the mucosa, causing nausea, vomiting, dyspnea and abdominal pain within minutes. Refractory hypotension, cardiac failure, and severe metabolic acidosis within a matter of hours are the hallmarks of toxicity and the major cause of mortality.

Exact mechanism of AlP toxicity is not clearly understood, however, major mechanisms of toxicity are: (a) mitochondrial toxicity characterized by cytochrome C oxidase inhibition, subsequently interfering with electron transfer between complex III to complex IV resulting in energy depletion, (b) oxidative injury and lipid peroxidation causing cellular injury, and (c) oxidative mechanisms causing protein denaturation and cellular membrane injury.

Treatment is based on best supportive care. Reducing absorption of AlP is one of the possible approaches to reduce mortality. Gastric lavage with potassium permanganate, sodium bicarbonate, and activated charcoal was routinely performed to counterbalance the toxic effects in AlP toxicity in the past. Potassium permanganate can change phosphide to less toxic compounds and bicarbonate can reduce gastrointestinal secretions which would reduce PH3 release. In addition, activated charcoal can absorb phosphine gas. Although the exact time of toxicity was not known, we performed gastric lavage with potassium permanganate and sodium bicarbonate. Rapid use of activated charcoal and gastric lavage with potassium permanganate and sodium bicarbonate can potentially be used in patients with AlP toxicity.

Another point to consider in treating AlP toxicity is to counter oxidative injury using antioxidants. One such study compared the administration of vitamin E against placebo, in which vitamin E administration significantly increased blood pressure and reduced mortality in treatment group. Taghaddosinejad et al. demonstrated possible protective effects of n-acetyl cysteine on cardiovascular complications by a similar mechanism. We used n-acetyl cysteine in the present case as an antioxidant to counter oxidative injury. Even though AlP poisoning is not solely due to increased oxidation status, antioxidants seem to have a beneficial role in treatment and should be considered in treatment algorithm.

The most common symptom and cause of mortality in AlP poisoning is severe hypotension. It is commonly refractory to massive crystalloid administration and vasoactive agents such as norepinephrine or dopamine. We administered a crystalloid solution with norepinephrine infusion in this case and fortunately, the patient recovered from hypotension quickly. However, even with the use of vasoactive agents, hypotension may persist due to insufficient vascular integrity. Volume expanders such as hydroxyethyl starch may be successfully used to prevent mortality in such cases, however, possible side effects such as renal failure and coagulopathy should be kept in mind.

Other major causes of mortality in AlP toxicity are severe metabolic acidosis and cardiogenic shock, which are possibly caused by cytochrome C oxidase inhibition and generalized tissue hypoperfusion. Cardiogenic shock in AlP poisoning is mainly characterized by pump failure. Tissue hypoperfusion and acidosis may further reduce cardiac output as well. In addition, common electrolyte abnormalities such as hypokalemia and hypocalcemia also influence the cardiac functions. Hyperlactatemia, a result of energy insufficiency and tissue hypoperfusion,
was shown to be a prognostic marker in acute AlP poisoning.\textsuperscript{11} Our patient did not develop acidosis, electrolyte abnormalities or cardiogenic shock, possibly due to rapid recovery. When it occurs, a range of options exist to treat cardiogenic shock, such as intra-aortic balloon pump, extracorporeal membrane oxygenation, digitalis, trimetazidine and glucose/insulin infusions.\textsuperscript{11-13} Sodium bicarbonate treatment may be arguably helpful in such cases to counteract severe acidosis, however, improving cardiac output to reduce hypoperfusion and related hyperlactatemia, should be the main purpose.

Herein, we reported an incidental case of AlP poisoning. AlP is a widely available pesticide and a common cause of toxicity and to date, there is no specific antidote or treatment for AlP toxicity. Fortunately, our patient responded well to conventional supportive care. However, literature is controversial regarding the treatment approach in refractory cases. In Turkey, AlP tablets are widely accessible and are sold without any restrictions. However, there are few local case reports in the literature. Preventive strategies, such as promoting local legislations to ban or restrict AlP sale should be considered to reduce incident of suicidal ingestions or possible biochemical terrorism acts in regions of high incidence. Additionally, clinicians should also be aware that AlP is a widely available and highly toxic compound that has no specific antidote and toxicity needs to be urgently treated with best supportive care. Inhalational toxicity may occur under extreme conditions, as presented in this case report. Volume expanders to treat severe hypotension, antioxidants to counter oxidative status, and treatment options to increase cardiac output such as intra-aortic balloon pump, extracorporeal membrane oxygenation, and digitalis, trimetazidine and glucose/insulin infusion should be kept in mind in patients with severe AlP toxicity.

REFERENCES

1. Halvaei Z, Tehrani H, Soltaninejad K, et al. Vitamin E as a novel therapy in the treatment of acute aluminum phosphate poisoning. Turk J Med Sci 2017;47:795-800.

2. Mehrpour O, Singh S. Rice tablet poisoning: A major concern in Iranian population. Hum Exp Toxicol 2010;29:701-2.

3. Moghadamnia AA. An update on toxicology of aluminum phosphate. Daru 2012;20:25.

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

5. Marashi SM, Nasri Nasrabadi Z, Jafarzadeh M, et al. Hydroxyethyl starch could save a patient with acute aluminum phosphate poisoning. Acta Med Iran 2016;54:475-8.

6. Farahani MV, Soroosh D, Marashi SM. Thoughts on the current management of acute aluminum phosphate toxicity and proposals for therapy: an evidence-based review. Indian J Crit Care Med 2016;20:724-30.

7. Mehrpour O, Gurjar M. Cardiogenic shock: the main cause of mortality in acute aluminum phosphate poisoning. Indian J Crit Care Med 2017;21:246-7.

8. Hassanian-Moghaddam H, Zamani N. Thoughts on the current management of acute aluminum phosphate toxicity and proposals for therapy: an evidence-based review. Indian J Crit Care Med 2017;21:61-2.

9. Soltani M, Shetab-Boushehri SF, Shetab-Boushehri SV. Chemical reaction between boric acid and phosphine indicates boric acid as an antidote for aluminum phosphate poisoning. Sultan Qaboos Univ Med J 2016;16:e303-9.

10. Taghaddosinejad F, Farzaneh E, Ghazanfari-Nasrabad M, et al. The effect of N-acetyl cysteine (NAC) on aluminum phosphate poisoning inducing cardiovascular toxicity: a case-control study. Springerplus 2016;5:1948.

11. Hassanian-Moghaddam H, Zamani N, Rahimi M, et al. Successful treatment of aluminium phosphate poisoning by extracorporeal membrane oxygenation. Basic Clin Pharmacol Toxicol 2016;118:243-6.

12. Hassanian-Moghaddam H, Zamani N. Therapeutic role of hyperinsulinemia/euglycemia in aluminum phosphate poisoning. Medicine (Baltimore) 2016;95:e4349.

13. Engebretsen KM, Kaczmarek KM, Morgan J, et al. High-dose insulin therapy in beta-blocker and calcium channel-blocker poisoning. Clin Toxicol (Phila) 2011;49:277-83.
Случайное отравление фосфидом алюминия: клинический случай и текущее лечение

Йозлем Чакан1, Гокхан Тазегул1, Айча Гюмюш2, Мелике Дженгиз2, Атилла Рамазаноглу2

1 Кафедра внутренних болезней, Медицинский университет Акдениз, Анталия, Турция
2 Кафедра анестезиологии и реанимации, Медицинский университет Акдениз, Анталия, Турция

Фосфид алюминия (ФА) представляет собой дешёвый родентицид, инсектицид и фумигант с массовым применением. Большинство случаев интоксикаций, описанных в литературе, относятся к суицидальному проглатыванию, но следует отметить, что ФА также может вызывать случайные ингаляционные интоксикации. После проглатывания или вдыхания в течение нескольких минут развиваются тошнота, рвота, диспнея и боль в животе. Отличительными признаками токсичности являются рефрактерная гипотензия, сердечная недостаточность и острый метаболический ацидоз, которые развиваются в течение нескольких часов и являются основными причинами смертности. В Турции таблетки ФА широко доступны и продаются без ограничений. Независимо от этого, в литературе описано небольшое количество местных клинических случаев. Кроме того, о случайных интоксикациях ФА сообщается исключительно редко.

Здесь мы представляем случай 25-летнего пациента, получившего случайно отравление ФА. Он был найден в бессознательном состоянии в зернохранилище, обработанном таблетками ФА. У него были установлены гипотония и тахикардия. Анализ газов артериальной крови не показывает метаболический ацидоз. Он был немедленно интубирован и принят в отделение интенсивной терапии. В качестве поддерживающей терапии назначены кристаллоидный раствор, н-ацетилцистеин и раствор норадреналина. Через 36 часов он был экстубирован и выписан без каких-либо осложнений.

Не существует конкретного антидота или лечения интоксикации ФА. В литературе встречаются противоречивые мнения в отношении подхода к лечению. Ингаляционная интоксикация может возникать в экстремальных условиях, как в данном клиническом случае. Следует предпринимать превентивные меры с целью сокращения числа инцидентов. Клиницисты должны также знать, что ФА широко распространён и является высокотоксичным веществом, для которого не существует специфического антидота, а необходимо своевременное лечение интоксикации с применением наиболее подходящей поддерживающей терапии.