World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(RESEARCH ARTICLE)

Check for updates

A SIGNIFICANT TOXIN: ALUMINUM PHOSPHIDE

Gul B [1,*] and Ozturan I [2]

[1] Department of Nursing, Faculty of Health Sciences, Firat University, Elazig, Turkey.
[2] Department of Pharmacology and Toxicology, Faculty of Veterinary Medicine, Firat University, Elazig, Turkey.
* Corresponding Author

ORCID Details
Gul B: https://orcid.org/0000-0002-9122-8953
Ozturan I: https://orcid.org/0009-0002-8606-9932

World Journal of Biology Pharmacy and Health Sciences, 2026, 27(03), 015–019

Publication history: Received on 26 July 2026; revised on 01 September 2026; accepted on 03 September 2026

Article DOI: https://doi.org/10.30574/wjbphs.2026.27.3.0460

ABSTRACT

Aluminum phosphide is a highly toxic fumigant used to control pests in agricultural products. It plays a critical role in food preservation and pest control. Its low cost and high efficiency ensure the safe transport of grains and feed in silos, ships, or trains. In the gastrointestinal tract, it rapidly transforms into the highly toxic phosphine gas (PH_3). Phosphine gas blocks the mitochondrial respiratory chain by inhibiting cytochrome oxidase activity and adenosine triphosphate (ATP) production. Its high mortality rate and lack of a specific antidote make treatment difficult. Early diagnosis, supportive treatment, and control of oxidative stress are critical in reducing mortality. This review provides general information about aluminum phosphide, as well as information on its mechanism of action, symptoms, diagnosis, and treatment protocols in cases of poisoning.

Keywords: Aluminum Phosphide, Toxicity, Treatment, Mechanism of action, Pestisid

1 INTRODUCTION

Poisoning can occur as a result of damage to living organisms by various factors such as some plants, foods, drugs, chemicals, toxic gases, metals and minerals, solvents, and pesticides. Pesticides, which have become widespread in agricultural production to increase productivity, have become a double-edged sword, leading to serious health problems and loss of life worldwide. This situation also brings about social and economic negative consequences in many societies [1]. Aluminum phosphide, preferred in the agricultural sector since the 1940s, plays a critical role in food preservation and pest control. Thanks to its low cost and high efficiency, it ensures the transportation of grains and feeds in silos, ships, or trains without spoilage [2]. Aluminum phosphide, considered the most dangerous and toxic member of the metal phosphide family, causes severe poisoning cases upon intentional or accidental exposure [3]. Aluminum phosphide is generally found in tablet, powder, and granular form and releases the highly toxic phosphine gas (PH_3) when it comes into contact with moisture, water, or stomach acid.

1.1 Chemical structure

Aluminum phosphide is a crystalline compound with a dark gray or yellow color (molecular weight: 57.95 g/mol). Approximately 56% of each tablet consists of aluminum phosphide, while the remaining 44% is aluminum carbonate. The toxic effects of aluminum phosphide are due to the PH_3 released when it reacts with stomach acid. Phosphine gas is rapidly absorbed through inhalation, ingestion, and skin or mucosal contact [4]. The chemical reactions that occur can be summarized as follows:

$$AlP + 3H_2O \rightarrow Al(OH)_3 + PH_3$$

$$AlP + 3HCl \rightarrow AlCl_3 + PH_3$$

Aluminum phosphite is available in tablet or pellet form. Aluminum carbonate is added to the mixture to prevent spontaneous combustion of phosphine gas released upon contact with moisture. Each 3 g tablet produces 1 g of phosphine, and each 0.6 g pellet releases 0.2 g of PH_3 upon contact with moisture. This reaction leaves behind a non-toxic grayish aluminum hydroxide residue [5]. Phosphine, which is colorless and odorless in its pure form, emits a pungent aroma similar to garlic or spoiled fish due to its components when it interacts with the atmosphere. However, even if the gas's odor is not noticeable, its presence in the environment at levels dangerous to human and animal health poses a critical risk.

1.2 Mechanism of action

Ingested aluminum phosphide rapidly transforms into the highly toxic PH_3 gas in the gastrointestinal tract. Phosphine gas blocks the mitochondrial respiratory chain by inhibiting cytochrome oxidase activity and adenosine triphosphate (ATP) production [6]. This biochemical effect occurs by interfering with the electron transport chain in the cells' energy powerhouses; the cessation of cytochrome c oxidase enzyme activity leads to cellular energy deficiency. The decline of ATP levels to critical levels causes impairment of vital organ functions, especially the heart.

PH_3, increases the production of reactive oxygen species (ROS), inhibiting enzymatic antioxidants such as catalase (CAT), glutathione reductase, and superoxide dismutase (SOD), thus creating a significant oxidative stress environment. An imbalance in redox triggers lipid peroxidation, which damages various cellular structures and accelerates cell death [7]. This mechanism includes cellular energy deficiency resulting from decreased ATP production, ion pump dysfunction, cell membrane damage, and necrosis and apoptosis processes.

Weakening of the activity of mitochondrial complexes I, II, and IV halts ATP synthesis, and the accompanying increase in ROS triggers the apoptosis pathway, accelerating cellular death. Aluminum phosphide causes cellular destruction by both paralyzing energy production and increasing structural damage [7].

When aluminum phosphide interacts with body fluids or stomach acid, a rapid release of phosphine gas begins. The first 24 hours following exposure are considered the most critical period; cardiovascular collapse is the most common cause of death during this time. This fatal condition is based on the cessation of ATP synthesis and the uncontrolled increase in ROS damaging the heart tissue.

2 TOXICITY

Aluminum phosphide poisoning can occur through oral and inhalation routes. When ingested orally, it is rapidly absorbed from the gastrointestinal tract, reacts with stomach acid, and the phosphine gas released enters the bloodstream. It causes significant toxic effects, primarily on the heart, but also on the liver, kidneys, lungs, and central nervous system. Aluminum phosphide poisoning leads to systemic collapse resulting in simultaneous loss of function in multiple organs [8]. The clinical picture of acute aluminum phosphide poisoning is characterized by severe metabolic acidosis resulting from a severe disruption of the body's acid-base balance, and resistant cardiogenic shock that does not respond to conventional treatments [9]. In poisoning cases, the primary factor leading to loss of life in the first 24 hours, considered the critical threshold, is the complete collapse of the cardiovascular system or sudden cardiac arrest [10]. Therefore, early recognition of congestive heart failure, arrhythmias, tachycardia, treatment-resistant hypotension, and electrocardiogram (ECG) changes is of great clinical importance [3].

For the heart muscle to maintain its mechanical function, cardiomyocytes must consume a high amount of energy continuously. In addition to energy synthesis and calcium balance, mitochondria play a decisive role in programmed cell death (apoptosis) or uncontrolled tissue loss (necrosis); therefore, maintaining mitochondrial integrity is vital for cardiomyocyte survival [11].

3 CLINICAL SYMPTOMS

The clinical picture following poisoning begins with vomiting, severe abdominal cramps, diarrhea, and a general state of restlessness. The main target organ is the circulatory system, and the primary cause of death is cardiovascular toxicity. Due to phosphine exposure, abnormalities in the ECG, histopathological changes, severe hypotension, left ventricular hypokinesis, changes in QT dispersion, and congestive heart failure develop [12]. In the cardiovascular system, weak (thread-like) pulse, tachycardia, tachypnea, metabolic acidosis, and treatment-resistant severe hypotension can be observed. Systemic damage resulting from aluminum phosphide exposure is characterized by uncontrolled bleeding, acute renal failure, disseminated intravascular coagulation disorders, and life-threatening arrhythmias. ECG shows serious electrical irregularities such as ST segment changes, prolongation of PR and QRS intervals, heart blocks, and fibrillation; however, in some cases, myocardial damage has been found to be reversible [13].

Poisoning can also lead to a severe condition characterized by fluid accumulation in the lungs (edema), dyspnea, and cyanosis due to hypoxia. Liver inflammation (hepatitis), disseminated intravascular coagulation disorders, and acute tubular necrosis in the kidney are among the rare but serious complications. In the clinical course, life-threatening conditions such as pericarditis, heart failure, acute gastrointestinal bleeding, and respiratory arrest can be observed.

4 DIAGNOSIS

The diagnosis of poisoning is made by evaluating clinical findings, anamnesis, and specific laboratory analyses together. The most decisive data in the diagnostic process are the patient's history of ingesting the substance and the pungent, garlic-like odor on their breath [14, 15].

Silver nitrate test: The darkening of paper moistened with a silver nitrate solution after contact with the patient's breath is a rapid preliminary indication of the presence of phosphine gas [15].

Biochemical analysis: Evaluation can be made for electrolyte imbalances and metabolic acidosis. For a definitive diagnosis, the detection of phosphine gas in gastric contents or blood samples is essential.

Clinical agreement: The clinical picture consistent with aluminum phosphide exposure supports the diagnosis [16].

5 TREATMENT PROTOCOL

Despite all medical interventions, the mortality rate in aluminum phosphide poisoning remains above 50%, and currently there is no specific antidote for this poisoning. The basis of treatment is a supportive approach; it is thought that some supportive agents can mitigate the destructive oxidative damage of aluminum phosphide. Among antioxidants and vitamins, melatonin, N-acetylcysteine, glutathione, sodium selenite, and vitamins C and E stand out. Magnesium sulfate, L-carnitine, boric acid, and coconut oil are considered supportive compounds. Triiodothyronine, liothyronine, vasopressin, and milrinone can be used for hormonal and cardiovascular support. These approaches aim to limit the cellular toxicity caused by aluminum phosphide [17]. Various positive inotropic agents are being investigated for the treatment of cardiotoxicity in aluminum phosphide poisoning. These agents include digoxin and dopamine; Dobutamine, amrinone, and enoximone are also being investigated as potential treatment options due to their inotropic properties [18]. Prospective studies have shown that left ventricular ejection fraction (LVEF) and global left ventricular hypokinesis are determinants in predicting the probability of survival; therefore, close monitoring of echocardiographic parameters is recommended [19].

It has been suggested that the use of sodium bicarbonate and coconut oil in gastric lavage may reduce phosphine release [20]. Sevelamer, which is thought to prevent phosphine gas formation by binding to aluminum phosphide groups due to its anion-exchange properties, has shown survival-enhancing and organ-damage-reducing effects in experimental studies [21]. Furthermore, melatonin stands out as a cardioprotective agent due to its ability to penetrate deeply into tissues by overcoming various morphological and physiological barriers, minimizing cellular damage in combating intense oxidative stress, and being directly synthesized in mitochondria [22]. The phenolic content of Moringa oleifera is reported to reduce cardiac damage by increasing oxidative stress defense enzymes and preventing lipid membrane peroxidation [23].

Dihydroxyacetone (DHA) provides significant support against energy depletion caused by aluminum phosphide by supporting ATP synthesis via the glycolytic pathway. Animal studies have shown that DHA use significantly reduces mortality; Clinical case reports indicate that DHA in addition to standard treatment improved symptoms [24, 25].

6 CONCLUSION

Aluminum phosphide is widely used, particularly in grain storage facilities, to eliminate harmful organisms. When it comes into contact with moisture or stomach acid, it releases highly toxic phosphine gas, which disrupts cellular respiration mechanisms, leading to severe toxic conditions that can result in multiple organ failure, severe metabolic disorders, and death. The inhibition of mitochondrial respiration by phosphine gas reduces ATP production, creates a significant oxidative stress environment, and causes cellular damage through lipid peroxidation. This process leads to cardiomyocyte death, resistant hypotension, and fatal arrhythmias. Aluminum phosphide poisoning remains a significant public health problem, especially in developing countries. Its high mortality rate and the lack of a specific antidote make treatment difficult. Cardiovascular system involvement is the leading cause of death. Early diagnosis, supportive treatment, and control of oxidative stress are critical in reducing mortality.

The speed of intervention is the most critical factor determining the prognosis in aluminum phosphide poisoning; It is essential to initiate medical intervention without delay to limit cardiac damage. Current research focuses on positive inotropic agents, antioxidant compounds, and strategies to reduce cellular damage. The cardiac protective potential of agents that support mitochondrial repair is attracting increasing attention. We believe that future studies developing and expanding mitochondrial protectors and antioxidant applications may be promising in the treatment management of aluminum phosphide poisoning.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

The authors declare no conflicts of interest regarding this manuscript.

REFERENCES

[1] Elhawary AE, Lashin HI, Fnoon NF, Sagah GA. Evaluation of the rate and pattern of suicide attempts and deaths by self-poisoning among Egytians before and during the COVID-19 pandemic. Toxicology Research. 2023; 12(6):1113-25.

[2] Hassanian-Moghaddam H. Phosphides and phosphine: Mechanisms for toxicity and range of the problem. Clinical Toxicology. 2014; 52(4),397-8.

[3] Sobh ZK, Kholief M, Sobh EK, Balah MIF. Exploring research gaps and trends in the management of acute phosphide poisoning: a systematic review. Critical Reviews in Toxicology. 2023; 53(3):181-206.

[4] Sudakin DL. Occupational exposure to aluminiumphosphide and phosphine gas? A suspectedcase report and review of the literature. Human & Experimental Toxicology. 2005; 24:27-33.

[5] Singh D, Dewan I, Pandey AN, Tyagi S. Spectrum of unnatural fatalities in the Chandigarh zone of north-west India- a 25 year autopsy study from a tertiary care hospital. Journal of Clinical Forensic Medicine. 2003; 10:145-52.

[6] Ranga GS, Dwivedi S, Agarwal M, Kumar D. Aluminium phosphide poisoning in a young adult: A suicidal cardiotoxin simulating myocardial ischaemia. Journal of the Indian Academy of Clinical Medicine. 2004; 5:369-72.

[7] Naddafi M, Eghbal MA, Khansari GM, SAttari MR, Azarmi Y, Samadi M, Mehrizi AA. Sensing of oxidative stress biomarkers: The cardioprotective effect of taurine & grape seed extract against the poisoning induced by an agricultural pesticide aluminum phosphide. Chemosphere. 2022; 287:132245.

[8] Abd-Allah MA, Abdalla Ahmed A, Mohamed NA, Rady MM, Farrag AA, Nasser GA, Salama K, Elfakharany YM. Updates on toxicology of Aluminum Phosphide and different management protocols. Zagazig University Medical Journal. 2022; 28(6):1176-83.

[9] Farrar RA, Justus AB, Masurkar VA, Garrett PM. Unexpected survival after delibrate phosphine gas poisoning: an Autralian experience of extracorporeal membrane oxygenation rescue in this setting. Anaesthesia and Intensive Care. 2022; 50(3):250-4.

[10] Kamaloddini MH, Zohoorian P, Foroughian M, Hemmati Awli S, Teimouri A. Successful Treatment of Acute Aluminum Phosphide Poisoning: Possible Benefit of Olive Oil - A Case Report. Updates in Emergency Medicine. 2021; 1(1):47-51.

[11] Bisaccia G, Ricci F, Gallina S, Di Baldassarre A, Ghinassi B. Mitochondrial dysfunction and heart disease: Critical appraisal of an overlooked association. International Journal of Molecular Sciences. 2021; 22(2):614.

[12] El-Sarnagawy GN, Shaban AE, Lashin HI. Validation of Corrected and Dispersed QT as Predictors of Adverse Outcomes in Acute Cardiotoxicities. Cardiovascular Toxicology. 2022; 22(1):1-13.

[13] Katira R, Elhence GP, Mehrotra ML. A study of aluminum phosphide (AlP) poisoning with special reference to electrocardiographic changes. Journal of the Association of Physicians of India. 1990; 38: 471-3.

[14] Chug SN, Ram S, Chugh K, Malhotra KC. Spot diagnosis of aluminium phosphide ingestion: an application of a simple test. Journal of the Association of Physicians of India. 1989; 37:219-20.

[15] Mital HS, Mehrotra TN, Dwivedi KK, Gera M. A study of aluminum phosphide poisoning with special reference to its spot diagnosis by silver nitrate test. The Journal of the Association of Physicians of India. 1992; 40:473-4.

[16] 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; 20:454-61.

[17] Karimani A, Mohammadpour AH, Zirak MR, Rezaee R, Megarbane B, Tsatsakis A, Karimi G. Antidotes for aluminum phosphide poisoning-An update. Toxicology Reports. 2018; 5:1053-9.

[18] Bogale DE, Ejigu BD, Muche TA. Clinical profile and treatment outcome of aluminum phosphide poisoning in felege hiwot referral hospital, Northwest Ethiopia: a retrospective study. Open Access Emergency Medicine. 2021; 13:239-48.

[19] Elgazzar FM, Shama MA, Shoeib O, Abd-Elhameed A, Hafez ASA. The Role of Echocardiographic Findings in Estimating Survival Probability of Intensive Care Unit Admitted Aluminum Phosphide Poisoned Patients. Journal of Medical Toxicology. 2022; 18(2): 128-38.

[20] 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. 2015; 19(2):109-12.

[21] Heidari R, Mohammadi HR, Goudarzi F, Farjadian F. Repurposing of sevelamer as a novel antidote against aluminum phosphide poisonin: An in vivo evaluation. Heliyon. 2023; 9: e15324.

[22] Chrustek A, Olszewska-Słonina D. Melatonin as a powerful antioxidant. Acta Pharmaceutica. 2021; 71(3): 335-54.

[23] Olvera-Aguirre G, Mendoza-Taco MM, Moo-Huchin VM, Lee-Rangel HA, Roque-Jimenez JA, Gomez-Vazquez A, Dzib-Cauich DA, Vargas-Bello-Perez E, Chay-Canul A. Effect of extraction type on bioactive compounds and antioxidant activity of Moringa oleifera Lam. leaves. Agriculture. 2022; 12(9): 1462.

[24] Niknahad H, Heidari R, Hashemi A, Jamshidzadeh A, Rashedinia M. Antidotal effect of dihydroxyacetone against phosphinepoisoning in mice. Journal of Biochemical and Molecular Toxicology. 2021; 35(11): e22897.

[25] Oghabian Z, Ahmadi J, Pakravan S, Dabaghzadeh F, Heidari MR, Tajaddini S, Karami-Mohajeri S. Successful treatment of aluminium phosphide poisoning bydihydroxyacetone: A two-case report study. Journal of Clinical Pharmacy and Therapeutics. 2020; 45(5):1194-8.