TURKISH JOURNAL OF EMERGENCY MEDICINE

Invited Review Article

Access this article online

Quick Response Code:

Website: https://turkjemergmed.com/

DOI: 10.4103/tjem.tjem_49_25

Aluminum phosphide: Toxicological profiles, health risks, environmental impact, and management protocols: A review

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Abstract:

Aluminum phosphide (AlP) is a common pesticide known for extremely negative environmental, health, and work-related outcomes. Its high availability and easy accessibility have led it to become the chosen method of suicide in many low- and middle-income countries. When AlP reacts with moisture or water, it releases phosphine gas, which is quickly absorbed by the body and leads to severe toxic effects, even death. Occupational and environmental health risks are particularly high in cases of large-scale fumigation or accidental exposure. In Türkiye, two people, one of whom was a child, died due to AlP accidents that affected workplaces and the environment and caused hospitalizations in 2023. In 2024, further suspected cases have been reported, highlighting the ongoing risk. First responders, particularly emergency department team, paramedics, and firefighters, are at significant risk of exposure when managing these cases. The lack of awareness and appropriate protective measures during initial intervention can lead to secondary exposure, worsening the crisis. Medical staff taking care of victims are also at risk of being exposed, further emphasizing the need for stringent safety precautions. Besides, this pollution might cause irreversible damage to soil and water. Thus, this review provides insight into the physical and chemical properties, mechanism of toxicity, current treatment modalities, health–environmental effects, and preventive measures. Given its high toxicity and frequent usage, increased awareness and preparedness among first responders and healthcare professionals are essential. This is a lesson in practice for better safety protocols and emergency response to mitigate health hazards and environmental impacts.

Keywords:

Aluminum phosphide, environmental effects, health effects, management, occupational health and safety, phosphine, toxicology

Submitted: 05-02-2025
Revised: 20-03-2025
Accepted: 26-03-2025
Published: 01-07-2025

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Introduction

Pesticides pose significant risks to human health and the environment, with both suicidal and accidental exposures contributing to major public health challenges. The global incidence of accidental acute pesticide poisoning has surged, increasing from 25 million cases in 1990 to approximately 385 million cases annually today, resulting in an estimated 11,000 deaths per year.[1] Accidental pesticide poisoning impacts nearly 44% of the global farming population. Furthermore, pesticides are frequently used for suicides, particularly in low- and middle-income countries where three-quarters of global suicides occur (mostly Asia).[2] This constitutes a critical global public health issue, causing

How to cite this article: Çakmakcı Karakaya S, Yavuz CI. Aluminum phosphide: Toxicological profiles, health risks, environmental impact, and management protocols: A review. Turk J Emerg Med 2025;25:178-90.

at least 110,000–168,000 deaths annually.[3-5] Accidental exposure can also result in severe clinical conditions and fatalities.

Among the most widely used pesticides are organophosphates and phosphides, particularly aluminum phosphide (AlP), which is prevalent in low- and middle-income countries. AlP is a common fumigant used to protect stored agricultural products and buildings from pests. It is responsible for approximately 15,000 poisoning cases annually in India, with a mortality rate of 67%.[6] Its frequent use in suicides[7] highlights the urgent need to address the problems associated with its widespread availability, particularly in developing countries.

Characteristic Features

AlP is a dark gray or dark yellow, crystalline (sand-like) metal phosphide in solid form.[8] It remains stable up to 1832°F (1000°C) but is highly sensitive to moisture. Since the 1940s, AlP has been utilized as a fumigant to protect agricultural products, buildings, and containers from pests and, occasionally, as a rodenticide in nests. It is typically formulated as tablets, pellets, or powder sachets with ammonium compounds to control gas release and prevent flammability.

This formulation is encased in a plastic matrix to regulate moisture exposure and manage gas emission.[9] In Türkiye, AlP is widely used against stored-product pests. It is sold in 3-gram tablets containing 56% AlP, which can release 1 g of phosphine (hydrogen phosphide, PH_3) gas when reacting with moisture.[10] When AlP pellets react rapidly with water or moisture, they release highly toxic PH_3 gas, a fumigation agent, along with ammonia and CO_2.[11] The gas formation rate depends on ambient temperature and humidity. PH_3 is a low-molecular weight, colorless, highly toxic, and flammable gas released through rapid chemical reactions, allowing deep penetration into materials like grain piles.[9] Pure PH_3 is odorless, but commercial PH_3 has a garlic or rotting fish odor. However, its odor threshold of 0.15 ppm is not an adequate warning for hazardous concentrations.[12,13]

Paths of phosphine release and regulatory restrictions

Being denser than air, PH tends to remain near the ground, with dispersion influenced by wind and geography. To mitigate risks, the U.S. EPA recommends notifying individuals within a 250-m radius before application. PH_3 degrades rapidly in air, water, and soil, with a half-life of 5 h in daylight and 28 h in darkness due to photoreaction,[14,15] which results in the formation of nonvolatile phosphorus compounds.[16] It does not bioaccumulate and is unlikely to contaminate crops. As PH_3 gas is denser than air, it tends to remain near the ground, with dispersion influenced by geography and wind. To mitigate risks, The United States Environmental Protection Agency (U. S. EPA) recommends notifying individuals within a 250-m radius before application.[17]

AlP is freely available in some countries and widely used in Central Asia, particularly in India and Iran.[18] The European Union regulates AlP under Commission Implementing Regulation (EU) No. 1034/2013, requiring usage by trained professionals, safety measures, and risk mitigation procedures.[19] Despite these restrictions, AlP poses risks for misuse, including chemical terrorism.[20] AlP poisoning has been extensively reported since the 1980s,[20] becoming a leading cause of pesticide-related deaths in India, accounting for 65% of poisoning cases.[21] Fatal cases are common in India, Iran, Egypt, Morocco, and Sri Lanka,[20,22] but rare in Europe due to sales restrictions.[23] In Türkiye, sales are restricted to licensed professionals, minimizing misuse. These restrictions have limited the misuse of AlP for suicide, though fatal accidents have occurred due to illegal use. In May 2023, AlP unloaded at Safiport Port in Kocaeli ignited for unknown reasons, releasing significant smoke.[24] The fire was extinguished after 6 h by emergency response teams. In June 2023, Somali nationals in Ankara used AlP illegally for bedbug control, resulting in two deaths of a mother and a child and 10 hospitalizations.[25] In October 2023, an AlP-related explosion occurred at a fumigation company's warehouse in Kocaeli due to water contact. The incident led to the evacuation of 43 individuals, but no fatalities occurred. The workplace had incomplete licensing documentation.[24]

Exposure Limits

The National Institute for Occupational Safety and Health (NIOSH) sets an average exposure limit of 5 mg/m^3 for a 10-h work shift, while the American Conference of Governmental Industrial Hygienists sets it at 5 mg/m^3 for an 8 h.[26] Other values include the lowest lethal concentration for AlP (LCLo) (human): 2,800 mg/m^3 (inhalation)[27] and the median lethal dose (LD_{50}) (human): 20 mg/kg (inhalation).[8] PH_3 occupational exposure limits are listed in Table 1, with regional variations.[15] Long-term exposure to 3 ppm is safe, but 30 min at 500 ppm or brief exposure to 1000 ppm (1400 mg/m^3) can be lethal.[28] Both AlP and PH_3 are classified as "super toxic" with a probable oral lethal dose below 5 mg/kg. The Turkish regulation sets PH_3 exposure limits: TWA (time-weighted average measured or calculated for a specified reference period of 8 h) of 0.1 ppm (0.14 mg/m^3) and STEL (upper exposure

Table 1: Occupational exposure limit values for phosphine

	TWA (8 h), ppm (mg/m³)	STEL (15 min), ppm (mg/m³)
NIOSH REL	0.3 (0.4)*	1 (1)
OSHA PEL	0.3 (0.4)	
ACGIH TLV	0.3	1
NIOSH IDLH	50	

*10 h. 1 ppm=1.39 mg/m³. ACGIH: American Conference of Governmental Industrial Hygienists, IDLH: Immediately dangerous to life or health, NIOSH: National Institute for Occupational Safety and Health, OSHA: Occupational Safety and Health Administration, PEL: Permissible exposure limits, ppm: Parts per million, REL: Recommended exposure limit, STEL: Short-term exposure limit, TLV: Threshold limit value

limit value that should not be exceeded for a period of 15 min) of 0.2 ppm (0.28 mg/m³).[29]

Health Effects

Clinical history and findings are critical in suspected AlP poisoning, as routine laboratory tests are unavailable. AlP may be detected in stomach contents or breath using silver nitrate test papers, though this method is rarely employed.[20]

AlP-related toxicity and mechanisms

AlP can irritate the skin, eyes, and mucous membranes.[8] AlP is highly toxic when ingested or inhaled, producing PH_3 gas upon reaction with water or acid in the gastrointestinal (GI) tract. PH_3 gas enters systemic circulation, primarily affecting the liver and lungs.[22] This mechanism is largely responsible for the toxicity,[11] and a fatal outcome can occur quickly depending on the dose.[20] PH_3 retention in the lungs causes congestion, hemorrhage, atelectasis, and alveolar damage.[22,30] Since AlP is solid under normal conditions, skin and eye poisoning are rare.[31] Inhalation of PH_3 gas, formed by moisture on metal phosphides, is another exposure route. PH_3 diffuses systemically and is exhaled, while AlP is primarily excreted in urine as hypophosphite.[32]

PH_3 inhibits protein production[15] and mitochondrial cytochrome-c oxidase, reducing oxidative phosphorylation by up to 70%.[33] PH_3 causes mitochondrial membrane depolarization and generates free radicals, causing lipid peroxidation, protein denaturation,[34] and cellular damage, leading to organ dysfunction.[35] Organs with high oxygen demand, such as the heart, lungs, kidneys, and liver, are particularly vulnerable.[36] In the heart, PH_3 disrupts mitochondria, induces oxidative damage, and alters cell permeability.[37] Free oxygen radicals contribute to neutrophil adhesion in coronary arteries and vasoconstriction,[38] while tissue hypoperfusion and acidosis may lead to cardiac dysfunction.[39] Postmortem findings include myocyte vacuolization and degeneration.[40] In addition, PH_3 reacts with hemoglobin, forming Heinz bodies and reducing heme capacity.[41]

Liver damage is typically mild, but fatal cases show hepatocyte vacuolization, sinusoidal congestion, and leukocyte clusters.[42] While the heart is the primary target, symptoms such as nausea, vomiting, arrhythmias, cyanosis, and pulmonary edema indicate systemic effects on the lungs, GI tract, and kidneys.[20,22]

Acute health effects

Acute AlP exposure effects appear within 10–15 min,[18,43] varying by exposure route. Respiratory exposure causes sore throat, cough, headache, dizziness, nausea, and garlic-smelling breath; dermal exposure leads to redness and burning; ocular exposure results in redness and pain; ingestion causes nausea, vomiting, abdominal pain, headache, convulsions, and shock. Symptoms can escalate to cardiovascular collapse, arrhythmia, acute respiratory distress syndrome (ARDS), and neurological issues like coma.

Early signs may include cardiovascular collapse, arrhythmia, ARDS, convulsions, coma, and central nervous system (CNS) depression.[23] Less common complications include hemolysis, adrenal insufficiency, pancreatitis, glucose imbalances, methemoglobinemia, hemolytic anemia, disseminated intravascular coagulation, and electrolyte disturbances.[44]

Hemolysis may occur in patients with glucose-6-phosphate dehydrogenase (G-6-PD) deficiency.[45,46] AlP disrupts endovascular and erythrocyte membranes via free radicals, causing hemolysis and forming methemoglobin (Met-Hb), which reduces oxygen delivery. Met-Hb effects depend on levels: up to 20% alters skin and blood color; over 20% causes neurological and cardiac symptoms; and above 70% is fatal. Rarely, metabolic acidosis triggers severe hemolysis.[47]

PH_3 inhalation leads to respiratory irritation, cardiovascular and CNS depression, and severe abdominal pain. Symptoms vary with dose, appearing immediately or within hours.[9,15] Cardiovascular complications may cause death within 12–24 h, while liver damage symptoms are delayed by 48–72 h. Deaths after 24 h often result from liver/kidney failure, with pulmonary edema appearing up to 72 h postexposure.[15]

Mild poisoning causes fatigue, tinnitus, nausea, chest pressure, and restlessness, resolving in fresh air.[9] Severe poisoning may cause extreme fatigue, vomiting, abdominal pain, vertigo, chest pain, and dyspnea. Extremely high concentrations result in severe dyspnea, cyanosis, agitation, ataxia, anoxia, unconsciousness, or death, occurring immediately or days later due to pulmonary edema, respiratory failure,

or brain edema. Dose and exposure type determine severity:[15,48]

Ocular exposure

PH_3 has no known ocular or systemic toxicity from eye exposure. Direct contact with liquefied or compressed PH_3 gas may cause frostbite.

Ingestion exposure

PH_3 is gaseous at room temperature, making ingestion unlikely. However, GI exposure to AlP can cause hematemesis, vomiting, and epigastric discomfort. Endoscopic studies show corrosive esophageal and gastric lesions, with possible severe erosions[23] and late complications such as esophageal strictures, fistulas, and dysphagia.[49,50] If untreated, aspiration may lead to lung infections, causing morbidity or death.[50]

Inhalation exposure

Mild-to-moderate exposure

Symptoms include lung irritation, chest pain, green sputum, dyspnea, and pulmonary edema, alongside cardiovascular issues such as hypotension and arrhythmias. Neurological symptoms such as headache, dizziness, gait disturbances, and diplopia may occur due to brain hypoperfusion. GI symptoms include nausea, vomiting, abdominal pain, and liver damage, accompanied by sweating and cyanosis.[51] GI effects include nausea, vomiting, abdominal pain, and liver damage, with general symptoms such as sweating and cyanosis.

Severe exposure

This may result in pulmonary edema, cardiovascular collapse, ventricular arrhythmias, heart failure, shock, acute hypoxic encephalopathy, seizures, coma, and potentially death.

Dermal exposure

PH_3 gas does not usually cause adverse skin effects or systemic toxicity. Contact with liquefied or compressed PH_3 gas can result in frostbite.

Chronic health effects

While most survivors of acute PH_3 exposure recover without permanent disability, some report damage due to inadequate blood supply to the heart and brain.[15] Chronic effects from AlP exposure may appear months or years later, including hepatotoxicity and nephrotoxicity.[23]

Cancer risk

The International Agency for Research on Cancer has not classified AlP as carcinogenic.[52] While aluminum production is a Group 1 carcinogen, aluminum itself is not classified as carcinogenic. PH_3 remains unclassified for human carcinogenicity (EPA Group D).[53]

Reproductive system effects

Current evidence does not indicate adverse effects of AlP or PH_3 gas on human reproduction or development. Some studies suggest no significant impacts.[54]

Other chronic effects

Subacute exposure over days may lead to reactive airway dysfunction syndrome months later.[15] Repeated exposure may cause bronchitis, with symptoms including cough, sputum production, and breathlessness.[26] Aspiration of chemicals can result in pneumonitis or bacterial pneumonia.[55] Liver damage may lead to jaundice, proteinuria, and renal failure.[15]

Health effects of chronic and repeated exposure

Although PH_3 is classified under Group D for carcinogenicity, chronic exposure has been linked to chromosomal damage.[15] Chronic exposure to very low concentrations can cause anemia, bronchitis, GI disorders, kidney failure, vision, speech, and motor disorders. These adverse health effects may be of greater concern for children exposed to similar levels. Toothache, jaw swelling, and spontaneous breaking of bones (Phossy jaw) are also consequences of chronic exposure.[56]

Diagnostic and Adjunctive Tests

Accurate diagnosis of AlP poisoning relies on clinical suspicion, thorough patient history, and crime scene investigation, coupled with timely communication with healthcare facilities. Although limited chemical and analytical tests exist, they are not routine in emergency settings. The silver nitrate test serves as a simple spot test for detecting PH_3 gas in gastric fluid or breath.[57] Ammonium molybdate testing is another option for stomach contents, offering both qualitative and quantitative results.[58] Gas chromatography is the most sensitive method for detecting PH_3 in blood or air.[59] Key tools:

- Electrocardiogram (ECG): This may show sinus tachycardia, ST segment abnormalities, inverted T waves, myocardial infarction, atrioventricular block (particularly right bundle branch), or complete heart block. ECG typically normalizes within 10–25 days if the patient survives the initial 24 h
- Chest X-ray: Pulmonary edema and pleural effusion
- Blood sugar monitoring: This may indicate hypoglycemia
- Arterial blood gas (ABG)/venous blood gas analysis: This can reveal metabolic acidosis, methemoglobinemia, or both, often accompanied by respiratory alkalosis
- Liver and kidney function tests: Crucial for diagnosing

AlP poisoning.[32,60] Elevated serum aspartate and alanine aminotransferase levels are common in cases of liver involvement[61]
- Serum electrolytes: This may show imbalances
- Blood tests: Leukopenia, anemia, or intravascular hemolysis
- Cardiac markers: Creatine phosphokinase (CK), CK-myocardial band (MB), troponin-T (TnT), and troponin-I (TnI) can identify myocardial damage.[62,63]

For individuals with occupational exposure, annual pulmonary function tests, as well as liver and kidney function tests, are recommended. Chest X-rays should be performed if symptoms develop, or overexposure occurs.[26] A comprehensive history and physical examination are essential in workplace evaluations.

Differential Diagnosis

The clinical presentation of AlP poisoning may mimic infectious, inflammatory, or metabolic conditions, potentially delaying diagnosis and treatment. Mild cases may resemble upper respiratory infections, while severe cases could be confused with other pesticide poisonings, acute coronary syndrome, myocarditis, cardiopulmonary edema, pneumonia, or ARDS.[64] Zinc phosphide poisoning presents with similar symptoms but tends to progress more slowly and has a lower mortality rate.[65,66]

Treatment Methods

First aid

Initial interventions should be performed on-site.[26,43] The local poison information center should be contacted for guidance. Victims should move to fresh air immediately, as mild symptoms may subside there, though first aid must continue until professional help arrives.[9] Medical help should be requested immediately by calling appropriate emergency services or be referred to the hospital.

Ocular contact

Rinse eyes with water for at least 15 min, lifting eyelids. Remove contact lenses if possible.

Dermal contact

Wear gloves, remove contaminated clothing, and wash skin with soap and water.

Respiratory contact

Move the victim to fresh air and rest in an upright position. Avoid mouth-to-mouth resuscitation; use rescue breathing if needed. Perform cardiopulmonary resuscitation if necessary.

Oral contact

Rinse mouth and avoid inducing vomiting.

Treatment

Treatment should begin immediately after taking a brief history and conducting a clinical examination, without waiting for a confirmed diagnosis. Healthcare personnel should wear full-face masks and latex gloves, as small-pore masks without canisters do not protect against PH_3 exposure (see respiratory protection).

Patient stabilization

Airway and breathing

If the patient is conscious and responds normally, the airway is likely open.[67] Untreated airway spasms or obstructions can quickly lead to arrest, so obstructions should be cleared by suctioning. Ventilation support may be required if breathing is inadequate, using a bag valve mask operated by trained personnel.

Circulation

Monitor capillary refill time, pulse, and skin color for signs of poor circulation, such as sweating and confusion. If a stethoscope is available, heart auscultation should be performed. Check blood pressure and ECG signals if possible. In cases of hypotension, lay the patient flat with legs slightly raised, secure IV access, and begin supportive fluid therapy, following guidelines.[67]

Decontamination

Gastric lavage

Early gastric lavage (GL) with potassium permanganate (1:10,000), which oxidizes PH_3 to nontoxic phosphate,[68] or a combination of coconut oil and sodium bicarbonate, is recommended.[20,22,23] However, potassium permanganate may cause hemolysis and methemoglobinemia in some patients.[47]

Paraffin oil (50 ml) can create a protective barrier that reduces PH_3 absorption, decreasing gastric symptoms and complications, and its effectiveness, along with sodium bicarbonate, was demonstrated in a recent randomized controlled trial, showing significant reductions in gastric symptoms and complications.[69] A meta-analysis showed paraffin oil reduced mortality risk by 41%, intubation by 41%, and vasopressor need by 29%, with similar benefits observed for coconut oil.[70]

For oral solid phosphide ingestion, GL with activated charcoal is suggested, though some argue it may be ineffective due to AlP's molecular weight being below the absorption limit of activated charcoal.[71] Water-based treatments are not recommended for gastric decontamination.[72]

Elimination

Coconut oil

Coconut oil may block the systemic absorption of PH_3 by coating the stomach wall.[73] In a study, GI lavage with diluted potassium permanganate, coconut oil, and sodium bicarbonate was used on seven patients with severe hemodynamic instability, with four patients surviving, suggesting coconut oil's potential effectiveness in the absence of a specific antidote.[74]

Antioxidants

Due to the oxidative effects of PH_3 toxicity, antioxidants like IV magnesium sulfate and N-acetylcysteine (NAC) are recommended.[22,23] A meta-analysis on acute AlP poisoning showed NAC reduced mortality by 50% and decreased mechanical ventilation needs, though not significantly.[75]

Other antioxidants, such as Vitamins C, E, and NAC, also showed efficacy in case reports and clinical trials.[76] In a randomized controlled study, 36 AlP-poisoned patients receiving 400 mg IM Vitamin E twice daily showed significantly reduced mechanical ventilation need (30% vs. 62%) and mortality rate (15% vs. 50%) compared to controls.[77] Melatonin may counteract AlP-induced cardiotoxicity by reducing mitochondrial damage and apoptosis through limiting mitochondrial permeability, impairing caspase activation and inhibiting cytochrome-c release.[78] Coenzyme Q10 (CoQ10) has shown promise in reducing oxidative stress, enhancing mitochondrial function, and protecting liver cells in AlP toxicity cases.[79] Resveratrol[80] and cerium oxide (CeO_2) nanoparticles[81] demonstrated protective effects in cardiac myocyte cells by reducing oxidative stress and apoptosis, indicating potential therapeutic value for AlP cardiotoxicity. Injecting healthy mitochondria in AlP-exposed rats improved survival rates and reduced oxidative damage, suggesting mitochondrial transplantation as a potential treatment.[82] At concentrations of 125 and 250 μg/kg, the survival rates were boosted to 40% and 56.25%, respectively, over 30 days.

Lipid sink theory

Since PH_3 may also have fat-soluble properties, IV lipid emulsion therapy, based on the lipid sink theory,[83] showed clinical and biochemical benefits in AlP poisoning by improving survival time, blood pressure, and metabolic acidosis, though mortality impact remains unclear.[84] The study indicated positive trends such as a decreased need for intubation and mechanical ventilation in the intervention groups.

Symptomatic and supportive treatment approaches

Since there is no specific treatment for AlP poisoning, symptomatic and supportive care are essential, with timing significantly impacting prognosis. Mild cases may require rest and warmth, while severe cases often need intensive care unit (ICU) monitoring.[9]

Mortality rates range from 30% to 100%, depending on dosage.[23] Although the survival rate is generally very low for poisonings of ≥ 1500 mg, few patients survive as a result of poisonings of ≥ 9000 mg.[20,85] Early emesis and supportive therapy have improved survival in some cases of high-dose poisoning.[20]

For complications, standard therapy for seizure, blood transfusions, antibiotics for aspiration pneumonia, and hydrocortisone for adrenal insufficiency may be needed. A 17-year-old girl who ingested 6 g of AlP for suicide, despite AlP doses of 150–500 mg having high mortality rates, survived severe symptoms including metabolic acidosis and shock with hydrocortisone infusion treatment.[60] A 40-year-old male who accidentally ingested 3 g of AlP received IV fluids and intermittent dialysis, which eliminated the need for norepinephrine, and was later extubated and discharged.[86] Patients should be closely monitored for arrhythmias, acidosis, shock, and ARDS, with prompt intervention as these conditions arise. Mortality is primarily due to arrhythmias within the first 24 h and to acidosis, shock, and ARDS thereafter.

Symptomatic patients should receive oxygen for shortness of breath, with 72-h monitoring and chest examinations. Hypoxia may be managed with oxygen, early positive pressure ventilation, or intubation to minimize pulmonary edema.[87] In a rare case, hyperbaric oxygen therapy led to recovery from AlP poisoning, though further research is needed.[88] Aerosol bronchodilators are recommended for acute bronchospasm, with consideration for myocardial health.[32,89,90]

In cases of toxic myocarditis with cardiogenic shock, one patient survived with an intra-aortic balloon pump after resistance to IV fluids and inotropic treatments.[89,90] Another 19-year-old patient with severe lactic acidosis and cardiac dysfunction was successfully treated with extracorporeal membrane oxygenation, though this complex treatment is reserved for high-risk cases.[91] Continuous renal replacement therapy was effective in two patients with shock, while hemodialysis proved beneficial for renal failure and severe acidosis, despite limited PH_3 removal.[20,92]

A recent systematic review evaluating the role of exogenous insulin euglycemia therapy in symptomatic AlP poisoning found that insulin administration reduced mortality, required lower vasopressor doses, and

decreased the need for intubation.[93] In addition, it was associated with improved systolic blood pressure (SBP) and favorable biochemical changes (e.g., increased bicarbonate and decreased lactate levels), making it a safer alternative to vasopressor-only treatment. Hypoglycemia, hyperglycemia, and hypokalemia were the most frequently reported adverse events, but they were generally manageable. Despite variability in insulin regimens across studies, findings consistently demonstrated hemodynamic stabilization and improved survival in AlP poisoning. An initial bolus of 1 IU/kg, followed by infusion rates of 0.2–0.5 IU/kg/h, was linked to faster blood pressure stabilization, whereas lower bolus doses (0.1–0.2 IU/kg) required longer infusion durations to achieve similar effects. In refractory hypotension cases (SBP <90 mmHg), dose escalation up to 3 IU/kg/h further improved circulatory function, while higher doses (up to 10 IU/kg) correlated with progressive SBP increases, reinforcing a dose-dependent response. Prolonged insulin infusion was also associated with reduced mortality, with each additional hour of therapy showing incremental survival benefits.

The pathophysiological basis for insulin's benefit in AlP poisoning lies in its ability to counteract mitochondrial dysfunction and metabolic failure. PH_3 gas inhibits cytochrome c oxidase, leading to oxidative phosphorylation failure, metabolic starvation, and severe refractory hypotension, which is the primary cause of mortality. The myocardium is particularly vulnerable, experiencing cell membrane dysfunction, oxidative stress, and impaired glucose utilization, exacerbating circulatory collapse. Insulin, a key regulator of cardiac metabolism, shifts myocardial energy utilization from β-oxidation to glycolysis, thereby enhancing cardiac contractility and tissue perfusion. Given its established role in conditions such as beta-blocker and calcium channel blocker toxicity, diabetic ketoacidosis, and metabolic crises, insulin euglycemia therapy has emerged as a promising adjunct in AlP poisoning management. With current knowledge, exogenous insulin appears beneficial in AlP poisoning. However, high-quality randomized controlled trials are still needed to establish standardized protocols and confirm its clinical efficacy with confidence.

In a study evaluating the effect of Glucose-Insulin-Potassium (GIK) infusion therapy on ALP poisoning, 20% of 15 ALP poisoning cases treated with GIK resulted in death, while 80% survived.[94] When compared with previous case results, it was stated that GIK treatment provided hemodynamic stabilization and allowed patients to be followed up for a longer period. It was observed that patients transferred to acute unit survived for a longer period. Normally, low blood pressure is a poor prognostic factor in ALP poisoning, but here it can be considered that GIK may help stabilize hypotension. Nevertheless, GIK treatment cannot be considered a completely rescue treatment but can be used within the scope of supportive treatment.

Dihydroxyacetone was tested on 76 AlP poisoning patients, with a survival rate of 65.5% in the dihydroxyacetone group versus 33.3% in the control group.[95] Dihydroxyacetone improved biomarkers without significant adverse effects, suggesting its potential as an effective adjunct therapy.

Sobh *et al*. analyzed acute AlP poisoning management among Egyptian physicians, revealing significant variability in approaches.[96] Noradrenaline was used by 90.7% without standard dosing; 84.1% employed oil (primarily paraffin oil) for GI decontamination, with sodium bicarbonate, proton pump inhibitors, IV magnesium sulfate, and antioxidants also commonly used. Using oil improved outcomes by 4.62 times. Clinical toxicologists achieved higher success rates, often employing oil and magnesium sulfate. The study highlights inconsistent treatment protocols and suggests that toxicologists' methods may significantly improve outcomes.

Additional recommendations post-intervention

The affected person should avoid work, especially fumigation tasks, for 48 h to allow for toxin elimination.[9] Complete abstinence from alcohol is also strongly advised after poisoning.

Prognostic Factors

Dose is a key predictor of outcome. The lethal dose of AlP is 150–500 mg, with mortality rates between 70% and 100%.[60] Mortality in adults rises to 30%–100% with AlP intake of 500 mg or more,[23] and survival decreases with higher doses. Non-survivors show lower ABG levels and poor treatment response.

In Ethiopia, 26% of poisonings were due to metal phosphides, mainly AlP, causing 55% of deaths.[97] AlP poisoning often leads to ICU admission and mechanical ventilation, reflecting its high fatality rate.

Saleh *et al*. reported that hypotension, cardiogenic shock, and palpitations were the most common symptoms in 60 AlP patients, with a mortality rate of 92%.[98] Poor prognosis is associated with cardiogenic shock, arrhythmias, and metabolic acidosis.

In Egypt, elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels (≥72 pg/ml) predicted mortality in AlP poisoning cases, whereas TnI and CK-MB

did not.[99] These findings suggest that NT-proBNP could serve as an early marker of poor prognosis in AlP poisoning cases.

Higher blood PH_3 levels correlate with higher mortality; levels ≤1.067 mg suggest survival.[100] Swallowing small amounts, expired tablets,[101] or early supportive care can improve survival.[20]

Mehrpour *et al.* identified factors associated with poor prognosis in AlP poisoning, including hyperglycemia, high simplified acute physiology score (SAPS II) and acute physiology and chronic health evaluation (APACHE II) scores, hypotension, acidosis, low prothrombin rate, leukocytosis, methemoglobinemia, hyperuricemia, ECG abnormalities, low Glasgow coma scale, acute kidney failure, use of vasoactive drugs, the absence of postingestion vomiting, and mechanical ventilation need.[23]

A case report described a unique instance of AlP toxicity in a 30-year-old male with G-6-PD deficiency, suggesting a protective effect against severe AlP complications.[102] The patient, who showed jaundice and anemia, recovered with supportive care, indicating G-6-PD deficiency may help counteract oxidative damage in AlP poisoning.

Smoking and alcohol intake may exacerbate health issues caused by AlP, as smoking increases risks for respiratory and cardiovascular diseases, and excessive alcohol can damage the liver.[26] Quitting smoking and consulting a doctor before taking phosphorus supplements are recommended for risk reduction. Given that more than moderate alcohol consumption can cause liver damage, alcohol intake could exacerbate the liver damage caused by AlP.

A study with 94 acute AlP poisoning cases found that high shock index (SI) and modified SI (MSI) were significantly linked to ICU admission and mortality.[103] SI and MSI correlated with poisoning severity and predicted ICU admission and mortality at cutoffs > 1.14 and > 1.47/1.5, respectively, proving their usefulness in assessing AlP poisoning severity.

Environmental and Ecological Impacts

AlP is unstable in most environments, decomposing upon contact with atmospheric moisture or soil into aluminum hydroxide and PH_3, the toxic substances in these pesticides.[17] Aluminum hydroxide residues can form natural mineral phases, while PH_3 oxidizes into inorganic phosphates and other phosphorus oxyacids. PH_3 below the soil surface is rapidly adsorbed and degraded, with interaction types varying by soil.[104-106] The risk of water contamination is low, as PH_3 degrades within days.[17] PH_3 near the soil surface diffuses into the atmosphere for photodegradation, while subsurface PH_3 binds to soil and oxidizes to phosphates.

AlP is generally considered low risk to nontarget organisms and water bodies, except for endangered species exposed to these chemicals in their habitats. Label restrictions, environmental fate, and aquatic toxicity data indicate minimal risk to aquatic species, habitats, and terrestrial plants.[107] However, AlP poses high risks to burrowing terrestrial vertebrates and invertebrates exposed to phosphine gas during treatment. Applicators can minimize risks by inspecting nests and ensuring the presence of target animals before application. There is no secondary risk to nontarget species, as PH_3 dissipates quickly and does not accumulate in target animals. Labels include restrictions to limit exposure and risks to nontarget terrestrial species.

Decontamination

Decontamination aims to safely and effectively remove toxic substances, ensuring safety for individuals and equipment. Care is needed, as absorbed agents may release as gases from clothing and skin.[15]

Decontamination corridor

Recommendations for first responders include:

- Position the decontamination corridor upwind and uphill of the hot zone. Establish two corridors: One for entering and another for exiting the hot zone to the cold zone. Ensure the exit zone is also upwind and uphill
- Workers in the decontamination area must wear appropriate personal protective equipment (PPE). Provide a detergent-water solution (8 <pH <10.5) for decontamination, along with soft brushes to clean PPE. Use durable, labeled polyethylene bags for disposing of contaminated PPE.

Individual decontamination

Effective methods for individual decontamination include:

Decontamination of the first aid team

- Wash the PPE of the first aid team with soap and water using a soft brush, moving downward (head to toe), ensuring all areas, including clothing folds, are cleaned. Rinse with cold or lukewarm water until contaminants are removed
- Remove PPE by rolling it down from top to bottom (head to toe) without pulling it over the head. Remove the self-contained breathing apparatus last
- Place all PPE in durable, labeled polyethylene bags.

Decontamination of patient(s)

- Move patients to the decontamination corridor from the contaminated area

- Undress patients (at least to their underwear) and place their belongings in labeled polyethylene bags
- Wash the patient's skin with soap and cold or lukewarm water, taking care not to damage the skin. Cover any wounds before decontamination
- Protect patients from shock and heat loss by covering them
- Transfer patients to an area for emergency medical treatment.

Environmental decontamination

Guidelines for environmental or spill decontamination:

- Avoid contact with or walking through the spill; wear appropriate PPE if necessary
- Keep flammable materials (e.g., wood, paper, and oil) away from the spill. Use water spray to reduce or direct vapors but avoid contact between water and the spilled substance
- Prevent water from reaching the spill or leak source
- If safe, stop the leak and position leaking containers to release gas instead of liquid
- Block entry into waterways, sewers, basements, or confined spaces
- Isolate and ventilate the area until the gas dissipates.

Prevention Methods

To reduce risks from toxic substances:

1. Minimize exposure: Use safer alternatives where possible.
2. Environmental controls: Ensure adequate ventilation in enclosed spaces.
3. Personal protection: Proper PPE (respirators, gloves, and protective clothing) should be used when handling phosphine-releasing compounds.
4. Emergency preparedness: Training for healthcare providers and first responders on early recognition, decontamination, and treatment protocols for AlP poisoning.
5. Regulation and education: Restrict access to hazardous pesticides and ensure awareness among agricultural and industrial workers.

General precautions

The use of highly toxic substances should be minimized whenever possible. If exposure is unavoidable, strict adherence to safety guidelines is essential. Key precautions include:[9,26]

1. Ensure proper ventilation in enclosed spaces where phosphine gas may accumulate
2. Use appropriate protective equipment (e.g., gloves, respirators, and protective clothing) when handling AlP
3. Avoid direct contact – wear gloves when handling AlP tablets or pellets
4. Do not rely on odor detection – use gas monitoring devices to confirm phosphine levels
5. Prevent ingestion or inhalation – prohibit smoking, eating, or drinking in areas where AlP is used
6. Emergency preparedness – ensure respiratory protection is available in case of unexpected gas exposure
7. Immediate decontamination – wash exposed skin immediately and at the end of the work shift
8. Post warning signs – inform all personnel and bystanders of fumigation activities
9. Avoid residual exposure – ensure thorough ventilation after fumigation or grain turnover before re-entry.

Workplace controls and practices

Engineering controls are the most effective way to reduce exposure to hazardous substances like AlP. Whenever possible, a less toxic alternative should be used. Key safety measures include:[26] ventilation and containment (ensure local exhaust ventilation in enclosed spaces where phosphine gas may accumulate), isolation (restrict access to areas where AlP is used and implement enclosed handling processes to limit exposure), protective measures (while engineering controls are preferable, respiratory masks and PPE should be used in high-risk situations), and exposure assessment (employers must evaluate substance toxicity, emission levels in the workplace, risk of skin, or ocular exposure).

AlP should be transferred via automated systems when possible. Before entering a potentially contaminated area, gas concentration levels must be checked to prevent explosive conditions.

Good work practices for exposure reduction include:[26]

1. Change contaminated clothing immediately to prevent secondary exposure
2. Do not bring work clothes home – they should be cleaned by trained personnel
3. Provide emergency decontamination facilities, including eyewash stations and safety showers
4. Immediately wash exposed skin to remove any residual chemical
5. Strict hygiene policies: Prohibit eating, drinking, or smoking in areas where AlP is handled
6. Minimize dust exposure by using vacuum cleaners instead of dry sweeping.

Personal protective equipment

While workplace controls are the primary means of exposure prevention, PPE is essential in high-risk scenarios, such as outdoor applications, confined spaces, or emergency response situations.[26] The Occupational Safety and Health Administration (OSHA) recommends that employers assess exposure risks, select appropriate PPE, and ensure proper training on usage and limitations.[108]

Clothing

- Minimize skin contact with AlP by wearing protective gloves and clothing
- Ensure protective suits, gloves, footwear, and headgear are clean and readily available before handling PH_3.

Eye protection

- Use goggles or face shields when handling AlP powders or in high-risk exposure areas.

Respiratory protection

- Proper respiratory protection is essential in high-exposure scenarios to PH_3. Emergency responders should follow these guidelines
- Use NIOSH-/OSHA-approved respirators, selecting appropriate models based on exposure levels and emergency response needs[48]
- Air-supplied or self-contained breathing apparatus (SCBA) is required for high-risk situations
- Respiratory protection guidelines based on PH_3 exposure.[48]

Up to 3 ppm:

- Any air-supplied respirator.

Up to 7.5 ppm:

- Air-supplied respirator in continuous flow mode.

Up to 15 ppm:

- Chin-style air-purifying respirator with canisters for PH_3 and a full-face mask
- Self-contained breathing apparatus with a full-face mask
- Supplied air respirator with full-face protection.

Up to 50 ppm:

- Supplied-air respirator operating in pressure-demand or positive-pressure mode.

Emergency or immediately dangerous to life or health conditions:

- Self-contained breathing apparatus with a full-face mask operating in pressure-demand or positive-pressure mode
- Supplied air respirator with full-face protection and an auxiliary positive-pressure device.

Conclusion

Acute AlP poisoning remains a significant global health and environmental challenge. Advances in understanding its toxicity and clinical effects have improved management, but a specific antidote is still unavailable. Various treatment approaches require further validation. Preventive measures, such as restricting access to phosphide compounds, banning their use as pesticides, and educating healthcare professionals on early recognition and management of phosphide poisoning, are essential to mitigate human poisoning risks.

Authors' contributions

SÇK developed the concept, conducted the literature search, created the table, and wrote and reviewed the manuscript. CIY contributed to the concept, performed the literature search, edited and reviewed the manuscript, and provided appropriate advice on the content of the paper. Both authors contributed to writing the manuscript and read and approved the final version.

Conflicts of interest

None declared.

Funding

None.

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