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Update in the Management of Acute Aluminum Phosphide Poisoning: A Narrative Review

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Abstract

Background: Aluminum phosphide is frequently used as a rodenticide and to preserve grain. There is a significant risk of aluminum phosphide self-poisoning because of its simple accessibility and lack of control. The fatality rate from aluminum phosphide intoxication is significant, and there is currently no known counteragent. There aren't many studies on aluminum phosphide poisoning in Ethiopia, despite its high frequency and fatality rate. This narrative review's aim is to elaborate existing and new approaches to treating acute aluminum phosphide poisoning.

Method: Eligibility criteria were set prior to the search. A comprehensive literature review was performed searching PubMed, Cochrane library and Google Scholar. The major focuses were on recent studies, clinical trials, and systematic reviews with both qualitative and quantitative aspects.

Results: Patients receiving glucose insulin and potassium therapy (GIK) had consistently lower mortality rates and fewer case fatalities than those receiving basic supportive care alone, according to prospective observational studies and randomized controlled trials (RCTs). By improving myocardial carbohydrate absorption and metabolism, GIK infusion helps the strained heart overcome the energy imbalance caused by phosphine poisoning. In the evaluated research, oil-based gastric lavage with agents like coconut oil or paraffin oil showed better results than conventional water-based lavage (e.g., with potassium permanganate). Numerous case studies demonstrate effective patient recoveries, enhanced cardiac function, and neurological outcomes, demonstrating the critical role of extracorporeal membrane oxygenation (ECMO) plays in saving lives. Antioxidants such as N-acetylcysteine (NAC), however, showed less consistent results. When paired with additional medications like Coenzyme Q10 or Vitamin E, several studies suggested possible advantages including increased survival rates, shorter hospital stays, or better results. L-carnitine also showed a non-significant tendency toward decreased mortality. Other cutting-edge treatment options investigated for acute aluminum phosphide toxicity include Trimetazidine, liothyronine, and boric acid.

Conclusion: The fatality rate from aluminum phosphide intoxication is significant, and there is currently no known counteragent. While antioxidants like N-acetylcysteine have shown conflicting results and require more research, therapeutic approaches like glucose-insulin-potassium (GIK) infusion oil-based gastric lavage have demonstrated a considerable reduction in death rate.

Keywords: Aluminium Phosphide; Glucose-insulin-potassium; Coconut Oil

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Abbreviations and Acronyms

ALP: Aluminium Phosphide; ARDS: Acute Respiratory Distress Syndrome; ECG: Electrocardiogram; ECMO: Extracorporeal Membrane Oxygenation; EF: Ejection Fraction; FHRH: Felege Hiwot Referral Hospital; GI: Gastrointestinal; GIK: Glucose-insulin-potassium; ICU: Intensive Care Unit; NAC: N-acetylcysteine; PH_3: Phosphine; RCT: Randomized Clinical Trial; ROS: Reactive Oxygen Species; TAC: Total Antioxidant Capacity; VA ECMO: Veno-arterial Extracorporeal Membrane Oxygenation; VV ECMO: Veno-venous arterial Extracorporeal Membrane Oxygenation

Introduction

According to estimates from the World Health Organization, suicide claims the lives of about

720,000 individuals annually. Low- and middle-income nations account for 77% of all suicides [1]. One typical way that people commit suicide is by ingesting pesticides. Aluminum phosphide (ALP) is a solid fumigant that is widely used for grain preservation and is an inexpensive and widely accessible pesticide [2]. Pesticide poisoning causes more fatalities than other poisonings. Approximately 20% of pesticide cases require mechanical breathing, and almost half of those treated in intensive care units did not survive [3].

Under the brand names Celphos, Alphos, Synfume, Phostek, Phostoxin, Phosfume, and Quickphos, ALP is sold as dark-gray, 3-gram tablets. About 44% of the tablet is made up of inert ingredients (ammonium carbonate) to prevent tablet disintegration, with the remaining 56% being aluminum phosphide. One of the often utilized agents for self-poisoning which is easily accessible on the market and is not restricted in developing nations [4].

The deadly dose of ALP is around 500 mg [5]. Each tablet contains pure ALP (active component 56%) and ammonium carbamate/ carbonate/urea (inert ingredient 44%), which produce CO_2 and NH3 gasses to prevent the self-ignition of phosphine gas. High fatality rates up to 100% [6], 53.85 in Indian [7], 31.2% at Felege Hiwot Referral Hospital (FHRH) in Ethiopia [8] in cases of aluminum phosphide poisoning have been documented. The outcome may be affected by variables like the quantity taken, the tablet's expiration date, and the interval between ingestion and the start of first-aid procedures [6].

ALP poisoning does not have a specific antidote. The literature describes a wide range of strategies, including hemodynamic supportive care, hyperbaric oxygen therapy, or stomach irrigation with various substances to decrease absorption of harmful molecules, and preventing oxidative damage using antioxidants. However, all these approaches are non-specific and have limited efficacy [9].

In Ethiopia, aluminum phosphide is used as a fumigant to keep pests at bay and preserve grain. There is a significant risk of self-poisoning with aluminum phosphide because of its simple accessibility and lack of control. Serious multi-organ failure results from the toxicological effects of phosphine gas emission. ALP has no known counteragent. ALP use for self-suicide has increased; the aim of this review is to narrate the recent advancement with good outcome in management of acute aluminum phosphide poisoning, April 2025.

Mechanisms of ALP toxicity

Phosphine gas ($PH_{3)}$ is produced from contact between ALP with water or hydrochloric acid in the gastrointestinal (GI) tract. 3 g tablet release about 1 g of PH_3 [10]. The toxic effects of the AlP are due to deadly phosphine gas liberated [11, 12]. However, the full and precise mechanisms of intoxication remain unclear [13].

Phosphine gas prevents the synthesis of proteins and enzymes in cells. Electron electron transport chain disruption of disrupting the electron transport chain through blockage of cytochrome c oxidase non-competitively, oxidative phosphorylation and cellular respiration are inhibited, which results in an excess of peroxide radicals and lipid peroxidation. In addition it leads to suppression of Glutathione,superoxide dismutase and catalase cause malfunction in cell membranes [14, 15]. Blockage of cytochrome c oxidase resulting in low ATP production followed by metabolic shutdown [10]. Inflammation is also implicated as a contributing factor to AlP-induced cardiotoxicity and Phosphine release is also linked to the activation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase (NOX) enzymes, which has been found to be a major source of intracellular ROS [15].

PH3 react with hemoglobin, increases in Heinz bodies (denatured hemoglobin) and hemichrome production are also noted, which lowers heme capacity. It is well known that under stress, the heart switches from b-oxidation to glycolysis for energy metabolism. This situation is made worse by ALP poisoning, which reduces glucose uptake and metabolism within cells [16]. ALP changes the fluidity of the mitochondrial membrane, which causes the outer membrane to rupture and the mitochondria to enlarge. This causes the release of pro-apoptotic substances, such as cytochrome c, which in turn causes apoptosis [15]. Imbalanced redox status leads to lipid peroxidation, which causes damage to many cellular structures and increases cellular death [17].

There is no large-scale, or systematically detailed research specifically focused on post-mortem findings in ALP poisoning but Organs with higher oxygen needs, such as the heart, lung, kidney, and liver, are more vulnerable to PH_3-induced damage involving the formation of oxygen free radicals, as demonstrated by case study on post-mortem histopathological investigations [21]. When the body opened, the phosphine gas smelled like fish or garlic. There were petechial hemorrhages clogging every single organ. Alveolar rupture, gastrointestinal mucosal congestion, and petechial hemorrhages on the liver and brain surface were all seen in the edematous and crowded lungs [22].

Clinical manifestations of AIP toxicity

The symptoms are immediate, vague, and contingent on the dose, entrance point, and amount of time after poisoning. Patients frequently experience dyspnea and airway discomfort following inhalation exposure. Dizziness, headache, nausea, vomiting, diarrhea, ataxia, numbness, paresthesia, tremor, muscle weakness, diplopia, jaundice, and tightness in the chest are possible additional symptoms. Acute respiratory distress syndrome (ARDS), cardiac failure, cardiac arrhythmias, convulsions, coma, and late manifestations of hepatotoxicity and nephrotoxicity are all possible outcomes of severe inhalation toxicity [18]. Toxic features typically appear a few minutes after intake. Common clinical symptoms of moderate poisoning include headache, nausea, diarrhea, and abdominal pain or discomfort. These individuals typically recover. However, the signs and symptoms of the respiratory, neurological, cardiovascular, and gastrointestinal systems first arise in moderate to severe ingestional poisoning. Later, disseminated intravascular coagulation and indicators of hepatic and renal failure may also occur [19].

ALP poisoning's cardiovascular complications include palpitations, thready pulses, acidosis, and severe hypotension. A number of ECG alterations, including PR, QRS interval, and ST segment elevation/depression are noted. There have been reports of prolongation, total cardiac block, ectopic pacemaking, fibrillation, and finally unresponsive shock. Until cerebral anoxia due to shock causes hemorrhage, severe renal failure, disseminated intravascular coagulation, and arrhythmias, patients maintain mental clarity [20].

Diagnosis of ALP toxicity

ALP-compatible clinical suspicion or history (self-report or by attendants) are typically used to diagnose ALP poisoning. The pills are sometimes called "rice tablets" in some contexts. If there is a history of rice tablet consumption, it should be distinguished from other kinds of rice tablets that are composed of herbal items [23]. By

using silver nitrate to detect phosphine in exhaled air or in stomach aspirate, the diagnosis of ALP consumption can be verified. In this test, diluted stomach contents are heated in a flask, and a strip of filter paper impregnated with freshly made silver nitrate is exposed close to the flask's opening. The paper turns black when phosphine is present. Another way to do the test is to have the patient breathe into a mask made of silver nitrate paper. The breath test is unreliable, though. Because phosphine can change into phosphorus pentoxide, people receiving oxygen may experience a false negative result. If hydrogen sulfide is present in the air, a false-positive result could happen. Phosphine detector tubes are another tool for detecting phosphine in breath. Commercially available detection tubes and bulbs are used for spot sampling of phosphine in air. Gas chromatography, on the other hand, is the most sensitive and specific method for detecting traces of phosphine in air [24].

Current management of ALP poisoning

In treatments of ALP early detection and gastrointestinal decontamination are essential. To lessen the absorption of the toxin, this often involves stomach lavage with potassium permanganate ($KMnO_4$), activated charcoal, and coconut oil. After early stabilization, ongoing supportive and symptomatic care is essential. In more severe cases, metabolic acidosis is treated with sodium bicarbonate or hemodialysis, while cardiac arrhythmias are treated with magnesium sulfate ($MgSO_4$). In shock, vasoactive drugs and, if required, advanced cardiac support, like pacemakers or intra-aortic balloon pumps, are required. As needed, respiratory support should be given, and it's critical to keep blood glucose and electrolyte balance. Acetyl-L-carnitine, vasopressin, and steroids are a few possible treatments were used and reported previously [25]. Melatonin, sodium selenite, vitamin E, N-acetylcysteine (NAC), vitamin C, triiodothyronine, liothyronine, milrinone, Laurus nobilis L., 6-aminonicotinamide and boric acid are additional suggestive agents that may use for AlPpoisoning [26]. However, the treatments have remained controversial, and more studies are required to confirm their efficacy [27].

Methods and Materials

Inclusion criteria

Eligibility criteria were set prior to the search. All studies that report the percentage mortality outcome of therapy in patients treated for ALP poisoning. Research articles on human subjects. Only study conducted in English language from 2015 to April 14, 2025 included.

Exclusion criteria

A literature which does not have a full access freely and studies with insufficient, not provide relevant or unclear information and didn't show mortality outcome while treated for ALP poisoning are exclude.

Search strategy and Data extraction

Search terms: (aluminum phosphide poisoning OR aluminum phosphide toxicity OR aluminum phosphide ingestion OR rice tablet OR Metallophosphide Poisonin OR Fumigant OR "Rodenticide" OR "Rat Poisoning" OR "Insecticide") AND (management OR therapy OR treatment) were used to conduct a literature review across PubMed, the Cochrane Library, and Google Scholar in order to find published studies published before from 2015 to April 14, 2025.

Studies that could help us achieve our aim are chosen those that fit our inclusion criteria after reviewing the abstract. Then, researches imported all of the extracted documents' full citations into Mendeley and looked closely at the entire study to find the data required for the final data synthesis to address the objectives.

Major Pharmacological and Toxicological Innovations in Management of ALP Poisoning

Glucose-Insulin-Potassium Infusion

By administering glucose, insulin, and potassium, hyperinsulinemia euglycemia (HIE) promotes the myocardial absorption of carbohydrates, the heart's preferred fuel substrate during stressful situations. Correcting hypoinsulinemia caused by beta-blockers or calcium antagonists is thought to be the process, which improves cell carbohydrate metabolism, raises peripheral vascular resistance and cardiac contractility, and corrects acidosis. Bradycardia and conduction block are not anticipated to improve with the treatment. In 2008, HIE was first proposed as a potential therapy for ALP toxicity. Despite having only been studied on five individuals at the time of publication, this medication is currently advised for ALP poisoning [25]. Similar metabolic abnormalities are also seen in acute aluminum phosphide toxicity. Insulin resistance and hyperglycemia are caused by phosphate-induced free radical production and oxidative stress, and hyperglycemia is a poor prognostic factor in aluminum phosphide poisoning [27]. Table 1 shows that treatment with GIK infusion was linked to a lower case fatality rate than supportive care alone.

Antioxidants

Researches looked at the protective benefits of different antioxidants, such as N-acetylcysteine, L carnitine, vitamin E, and vitamin C, after ALP poisoning due to the rise in oxidative stress [28]. is N-acetylcysteine (NAC) is popular pharmacological antioxidant [29]. It restores glutathione storage by providing cysteine, an essential precursor in the manufacture of glutathione. NAC also binds to the toxic metabolites and scavenges free radicals. It also enhances oxygen delivery to tissues, changes microvascular tone, and promotes mitochondrial ATP generation to improve blood flow and oxygen delivery to the liver and other vital organs [30].

The results for antioxidants such as N-acetylcysteine (NAC) were less consistent. When paired with additional medications like Coenzyme Q10 or Vitamin E, several studies suggested possible advantages including increased survival rates, shorter hospital stays, or better results. Similarly, as seen in table 2, L-carnitine exhibited a non-significant trend towards decreased mortality.

Oil based gastric lavage

During ALP poisoning the stomach is cleaned with sorbitol, liquid paraffin, coconut oil, potassium permanganate, activated charcoal, and antacids. Supportive therapy involves the use of vasoactive drugs, magnesium sulphate ($MgSO_4$), soda bicarbonate, and ventilator support [31]. Oil-based gastric lavage, using agents like coconut oil or paraffin oil, demonstrated positive outcomes compared to traditional water-based lavage (e.g., with potassium permanganate). Coconut oil has two benefits: first, it forms a barrier on mucosal membranes to block phosphine absorption; second, it dilutes HCL, which prevents phosphine from breaking down from pellets [32].

Extracorporeal membrane oxygenation

Extracorporeal membrane oxygenation (ECMO) is a life-sustaining technique that oxygenates and eliminates carbon dioxide

Table 1: The role of Glucose-insulin-potassium infusion in ALP poisoned patients' outcomes from Studies Reviewed 2015 to April 2025.

Author	Publication year	Study design	Sample size	Outcome of therapy
Pannu AK, et al.	2020	Prospective randomized controlled trial	60 patients	In comparison to supportive care alone, GIK infusion treatment was linked to a considerably decreased case fatality rate (46.7% *versus* 73.3%; p-value 0.03).
Hassani an-Moghadda, et al.	2016	Prospective intervention al study	88 patients	Better results were linked to GIK (72.7% *vs*. 50% mortality; P = 0.03).
Ullah, et al.	2024	Cross- sectional study	15 patients	The mortality rate dropped by more than 53% as a result of GIK therapy.
Mazaheri tehrani, et al.	2023	RCT	76 patients	Compared to the control group, the group's mortality rate was 26% lower (p-value: 0.058). In contrast to supportive therapy, the case group receives GIK administration together with vitamin E and N-acetyl cysteine.
Adel, et al.	2023	RCT	108 patients	A considerable reduction in mortality was associated with insulin-euglycemia therapy (64.8 and 96.3 percent, respectively; P value <0.001).

Table 2: The role of Antioxidants (N-acetylcysteine (NAC), L-carnitine and vitamin E) in ALP poisoned patient outcomes from studies reviewed 2015 to April 2025.

Author	Publication year	Study design	Sample size (patients)	Outcome of therapy
AbdEl-khalek, et al.	2025	RCT	96	The mortality rate and the need for mechanical ventilation between control group, the NAC group were comparable (P=0.601 and 0.505, respectively) but The NAC group experienced a considerably shorter hospital stay
Emam, et al.	2020	RCT	60	(80%) survived in the NAC-treated group and (56.7%) survived in non NAC-treated group
Goharbari, et al.	2018	Prospective-retrospective	60	Patients treated with Coenzyme Q10 and N- acetylcysteine (NAC) 73.3 percent survival rate, while Those who used the usual protocol had a 50 percent survival rate.
Bhalla, Jyothinath, et al.	2017	Prospective intervention	50	NAC administration did not confer any survival benefit. Overall mortality in the study population was 88%. In NAC group, the unfavorable outcome was seen in 87.5% Whereas 88.5% placebo group.
El- Ebiary, et al.	2017	RCT	30	Group A (NAC treatment) had a 66.7% survival rate, while Group B (no NAC) had a 20% survival rate.
Taghad dosinejad, et al.	2016	RCT	63	No significant difference in mortality between case (treated with NAC) and control groups made.
Halvaei, et al.	2017	RCT	36 (divided into 2 groups)	Group receiving vitamin E in addition to conventional therapy had a considerably decreased death rate (15% *vs*. 50%, P = 0.02)
Elgazzar, Keshk, et al.	2019	RCT	50	Deaths among patients receiving L-carnitine therapy (group B) was, however, non-significantly lower than that of group A (60% and 80%, respectively, p >0.05).

Table 3: Shows the role of oil-based gastric lavage (coconut oil, paraffin oil) in the outcomes of ALP-poisoning patients from Studies Reviewed 2015 to April 2025.

Author	Publication year	Study design	Sample size (patients)	Outcome of therapy
Dayananda, et al.	2018	RCT	50	Use of coconut oil reduce (60%) the mortality rate in Group A (30%) was lower (p>0.03)
Helal, et al.	2022	RCT	62	Use of paraffin oil no significance mortality difference is observed but severity of ALP poisoning is decreased significantly
Darwish, et al.	2020	RCT	30 with km no4, 30 with paraffin, 30 paraffin with Co Q10	Mortality rates for KMNO4 (water-based solution) 73.33%, paraffin oil (oily solution) 36.67% and paraffin oil (oily solution) with Coenzyme Q10 were % and 23.33%, respectively.

from the blood in cases of severe cardiac or respiratory failure. Veno-arterial (VA) ECMO supports respiratory and/or cardiac function, while veno-venous (VV) ECMO promotes respiratory function. These are the two main types of ECMO [33]. In circumstances of ALP poisoning with severe cardiogenic shock and significant mortality, research have demonstrated that ECMO dramatically increases survival; as seen in table 4.

Other Pharmacological and Toxicological Innovations in Management of ALP Poisoning

Liothyronine

The main reason thyroid hormones are utilized to treat and manage ALP poisoning is because of their cytoprotective qualities. The toxicity of ALP may even be causing tissue hypothyroidism. There are two ways that thyroid hormones impact mitochondria. Thyroid hormones also have certain antiapoptotic effects on cells, and one occurs rapidly, within minutes or hours after hormone therapy [34]. Oral liothyronine significantly enhanced systolic blood pressure, arterial PH, and total thiol molecules while reducing lipid peroxidation, boosting catalase activity, and maintaining total antioxidant capacity [21]. A randomized, non-blind, case-controlled clinical experiment was conducted at Baharloo Hospital in Tehran, Iran, where 24 acute ALP-poisoned patients received 50 µg of lithyronine following gastric lavage. In the case group, the death rate decreased by 25%, while in the control group, it decreased by 33.3% [34].

Trimetazidine

An anti-ischemic medication called trimetazidine (Vastared) 35 mg retard tablet is demonstrating promise in the treatment of

Table 4: The role of ECMO ALP poisoned patients' outcomes from Studies Reviewed 2015 to April 2025.

Author	Publication year	Study design	Sample size (patients)	Outcome of therapy
Merin, et al.	2015	Case report (family)	4 children	Two children recovered without any brain impairment following a successful ECMO deployment; in cases of deadly ALP poisoning, ECMO provided hemodynamic support.
Shad, et al.	2019	retrospective study	107	In-hospital mortality decreased from 84.4% to 40% with ECMO added to standard care (OR: 0.47; 95% CI 0.31–0.73).
Jain, et al.	2020	Case report	1 patient	After five days on ECMO, the patient's EF increased from less than 5% to 55%, indicating a notable cardiac improvement.
Rao, et al.	2020	Case report	1 patient	ECMO, hemodialysis, magnesium, trimetazidine, N-acetyl cysteine, inotropes, and blood products were all used to successfully sustain him. Many young lives can be saved by early detection and ECMO implementation, which has no neurological aftereffects.
Kumar, et al.	2021	Case report	1 patient	She experienced multiorgan failure after taking 5g of a medication, and VA-ECMO was able to properly manage her delayed presentation (>24 hours).
Sharma, et al,	2023	Case report	1 patient	Survival and an undamaged neurological outcome were the outcomes of prompt VA-ECMO commencement.
Moshiri, et al.	2024	Systematic review and meta-analysis	198 reported cases underwent ECMO. 24 ECMO cases (from 17 case reports, etc.)	The mortality rate for subjects treated with ECMO was 23% [95% (CI): 7%–39%]; this was significantly lower than the mortality rate for cases treated conventionally, which was 60% [95% CI: 39%–63%]; P < 0.001).

ALP poisoning, particularly in the control of related cardiovascular symptoms [35]. Trimetazidine prevents further damage to the myocardium by enhancing glucose oxidation through the inhibition of beta-oxidation of fatty acids. Magnesium and trimetazidine aid in the suppression of arrhythmia [36].

Boric acid

Because boric acid is a lewis acid, it efficiently "traps" phosphine gas, which behaves like a Lewis base. This trapping response may neutralize phosphorus, reducing its detrimental effects on the body. Based on this mechanism, boric acid is being evaluated as a therapeutic drug in situations of ALP poisoning. Boric acid shows promise as an antidote for ALP poisoning, and rats intoxicated with ALP and treated with boric acid show a significant improvement in histopathological changes, a significant improvement in biochemical parameters like liver and cardiac markers (SGOT, SGPT, and cardiac troponin), and a significant increase in survival time [37].

Phosphine and boric acid combine to generate a gaseous adduct. Activated charcoal was found to drastically reduce the volume of released PH_3 gas, whereas boric acid was shown to dramatically reduce the rate of gas evolution. A lethal volume of PH_3 gas was produced in 6.5–21 minutes [38].

Discussion

GIK infusion therapy is a promising treatment option since it can counteract the negative effects of ALP poisoning by improving cardiac function through increased uptake of carbohydrates. In Tanta University Poison Control Center carried out two-year retrospective cohort research on ALP poisoning victims. The survival of those with a Glu-K ratio > 37.07 was considerably shorter (0.38 days) than that of those with a Glu-K ratio < 37.07 (3 days), indicating a significant increase in mortality among those with a high Glu-K ratio [39]. research conducted Pakistan detect 73% mortality while after addition of GIK protocol mortality is reduced to 20%. Consequently, it was shown that GIK therapy led to a more than 53% reduction in mortality [40]. Study conducted in north India found GIK infusion treatment was associated with a significantly lower in-hospital case fatality rate (46.7% *versus* 73.3%; p-value 0.03) when compared to supportive care alone. There was a 26.6% decrease in mortality [27].

Prospective interventional trial conducted in Tehran. GIK was associated with superior outcomes (72.7% *vs*. 50% mortality; P = 0.03). 95% CI = [1.004, 1.087]; odds ratio [1.045]. After starting GIK, the chance of death dropped by 4.5% every hour [29]. Insulin-euglycemia therapy was associated with a significant reduction in mortality (64.8 percent and 96.3 percent, respectively; P value <0.001) when compared to the control group [25]. Combination of Hyper Insulin Euglycemia with antioxidants (vitamin E and N-acetyl cysteine) therapy carried in Iran on a total of seventy-six toxic individuals were recruited and divided into two groups. Compared to the control group, the group's mortality rate was 26% lower (p-value: 0.058) [41].

Randomized control trial on role of Antioxidants (NAC) on the treatment of acute ALP intoxication, 60 intoxicated individuals participated in, with the median [interquartile range (IQR)] being significantly lower 4.6 [1.9–10.6] *vs*. 6.8 [3.5–17.4]), (P<0.05*): 24 (80%) of the poisoned patients survived in the NAC-treated group, while 17 (56.7%) survived in the NAC-non-treated group [42]. However, study which evaluate 63 individuals poisoned by ALP, a case-control study was carried out. For 20 hours, the case group was infused with 300 mg/kg of NAC. NAC prevented the ALP patients' heart rates in the case group from wildly varying. 17 patients died as a result of the outcomes (10 in the case group and 7 in the control group). There was no discernible difference [43].

Pilot research for a prospective intervention in India. Acute ALP poisoning had an 88% fatality rate. 87.5% of the NAC group and 88.5% of the placebo group experienced adverse effects. The two groups' results did not differ in a way that was statistically significant [44]. Two equal groups of thirty acute ALP-intoxicated patients were randomly assigned. There were notable differences in mortality between groups A and B. Group B had a 20% survival rate after not receiving NAC medication, but Group A had a 66.7% survival rate after receiving NAC treatment [45]. 96 individuals with acute ALP poisoning were divided into two groups for a RCT the standard care group (control group) and the standard care plus NAC infusion group (NAC group).The mortality rate and the need for mechanical ventilation were comparable in both groups (P = 0.505 and 0.601, respectively) [28].

In the RCT, 36 people had acute ALP poisoning. The treatment group that received vitamin E in addition to conventional therapy had a significantly lower death rate (15% *vs*. 50%, P = 0.02) than the

control group that received only conventional care [46]. L-carnitine was used as an antioxidant to treat severe acute ALP poisoning in a randomized controlled clinical trial. Two equal groups, A and B, were randomly selected from among fifty patients who had acute ALP poisoning. Group B received L-carnitine therapy in addition to the usual course of treatment for Group A. Additionally, compared to group A. The mortality rate among L-carnitine therapy patients (group B) was not statistically different from that of group A (60% and 80%, respectively, p >0.05) [47].

N-acetylcysteine (NAC) and coenzyme Q10 have antioxidant and cardioprotective qualities. Sixty individuals with acute ALP poisoning were the subjects of a prospective-retrospective (hybrid design) study. A group which received (new protocol) received the NAC and CoQ10 regimen. Compared to patients who followed the standard regimen, those who employed the new technique had a 73.3 percent survival rate compared to 50 % who received the standard protocol [48].

Four antioxidants N-acetyl cysteine (NAC), L-carnitine, vitamin E, and co-enzyme Q10 (CoQ10) were employed in a meta-analysis of ten publications, outcome of treatment using antioxidant therapy in conjunction with traditional supporting measures. According to subgroup analysis, antioxidants significantly lower the mortality of acute ALP poisoning by about three times (OR = 2.684, 95% CI: 1.764–4.083; P <.001), NAC significantly lowers death by nearly three times (OR = 2.752, 95% CI: 1.580–4.792; P <.001), and vitamin E significantly lowers mortality by nearly six times (OR = 5.667, 95% CI: 1.178–27.254; P =.03) [49].

The impact of oil-based gastric lavage on the course of acute ALP poisoning has been the subject of systematic reviews and meta-analyses. mortality (RR = 0.59 [95% CI: 0.45, 0.76], p <.001), when paraffin oil was used. Paraffin oil significantly extended survival time (SMD = 0.72 [95% CI: 0.32, 1.13], p <.001) and longer survival time (SMD = 0.83 [95% CI: 0.06, 1.59], p =.03) were both substantially correlated with coconut oil. Regarding the rate of intubations and survival duration, coconut oil demonstrated certain advantages. It is advised to use paraffin oil for decontamination [50].

RCT in 62 ALP-intoxicated people, 31 in the control group's stomach was decontaminated using saline and sodium bicarbonate while the intervention group's stomach was decontaminated using paraffin oil and sodium bicarbonate 8.4%. The intervention group's mortality rate was non-significantly lower than that of the control group [50]. The prognosis of patients who received liquid paraffin oil as an alternative method of GIT cleaning significantly improved. Thirty patients each group was treated with various stomach decontamination solutions. The mortality rates for paraffin oil (oily solution) with Coenzyme Q10 were 23.33%, KMNO4 (water-based solution) was 73.33%, and paraffin oil (oily solution) was 36.67%. When CoQ10 is added to paraffin oil, the acute ALP poisoning outcome is considerably improved. Therefore, as a pre-hospital treatment or immediately following hospitalization, rapid oral absorption of any accessible oil could greatly improve the outcome [51].

In cases of severe aluminum phosphide poisoning, which are marked by cardiogenic shock and high mortality, extracorporeal membrane oxygenation (ECMO) can save lives. A systematic review and meta-analysis revealed that patients receiving ECMO had a significantly lower mortality rate of 23% compared to 60% in patients receiving conventional treatment (P <0.001) [52] and another retrospective study showed that adding ECMO to conventional treatment decreased in-hospital mortality from 84.4% to 40% [53].

Through pathways including mitochondrial support, antioxidant effects like lowering lipid peroxidation, and anti-apoptotic actions, the thyroid hormone liothyronine is being studied for its cytoprotective qualities, which could potentially offset toxin-induced tissue hypothyroidism [34, 54]. By improving cardiac energy metabolism through increased glucose oxidation and possibly helping to reduce arrhythmias, the anti-ischemic medication trimetazidine shows promise [36].

Conclusion and Future Direction

In conclusion, there is no known antidote for aluminum phosphide intoxication, which has a significant fatality rate. IV fluids, sodium bicarbonate, vasopressors, sodium bicarbonate, and steroids are all part of aggressive early resuscitation and supportive therapy for symptoms. Trimetazidine, ECMO and liothyronine, along with the early administration of IV magnesium sulphate, could be recommended as a crucial treatment in addition to the traditional supportive therapy used for acute aluminum phosphide intoxication to improve patient survival. While antioxidants like N-acetylcysteine have shown conflicting results and require more research, therapeutic approaches like glucose-insulin-potassium (GIK) infusion oil-based gastric lavage have demonstrated a considerable reduction in death rate.Given the high fatality rate of ALP phosphide, a combination of therapies must be administered to lower the death rate.

Using this new insight and expanding the research mainly focus on the way to absorb phosphine gas which is released from ALP tablets necessary to revise the management procedure in order to lower the number of fatalities from ALP poisoning.

Strength and Limitations

Strength

- This review's therapies point to possible improvements in management techniques.
- This review focus on mechanism of reducing mortality associated with ALP poisoning.
- The review narrates promising therapies like GIK infusion, oil based gastric lavage, ECMO therapies and outcomes associated with combination of therapies.

Limitations

- The scope is further limited because it is a narrative study and introduces selection bias research without complete open access.
- The primary focus of this review is on human studies, and the death rate is used to illustrate the results.
- The findings lack the statistical rigor of a systematic review and are primarily descriptive and qualitative.
- Due to the limited availability of high-quality RCT data. Variety of study designs, such as prospective studies, observational data, and case control study designs, in addition to RCTs is gathered and presented in this review due to unavailability adequate data from RCT.

Declarations

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Authors declare that there is no competing interest.

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MA and KDA have conducted the review and wrote the original draft and involved in conceptualization, and designing the methodology. KDA have supervised the review. authors have approved the final version and agreed to be accountable for the research presented.

References

1. Suicide [Internet]. [cited 2025 Sep 9]. Available from: https://www.who.int/news-room/fact-sheets/detail/suicide
2. Khosla SN, Handa R, Khosla P. Aluminium Phosphide Poisoning. Trop Doct [Internet]. 1992 [cited 2025 Sep 9]; 22(4): 155–7. Available from: https://pubmed.ncbi.nlm.nih.gov/1440881/
3. Dorooshi G, Samsamshariat S, Gheshlaghi F, Zoofaghari S, Hasanzadeh A, Abbasi S, et al. Comparing Sequential Organ Failure Assessment Score, Acute Physiology and Chronic Health Evaluation II, Modified Acute Physiology and Chronic Health Evaluation II, Simplified Acute Physiology Score II and Poisoning Severity Score for Outcome Prediction of Pesticide Poisoned Patients Admitted to the Intensive Care Unit. J Res Pharm Pract . 2023 Apr; 12(2): 49–57.
4. Gurjar M, Baronia AK, Azim A, Sharma K. Managing aluminum phosphide poisonings. J Emerg Trauma Shock. 2011 Jul [cited 2025 Sep 9]; 4(3): 378. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3162709/
5. Karimani A, Mohammadpour AH, Zirak MR, Rezaee R, Megarbane B, Tsatsakis A, et al. Antidotes for aluminum phosphide poisoning – An update. Toxicol Rep .2018 Jan 1 [cited 2025 Sep 9]; 5: 1053. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC6214862/
6. Nada MAA, Elfeky AKEE, Darweesh HAM, Mohamed FKI, Sharif LS, Kandil FS, et al. Correction to: The effect of educational programs on parents' knowledge, behavior, and practices regarding aluminum phosphide poisoning and its first-aid measures (BMC Pediatrics, (2024), 24, 1, (846), 10.1186/s12887-024-05231-x). BMC Pediatr. 2025 Dec 1 [cited 2025 Sep 9]; 25(1): 1–1. Available from: https://bmcpediatr.biomedcentral.com/articles/10.1186/s12887-025-05404-2
7. Aggarwal N, Sawlani KK, Chaudhary SC, Usman K, Dandu H, Atam V, et al. Study of pattern and outcome of acute poisoning cases at tertiary care hospital in North India. J Family Med Prim Care . 2023 Sep [cited 2025 Sep 10]; 12(9): 2047. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10657109/
8. 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 Emerg Med. 2021 [cited 2025 Sep 10]; 13: 239–48. Available from: https://pubmed.ncbi.nlm.nih.gov/34163261/
9. Çakın Ö, Tazegul G, Gümüş A, Cengiz M, Ramazanoğlu A. Incidental Aluminum Phosphide Poisoning: Case Report and Current Management. Folia Med (Plovdiv) [Internet]. 2018 Sep 1 [cited 2025 Sep 9]; 60(3): 464–7. Available from: https://pubmed.ncbi.nlm.nih.gov/30355847/
10. Anand R, Binukumar BK, Gill KD. Aluminum phosphide poisoning: An unsolved riddle. Journal of Applied Toxicology [Internet]. 2011 Aug 1 [cited 2025 Sep 9]; 31(6): 499–505. Available from: https://doi.org/10.1002/jat.1692
11. Moghadamnia AA. An update on toxicology of aluminum phosphide. DARU Journal of Pharmaceutical Sciences [Internet]. 2012 [cited 2025 Sep 9]; 20(1): 25. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3555759/
12. Sobh ZK, Abd-Elhameed A. The therapeutic benefit of antioxidants on the outcome of acute aluminum phosphide poisoning: a systemic review and meta-analysis. Toxicol Res (Camb) [Internet]. 2023 Jun 1 [cited 2025 Sep 9]; 12(3): 345. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10311134/
13. Haghi Aminjan H, Abtahi SR, Hazrati E, Chamanara M, Jalili M, Paknejad B. Targeting of oxidative stress and inflammation through ROS/NF-kappaB pathway in phosphine-induced hepatotoxicity mitigation. Life Sci [Internet]. 2019 Sep 1 [cited 2025 Sep 9]; 232: 116607. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0024320519305338?via%3Dihub
14. Hashemi-Domeneh B, Zamani N, Hassanian-Moghaddam H, Rahimi M, Shadnia S, Erfantalab P, et al. A review of aluminium phosphide poisoning and a flowchart to treat it. Arh Hig Rada Toksikol. 2016 Sep 1; 67(3): 183–93.
15. Mostafa EMA, Atta R, Maher SA, El-Kherbetawy MK, Ameen AM. Quercetin and its potential therapeutic effects on aluminum phosphide-induced cardiotoxicity in rats: Role of NOX4, FOXO1, ERK1/2, and NF-κB. Tissue Cell [Internet]. 2024 Dec 1 [cited 2025 Sep 9]; 91: 102622. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0040816624003239?via%3Dihub
16. Potter WT, Rong S, Griffith J, White J, Garry VF. Phosphine-mediated Heinz body formation and hemoglobin oxidation in human erythrocytes. Toxicol Lett [Internet]. 1991 Jun 1 [cited 2025 Sep 9]; 57(1): 37–45. Available from: https://www.sciencedirect.com/science/article/abs/pii/037842749190117O?via%3Dihub
17. Aghebat-Bekheir S, Abdollahi M. Discovering the most impactful treatments for aluminum phosphide cardiotoxicity gleaned from systematic review of animal studies. Hum Exp Toxicol [Internet]. 2024 Jan 1 [cited 2025 Sep 9]; 43. Available from: https://scholar.google.com/scholar_url?url=https://journals.sagepub.com/doi/pdf/10.1177/09603271241290922&hl=en&sa=T&oi=ucasa&ct=ufr&ei=8Ku_aNKGOsfOieoP8bzKoAI&scisig=AAZF9b9DioXEF9tPxTBLQ0ir__tu
18. Sudakin DL. Occupational exposure to aluminium phosphide and phosphine gas? A suspected case report and review of the literature. Hum Exp Toxicol [Internet]. 2005 Jan [cited 2025 Sep 10]; 24(1): 27–33. Available from: https://pubmed.ncbi.nlm.nih.gov/15727053/
19. Anbalagan LC, Arora N, Pannu AK. Management of Acute Aluminum Phosphide Poisoning: Has Anything Changed? Drug Metab Lett. 2021 Aug 16; 14(2): 106–16.
20. Singh Y, Joshi SC, Satyawali V, Gupta A. Acute aluminium phosphide poisoning, what is new? The Egyptian Journal of Internal Medicine 2014 26:3 [Internet]. 2015 Aug 11 [cited 2025 Sep 10]; 26(3): 99–103. Available from: https://ejim.springeropen.com/articles/10.4103/1110-7782.145298
21. Sinha US, Kapoor AK, Singh AK, Gupta A, Mehrotra R. Histopathological changes in cases of aluminium phosphide poisoning. Indian J PatholMicrobiol [Internet]. 2005 Apr 1 [cited 2025 Sep 9]; 48(2): 177–80. Available from: https://europepmc.org/article/MED/16758658
22. Meena MC, Mittal S, Rani Y. Fatal aluminium phosphide poisoning. InterdiscipToxicol [Internet]. 2015 Jun 1 [cited 2025 Sep 10]; 8(2): 65.

Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC4961899/

23. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. Arh Hig Rada Toksikol [Internet]. 2012 Mar 1 [cited 2025 Sep 10]; 63(1): 61–73. Available from: https://pubmed.ncbi.nlm.nih.gov/22450207/

24. Singh S, Bhalla A. Aluminum phosphide poisoning. Journal of Mahatma Gandhi Institute of Medical Sciences [Internet]. 2015 [cited 2025 Sep 10]; 20(1): 15. Available from: https://journals.lww.com/mgim/fulltext/2015/20010/aluminum_phosphide_poisoning.4.aspx

25. Hashemi-Domeneh B, Zamani N, Hassanian-Moghaddam H, Rahimi M, Shadnia S, Erfantalab P, et al. A review of aluminium phosphide poisoning and a flowchart to treat it. Arh Hig Rada Toksikol. 2016 Sep 1; 67(3): 183–93.

26. Karimani A, Mohammadpour AH, Zirak MR, Rezaee R, Megarbane B, Tsatsakis A, et al. Antidotes for aluminum phosphide poisoning – An update. Toxicol Rep [Internet]. 2018 Jan 1 [cited 2025 Sep 10]; 5: 1053. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC6214862/

27. Pannu AK, Bhalla A, Gantala J, Sharma N, Kumar S, Dhibar DP. Glucose-insulin-potassium infusion for the treatment of acute aluminum phosphide poisoning: an open-label pilot study. Clin Toxicol (Phila) [Internet]. 2020 Oct 2 [cited 2025 Sep 10]; 58(10): 1004–9. Available from: https://pubmed.ncbi.nlm.nih.gov/32129101/

28. Shaker HO, Rageh OES, Alnajar M, Alshamaly NF, Abdelmaged WA, Abd-ElGawad M. Efficacy of intravenous N acetylcysteine as an adjuvant therapy in the treatment of acute aluminum phosphide Poisoning: a systematic review and meta-analysis. BMC PharmacolToxicol [Internet]. 2023 Dec 1 [cited 2025 Sep 12]; 24(1). Available from: https://pubmed.ncbi.nlm.nih.gov/37924139/

29. Aldini G, Altomare A, Baron G, Vistoli G, Carini M, Borsani L, et al. N-Acetylcysteine as an antioxidant and disulphide breaking agent: the reasons why. Free Radic Res [Internet]. 2018 Jul 3 [cited 2025 Sep 12]; 52(7): 751–62. Available from: https://pubmed.ncbi.nlm.nih.gov/29742938/

30. Atkuri KR, Mantovani JJ, Herzenberg LA, Herzenberg LA. N-Acetylcysteine—a safe antidote for cysteine/glutathione deficiency. Curr OpinPharmacol [Internet]. 2007 Aug 1 [cited 2025 Sep 12]; 7(4): 355–9. Available from: https://www.sciencedirect.com/science/article/abs/pii/S1471489207000896?via%3Dihub

31. Bajpai DrSR. Aluminium Phosphide Poisoning: Management and Prevention. Journal of Indian Academy of Forensic Medicine [Internet]. 2010 Dec [cited 2025 Sep 12]; 32(4): 352–4. Available from: DOI:10.1177/0971097320100422

32. VP D, MN SK. Coconut Oil as an Adjuvant to Magnesium Sulphate Therapy in Acute Aluminum Phosphide Consumption. Int J Anesthesiol Res. 2018 Feb 27; 509–14.

33. Manert W, Haller M, Briegel J, Hummel T, Kilger E, Polasek J, et al. Veno-Venose Extrakorporale Membranoxygenierung (ECMO) Mit Heparinbeschichtetem Bypass-System. Eine Effektive Erweiterung Bei Der Behandlung Des Akuten Lungenversagens (ARDS). Anaesthesist. 1996; 45(5): 437–48.

34. Goharbari MH, Taghaddosinejad F, Arefi M, Sharifzadeh M, Mojtahedzadeh M, Nikfar S, et al. Therapeutic effects of oral liothyronine on aluminum phosphide poisoning as an adjuvant therapy: A clinical trial. Hum Exp Toxicol [Internet]. 2018 Feb 1 [cited 2025 Sep 12]; 37(2): 107–17. Available from: https://pubmed.ncbi.nlm.nih.gov/29233028/

35. El Shehaby DM, Sayed SA, Abd El-Kareem DM, Elsherif R, Almaz D. Trimetazedine with hyperinsulinimea–euoglycemia, N-acetyl cysteine, and vitamin C: A new approach concept for management of aluminum phosphide poisoning. J Biochem Mol Toxicol [Internet]. 2022 Jan 1 [cited 2025 Sep 12]; 36(1): e22931. Available from: https://doi.org/10.1002/jbt.22931

36. Himaaldev GJ, Rao CC. STEMI in Young Befogged by Aluminum Phosphide Toxicity—Role of ECMO as Salvage Therapy and Trimetazidine and Magnesium to Suppress Arrhythmias. Indian J Crit Care Med [Internet]. 2020 Aug [cited 2025 Sep 12]; 24(8): 727. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7519610/

37. Soltani M, Shetab-Boushehri SF, Mohammadi H, Shetab-Boushehri SV. Proposing boric acid as an antidote for aluminium phosphide poisoning by investigation of the chemical reaction between boric acid and phosphine. Journal of Medical Hypotheses and Ideas [Internet]. 2013 [cited 2025 Sep 12]; 7(1): 21–4. Available from: https://www.researchgate.net/publication/257747302_Proposing_Boric_Acid_as_an_Antidote_for_Aluminium_Phosphide_Poisoning_by_Investigation_of_the_Chemical_Reaction_Between_Boric_Acid_and_Phosphine

38. Soltani M, Shetab-Boushehri SF, Shetab-Boushehri S V. Chemical Reaction between Boric Acid and Phosphine Indicates Boric Acid as an Antidote for Aluminium Phosphide Poisoning. Sultan Qaboos Univ Med J [Internet]. 2016 Aug 1 [cited 2025 Sep 12]; 16(3): e303. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC4996292/

39. El-Sarnagawy GN, Hafez ASAF, Ghonem MM. Role of serum glucose/potassium ratio in assessing poisoning severity and adverse outcomes in patients with acute aluminum phosphide poisoning. Toxicol Rep [Internet]. 2025 Jun 1 [cited 2025 Sep 16]; 14. Available from: https://pubmed.ncbi.nlm.nih.gov/40026479/

40. Ullah A, Jan S, Shahzad H, Dar MR, Khan S, Ahmad K, et al. Therapeutic Effect of Glucose-Insulin-Potassium (GIK) Infusion Therapy in the Treatment of Acute Aluminum Phosphide Poisoning: An Institutional Study. Cureus [Internet]. 2024 Dec 22 [cited 2025 Sep 16]; 16(12). Available from: https://pubmed.ncbi.nlm.nih.gov/39840180/

41. Mazaheri Tehrani B, Toreyhi H, Jolfaei P, Soltaninejad K, Rahimi M, Amiri H, et al. The effect of hyperinsulinemia-euglycemia on acute aluminum phosphide poisoning: a clinical trial. Asia Pac J Med Toxicol [Internet]. 2023 Oct 1 [cited 2025 Sep 16]; 12(3): 97–102. Available from: https://apjmt.mums.ac.ir/article_23748.html

42. Emam NM, Hamed SS, Alsaied D, Ahmed M. Could N. Acetyl Cysteine Prolong Survival Time in Acute Aluminum Phosphide Poisoning Among Egyptian Patients? Egyptian Society of Clinical Toxicology Journal [Internet]. 2020 Dec 1 [cited 2025 Sep 16]; 8(2): 1–12. Available from: https://esctj.journals.ekb.eg/article_126953.html

43. Taghaddosinejad F, Farzaneh E, Ghazanfari-Nasrabad M, Eizadi-Mood N, Hajihosseini M, Mehrpour O. The effect of N-acetyl cysteine (NAC) on aluminum phosphide poisoning inducing cardiovascular toxicity: a case-control study. Springerplus [Internet]. 2016 Dec 1 [cited 2025 Sep 16]; 5(1). Available from: https://pubmed.ncbi.nlm.nih.gov/27917341/

44. Bhalla A, Jyothinath P, Singh S. Antioxidant Therapy in Patients with Severe Aluminum Phosphide Poisoning: A Pilot Study. Indian J Crit Care Med [Internet]. 2017 Dec 1 [cited 2025 Sep 16]; 21(12): 836. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC5752792/

45. El-Ebiary AA, Abuelfad AA. N-acetylcysteine as an Adjuvant in The Treatment of Acute Aluminum Phosphide Poisoning: A Randomized Clinical Trial. Ain Shams Journal of Forensic Medicine and Clinical Toxicology [Internet]. 2017 Jan 1 [cited 2025 Sep 16]; 28(1): 38–46. Available from: https://ajfm.journals.ekb.eg/article_18278.html

46. Halvaei Z, Tehrani H, Soltaninejad K, Abdollahi M, Shadnia S. Vitamin E as a novel therapy in the treatment of acute aluminum phosphide poisoning. Turk J Med Sci [Internet]. 2017 [cited 2025 Sep 16]; 47(3): 795–800. Available from: https://pubmed.ncbi.nlm.nih.gov/28618724/

47. Elgazzar FM, Keshk WA, Khalifa HK. Early L-carnitine Therapy in Severe Acute Aluminum Phosphide Poisoning: A Randomized Controlled Clinical Trial. The Egyptian Journal of Forensic Sciences and Applied Toxicology [Internet]. 2019 Dec 1 [cited 2025 Sep 16]; 19(4): 147–64. Available from: https://ejfsat.journals.ekb.eg/article_67562.html

48. Henaidy MF, Ghanem M, Shehata SF, Shouair AM, Mabrouk RR. Therapeutic effects of NAC and CoQ10 on aluminium phosphide

poisoning as an adjuvant therapy: A Pilot study. Toxicol Rep [Internet]. 2025 Jun 1 [cited 2025 Sep 16]; 14: 101907. Available from: https://www.sciencedirect.com/science/article/pii/S2214750025000253

49. Sobh ZK, Abd-Elhameed A. The therapeutic benefit of antioxidants on the outcome of acute aluminum phosphide poisoning: a systemic review and meta-analysis. Toxicol Res (Camb) [Internet]. 2023 Jun 1 [cited 2025 Sep 16]; 12(3): 345. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10311134/

50. De Santi O, Orellana MJ, Di Niro CA, Lashin HI, Greco V. The adjuvant effect of oil-based gastric lavage on the outcome of acute Aluminum phosphide poisoning: a systematic review and meta-analysis. Toxicol Res (Camb) [Internet]. 2024 Apr 1 [cited 2025 Sep 16]; 13(2). Available from: https://pubmed.ncbi.nlm.nih.gov/38496382/

51. Darwish RT, Sobh ZK, Hamouda EH, Saleh EM. The efficacy of Coenzyme Q10 and liquid paraffin oil in the management of acute aluminum phosphide poisoning. Toxicol Res (Camb) [Internet]. 2020 [cited 2025 Sep 16]; 9(4): 444. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC8059132/

52. Moshiri M, Dorooshi G, Etemad L, Feizi A, Rahimi A, Gheshlaghi F, et al. Effect of extracorporeal membrane oxygenation on mortality rate of aluminum phosphate poisoning: A systematic review and meta-analysis. J Res Med Sci [Internet]. 2024 Oct 1 [cited 2025 Sep 16]; 29(1). Available from: https://pubmed.ncbi.nlm.nih.gov/39744188/

53. Mohan B, Gupta V, Ralhan S, Gupta D, Puri S, Mahajan R, et al. Impact of extra-corporeal membrane oxygenation on outcome of aluminium phosphide poisoning complicated with myocardial dysfunction. Clin Toxicol (Phila) [Internet]. 2019 Nov 2 [cited 2025 Sep 16]; 57(11): 1095–102. Available from: https://pubmed.ncbi.nlm.nih.gov/30856020/

54. Goharbari MH, Taghaddosinejad F, Arefi M, Sharifzadeh M, Mojtahedzadeh M, Nikfar S, et al. Therapeutic effects of oral liothyronine on aluminum phosphide poisoning as an adjuvant therapy. Hum Exp Toxicol [Internet]. 2018 Feb 1 [cited 2025 Sep 12]; 37(2): 107–17.