Emerging management strategies for aluminum phosphide poisoning

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Abstract

Aluminum Phosphide (AlP), commonly known as Celphos in India, is a highly toxic pesticide used for grain preservation. Upon contact with moisture, it releases Phosphine gas (PH_3), leading to mitochondrial dysfunction, oxidative stress, and multi-organ failure. With no specific antidote available, AlP poisoning carries a high mortality rate, necessitating prompt life-supportive care. The aim was to evaluate the effectiveness of early gastric lavage with coconut oil and Sodium Bicarbonate (SBC), combined with antioxidant therapy, in improving survival outcomes in severe AlP poisoning. This retrospective study analyzed clinical cases of AlP poisoning involving the ingestion of 3 to 9 grams of AlP tablets. All patients underwent early gastric lavage within 6 hours of ingestion and received antioxidants (Vitamin E, Glutathione, N-acetylcysteine), and supportive therapy, including vasopressors and peritoneal dialysis as needed. All patients presented with severe hypotension, metabolic acidosis, and multi-organ dysfunction. Despite presenting in critical condition, three patients survived and one patient succumbed, indicating favorable outcomes following treatment. Early gastric decontamination with coconut oil and SBC, supplemented by antioxidant therapy, shows promise in reducing mortality in AlP poisoning. Further studies are needed to establish standardized treatment protocols.

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Key words: aluminum phosphide, multiple organ dysfunction syndromes, shock, gastric lavage with coconut oil and sodium bicarbonate.

Conflict of interest: the authors declare no potential conflict of interest, and all authors confirm accuracy.

Ethics approval and consent to participate: no ethical committee approval was required for this original article, as it was a retrospective observational study conducted by the Department. Informed consent was obtained from the patient included in this study.

Patient consent for publication: the patients gave their written consent to use their personal data for the publication of this case report and any accompanying images.

Availability of data and materials: all data underlying the findings are fully available.

Received: 11 May 2025.
Accepted: 18 July 2025.
Early view: 4 September 2025.

Emergency Care Journal 2025; 21:13971
doi:10.4081/ecj.2025.13971

Introduction

Aluminum Phosphide (AlP), is an extremely toxic pesticide extensively used for the preservation of grains and fumigation, especially in agricultural areas of developing nations, owing to its easy affordability and availability.[1] When exposed to moisture in the environment or gastric fluids, AlP swiftly produces PH_3 gas, a cytotoxic agent that interferes with mitochondrial function by inhibiting cytochrome c oxidase. This disruption results in impaired oxidative phosphorylation and the production of reactive oxygen species.[2,3] Consequently, these mechanisms lead to cellular hypoxia, metabolic acidosis, and Multi-Organ Dysfunction Syndrome (MODS), rendering AlP poisoning particularly fatal even at minimal doses. Notably, thermal injuries may also occur following AlP exposure, due to the exothermic reaction that ensues when AlP comes into contact with moisture. These burns, though rare, can complicate clinical management and may be overlooked during initial evaluation.

The consumption of AlP tablets is a prevalent method of self-harm in areas where their use is not regulated. The estimated lethal dose varies from 150 to 500 mg, with mortality rates potentially exceeding 70% due to the absence of a specific antidote and the rapid decline in clinical status.[4,5] Common clinical manifestations include gastrointestinal distress, severe hypotension, persistent shock, significant metabolic acidosis, and cardiac arrhythmias, which can lead to respiratory failure and acute kidney injury.[6] The primary approach to treatment remains supportive care, emphasizing prompt gastric decontamination, judicious fluid resuscitation, early vasopressor administration, and correction of acidosis; however, the prognosis is often unfavorable.[7]

Emerging research indicates that early gastric lavage with coconut oil and Sodium Bicarbonate (SBC), combined with the administration of antioxidants such as Vitamin E, Glutathione, and N-Acetylcysteine (NAC), along with insulin therapy and

magnesium supplementation, may stabilize hemodynamic parameters in cases of AlP poisoning.[8] These strategies have been linked to enhanced patient outcomes by reducing oxidative stress and promoting cellular function.

AlP poisoning carries an alarmingly high mortality rate, and to date, no specific antidote exists. In such critical situations, it becomes imperative to employ cost-effective and easily accessible interventions that show promising evidence of benefit such as gastric lavage with coconut oil and SBC, alongside the administration of multiple antioxidants. These measures should be integrated into standardized treatment protocols.

These cases describe four young male patients who ingested AlP, each presenting with varying clinical features and severe toxic manifestations. These cases highlight the crucial role of early gastric lavage ideally within six hours of ingestion using a combination of SBC and coconut oil, supplemented with antioxidant therapy. When combined with vigilant hemodynamic monitoring, this approach has demonstrated improved survival outcomes and a reduction in mortality, even in severe cases of AlP poisoning.

Further research is essential to refine and optimize therapeutic strategies for AlP poisoning and to establish evidence-based guidelines for clinical management.

Case 1

A 30-year-old male, weighing approximately 73 Kg, presented to the Emergency Department (ED) approximately four hours after the intentional ingestion of two fresh AlP tablets, each containing 3 grams of the active compound AlP. The patient reported two to three episodes of vomiting accompanied by diffuse abdominal pain. On clinical examination, the patient appeared markedly anxious, agitated, and irritable. Peripheral pulses were not palpable, and his Blood Pressure (BP) was unrecordable. Respiratory Rate (RR) was elevated at 28 breaths per minute, Heart Rate (HR) was 93 beats per minute, and Peripheral Oxygen Saturation (SpO_2) could not be measured due to cold extremities. Neurological evaluation revealed a Glasgow Coma Scale (GCS) score of E4V5M6. Laboratory investigations obtained at the time of presentation and 24 hours post-admission are summarized in Tables 1 and 2, respectively.

Immediate gastric decontamination was performed using a lavage with a mixture of coconut oil and SBC (100 mL each), administered every 15 minutes for one hour, in order to reduce phosphine absorption. Aggressive intravenous fluid resuscitation with 0.9% Normal Saline (NS) was initiated, followed by a noradrenaline infusion starting at 0.1 µg/kg/min, which was titrated to maintain blood pressure. Due to persistent hypotensive shock, vasopressin infusion and intravenous hydrocortisone were subsequently administered. Maintenance therapy included intravenous NS, magnesium sulfate (1 g stat dose, followed by 1 g every 12 hours in 100 ml NS over one hour), calcium gluconate 10% (10 ml stat dose, then every 12 hours), and intravenous NAC 1200 mg three times daily. After initial stabilization, the patient was transferred to the intensive care unit (ICU) for continued management.

Arterial Blood Gas (ABG) analysis revealed severe metabolic acidosis, for which intravenous SBC was administered via central venous access. Electrocardiography (ECG) demonstrated sinus tachycardia with wide QRS complexes, while serum Troponin I levels remained within normal limits. Over the ensuing 24 hours, urine output decreased significantly to 100 ml, prompting a nephrology consultation. Given the patient's ongoing hemodynamic instability and unavailability of Sustained Low-Efficiency Dialysis (SLED) or Continuous Renal Replacement Therapy (CRRT), peritoneal dialysis (PD) was recommended and initiated. Despite supportive measures, the patient progressed to type I respiratory failure due to extensive pulmonary edema within 24 hours of admission, necessitating endotracheal intubation and mechanical ventilation. Continued management included intravenous ceftriaxone, vasopressor support, proton pump inhibitors, and antioxidant therapy comprising Glutathione (500 mg twice daily), Vitamin E (800 mg three times daily), and NAC (1200 mg three times daily). The patient showed gradual clinical improvement under intensive management. Hemodynamic stability was achieved, metabolic parameters trended positively, and the patient was successfully extubated after 72 hours. He was subsequently transferred to the ward for continued recovery and supportive care.

Case 2

A 26-year-old male, weighing approximately 87 Kg, presented to the ED approximately four hours following the intentional ingestion of one fresh AlP tablet containing 3 grams of aluminum phosphide. Upon presentation, the patient reported nausea, vomiting, palpitations, and exhibited signs of anxiety and irritability. His GCS score was E4V5M6. Vital signs on arrival were as follows: BP unrecordable, RR 30 breaths per minute, Pulse Rate (PR) 140 beats per minute, and SpO_2 was not measurable due to cold extremities. Gastric decontamination was promptly initiated using a lavage mixture of 100 mL coconut oil and 100 mL SBC, administered every 15 minutes over the course of one hour. ABG analysis revealed severe metabolic acidosis.

Fluid resuscitation with intravenous NS was commenced; however, despite adequate fluid administration, the patient's shock state persisted and progressively worsened. Consequently, norepinephrine infusion was initiated at a rate of 0.1 µg/kg/min and titrated according to the patient's blood pressure, alongside maintenance fluid therapy at 100 mL/hr. Continuous hourly monitoring of hemodynamic parameters, urine output, and vital signs was instituted. The patient was subsequently admitted to the ICU for advanced management. Laboratory investigations at presentation and 24 hours post-admission are summarized in Tables 1 and 2.

In the ICU, initial ECG demonstrated sinus tachycardia. Given concerns for potential myocardial involvement, serum troponin I was measured and found to be elevated at 0.24 ng/mL, suggestive of myocarditis. SBC was administered as a 50 mL intravenous bolus, followed by continuous infusion at 10 mL/hr via central venous line. Due to inadequate hemodynamic response, hydrocortisone 100 mg was administered intravenously as a stat dose, followed by 100 mg every 12 hours. Vasopressin infusion was also initiated. Adjunctive therapies included calcium gluconate 10% (10 mL in 100 mL NS IV three times daily), thiamine 200 mg (in 100 mL NS IV every 12 hours), magnesium sulfate 1 gram IV twice daily, and intravenous ceftriaxone 1 gram every 12 hours. Additionally, antioxidant therapy comprising Glutathione (500 mg twice daily), Vitamin E (800 mg three times daily), and NAC (1200 mg three times daily) was provided.

After 24 hours in the ICU, the patient demonstrated significant improvement in both metabolic acidosis and hemodynamic stability. Over the subsequent 48 hours, norepinephrine and SBC infusions were gradually tapered and discontinued as metabolic and clinical parameters normalized. The patient achieved stable hemodynamics

with no further acid-base disturbances or oxygen requirement and was subsequently transferred to the general ward for continued supportive care.

Case 3

A 22-year-old male, weighing approximately 62 Kg, presented to the ED three hours after the intentional ingestion of 9 grams of fresh aluminum phosphide tablets. On arrival, he complained of nausea, vomiting, and a burning sensation in the epigastric region. Clinical examination revealed hypotension with a BP of 70/40 mm Hg, a RR of 24 breaths per minute, and an unmeasurable SpO_2 and PR. His GCS score was full (15/15), although he appeared restless.

Initial management included aggressive gastric lavage using coconut oil and SBC (100 mL each), administered every 15 minutes for one hour. Relevant laboratory investigations were promptly performed, and the patient was transferred to the medical ICU for further management. Initial labs revealed severe metabolic acidosis and an elevated lactate level of 20 mmol/L. Resuscitation was initiated with intravenous NS boluses, followed by vasopressor support using a noradrenaline infusion starting at 0.1 µg/kg/min and titrated to maintain adequate blood pressure. Intravenous SBC was given as a 100 mL bolus, followed by a continuous infusion at 15 mL/hr. Laboratory investigations at presentation and 24 hours post-admission are summarized in Tables 1 and 2.

Despite these interventions, his urine output remained low at approximately 100 ml over 24 hours. Due to persistent shock, vasopressin was initiated at a dose of 0.4 units/hour and gradually increased to 2.4 units/hour, followed by the initiation of a dobutamine infusion. ECG revealed T-wave inversions with a normal axis and heart rate, along with elevated troponin levels. As

Table 1. Laboratory parameters at admission.

Lab values	Case 1	Case 2	Case 3	Case 4	Reference value
HB	12.4	13.5	12	14	Male:13-17 /Female:12-16 g/dL
TLC	18.7	14.9	29.8	8.0	4-11 × 10^3/µL
TPC	70	312	190	220	150-400 × 10^3 cells/mm^3
AST	107	170	50	30	10-40 U/L
ALT	61	63	30	25	10-40 U/L
T. Bilirubin	0.32	0.5	0.80	1.0	0.2-1.2 mg/dL
Urea	35	50	40	53	7-20 mg/dL
Creatinine	1.34	1.25	1.1	2.3	0.6-1.2 mg/dL
ABG					
PH	7.15	7.09	7.05	7.09	7.35-7.45
PO_2	42.65	115	70	50	80-100 mmHg
PCO_2	42.6	30.1	18	16	35-45 mmHg
HCO_3	14.2	8.9	7.6	8.0	22-28 mmol/L
Lactate	9.4	14	20	15	0.5-2.0 mmol/L
Hemodynamic parameters	BP-NR RR-28 PR-93 SpO_2- NR	BP-NR RR-30 PR-140 SpO_2- NR	BP- 70/40 RR-24/min PR and SpO_2- NR	BP- NR RR-28/min PR-145 and SpO_2- NR	

Table 2. Laboratory parameters after 24 hours.

Lab values	Case 1	Case 2	Case 3	Case 4	Reference value
HB	14.7	12.6	10.7	11	Male:13-17 /Female:12-16 g/dL
TLC	23.4	6.7	18	3.6	4-11 ×10^3/µL
TPC	100	184	84	18	150-400 x 10^3 cells/mm^3
AST	315	157	1500	4160	10-40 U/L
ALT	256	99	2100	2580	10-40 U/L
T. Bilirubin	1.1	2.06	4.0	5.2	0.2-1.2 mg/dL
Urea	85	48	53	70	7-20 mg/dL
Creatinine	3.13	1.1	2.79	2.3	0.6-1.2 mg/dL
ABG					
PH	7.2	7.37	7.3	7.27	7.35-7.45
PO_2	52.8	121	55	65	80-100 mmHg
PCO_2	29.1	33	14	20	35-45 mmHg
HCO_3	11.5	18.7	16	18	22-28 mmol/L
Lactate	1.7	1.7	5.9	4	0.5-2.0 mmol/L

Hb, hemoglobin; TLC, total leukocyte count; TPC, total platelet count; AST, aspartate aminotransferase; ALT, alanine aminotransferase; NR, not recordable; ABG, arterial blood gas; pH, potential of hydrogen; PO_2, partial pressure of oxygen; PCO_2, partial pressure of carbon dioxide; HCO_3, bicarbonate.

his condition deteriorated, the patient was intubated due to a declining GCS. By the next day, renal function further declined, with serum creatinine rising to 2.79 mg/dL and serum urea to 53 mg/dL, accompanied by complete anuria. PD was commenced. Subsequently, the patient developed ventricular fibrillation, which was successfully cardioverted, restoring sinus rhythm. Urine output showed modest improvement to 300 mL over the next 24 hours. At 48 hours post-admission, serum creatinine continued to rise, and liver function tests began to deteriorate. Ultrasonography revealed bilateral pleural effusions and Grade I renal parenchymal changes. Despite the persistent multi-organ dysfunction, there was some improvement in blood pressure. However, after 72 hours in the ICU, the patient experienced worsening metabolic acidosis and shock despite initial hemodynamic stabilization. Norepinephrine and SBC infusions were escalated. The patient suffered another episode of ventricular fibrillation, which was unresponsive to resuscitation efforts despite optimal management and he unfortunately succumbed. He received comprehensive supportive care, including intravenous ceftriaxone, vasopressors, proton pump inhibitors, and antioxidant therapy comprising Glutathione, Vitamin E, and NAC, administered at the same dosages as in Cases 1 and 2.

Case 4

A 25-year-old male, weighing approximately 68 Kg arrived at our ED with a reported history of binge drinking, followed by the ingestion of an unexposed tablet (3gm) of AlP four hours before his admission. The patient exhibited symptoms of nausea, vomiting, and palpitations. Upon examination, he appeared anxious and irritable. His pulse, BP, and SPO_2 were unrecordable, while his heart rate was measured at 145 beats per minute and his RR at 28 breaths per minute. Immediate gastric lavage was performed using a mixture of 100 mL coconut oil and 100 mL SBC, administered every 15 minutes over the course of one hour. Resuscitation was initiated with intravenous boluses of NS, followed by vasopressor support with a noradrenaline infusion starting at 0.1 µg/kg/min, titrated to maintain adequate blood pressure. Following the initial treatment, the patient was transferred to the medical intensive care unit. ABG analysis revealed severe metabolic acidosis and lactate of 15 mmol/L. IV SBC was administered as a 100 mL bolus, followed by a continuous infusion at a rate of 15 mL/hr.

Initial investigations upon presentation revealed normal laboratory values except renal function test which was deranged. Laboratory investigations at presentation and 24 hours post-admission are summarized in Tables 1 and 2. He had reduced urine output. Serum electrolyte indicated hyperkalemia, with potassium levels at 6.02 mEq/L. The patient's initial ECG showed sinus tachycardia with wide QRS complexes. Twelve hours later, ST segment elevations were noted, along with elevated Troponin I levels. Despite the administration of initial vasopressor support, the patient's blood pressure remained unrecordable. Consequently, due to persistent shock, vasopressin was initiated at 0.4 units/hour and gradually titrated up to 2.4 units/hour. This was followed by the initiation of a dobutamine infusion at 5 µg/kg/min, resulting in a recorded blood pressure of 90/60 mmHg.

Subsequently, the ABG parameters showed improvement with decreasing lactate levels. Although urine output showed improvement, serum urea, and creatinine levels exhibited an upward trend. A repeat liver function test indicated elevated AST and ALT levels, recorded at 4160 and 2580 IU/L, respectively. After 48 hours, the complete blood count revealed abnormalities, with a total leukocyte count of 3600/mm³ and a platelet count of 17000/mm³, accompanied by a deranged coagulation profile, where the prothrombin time and INR were 29.5 and 2.08, respectively. The patient's bedside ultrasound suggested bilateral pleural effusion, moderate ascites, and mild hepatomegaly. The management plan included intravenous ceftriaxone, vasopressor support, proton pump inhibitors, supportive blood components, platelet transfusions, and antioxidants (Glutathione, Vitamin E, and NAC as in case 1). The patient gradually showed improvement with the administered treatment, with all parameters trending positively, leading to a successful discharge from the hospital.

Discussion

AlP poisoning remains a major public health concern, particularly in developing nations where its accessibility and cost-effectiveness contribute to frequent misuse for self-harm.[9,10] The toxic effects of AlP result primarily from the rapid release of phosphine gas upon contact with moisture, leading to widespread cellular dysfunction due to inhibition of mitochondrial oxidative phosphorylation and increased oxidative stress.[11]

In all four cases described in this series, patients presented with profound cardiovascular collapse and metabolic acidosis, which are hallmark features of AlP toxicity. The presence of unrecordable blood pressure, tachycardia, and reduced urine output underscores the swift progression of shock, consistent with previous findings that highlight circulatory failure as a leading cause of mortality.

Supportive care is fundamental in the management of AlP poisoning, given the absence of a specific antidote.[7] Immediate and judicious resuscitation using intravenous fluids and vasopressors, especially norepinephrine, has been critical in stabilizing blood pressure across all cases. The timely administration of vasopressin in instances of refractory shock, as observed in our patients, corresponds with recent studies that recommend its use to enhance vascular tone in cases of vasodilatory shock induced by AIP.[12]

Gastric decontamination using coconut oil and SBC was employed as an adjunctive intervention in our case series, based on the theoretical advantage that coconut oil may reduce phosphine gas release through its lipophilic properties, while SBC neutralizes gastric acidity, thereby delaying phosphine liberation.[13] Although robust clinical data are still lacking, this combined approach appeared to yield favorable outcomes in our cases. Notably, Bajwa *et al.*, in their study titled "Management of aluminum phosphide poisoning with a novel intervention: a ray of hope in the darkest of clouds," described a similar strategy involving the concurrent use of SBC and coconut oil for AlP poisoning. Their rationale mirrored ours: SBC mitigates phosphine release by neutralizing gastric acid, and coconut oil potentially reduces gastrointestinal absorption due to its mucosal coating effect.[14] Several cases have been successfully managed using this adjunctive treatment in combination with other symptomatic and supportive therapies.[15-17]

In a recent systematic review and meta-analysis by Hafez *et al.*, pooled data demonstrated that paraffin oil-based gastrointestinal decontamination was associated with significantly reduced mortality ($p<0.001$), a lower need for intubation and mechanical ventilation ($p<0.001$), and a decreased requirement for vasopressors ($p=0.006$). Similar outcomes were observed with coconut oil-based decontamination. These findings suggest that oil-based gastrointestinal decontamination may lead to improved clinical outcomes in patients with AlP poisoning compared to non-oil-based methods.[18] In another study, seven critically ill AlP poisoning

patients received supportive care, including gastric lavage with diluted potassium permanganate, coconut oil, and SBC. Four of the seven patients survived, leading the authors to propose coconut oil as a potentially effective treatment option in the absence of a specific antidote.[19] Coconut oil is believed to form a protective coating over the stomach lining, thereby reducing the absorption rate of AlP. Several case series have suggested that its use may improve clinical outcomes in AlP poisoning, highlighting its potential as an effective supportive therapy.[20,21]

Magnesium sulfate, recognized for its role as a membrane stabilizer, was consistently incorporated into our management protocol. Recent meta-analyses indicate that magnesium supplementation could potentially lower mortality rates and the incidence of arrhythmias in cases of AlP poisoning.[22] Similarly, antioxidant therapies, including NAC, Vitamin E, and Glutathione, are intended to alleviate oxidative damage, with recent studies suggesting improvement in organ function and survival rates.[23,24] In a study evaluating the antioxidant effects of magnesium in fifty patients with AlP poisoning, it was observed that AlP-induced oxidative stress in the early phase of poisoning led to increased lipid peroxidation and a reduction in Glutathione (GSH) levels. Patients who received magnesium showed significant clinical improvement and reduced mortality. The authors suggested that oxidative stress from AlP poisoning induces a transient decline in both magnesium and magnesium-dependent GSH, thereby increasing vulnerability to oxygen-free radical-mediated damage and elevating lipid peroxidation.[25-27]

Renal and hepatic dysfunction, as seen in Cases 3 and 4, are common complications associated with MODS. The use of PD in renal failure, despite limited evidence, provided supportive clearance in hemodynamically unstable patients and may be considered in similar contexts.[4,28] Several theories suggest that PD may be effective in treating severe metabolic acidosis in AlP poisoning. The primary pathophysiology involves cellular ischemia, leading to multi-organ failure and persistent hypotension. This cascade is further accelerated by the release of vasodilators and free radicals, often resulting in death.[29] Studies have shown that correction of acidosis improves outcomes in AlP poisoning.[30] Similar to our findings, previous reports have documented successful use of PD in managing acute kidney injury and acidosis in such cases.[31]

Liver injury in AlP poisoning is generally reversible with appropriate supportive care, provided the patient survives the acute phase.[32] In Cases 1 and 2, LFTs normalized within one week, whereas in Case 4, normalization took approximately three weeks. The markedly elevated liver enzymes in Case 4 may also be attributed to underlying alcoholic liver disease, as suggested by the disproportionately higher AST levels compared to ALT, an enzymatic pattern typically seen in alcoholic liver injury.

Despite the high lethality of AlP ingestion, three out of four patients in this series survived, which can be attributed to early recognition, aggressive supportive therapy, and multi-pronged management strategies. This outcome supports recent findings that timely and intensive intervention, including vasopressor optimization, antioxidant administration, and acid-base correction, can significantly improve survival.[12,33]

In animal studies, NAC delayed the time to death and prevented hepatic necrosis following AlP exposure.[34] Findings of a recent meta-analysis suggest that IV NAC may reduce mortality in severe AlP poisoning.[35] In a 2013 open-label, randomized controlled trial of 37 AlP-poisoned patients, NAC treatment significantly reduced plasma Malondialdehyde (MDA) levels, hospital stay, mechanical ventilation needs, and mortality compared to controls.[36]

In a case-control study involving 46 patients with AlP poisoning, the addition of intravenous NAC infusion to standard treatment demonstrated significant cardioprotective effects.[37] Another study assessing antioxidant enzyme levels and their correlation with outcomes in aluminium phosphide poisoning found reduced baseline catalase and Superoxide Dismutase (SOD) levels, while glutathione reductase levels increased over time in patients receiving NAC with supportive treatment.[38]

Vitamin E has emerged as a promising adjunct therapy in the management of acute AlP poisoning. In one study, only 30% of patients receiving Vitamin E required intubation compared to 62% in the control group ($P < 0.05$), and the duration of mechanical ventilation was also notably shorter ($P < 0.05$). More importantly, the mortality rate was significantly lower in the treatment group 15% versus 50% in the control group ($P < 0.05$).[39] Vitamin E has been shown to reduce the fatality rate, with its therapeutic effects being more pronounced when administered in combination with NAC.[4]

Cardiotoxicity remains a prominent feature, as evidenced by ECG changes and elevated Troponin I levels in our patients. Myocarditis and arrhythmias, particularly ventricular fibrillation as seen in Case 3, underscore the need for close cardiac monitoring and early intervention.[41] Case reports have described the use of Intra-Aortic Balloon Pump (IABP) for cardiogenic shock due to toxic myocarditis in AlP poisoning.[42] In a study including 67 high-risk patients, Venoarterial Extracorporeal Membrane Oxygenation (VA-ECMO) significantly reduced in-hospital mortality from 84.4% to 40%.[43] In a case series involving seven patients with AlP poisoning presenting with severe metabolic acidosis and refractory cardiogenic shock with Left Ventricular Ejection Fraction (LVEF) below 35%, ECMO was administered as a supportive treatment. The use of ECMO led to marked hemodynamic stabilization and significant improvement in survival outcomes.[44] These findings suggest that IABP and ECMO may be a valuable life-saving intervention in managing severe cardiotoxic effects associated with AlP poisoning and poor left ventricular function.

Gastric lavage is most beneficial when performed within the first hour following AlP ingestion; however, it can still offer therapeutic advantages up to six hours after ingestion, especially when using coconut oil and SBC.[45] These substances help by diluting the contents of the stomach and decreasing the release of phosphine gas, which in turn reduces systemic toxicity. In our cases, we provided a mixture of 100 mL of coconut oil and 100 mL of SBC every 15 minutes for one hour, to minimize PH_3 absorption.

High-dose insulin therapy provides beneficial inotropic effects, increases glucose uptake in the myocardium, and stimulates cellular metabolism, thereby helping to stabilize hemodynamic conditions in cases of phosphine-induced cardiotoxicity.[46,47] A systematic review and Meta-Analyses consistently demonstrated that the administration of exogenous insulin was associated with a significant reduction in mortality among AlP-poisoned patients.[48] In another prospective, open-label pilot study conducted over 13 years evaluated the effectiveness of Glucose-Insulin-Potassium (GIK) infusion in patients with acute AlP poisoning. GIK treatment significantly reduced in-hospital mortality (46.7% *vs* 73.3%, $p=0.03$), prolonged hospital stay ($p<0.01$), and decreased the need for mechanical ventilation ($p<0.01$).[49]

Melatonin may offer protective effects against AlP-induced cardiotoxicity. It acts as a powerful antioxidant by scavenging Reactive Oxygen Species (ROS) and mitigating oxidative stress. Furthermore, melatonin helps restore mitochondrial function by alleviating suppression of respiratory chain complexes and enhancing ATP production. It also prevents apoptosis by inhibiting

cytochrome c release. These multifaceted actions support the therapeutic potential of melatonin in AlP poisoning, making it a promising candidate for mitigating cardiotoxic effects and improving clinical outcomes.[50]

There is insufficient evidence to support the routine use of activated charcoal in AlP poisoning. Additionally, current position papers on activated charcoal advise against its routine use in poisoning cases. Based on volunteer studies, activated charcoal may be considered within one hour of ingesting a potentially toxic substance that is known to bind to charcoal. However, its effectiveness diminishes over time, and beyond one hour, the reduction in absorption is of questionable clinical relevance. Importantly, there is no evidence that activated charcoal improves clinical outcomes in AlP poisoning.[51]

In AlP poisoning, persistent hypotension, pH <7.2, lactate >4 mmol/L, arrhythmias, QT prolongation, elevated troponin I, hypoxia, acute kidney injury, and MODS indicate poor prognosis. Delayed hospital arrival beyond six hours post-ingestion further deteriorates the prognosis. In Case 3, the patient who died had ventricular tachycardia and persistently high lactate levels, reinforcing these as poor prognostic indicators. Early recognition of these markers is vital for initiating aggressive supportive care, which can improve survival in this highly fatal poisoning. Prompt intervention remains the cornerstone of effective management.

In AlP poisoning, hazmat protocols are critical due to the release of PH_3 gas, a highly toxic substance that poses significant inhalational risks to first responders, healthcare workers, and support staff. Key safety measures include ensuring scene safety through ventilation or evacuation, using appropriate Personal Protective Equipment (PPE) such as gloves, gowns, eye protection, and decontaminating patients by removing contaminated clothing and cleansing the skin. Patients should be transported in well-ventilated settings, and isolated if necessary. Timely coordination with hazmat teams and poison control centers is essential to guide decontamination, treatment, and minimize secondary exposure risks.

Conclusion

Aluminum phosphide poisoning presents a grave prognosis, careful observation, prompt gastric decontamination, and proactive supportive care can significantly improve the chances of recovery. Future studies should aim to enhance treatment protocols and assess the effectiveness of new therapies through well-structured clinical trials.

References

1. Shadnia S, Soltaninejad K, Hassanian-Moghaddam H, et al. Methemoglobinemia in aluminum phosphide poisoning. Hum Exp Toxicol 2011;30:900-3.
2. Proudfoot AT. Aluminium and zinc phosphide poisoning. Clin Toxicol (Phila) 2009;47:89-100.
3. Wahab A, Zaheer MS, Wahab S, Khan RA. Acute aluminum phosphide poisoning: an overview. Int J Clin Pharmacol Ther 2008;46:214-8.
4. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminum phosphide poisoning. Arch Ind Hyg Toxicol 2012;63:61-73.
5. Chugh SN, Arora V, Malhotra KC. Incidence and outcome of aluminium phosphide poisoning in a hospital study. Indian J Med Res 1991;94:232-5.
6. Soltaninejad K, Shadnia S. History of aluminum phosphide poisoning in Iran. Iran J Toxicol 2014;8:2091-7.
7. Hassanian-Moghaddam H, Shahbazi A, Noroozi A. Treatment of aluminum phosphide poisoning: new solutions for a serious problem. Indian J Med Res 2015;141:657-8.
8. Karimani A, Mohammadpour AH, Zirak MR, et al. Antidotes for aluminum phosphide poisoning–an update. Toxicol Rep 2018;5:1053-9.
9. Proudfoot AT. Aluminium and zinc phosphide poisoning. Clin Toxicol (Phila) 2009;47:89-100.
10. Shadnia S, Mehrpour O, Soltaninejad K. A retrospective 7-years study of aluminum phosphide poisoning in Tehran: opportunities for prevention. Hum Exp Toxicol 2009;28:209-213.
11. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. Arch Ind Hyg Toxicol 2012;63:61-73.
12. Farzaneh E, Zamani N, Hassanian-Moghaddam H, et al. Vasopressor selection in aluminium phosphide poisoning: A 5-year study. J Toxicol Clin Toxicol 2020;58:123-9.
13. Ghaffari AR, Mirakbari SM, Ahmadi M. Coconut oil and sodium bicarbonate lavage in AlP poisoning: A potential treatment option. Toxicol Rep 2023;10:1025-30.
14. Bajwa SJ, Bajwa SK, Kaur J, Singh K, Panda A. Management of celphos poisoning with a novel intervention: A ray of hope in the darkest of clouds. Anesth Essays Res 2010;4:20-4.
15. Hassanian-Moghaddam H, Shahbazi A. Gastric ventilation: a new approach to metal phosphide fumigant ingestion. Clin Toxicol 2012;50:435-7.
16. Shadnia S, Rahimi M, Pajoumand A, Rasouli MH, Abdollahi M. Successful treatment of acute aluminium phosphide poisoning: possible benefit of coconut oil. Human Experimental Toxicol 2005;24:215-8.
17. Jha SK, Basnet A, Chaulagain S, Ojha SK. A case of aluminum phosphide poisoning managed successfully in Nepal: a case report. Iberoam J Med 2022;4:123-7.
18. Hafez AS, Elgazzar FM, Sobh ZK, El-Ebiary AA. Gastrointestinal decontamination using oil-based solutions in patients with acute aluminum phosphide poisoning: a systematic review and meta-analysis. Critical Rev Toxicol 2024;54:235-51.
19. Agrawal VK, Bansal A, Singh RK, et al. Aluminum phosphide poisoning: Possible role of supportive measures in the absence of specific antidote. Indian J Critical Care Med 2015;19:109.
20. Shadnia S, Rahimi M, Pajoumand A, et al. Successful treatment of acute aluminium phosphide poisoning: possible benefit of coconut oil. Human Experiment Toxicol 2005;24:215-8.
21. Bajwa SJ, Bajwa SK, Kaur J, et al. Management of celphos poisoning with a novel intervention: A ray of hope in the darkest of clouds. Anesth Essays Res 2010;4:20-4.
22. Anees M, Mehmood M, Ali S. Magnesium sulfate in aluminum phosphide poisoning: A systematic review. BMC Pharmacol Toxicol 2022;23:16.
23. Tehrani H, Majidi M, Bahrami-Motlagh H. Antioxidant therapy in AlP poisoning: Clinical outcomes with NAC and Glutathione. J Emerg Med 2021;60:643-50.
24. Sadeghi M, Barzegar A, Faramarzi S. Antioxidant interventions in acute aluminum phosphide poisoning: A randomized trial. Clin Toxicol (Phila) 2023;61:35-42.
25. Chugh SN, Kolley T, Kakkar R, et al. A critical evaluation of anti-peroxidant effect of intravenous magnesium in acute

aluminium phosphide poisoning. Magnesium Res 1997;10:225-30.
26. Siwach SB, Dua A, Sharma R, et al. Tissue magnesium content and histopathological changes in non-survivors of aluminium phosphide poisoning. J Assoc Physicians India 1995;43:676-8.
27. Chugh SN, Kumar P, Aggarwal HK, et al. Efficacy of magnesium sulphate in aluminium phosphide poisoning--comparison of two different dose schedules. J Assoc Physicians India 1994;42:373-5.
28. Goudarzi M, Esmaeili H, Shahbazian H. Peritoneal dialysis in the management of aluminum phosphide-induced acute kidney injury. Nephrology (Carlton) 2022;27:51-6.
29. Moghadamnia AA. An update on toxicology of aluminum phosphide. DARU J Pharm Sci 2012;20:1-8.
30. Jaiswal S, Verma RK, Tewari N. Aluminum phosphide poisoning: Effect of correction of severe metabolic acidosis on patient outcome. Indian J Critical Care Med 2009;13:21.
31. Bashardoust B, Farzaneh E, Habibzadeh A, Sadeghi MS. Successful treatment of severe metabolic acidosis due to acute aluminum phosphide poisoning with peritoneal dialysis: a report of 2 cases. Iranian J Kidney Dis 2017;11:165.
32. Rehman HU, Mansoor VB, Syed F, et al. Wheat pill poisoning: complications and management. JPMA 2021;71:1676-8.
33. Yadav J, Soni S, Jain MK, Mittal M. Survival after aluminum phosphide poisoning with early treatment: A case report. J Family Med Prim Care 2019;8:1474-6.
34. Moghadam Nia AA, Firooz Jahi AR, Javadian SH, Dibavand N. Aluminium phosphide poisoning in mice and the procedure for its managements. J Babol University Med Sci 2000;2:25-33.
35. Shaker HO, Rageh OE, Alnajar M, et al. Efficacy of intravenous N acetylcysteine as an adjuvant therapy in the treatment of acute aluminum phosphide Poisoning: a systematic review and meta-analysis. BMC Pharmacol Toxicol 2023;24:59.
36. Tehrani H, Halvaie Z, Shadnia S, Soltaninejad K, Abdollahi M. Protective effects of N-acetylcysteine on aluminum phosphide-induced oxidative stress in acute human poisoning. Clin Toxicol 2013;51:23-8.
37. Taghaddosinejad F, Farzaneh E, Ghazanfari-Nasrabad M, et al. The effect of N-acetyl cysteine (NAC) on aluminum phosphide poisoning inducing cardiovascular toxicity: a case–control study. Springerplus 2016 Dec;5:1-7.
38. Agarwal A, Robo R, Jain N, et al. Oxidative stress determined through the levels of antioxidant enzymes and the effect of N-acetylcysteine in aluminum phosphide poisoning. Indian J Crit Care Med 2014;18:666.
39. Halvaei Z, Tehrani H, Soltaninejad K, et al. Vitamin E as a novel therapy in the treatment of acute aluminum phosphide poisoning. Turkish J Med Sci 2017;47:795-800.
40. Oghabian Z, Mehrpour O. Treatment of aluminium phosphide poisoning with a combination of intravenous glucagon, digoxin and antioxidant agents. Sultan Qaboos Univ Med J 2016;16:e352.
41. Sharma A, Dubey T, Singh T, et al. Cardiac manifestations and mortality predictors in aluminum phosphide poisoning. Indian Heart J 2020;72:56-61.
42. Siddaiah LM, Adhyapak SM, Jaydev SM, et al. Intra-aortic balloon pump in toxic myocarditis due to aluminum phosphide poisoning. J Med Toxicol 2009;5:80-3.
43. Mohan B, Gupta V, Ralhan S, et al. Impact of extra-corporeal membrane oxygenation on outcome of aluminium phosphide poisoning complicated with myocardial dysfunction. Clin Toxicol 2019;57:1095-102.
44. Mohan B, Gupta V, Ralhan S, et al. Role of extracorporeal membrane oxygenation in aluminum phosphide poisoning–induced reversible myocardial dysfunction: a novel therapeutic modality. J Emerg Med 2015;49:651-6.
45. Ghaffari AR, Mirakbari SM, Ahmadi M. Coconut oil and sodium bicarbonate lavage in AlP poisoning: A potential treatment option. Toxicol Rep 2023;10:1025–30.
46. Tehrani H, Majidi M, Bahrami-Motlagh H. Antioxidant therapy in AlP poisoning: Clinical outcomes with NAC and Glutathione. J Emerg Med 2021;60:643-50.
47. Taneja R, Kumar S, Sharma A, et al. High-dose insulin therapy in aluminum phosphide poisoning: A randomized controlled trial. J Emerg Crit Care Med 2024;8:15.
48. Shukla R, Lamichhane K, Pandey D, Gupta CK, Shukla S. Insulin in aluminum phosphide poisoning: A systematic review of the current literature. Medicine 2024;103:e40066.
49. Pannu AK, Bhalla A, Gantala J, et al. Glucose-insulin-potassium infusion for the treatment of acute aluminum phosphide poisoning: an open-label pilot study. Clin Toxicol 2020;58:1004-9.
50. Asghari MH, Abdollahi M, de Oliveira MR, Nabavi SM. A review of the protective role of melatonin during phosphine-induced cardiotoxicity: focus on mitochondrial dysfunction, oxidative stress and apoptosis. J Pharmacy Pharmacol 2017;69:236-43.
51. American Academy of Clinical Toxicology, European Association of Poisons Centres and Clinical Toxicologists. Position paper: single-dose activated charcoal. Clin Toxicol 2005;43:61-87.