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Refractory Cardiogenic Shock Following Aluminum Phosphide Poisoning: A Fatal Case during VA-ECMO Cannulation and Mini-Review of the Literature

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ABSTRACT

Aluminum phosphide (AIP) is a widely used pesticide known for its lethality when ingested, posing a significant challenge in clinical management because of its rapid onset of severe toxicity. Despite efforts to mitigate its effects, AIP poisoning remains associated with high mortality.

We present the case of a young male patient admitted to our emergency department after intentional ingestion of AIP. Despite initial stability, the patient rapidly deteriorated, necessitating aggressive hemodynamic support. Despite efforts, including vasopressor therapy and initiation of extracorporeal membrane oxygenation (ECMO), the patient ultimately experienced an arrhythmic cardiac arrest without return of spontaneous circulation and was pronounced dead.

This case highlights the devastating course of AIP poisoning and underscores the importance of early recognition and aggressive management. ECMO may be a promising therapeutic approach for such individuals, given the absence of a clearly identifiable antidote, providing valuable support to the cardiovascular system until the body can eliminate the toxin.

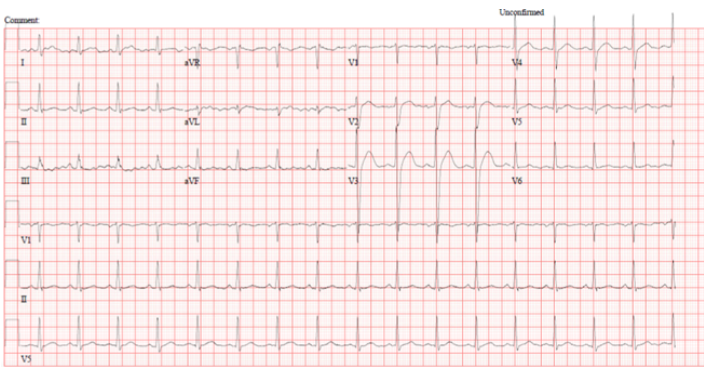
Keywords
Aluminum phosphide, ECMO, ECLS.

Case Presentation

During a routine shift in the resuscitation area, the team was alerted by Emergency Medical Services (EMS) that a young male in his 20s had deliberately ingested an UNKNOWN substance in a suicide attempt, resulting in persistent vomiting.

Upon arrival in the resuscitation room, a thorough evaluation was performed using the "A to E" protocol. The patient's blood pressure was 104/67 mmHg (mean arterial pressure 75 mmHg), and his heart rate fluctuated between 100 and 105 beats per minute. Although his oxygen levels were satisfactory, he was restless and continued to retch. He remained conscious, alert, and oriented, with a full GCS score of 15/15. Lung examination revealed symmetrical air entry bilaterally without added sounds. Additionally, the patient had reactive pupils measuring 4 mm in diameter, responding appropriately to light.

After gathering a detailed medical history, the patient reported experiencing substantial stress and depressive symptoms. The patient showed us a picture of the ingested substance, labeled as a fumigant-insecticide named Salphos, which contained 56% aluminum phosphide. According to the patient, he ingested a portion of the tablet, less than half, approximately 30 minutes before presenting to the emergency department. Following this disclosure, basic blood examinations were requested, and the initial Venous Blood Gas analysis showed a lactate level of 7.6 mmol/L with minimal electrolyte imbalances, notably a calcium level of 1.07 mmol/L. Furthermore, an electrocardiogram indicated a normal sinus rhythm, as shown in Figure 1.



Recognizing the team's limited knowledge of the toxic effects of aluminum phosphide, immediate consultation was sought with the on-call toxicologist. Emphasis was placed on the substance's high toxicity and the associated mortality risk, and the patient was advised to be promptly admitted to the Medical Intensive Care Unit (MICU) for vigilant observation.

Unfortunately, the patient's condition deteriorated rapidly within an hour, with a significant decline in blood pressure unresponsive to fluid resuscitation, necessitating initiation of a peripheral phenylephrine infusion. Subsequently, his level of consciousness decreased to a GCS of 8, prompting endotracheal intubation. Despite insertion of central and arterial lines and administration of maximal doses of phenylephrine and noradrenaline, the patient's blood pressure remained refractory. Consequently, he was transferred to the MICU for veno-arterial extracorporeal membrane oxygenation (VA ECMO) support.

Tragically, during the VA ECMO cannulation, the patient suffered a cardiac arrest with ventricular fibrillation as the initial rhythm. Despite 35 minutes of uninterrupted cardiopulmonary resuscitation (CPR), spontaneous circulation was not re-established, and the patient ultimately succumbed to the toxic effects of aluminum phosphide.

Background

AIP poisoning is a severe condition that affects individuals exposed to the toxicant. Currently, most cases are managed medically with supportive care until the toxicant is eliminated through the kidneys and lungs. Despite appropriate supportive care, many aluminum phosphide-poisoned patients do not recover, and there is no recognized treatment for this condition. The mortality rate from AIP poisoning is very high, and even among patients receiving critical care, it can range from 30 to 100%. In highly specialized institutions equipped with cutting-edge life support systems, this rate might be as high as 60 percent. Therefore, it is one of the leading global causes of poisoning deaths [1,2].

Discussion

Upon ingestion, solid phosphides, including aluminum phosphides, produce hazardous phosphine gas when they contact water, ambient moisture, or hydrochloric acid in the stomach. Although the precise mechanism remains incompletely understood, evidence suggests that phosphine acts at the mitochondrial level, disrupting enzyme and protein synthesis after systemic absorption. In addition to its corrosive properties, phosphine's toxicity involves the generation of highly reactive hydroxyl radicals. This process leads to cellular injury via lipid peroxidation. Previous reports have indicated decreased catalase levels and increased superoxide dismutase activity in individuals affected by AIP poisoning. The decline in glutathione concentration across various tissues in AlP poisoning further underscores cellular injury, as glutathione serves as a protective factor against oxidation by catalyzing the reduction of oxygen peroxide to oxygen and water [1,3].

Markers of oxidative stress, including reduced glutathione and malondialdehyde, have been observed to peak within 48 hours of poison exposure. Despite extensive research demonstrating phosphine-induced oxidative damage in animal models, there remains a scarcity of data on measuring total antioxidant capacity in patients with AlP poisoning. Furthermore, any reduction in plasma levels of thiol groups, integral components of the antioxidant defense system, may suggest the presence of oxidative stress in individuals poisoned with AlP. This imbalance can lead to the generation of free radicals and alterations in the antioxidant system [4].

The management and treatment of aluminum phosphide poisoning pose considerable clinical challenges, primarily because there is no definitive therapeutic regimen that consistently correlates with improved patient outcomes. This ambiguity arises predominantly from the variable severity observed across cases, influenced by the quantity of the ingested toxic substance. Consequently, a diverse array of therapeutic interventions has been advocated, with the aim of mitigating the deleterious effects of poisoning and improving patient prognosis [5].

During therapy, the primary aim is to ensure adequate oxygenation, ventilation, and circulation until phosphine is expelled; central among these interventions is gastric lavage. The efficiency of gut decontamination primarily depends on the duration of exposure to the poison and should be carried out as soon as feasible. This involves employing potassium permanganate at a concentration of 1:10,000 in conjunction with the administration of activated charcoal. This primary intervention seeks to curtail further absorption of the toxic agent within the gastrointestinal tract and oxidize phosphine to phosphate, the non-toxic form. Supplementing this approach, the intravenous administration of calcium gluconate at a concentration of 10% at a dosage of 1 gram every 6 hours, alongside intravenous magnesium sulfate at the same dosage and frequency, and sodium bicarbonate delivered via either intravenous or oral routes, represent recommended therapeutic modalities. These interventions are aimed at counteracting the toxic effects of aluminum phosphide poisoning and addressing associated metabolic aberrations. Furthermore, concurrent supportive measures constitute integral components of the therapeutic approach. These measures include ensuring adequate oxygenation, meticulous hemodynamic monitoring, and timely

replacement of fluids and electrolytes to preserve physiological equilibrium. Moreover, when respiratory compromise is imminent, endotracheal intubation becomes imperative to maintain airway patency and ensure optimal ventilation [6,7].

The primary contributor to mortality in acute aluminum phosphide poisoning is progressive severe left ventricular (LV) dysfunction, leading to circulatory collapse, impaired respiratory gas exchange, systemic hypotension, and metabolic acidosis. Reported differences in mortality rates across case reports may reflect the varying degrees of cardiovascular and respiratory impairment observed in individual cases. In patients with milder shock, supportive measures such as fluid administration, inotropic support, and intra-aortic balloon pump may aid survival until the phosphine toxin is eliminated. However, available data suggest that mortality rates approach 100% in patients with severe shock. Given that LV dysfunction and respiratory impairment in aluminum phosphide poisoning can potentially be reversed within 5-7 days, the use of extracorporeal life support systems (ECLS), such as VA ECMO, may be beneficial. VA ECMO can temporarily support both cardiovascular and pulmonary functions in patients with severe shock [8].

Several case reports have shown that ECLS is effective in managing patients poisoned by aluminum phosphide who develop shock unresponsive to inotropic or vasopressor therapy. In those patients, lactate levels ranged from 8.8 to 47 mmol/L, yet all survived. Each documented case presented with markedly impaired cardiac function, with left ventricular ejection fractions (LVEF) ranging from 20% to 30%. Despite this initial severe cardiac dysfunction, LVEF returned to normal levels at hospital discharge and/or during follow-up. ECLS support was sustained for up to 10 days, with a mean duration of 42.1 ± 26.9 hours among survivors compared with 67 ± 35.9 hours among non-survivors in the largest study. In a case series of seven patients with severe cardiovascular failure due to AlP poisoning, ECLS was initiated when lactate concentrations exceeded 15 mmol/L, and LVEF was significantly depressed (18%–32%). Remarkably, despite the severity of these cases, the survival rate was 71% (5 out of 7 patients), indicating that ECLS is a valuable intervention for managing AlP-related cardiac failure [9-13].

In one observational study, alongside conventional and supportive care, the implementation of ECMO therapy resulted in a notable reduction in in-hospital mortality among patients with AlP poisoning, decreasing from 86.7% to 33.3% ($p = 0.001$). Upon discharge, all patients in both study groups exhibited nearly normal left ventricular myocardial function, which was confirmed to have completely normalized during follow-up assessments. This outcome implies the reversible nature of myocardial dysfunction secondary to AlP poisoning. Survivors in this study received ECMO support for an average of 60 ± 35 hours, while the half-life of phosphine gas ranges from 5 to 24 hours. This correlation suggests that the observed cardiorespiratory failure is transient and aligns with the duration of phosphine gas's effects [14].

Conclusion

To conclude, the mortality rates in ALP poisoning vary due to differences in cardiovascular and respiratory impairment among individuals. Supportive measures may aid in patient survival in milder cases, but mortality rates can approach 100% in severe shock. Extracorporeal life support systems (ECLS), such as VA ECMO, may offer potential benefits by temporarily supporting cardiovascular and pulmonary functions. ECLS has shown effectiveness in managing ALP poisoning-related cardiac failure, with improved survival rates and normalization of cardiac function observed in patients. Therefore, it is highly advisable to consider the initiation of ECMO at an early stage, prior to the onset of cardiovascular collapse in cases of AIP poisoning.

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