CASE REPORT

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Successful Treatment of Acute Respiratory Distress Syndrome Following Cardiopulmonary Resuscitation in the Context of Aluminum Phosphide Poisoning: A Case Report

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Abstract

Aluminum phosphide (ALP) is a pesticide known for its high cellular toxicity, making it one of the common means of suicide in developing countries. We report the case of a 19-year-old female who presented to the emergency department after ALP poisoning. Initial emergency treatments were administered. Eight hours later, due to respiratory distress, she was intubated. Cardiac arrest occurred immediately after intubation, but successful resuscitation was performed. The patient subsequently developed acute respiratory distress syndrome. With proper ventilator adjustment on the third day, her hemodynamic status stabilized. A computed tomography (CT) scan taken after 72 hours showed improvement in lung opacities. The patient was successfully extubated, transferred to the general ward, and discharged following psychiatric counseling. Acute respiratory distress syndrome in the context of ALP poisoning represents a critical condition with a high mortality rate. Its primary differential diagnosis is pulmonary edema. Proper supportive care led to clinical improvement within six days of severe ALP poisoning.

Keywords: Acute respiratory distress syndrome; Aluminum phosphide; ARDS; Poisoning; Pulmonary edema.

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Introduction

Aluminum phosphide is a pesticide commonly used for grain storage due to its affordability and minimal adverse effects on foodstuffs. Despite its benefits, intentional ingestion of this substance is highly toxic, leading to its prohibition in many countries.[1,2] Predominantly, cases of aluminum phosphide poisoning have been reported in males aged 21 to 30 years.[3] The major manifestations of aluminum phosphide poisoning include chest pressure, headache, nausea, vomiting, severe hypotension, metabolic acidosis, and common compli-

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cations such as heart failure, arrhythmias, seizures, coma, liver failure, and kidney failure.[4,5] Additionally, involvement of the respiratory system, including pulmonary edema, acute respiratory distress syndrome, and pleural effusion, has also been reported.[6,7] We report a case of successful management of acute respiratory compromise following cardiopulmonary resuscitation in a patient suffering from acute aluminum phosphide poisoning.

Case Report

A 19-year-old female presented to the hospital's emergency department approximately 30 minutes after ingesting a 5-gram aluminum phosphide tablet. Upon arrival, the patient was conscious and complained of nausea and shortness of breath, with no prior medical history or medication use. The patient's vital signs are presented in Table 1. Arterial blood gas samples were obtained to assess the patient's condition, with results shown in Table 2. Blood pressure was monitored every 30 minutes, and arterial blood gas analysis was performed every 3 hours. Treatment included gastric lavage with sodium bicarbonate and the administration of standard therapeutic interventions: an initial 200 mg dose of hydrocortisone, followed by 200 mg every 6 hours; a loading dose of magnesium sulfate at 2 g, with subsequent 1 g doses every 6 hours; calcium gluconate starting at 2 g, then 1 g every 6 hours; and N-acetylcysteine at a dosage of 300 mg/kg over a 24-hour period. Normal saline was administered based on blood pressure, and sodium bicarbonate was administered based on arterial blood gas results.

Approximately 8 hours after admission, the patient exhibited tachycardia and respiratory distress (Table 1). To manage the tachycardia, intravenous diazepam (5 mg) and propranolol (1 mg) were given. Subsequently, the decision was made to intubate the patient. However, shortly after intubation, the patient suffered cardiac arrest. Fortunately, successful resuscitation was achieved within 5 minutes, restoring sinus rhythm. However, significant foamy pink secretion was noted in the endotracheal tube. Considering the patient's condition, a toxicology consultation was requested, and she was transferred to the intensive care unit (ICU). Upon her admission to the ICU, her condition was unstable (Tables 1 and 2). She received respiratory support through synchronized intermittent mandatory ventilation (SIMV) with a fraction of inspired oxygen (FiO2) set at 70%. The treatment administered over the past 8 hours included 6,500 mL of normal saline and 8 ampules of 7.5% sodium bicarbonate. Diagnosed with acute pulmonary edema, the patient was treated with furosemide and morphine, tailored to her clinical conditions, and fluid and serum restriction was considered.

To maintain a systolic blood pressure above 70 mmHg, norepinephrine was administered. For systolic blood pressure exceeding 80 mmHg, the glucose-insulin-potassium (GIK) treatment protocol was initiated. This protocol included the administration of 50 grams of glucose, 50 units of regular insulin, and 30 milliequivalents of potassium chloride over the first hour, followed by half of the initial dose for subsequent hours. Blood sugar levels were monitored hourly, and serum potassium levels were checked every 4 hours. All values remained within the normal range. After initiating treatment in the ICU, arterial blood gas analysis indicated an improvement in respiratory acidosis and a significant improvement in metabolic acidosis (Table 2).

Over the next 18 hours, the patient received a total of three doses of hydrocortisone, each measuring 200 milligrams; four doses of furosemide, each measuring 40 milligrams; and three doses of morphine, each measuring 5 milligrams. The patient's blood pressure remained above 80 mmHg after receiving the GIK treatment, eliminating the need for norepinephrine. During the examination on the morning of the following day, the patient's heart rate was 117 beats per minute, and the blood pressure was 61/92 mmHg. Despite a fraction of inspired oxygen (FiO2) of 70%, arterial oxygen saturation levels fluctuated between 88% and 91%. Rapidly decreasing the FiO2 led to a decrease in arterial oxy-

Table 1. Results of vital signs assessment for the patient from admission to the time of admission to the intensive care unit

Time since poisoning	Heart Rate	Blood Pressure	Respiratory Rate	Atrial Oxygen Saturation (Pulse Oximetry)
On Arrival	107	91/58	15	97%
After 8 Hours	144	101/73	26	88%
On Admission to ICU	138	85/52	Intubated (14)	85%

Table 2. Results of arterial blood gas analysis for the patient from admission to the second day of hospitalization

Time since poisoning	pH	HCO3	BE	PCO_2	Atrial Oxygen Saturation (Pulse Oximetry)
On Arrival	7.37	11.4	-12.6	21.1	97%
Admission to ICU	7.12	16.0	-13.5	49.2	80%
3 Hours After Admission to ICU	7.06	24.3	-5.9	87.7	80%
2nd Day of Admission	7.35	22.1	-3.3	39.2	78%

gen saturation levels. On the second day of hospitalization, arterial blood gas analysis revealed a significant decrease in arterial oxygen levels (the partial pressure of arterial oxygen/fraction of inspired oxygen ratio, PaO2/FiO2=105/0.7=150).

Therefore, an echocardiogram was performed to investigate further, which showed an ejection fraction of 60%. A computed tomography (CT) scan of the chest revealed bilateral pulmonary opacities in the central lung region. This finding was more consistent with cardiogenic pulmonary edema. However, given the acceptable ejection fraction along with a PaO2/FiO2 ratio of 150, a diagnosis of acute respiratory distress syndrome (ARDS) was considered more probable (Figure 1). Laboratory results indicated leukocytosis (white blood cell count [WBC]: 17,200) and a mild increase in liver enzymes (aspartate aminotransferase [AST]: 104 and alanine aminotransferase [ALT]: 105). Additionally, the patient developed a fever from the second day.

Given the clinical condition, empirical antibiotic therapy with ceftriaxone and clindamycin was initiated. To improve ventilation, the ventilator settings were adjusted by increasing the positive end-expiratory pressure to 8 mmHg and reducing the tidal volume to 350 ml. On the third day of admission, due to persistent fever and decreased oxygen saturation, a consultation with a pulmonary specialist was sought. The specialist recommended the culture of blood and tracheal secretions, discontinuation of ceftriaxone and clindamycin, and initiation of empirical treatment with meropenem 1000 mg every 8 hours, vancomycin 1000 mg every 12 hours, and levofloxacin 500 mg daily. Hydrocortisone was continued as previously ordered. The patient's blood pressure remained stable on the third day of ICU admission, and there was a significant increase in urine output. The patient's fluid intake and urinary output over the first three days are detailed in Table 3.

Table 3. Fluid intake and urinary output of the patient during the first three days in the ICU

Timeline as Documented in the ICU Chart	Fluid Intake (cc)	Urinary Output (cc)
1st Day of Admission		
10 (AM) -12 (PM)	1000	1000
12 (PM) -18 (PM)	500	2000
18 (PM) -6 (AM)	1800	5400
2nd Day of Admission		
6 (AM) -12 (PM)	500	300
12 (PM) -18 (PM)	500	400
18 (PM) -6 (AM)	2250	700
3rd Day of Admission		
6 (AM) -12 (PM)	400	400
12 (PM) -18 (PM)	400	1900
18 (PM) -6 (AM)	800	6200

After 72 hours, the patient became afebrile. Blood culture reported no significant growth of pathogenic bacteria, but Klebsiella species were detected in the tracheal secretions culture. A repeat CT scan of the chest showed improvement in bilateral lung opacities. Efforts were made to transition the patient to bilevel positive airway pressure (BiPAP) for ventilatory support, and the FiO2 was reduced to 40%. With improved arterial oxygen saturation (PaO2: 108), the endotracheal tube was removed. At this point, the patient's fever was under control, and vital signs were stable. Laboratory results showed a trend toward normalization, with improved leukocytosis and liver function tests (WBC: 12,200; AST: 36; ALT: 62). Over the next 48 hours, the patient's respiratory and hemodynamic statuses remained completely stable, with all tests reported within the normal range.

The patient was transferred to the toxicology department and was discharged after receiving psychiatric counseling. A follow-up examination 14 days after discharge revealed normal vital signs and paraclinical tests. Informed consent was obtained from the patient for reporting this case.

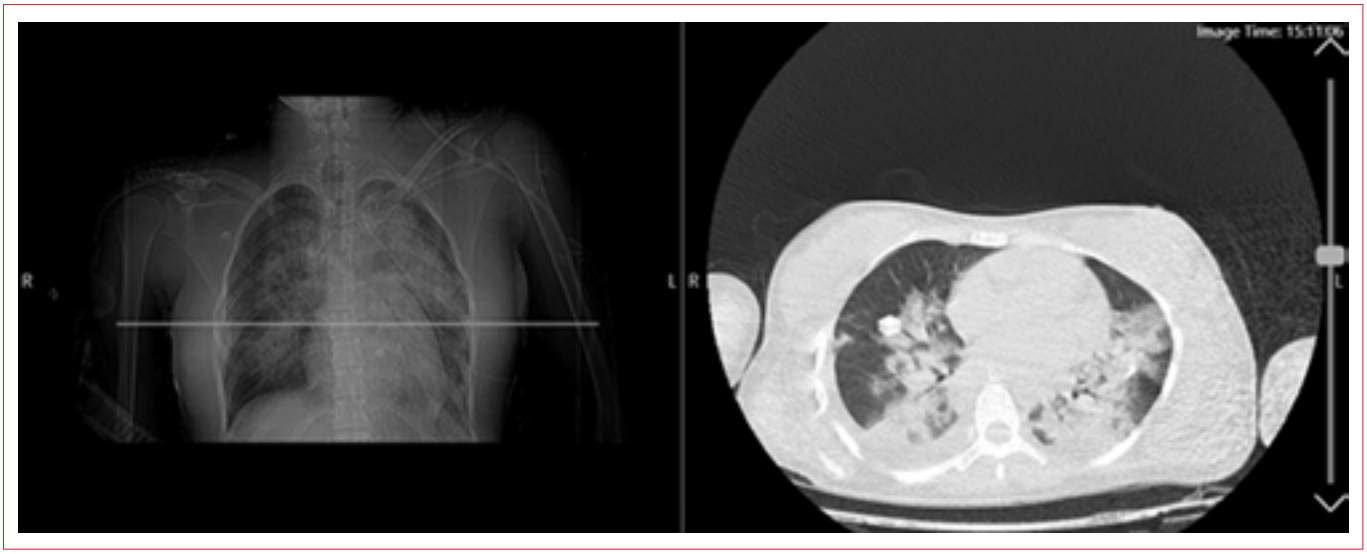

Figure 1. Bilateral pulmonary opacities demonstrating acute respiratory distress syndrome in the patient.

Discussion

This article reports a case of aluminum phosphide poisoning in a young woman who experienced a severe drop in blood pressure and metabolic acidosis. Initial treatment, including gastric lavage, began approximately 30 minutes after ingesting 5,000 milligrams of aluminum phosphide. The lethal dose for adults is reported to be between 150 and 500 mg.[8] However, recent studies suggest that gastric lavage with sodium bicarbonate may exacerbate the release of phosphine gas, potentially worsening the clinical presentation of poisoning. As a result, it is now recommended to use non-absorbable oils, such as castor oil, to reduce the toxin's contact with gastric contents.[9] Another notable observation was the occurrence of tachycardia and decreased arterial oxygen saturation about 8 hours after admission, despite a recorded systolic blood pressure above 100 mmHg, which should be considered a sign of vital instability due to reduced tissue perfusion. In a young patient, stimulation of the sympathetic system can help maintain blood pressure within the normal range by increasing the heart rate. In this case, it appears that the rapid administration of the beta-blocker medication, propranolol, may have led to cardiac arrest. Fortunately, successful cardiopulmonary resuscitation was rapidly performed on this patient. The cardiac arrest appears to be related to the administration of propranolol, rather than the toxic effects of aluminum phosphide, contributing to the successful resuscitation.

After resuscitation, signs of pulmonary edema were present. Investigation into the sodium bicarbonate levels and administered fluids indicated that excessive fluid volume was the main cause of this condition. Therefore, fluid restriction and the administration of furosemide and morphine were considered. Additionally, due to the risk of cardiac arrhythmias associated with vasopressor medications,[8] the GIK treatment protocol was used. The benefit of the GIK protocol is supported by studies suggesting that insulin promotes energy production from carbohydrates and restores calcium flux. These actions may improve myocardial contractility, vascular tone, and reduce the need for vasopressor drugs.[10,11] The improvement in metabolic acidosis indicated adequate tissue perfusion with the administered treatment. In this case, considering the rapid decrease in arterial oxygen saturation upon reducing the FiO2 and the presence of bilateral pulmonary opacities, despite adequate left ventricular systolic function, a diagnosis of ARDS was established. An important differential diagnosis is pulmonary edema. Our patient experienced hypotension and decreased systemic vascular resistance during the initial period of management in the emergency room. Tachycardia, a normal physiological response to this condition, deteriorated after administering propranolol, leading to cardiac arrest. Additionally, our patient was managed with excessive administration of intravenous fluids. After successful cardiopulmonary resuscitation, significant foamy pink secretion was observed in the endotracheal tube. Consequently, the diagnosis of pulmonary edema was made, and treatment with respiratory support (SIMV) and an FiO2 of 70%, as well as administration of furosemide and morphine, was initiated.

On the second day, decreasing the FiO2 rapidly led to a decrease in arterial oxygen saturation. An echocardiogram showed an ejection fraction of 60%. A CT scan of the chest revealed bilateral pulmonary opacities in the central lung region, a picture more consistent with cardiogenic acute pulmonary edema. Nevertheless, given the acceptable ejection fraction along with a PaO2/FiO2 ratio of 150, the diagnosis of acute respiratory distress syndrome could not be ruled out. It should be kept in mind that this condition is associated with high mortality in aluminum phosphide poisoning.[12] Based on the PaO2/FiO2 ratio, this patient was diagnosed with a moderate to severe condition.[13]

Phosphine gas inhibits catalase, induces superoxide dismutase, and reduces glutathione levels, leading to lipid peroxidation, cell membrane damage, extensive cellular injury, and disruption of ion channel function. Additionally, it directly damages the alveolar membrane, leading to the development of ARDS.[11] However, excessive fluid administration and the development of pulmonary infections are also common causes of ARDS.[13] Glucocorticoid medications may be effective in reducing mortality and the duration of mechanical ventilation in these patients.[14] Nevertheless, it should be remembered that the primary treatment for these patients involves respiratory support with advanced mechanical ventilation settings. Low tidal volume ventilation, which prevents excessive alveolar distension, can be more effective in rapidly improving this condition.[15]

In this case, hydrocortisone treatment, initiated as part of the management for aluminum phosphide poisoning, was continued during respiratory care in the ICU. Furthermore, considering the possibility of ventilator-associated pneumonia contributing to this condition and the increased risk of patient mortality, empirical antibiotic treatment was deemed reasonable. Additionally, to reduce lung injury, low tidal volume ventilation was adopted.

The administration of propranolol for tachycardia control in this patient led to sudden cardiac arrest, which responded well to resuscitation measures. However, the excessive administration of fluids and sodium bicarbonate resulted in the patient developing acute pulmonary edema. Indeed, assessment of fluid responsiveness was necessary to guide fluid therapy for our patient, but this critical consideration was overlooked in our patient's management. The combined glucose-insulin-potassium treatment protocol was effective in improving tissue perfusion during the acute phase of poisoning. Nonetheless, the development of ARDS necessitated prolonged care in the ICU. Early recognition of this condition and the adjustment of mechanical ventilation settings based on the patient's clinical condition contributed to the successful treatment and the patient's clinical improvement eight days after aluminum phosphide poisoning.

Conclusion

Acute respiratory distress syndrome in the context of aluminum phosphide poisoning represents a grave condition with a high mortality rate. Cardiogenic pulmonary edema can occur as a direct consequence of aluminum phosphide poisoning. Moreover, the administration of extensive fluid therapy during the acute phase of aluminum phosphide poisoning can further contribute to the development of pulmonary edema, presenting significant challenges in diagnosis and treatment. Additionally, the presence of cardiogenic pulmonary edema can exacerbate the progression of ARDS. The pulmonary fluid overload resulting from pulmonary edema can intensify the impairment of gas exchange inherent in ARDS, leading to a further decline in oxygenation. This complexity can pose a challenge in patient management, as the therapeutic approaches for treating pulmonary edema and ARDS can vary.

Informed Consent: Written informed consent was obtained from the patient for reporting this case.

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