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Intensive Care

A Reversible Toxic Myocarditis Due to Aluminum Phosphide Poisoning

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Abstract **Case Report**

Aluminum phosphide (Phostoxin®) is a fumigant pesticide whose intentional ingestion is a frequent method of suicide in several developing countries. Acute aluminum phosphide poisoning is a true medical emergency that can lead to severe myocardial injury and even multiorgan failure. No specific antidote is currently available. We report the case of a 24-year-old male admitted in a state of severe cardiogenic shock and coma following the voluntary ingestion of two Phostoxin® tablets (6 g). Initial echocardiography revealed a critically reduced left ventricular ejection fraction (LVEF) of 15%, consistent with acute toxic myocarditis. The patient underwent intensive hemodynamic resuscitation with dobutamine, norepinephrine, moderate fluid administration, and bicarbonate-based alkalinization. Mechanical ventilation was initiated from the outset. Clinical evolution was favorable, with return of urine output by 48 hours, correction of lactic acidosis, withdrawal of vasoactive drugs by Day 6 (norepinephrine) and Day 8 (dobutamine), successful ventilator weaning, and LVEF recovery to 45% by Day 10. He was subsequently transferred to psychiatric care for psychological follow-up. This case illustrates that favorable outcomes are possible even in cases of severe toxic myocarditis, provided that rapid and aggressive intensive care is implemented. Management relies on early vasopressor/inotrope support, volume resuscitation, alkalinization, and mechanical ventilation. Prognosis depends on how early diagnosis and treatment are established. Psychiatric follow-up is essential to prevent recurrence and ensure comprehensive care.

Keywords: Aluminum phosphide, Phostoxin, acute poisoning, toxic myocarditis, cardiogenic shock, intensive care, inotropes, myocardial recovery.

INTRODUCTION

Aluminum phosphide, marketed under the name Phostoxin® in many countries, is a solid fumigant pesticide widely used in Morocco for grain preservation, pest control, and rodent extermination. However, its use for suicidal purposes has turned it into a serious public health issue. Its ingestion is highly lethal and typically results in death within 12 to 24 hours, primarily due to cardiovascular toxicity. The lethal dose in adults is estimated at 150 to 500 mg. Currently, there is no specific antidote or effective treatment. Prognosis is heavily influenced by the extent of cardiac involvement, with mortality rates approaching 100% in cases of severe cardiogenic shock [1,2].

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We present the case of a 24-year-old male patient admitted to the Multidisciplinary Intensive Care Unit of Prince Moulay Abdellah Provincial Hospital in Salé following severe, isolated aluminum phosphide poisoning. The poisoning caused profound cardiogenic shock secondary to acute toxic myocarditis, from which the patient made a favorable recovery within a few days.

CASE REPORT

We describe the case of Mr. M.R., a 24-year-old man with no known medical, surgical, or psychiatric history, admitted to the emergency department in a state of shock and coma, 2.5 hours after ingesting two Phostoxin tablets (6 g total) in a suicide attempt following family conflict.

Twenty minutes after ingestion, he developed diffuse abdominal pain with associated vomiting. As he rapidly developed altered consciousness, his family rushed him to the emergency department, giving him milk beforehand in an attempt to mitigate symptoms. Upon arrival, the family presented the Phostoxin® tablets they had found near his bed.

At triage, the patient was tachycardic at 130 bpm, hypotensive (BP 60/30 mmHg), and exhibited signs of peripheral hypoperfusion including cold extremities, mottling, prolonged capillary refill, and oliguria (50 mL over 2 hours). Respiratory examination revealed a respiratory rate of 28 breaths per minute with oxygen saturation at 90% on pulse oximetry. Lung auscultation was unremarkable. Neurologically, his Glasgow Coma Scale (GCS) was 7 (motor response 4, eye opening 2, verbal response 1), with no signs of trauma or seizure activity. Pupils were equal and reactive; the neck was supple. He was afebrile at 36.7°C, and capillary blood glucose was 1.15 g/L.

Immediate management in the shock room included securing the airway through rapid-sequence orotracheal intubation, mechanical ventilation, isotonic saline fluid resuscitation, and administration of norepinephrine at 0.7 µg/kg/min alongside dobutamine at 15 µg/kg/min, targeting a mean arterial pressure (MAP) > 65 mmHg. A bedside FAST ultrasound revealed global left ventricular hypokinesia, consistent with cardiogenic shock due to aluminum phosphide ingestion. Urinary and gastric catheterization were also performed. After stabilization, the patient underwent toxicology screening and a cranial CT, which was unremarkable, and was then transferred to the intensive care unit.

In the ICU, invasive arterial pressure monitoring was established via a right radial artery catheter, along with central venous access via the right internal jugular vein for vasopressor infusion. Arterial blood gas analysis revealed severe metabolic acidosis with a pH of 6.9, $PaCO_2$ of 46 mmHg, bicarbonate (HCO_3^-) of 5 mEq/L, and lactate of 16 mmol/L. Laboratory workup showed hemoglobin at 11 g/dL, white blood cell count of 9,000/mm³, and platelets at 140,000/mm³. Electrolyte analysis revealed sodium 140 mEq/L, potassium 4.1 mEq/L, bicarbonate reserves at 6 mEq/L. Renal function was slightly impaired, with urea at 0.6 g/L and creatinine at 17 mg/L (estimated GFR 66 mL/min via Cockcroft formula). Troponin I was markedly elevated at 4.5 ng/mL (>100 times the normal range), LDH was 485 U/L, ALT was 110 IU/L (×3 normal), and AST was 280 IU/L (> ×9 normal), indicating hepatic cytolysis.

Transthoracic echocardiography revealed global left ventricular hypokinesia with an estimated ejection fraction of 15% (Simpson method). Right ventricular size and function were normal, with no elevated filling pressures or significant valvular disease. Pericardium was free of effusion. ECG showed sinus tachycardia without repolarization abnormalities. Chest X-ray revealed a right lower lobe opacity. Unfortunately, the toxicological samples were lost in transit and not processed by the Poison Control Center.

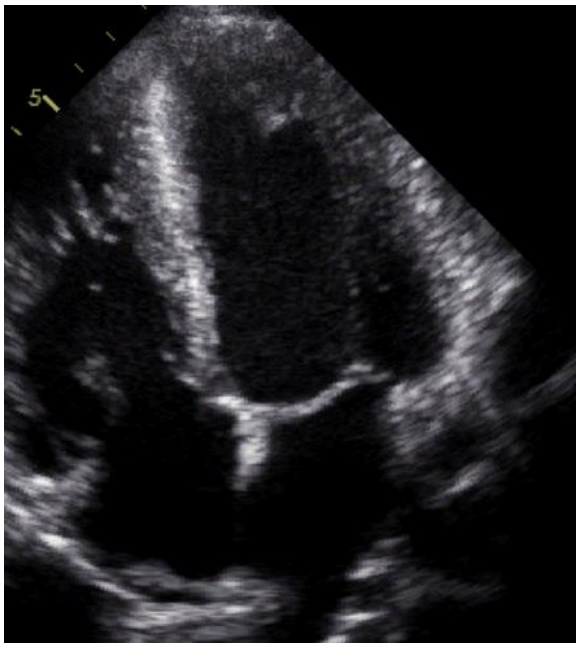


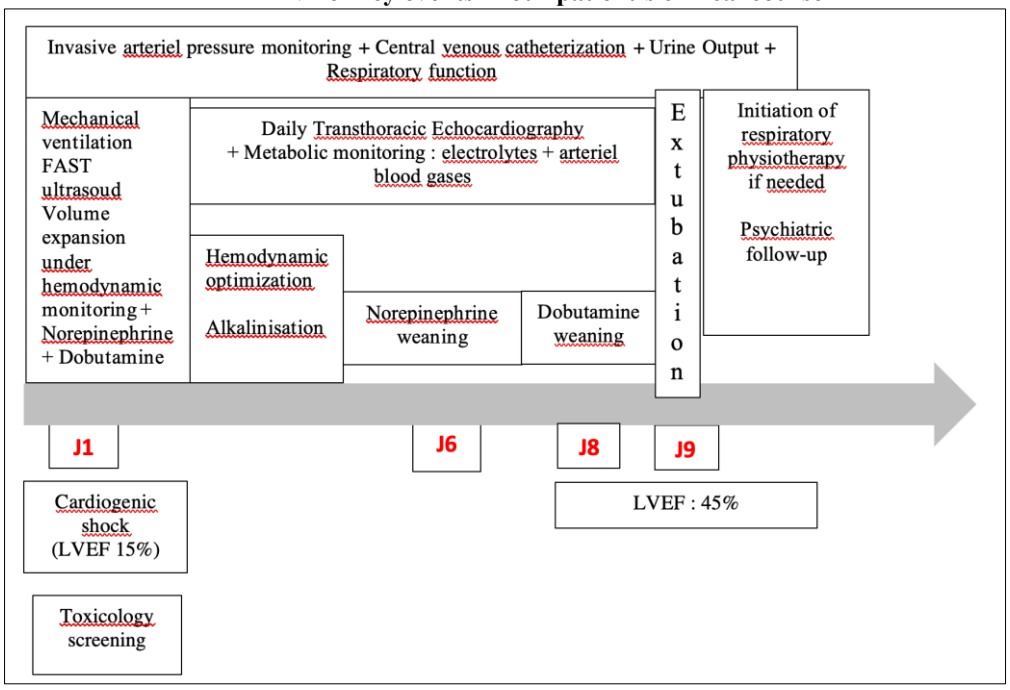
Figure 1: Apical four-chamber view on Transthoracic echocardiographic in our patient

CLINICAL COURSE

Under continuous administration of dobutamine at 20 µg/kg/min and norepinephrine at 1 µg/kg/min, with a target MAP of 70 mmHg, along with alkalinization using 14‰ sodium bicarbonate solution and mechanical ventilation, the patient began to show progressive improvement.

Urine output resumed after 48 hours, reaching 1 cc/kg/hour. Arterial blood gas normalized progressively, with pH rising to 7.34, $PaCO_2$ decreasing to 36 mmHg, bicarbonate increasing to 21 mEq/L, and lactate levels dropping to 1.4 mmol/L. Norepinephrine was weaned off on Day 6, dobutamine on Day 8, and the patient was extubated on Day 9. A follow-up echocardiogram performed on Day 10 revealed recovery of the left ventricular ejection fraction (LVEF) to 45%. The patient was transferred to a psychiatric hospital on Day 12 for ongoing psychological support.

TIMELINE of key events in our patient's clinical course



DISCUSSION

Aluminum phosphide is a rodenticide frequently used in suicide attempts in countries such as India, Iran, and parts of North Africa. The toxic dose in adults ranges from 150 to 500 mg [1,2]. Toxicity results from the release of phosphine gas (PH_3) upon contact with gastric moisture. Phosphine is rapidly absorbed within 10 to 15 minutes, making any delay in gastrointestinal decontamination largely ineffective unless initiated immediately. Phosphine exerts its toxic effects mainly by inhibiting mitochondrial cytochrome-c oxidase, thereby blocking oxidative phosphorylation. This leads to cytotoxic cellular hypoxia, massive oxidative stress, energy failure at the cellular level, and ultimately, severe multiorgan failure [3].

The clinical presentation is variable and generally begins within 10 to 15 minutes of ingestion. It often includes gastrointestinal symptoms (nausea, vomiting, abdominal pain), neurologic disturbances (altered consciousness, seizures), respiratory manifestations (dyspnea, acute respiratory distress), and most critically, cardiovascular compromise. Cardiac involvement may include toxic myocarditis, heart failure, and arrhythmias, progressing to refractory cardiogenic shock and death [4].

In this case, the patient presented with immediate cardiogenic shock, a LVEF of 15%, and significantly elevated cardiac enzymes, all indicating severe myocardial injury. Such left ventricular dysfunction is commonly seen in severe aluminum phosphide poisoning. Several studies report mortality exceeding 80% when cardiogenic shock is accompanied by severe metabolic acidosis (pH < 7.0) [4,5]. In our patient, arterial blood gas showed profound lactic acidosis, reflecting severe tissue hypoperfusion. Notably, early vomiting, which likely reduced systemic absorption of the toxin, may have contributed to the reversibility of myocarditis in this case.

Diagnosis is based on clinical history, presentation (cardiogenic shock in a young patient without underlying cardiac disease), and toxicology testing (to detect phosphine or its metabolites). In this case, although toxicology samples were lost, the diagnosis was supported by family history and the patient's subsequent admission of ingestion.

Management of aluminum phosphide poisoning is primarily supportive, as no specific antidote has proven effective to date [3]. Gastric lavage using saline is discouraged, as it may accelerate phosphine gas release [7]. The optimal solution for decontamination remains a topic of debate. Agents such as sodium bicarbonate, potassium permanganate, and even coconut oil have been suggested. Coconut oil, in particular, has been proposed due to its immiscibility with water and its theoretical ability to inhibit phosphine gas release. However, there is no conclusive evidence demonstrating the superiority of any solution for gastrointestinal decontamination. In this case, gastric lavage was not performed as the patient presented 2.5 hours after ingestion, beyond the window of likely benefit [4,7].

Treatment of cardiogenic shock from aluminum phosphide poisoning demands early, aggressive resuscitation as soon as cardiovascular compromise is evident. Any delay in initiating therapy may lead to irreversible cellular hypoxia and rapid multiorgan failure [8]. The primary goal is to restore adequate tissue

perfusion by optimizing arterial pressure and cardiac output.

In our patient, early administration of dobutamine (15 µg/kg/min) and norepinephrine (0.7 µg/kg/min) corrected profound hypotension and progressively improved systemic perfusion. These medications were titrated under continuous monitoring via radial arterial catheter. Dobutamine, a β1-agonist inotrope, was chosen for its ability to enhance myocardial contractility with a relatively low arrhythmogenic risk. Norepinephrine, primarily an α1-agonist, was used to increase systemic vascular resistance in cases of refractory hypotension, aiming to maintain MAP > 65 mmHg.

Initial fluid resuscitation was performed with isotonic saline, as lactated Ringer's was not available. Fluids were administered cautiously and guided by echocardiographic assessment. The absence of right ventricular dilation and low filling pressures, along with clinical findings, supported moderate volume expansion without risk of overload, effectively improving preload.

Severe metabolic acidosis was managed with 14‰ sodium bicarbonate solution. This intervention likely contributed to mitigating intracellular phosphine toxicity and supported myocardial recovery. Jaiswal's 2009 study demonstrated that although bicarbonate does not neutralize PH_3 or inhibit its release, it may counteract intracellular acidosis and improve outcomes by acting as an extracellular buffer, stabilizing membranes, limiting intracellular diffusion of the toxin, and enhancing renal elimination [8].

Due to deep coma (GCS 7) and hypoxia, orotracheal intubation was performed at admission. Protective mechanical ventilation was instituted with moderate PEEP and tailored FiO_2 to ensure adequate oxygenation. The patient was successfully weaned from ventilation on Day 9, after hemodynamic and metabolic stabilization.

Positive clinical progression was marked by the return of diuresis at 48 hours, gradual correction of lactic acidosis, weaning from norepinephrine by Day 6 and dobutamine by Day 8, and recovery of LVEF to 45% by Day 10. These findings support the potential reversibility of toxic myocarditis when early, intensive critical care is provided.

Several pharmacologic interventions have been proposed in the literature. N-acetylcysteine (NAC), a potent antioxidant known to replenish intracellular glutathione, has shown cardioprotective effects in animal studies by reducing phosphine-induced oxidative myocardial injury [11]. However, a 2016 case-control study by Taghaddosinejad failed to demonstrate a mortality benefit in humans [12].

Triiodothyronine (T3), administered at 3 µg/kg in animal models, has demonstrated efficacy in aluminum phosphide-induced myocarditis by improving electrophysiological parameters and reducing oxidative stress markers. T3 may also support mitochondrial activity and ATP production, enhancing cellular survival. To date, no human studies have evaluated its use [13].

Trimetazidine, a metabolic anti-ischemic and cardioprotective agent, has been suggested for its ability to preserve oxidative metabolism and limit oxidative stress. However, it has only been used in mild, non-severe toxic myocarditis cases, and current evidence does not support routine use [14].

High-dose insulin euglycemia therapy has shown promising results in toxic myocarditis from aluminum phosphide, likely due to improved cardiac contractility through modulation of energy metabolism and intracellular calcium dynamics [15].

Magnesium sulfate may offer benefit in treating aluminum phosphide-induced arrhythmias (tachyarrhythmias or bradyarrhythmias) through its membrane-stabilizing properties, regardless of serum magnesium levels [16].

In the most severe cases, mechanical circulatory support may be considered. Intra-aortic balloon pump (IABP) has been used successfully in cases of refractory cardiogenic shock, offering temporary hemodynamic support and facilitating myocardial recovery [10,17]. However, its availability is limited in many developing countries due to the need for cardiac surgery infrastructure.

Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) has gained traction in critical cases. Recent observational data suggest significantly reduced short-term mortality when implemented early alongside conventional treatments, although this requires rapid transfer to equipped centers [18]. In Morocco, access to ECMO remains limited.

In this case, mechanical support was not required due to early hemodynamic stabilization with inotropic therapy, underscoring the importance of intensive management during the initial hours post-ingestion.

CONCLUSION

Acute aluminum phosphide (Phostoxin®) poisoning constitutes a true medical emergency capable of inducing profound cardiac failure. This case demonstrates that even severe myocardial injury can have a favorable outcome if intensive, rapid, and appropriate management is initiated. Such management relies on early and aggressive resuscitation, including

targeted volume expansion, prompt use of vasopressors and inotropes (dobutamine and norepinephrine), correction of metabolic acidosis through alkalinization, and mechanical ventilatory support. Prognosis is determined by the timeliness of diagnosis and intervention. Furthermore, comprehensive care must include psychiatric evaluation and follow-up to prevent recurrence.

Conflicts of Interest: The authors declare that they have no conflicts of interest related to this article.

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