CASE REPORT

Fatal Aluminum Phosphide Poisoning Following Improper Hotel Fumigation: A Case Report

Hatalı Otel İlaçlamasına Bağlı Fatal Alüminyum Fosfit Zehirlenmesi: Olgu Sunumu

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ABSTRACT

Objective: *To report a fatal case of unintentional inhalational aluminum phosphide (ALP) poisoning and to highlight the diagnostic challenges, the lack of a specific antidote and the necessity for stricter regulatory oversight.*

Material and Method: *The case of a 38-year-old male, who was unintentionally exposed to ALP-containing pest control products at a hotel in Türkiye, was analyzed. The clinical progression, symptoms upon admission, and the medical management provided were reviewed.*

Results: *The patient presented with nonspecific symptoms such as dyspnea, nausea, diaphoresis, hypotension, and tachycardia. Despite aggressive supportive care, the patient's condition rapidly deteriorated into severe metabolic acidosis and cardiovascular collapse, resulting in a fatal outcome consistent with the known 70–100% mortality rate of ALP toxicity.*

Conclusion: *Inhalational ALP poisoning is highly lethal and difficult to diagnose early due to nonspecific symptoms. The absence of a specific antidote underscores the importance of strict regulatory oversight of fumigation practices and increased public awareness to prevent future catastrophic incidents.*

Keywords: *Aluminum Phosphide Poisoning, Fumigation, Substance exposure, Emergency, Pesticides, Toxic Inhalation.*

ÖZET

Amaç: *Bu çalışma, kazara solunumsal alüminyum fosfit (ALP) zehirlenmesine bağlı fatal bir olguyu sunmayı; tanısal zorluklara, spesifik bir antidotun eksikliğine ve daha sıkı yasal denetim gerekliliğine dikkat çekmeyi amaçlamaktadır.*

Gereç ve Yöntem: *Türkiye'de bir otelde, alüminyum fosfit içeren haşere kontrol ürünlerine kazara maruz kalan 38 yaşındaki erkek hasta analiz edildi. Hastanın klinik seyri, başvuru anındaki semptomları ve uygulanan tıbbi tedavi gözden geçirildi.*

Bulgular: *Hasta; dispne (nefes darlığı), bulantı, diyaforez (aşırı terleme), hipotansiyon ve taşikardi gibi non-spesifik semptomlarla başvurdu. Agresif destekleyici bakıma rağmen hastanın durumu hızla kötüleşerek şiddetli metabolik asidoz ve kardiyovasküler kollapsa ilerledi. Bu tablo, ALP toksisitesinin %70-100 arasında bilinen yüksek mortalite (ölüm) oranıyla uyumlu şekilde fatal sonuçlandı.*

Sonuç: *İnhalasyon yoluyla alüminyum fosfit zehirlenmesi oldukça ölümcüldür ve spesifik olmayan semptomlar nedeniyle erken teşhis edilmesi zordur. Spesifik bir antidotun bulunmayışı; fumigasyon (ilaçlama) uygulamaları üzerinde sıkı bir yasal denetimin ve gelecekteki benzer felaketleri önlemek amacıyla kamuoyu farkındalığının artırılmasının önemini vurgulamaktadır.*

Anahtar Kelimeler: *Alüminyum Fosfit Zehirlenmesi, İlaçlama, Kimyasal Maruziyet, Acil, Pestisit Zehirlenmesi, Toksik İnhalasyon.*

INTRODUCTION

Aluminum phosphide (ALP) is a potent fumigant, known as 'rice tablets' due to its widespread use in grain preservation. (1). When it comes into contact with moisture, ALP releases phosphine gas, a highly toxic and colorless (2). The primary mechanisms of toxicity include inhibition of mitochondrial oxidative phosphorylation, the generation of reactive oxygen species and the development of severe metabolic acidosis with subsequent cardiovascular collapse (3). Typically manifests with symptoms such as nausea, vomiting, abdominal pain, dyspnea, shock, arrhythmias and rapidly progressive multiorgan failure. A dose of 0.15–0.5 g is generally considered as the fatal dose while standard tablets typically weigh 3 grams each and no specific antidote exists (4). Here, we present the case of a 38-year-old man who, along with three family members, died after unintentional inhalational exposure to aluminum phosphide used during hotel pest control.

CASE PRESENTATION

A 38-year-old male patient presented to the emergency department with dyspnea, nausea, dizziness and excessive sweating. The patient had no known past medical history, took no regular medications, and denied alcohol or recreational drug use. He and his family had arrived in Türkiye from abroad four days ago. All family members reported consuming common meals including baked potatoes (kumpir) and mussels. He also stated that he did not notice any unusual odor in the hotel room. The symptoms had begun approximately two days prior and were shared by all four members of his family.

His vital signs were: blood pressure 93/77 mmHg, heart rate 134 beats/min, oxygen saturation 99% and normal body temperature. Physical examination revealed diaphoresis and mild confusion. On auscultation, diffuse bilateral coarse crackles were present. The patient exhibited orthopnea, and peripheral edema was absent. ECG demonstrated sinus tachycardia with no ischemic changes.

Arterial blood gas revealed pH 7.34, PCO_2 25.7 mmHg, and HCO_3^- 16 mmol/L, metabolic acidosis with respiratory compensation. Laboratory studies were notable for creatinine 2.23 mg/dL, urea 55 mg/dL, ALT 40 U/L, AST 23 U/L, CRP 13 mg/L, d-dimer 1.36 mg/L, and a troponin level of 105 ng/L. CT pulmonary angiography was performed due to the elevated d-dimer and worsening dyspnea, and pulmonary embolism was excluded.

His oxygen saturation fell to 80–84% despite a reservoir mask. Due to intolerance of non-invasive mechanical ventilation, he subsequently required endotracheal intubation. A repeat arterial blood gas revealed severe mixed acidosis with pH 6.48, PCO_2 126 mmHg, and HCO_3^- 8 mmol/L. At this stage, he was started on intravenous sodium bicarbonate.

Patient had three cardiac arrests, initially presenting as pulseless electrical activity. Cardiopulmonary resuscitation was administered for 20, 15, and 5 minutes, respectively, with return of spontaneous circulation achieved after each episode. He developed two episodes of ventricular fibrillation, both successfully treated with defibrillation. Post-resuscitation, the patient remained profoundly hypotensive with blood pressure levels hovering around 60/35 mmHg, indicating severe cardiogenic shock. To maintain hemodynamic stability, triple inotropic and vasopressor support was initiated, comprising norepinephrine (0.1–1.0 mcg/kg/min), epinephrine (0.05–0.5 mcg/kg/min), and dobutamine (5–20 mcg/kg/min). Urgent renal replacement therapy was commenced in the emergency department to manage refractory metabolic acidosis and electrolyte imbalances. After initial stabilization, the patient was transferred to the intensive care unit for advanced monitoring and organ support.

Upon admission to the intensive care unit, the patient's liver enzymes were markedly elevated (ALT 494 U/L, AST 593 U/L), and there was significant rhabdomyolysis (CK 15,971 U/L, LDH 1480 U/L). Renal function deteriorated further with creatinine 4.67 mg/dL and urea 99 mg/dL. Cardiac biomarkers increased, with troponin I 23,077.9 ng/L. Laboratory investigations further revealed significantly elevated procalcitonin >96.6 µg/L, Na 147 mmol/L, K 3.3 mmol/L, and Ca 7.5 mg/dL. Hematologic parameters demonstrated severe leukocytosis (WBC 22.68×10^9/L), thrombocytopenia (platelet count 30×10^9/L), and neutrophil predominance (NEU% 83.4%). Coagulation studies were markedly deranged, showing prolonged APTT (58 s), elevated D-dimer (35.2 mg/L), high fibrinogen (597 mg/dL), and prolonged PT (22.5 s) with an INR of 1.97. These findings were consistent with disseminated intravascular coagulation (ISTH DIC score = 7) and severe multiorgan dysfunction.

The patient died on the fourth day of his intensive care admission. It was reported that other family members, who had been admitted to different healthcare facilities, also died. The incident prompted public health investigation. Subsequent inquiries revealed that the hotel had undergone pest control treatment three days prior to the onset of symptoms, and the agent used was suspected to contain aluminum phosphide. Postmortem examination confirmed that the deaths were related to acute aluminum phosphide poisoning.

DISCUSSION

ALP is a highly toxic, solid and cheap pesticide commonly used to protect stored grains. When exposed to moisture, undergoes a chemical reaction that releases phosphine gas, which is the primary agent responsible for its lethal systemic toxicity (4). The primary mechanism of toxicity is the inhibition of mitochondrial cytochrome c oxidase, which disrupts oxidative phosphorylation and cellular respiration. This leads to cellular hypoxia and marked oxidative stress due to the accumulation of reactive oxygen species, resulting in widespread cellular and tissue injury (5).

Early symptoms are often nonspecific, including gastrointestinal distress (nausea, vomiting, and abdominal pain) along with autonomic signs such as dizziness, tachycardia, dyspnea and diaphoresis. Patients often develop severe metabolic acidosis, early cardiovascular collapse, and multiorgan failure. In our case, the patient demonstrated this fulminant progression. Overall mortality rates range from 70% to 100% (6).

Management is challenging because no specific antidote exists. Early aggressive resuscitation remains the most important factor for survival. Supportive care includes correction of acidosis, early resuscitation with fluids, vasopressor therapy (triple inotropic support if required) and mechanical ventilation (7). Although hemodialysis is not effective for phosphine clearance, it is vital for managing metabolic acidosis and renal failure (6). Magnesium sulfate may help stabilize cell membranes and counteract free radical injury. N-acetylcysteine, when added to standard treatment, has been associated with improved survival time in some studies (2).

In this case, the family had initially been discharged from another hospital with a presumed diagnosis of food poisoning, as no acute abnormality in laboratory test was detected. After returning to the hotel, they were likely exposed to a higher concentration of gas, leading to rapid deterioration. Inhalational cases can be difficult to identify because phosphine may lack a characteristic odor and exposure history may be unclear. Silver nitrate paper testing can theoretically support diagnosis by detecting phosphine in gastric aspirate or exhaled air (1). This case highlights the need for strict regulatory control, as improper fumigation practices can lead to unintentional mass poisoning.

CONCLUSION

Aluminum phosphide poisoning is a rapidly progressive and highly fatal condition, exposure may occur unknowingly through inhalation. This case demonstrates how an unrecognized fumigation source can lead to catastrophic outcomes, including multiorgan failure and death. Because early symptoms are nonspecific and no antidote exists, prompt recognition and aggressive supportive management are essential. The findings highlight the need for greater clinical awareness and strict regulation of fumigation practices to prevent similar cases.

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