

# Case Report: Successful Management of an Aluminum Phosphide Poisoned Patient Following Ventricular Tachycardia



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## ABSTRACT

Aluminum Phosphide (ALP) poisoning is regarded as fatal; however, it is commonly used in developing countries. In some countries, such as India and Iran, it is still being used to protect grain against pests at home. As yet, no known antidote is reported to be effective against ALP poisoning. ALP acts through blockage of cell respiration, alteration of cell permeability, and induction of oxidative stress. Some factors can worsen the prognosis of its intoxication, which include hyperglycemia on admission, ingestion of fresh tablets, cardiac abnormality, especially Ventricular Tachycardia (VT), severe metabolic acidosis, cardiogenic shock and other types of shocks, electrolyte disturbances, end-organ damage, and a need for mechanical ventilation. ALP kills its victims by induction of multi-organ failure.

In this study, we controlled the patient's VT using MgSO<sub>4</sub>, electric shock, amiodarone, and lidocaine. We also encountered the cardiogenic shock using norepinephrine, High-Dose Insulin (HDI), antioxidants, sodium bicarbonate, and normal saline. Moreover, myocardial ischemia, recurrent VTs, and adverse cardiac effects were protected via tight control of magnesium and other electrolytes.

## 1. Introduction

**A**

luminum Phosphide (ALP), commonly known as rice pill or wheat pill in Iran, an insecticide and rodenticide, which is used in the grain processing industries, is commonly recognized for its dramatic human intoxication, as it kills 37-100% of its vic-

tims [1]. With such a high mortality rate and its wide-spread use in developing countries, such as India and Iran, ALP poisoning is known as the main cause of death in toxicology wards and Intensive Care Units (ICUs) [2]. When ALP enters the body through ingestion or inhalation, it interacts with the water or hydrochloric acid molecules and produces the phosphine gas. This substance can act through blockage of the cell respiration, inhibi-

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tion of cytochrome, protein synthesis blockage, production of reactive oxygen species and alter the permeability of the cell membranes.

These mechanisms affect the heart, kidneys, liver, as well as lungs and lead to end-organ failures. Cardiac toxicity includes all kinds of arrhythmias e.g. Ventricular Tachycardia (VT), electrocardiogram (ECG) abnormalities, Heart Failure (HF), and myocarditis [3, 4]. VT in these patients is treated by amiodarone and Magnesium Sulfate (MgSO<sub>4</sub>), but it is still known as an indicator of poor prognosis among ALP poisoned patients [5]. Several case report studies have indicated the successful cardioversion from VT to the sinus rhythm using MgSO<sub>4</sub> and amiodarone [6]. Although using lidocaine could cardiovert patients with VT rhythm to sinus rhythm, in some other studies, it could not be cardioverted [7]. As there is a limited number of surveys to address the effectiveness of these drugs, efficacy and failure rate of them are under question.

Many studies have reported hypomagnesemia following ALP intoxication and suggested maintaining a eumagnesemic state as a cornerstone for the treatment of cardiac manifestations of ALP intoxication. It can affect through restoring the glutathione storage and suppressing ALP-induced oxidative stress. Due to its autonomic modulatory, antioxidant, and membrane stabilizer effects, it could significantly reduce the mortality rate in several human studies [8, 9].

This study presented a rare case of successful management of an Aluminium phosphide poisoned patient following VT and cardiogenic shock, who was discharged successfully from the hospital one month after intoxication with negligible complication.

### Case Presentation

A 13-year-old girl, with no history of surgical, medical, or psychological disorders, was admitted to the emergency ward of "Sina Hospital" (toxicology center of North-West Iran). Her mother had a complaint about the gradual loss of her daughter's consciousnesses, following vomiting and diarrhea after ingestion of two ALP tablets (6 g) at the school. The purpose of the act was uncertain. At the emergency ward, a blood pressure of 95/50mmHg, pulse rate of 115 bpm, a body temperature of 37°C, and blood sugar of 198mg/dL were recorded for her. Due to the loss of consciousness (GCS:7), she underwent Rapid sequence intubation; thus, we could secure her airways and prevent aspiration. At the emergency ward, sodium bicarbonate (100 mEq), calcium gluconate (1 g), vitamin C (1 g), vitamin D (300,000 IU),

vitamin E (400 IU), KCl (20 mEq), and serum normal saline (1 L) were added to her orders.

Subsequently, she was admitted to the toxicology ICU with the diagnosis of ALP poisoning. During her stay in ICU, she was sedated with fentanyl and ventilated with a mechanical ventilator. Because of severe hypovolemic and cardiogenic shock, we tried to correct metabolic acidosis and shock with norepinephrine (10 µg/min), hydrocortisone (200 mg stat and 100 mg every 6 h), normal saline (1 L stat and 1 L every 4 h), sodium bicarbonate (100 mEq in each 1 l serum), N- acetylcysteine (300 mg/kg in 1 L serum dextrose water 5% daily), magnesium sulfate (1 g every 6 h), calcium gluconate (1 g every 6 h on the first day and 1 g every 4 h in next days), and High-Dose Insulin (HDI) (1 u/kg regular insulin plus 1 g/kg glucose 50% stat and 0.5 u/kg/h regular insulin plus 0.5 g/kg/h glucose 50%) infusion via central venous line. Also, because of rapidly progressive hypovolemic shock, three units of iso-group Fresh Frozen Plasma (FFP) was administered on the first day.

Eight hours after admission, she experienced pulseless Ventricular Tachycardia (VT). Therefore, a 200 J unsynchronized electric shock was given to her. Also, lidocaine (100 mg, stat), 500cc normal saline serum, and 100 mEq sodium bicarbonate were infused; but an hour after recovery of the first VT, she experienced the second VT; thus, another 200 J unsynchronized electric shock was given again. Following the second electric shock, the sinus rhythm and her pulses came back. Afterward, amiodarone (150 mg in first 10 min, 360mg during the next 6 h and 540 mg during subsequent 18 h) was prescribed.

Ten days after admission, we noted inversion of the T-waves in her daily ECGs (leads: II, III, aVf, V3, V4, V5, V6) followed by Non-Sustained VT (NSVT). We kept her serum potassium levels at 4mEq/L, magnesium levels at 2mg/dL, and then a cardiology counsel was requested.

The cardiologist started 25mg of metoprolol, 20mg of atorvastatin, and 80mg of aspirin on a daily basis because the myocardium was suffering from ALP-induced hypoxia. Afterward, it was observed that her body temperature rose to 40°C, heavy tracheal discharges were produced, and coarse crackles were heard from her lungs. Chest X-ray was taken and consultation with the infectious diseases service was asked for, and antibiotic therapy was started, as they suspected Ventilator-Associated Pneumonia (VAP)-induced septicemia. We only detected enterococcus in her urine culture and Methicillin-Sensitive Staphylococcus Aureus (MSSA) in her blood culture for two subsequent times. In her tracheal aspi-

**Table 1.** Brief results of the lab tests of the Aluminum Phosphide (ALP) intoxicated patient

Test Name	Admission	10 <sup>th</sup> Day	Last Day
WBC (/mm <sup>3</sup> )	19100	12100	4700
RBC (/mm <sup>3</sup> )	4.84	3.16	3.4
Hb (g/dL)	14.8	9.4	9.8
Hct (%)	43.2	27.9	30.5
MCV (fL)	89.3	87.7	89.7
MCH (pg)	30.6	29.6	28.8
MCHC (g/dL)	34.3	33.7	32.1
Plt (/mm <sup>3</sup> )	134000	151000	238000
RDW CV	13.6	12	12.5
MPV (fL)	10.1	9.4	9.5
PT (sec)	16.8	12.8	12
INR	1.9	1.2	1.2
PTT (sec)	44	79	25
Urea (mg/dL)	35	26	16
Cr (mg/dL)	2.1	1.5	1
AST (IU/L)	60	42	13
ALT (IU/L)	18	19	15
Alk.P (IU/L)	92	90	104
CK (U/L)	1768	1070	39
CK-MB (IU/L)	124	56	17
LDH (IU/L)	788	645	474
Na (meq/L)	150	144	139
K (meq/L)	2.8	3.8	4.4
Ca (mg/dL)	7.4	8	9
Mg (mg/dL)	1.1	1.7	2.4
CTNI (qualit)	Negative	Negative	-
U/C	Enterococcus, spp		
B/C	Staphylococcus Aureus (MSSA)		
T/C	No bacteria growth in tracheal culture		

ration culture, no bacteria growth was detected. During her admission, serial Arterial Blood Gas tests (ABGs)

were taken to control the metabolic acidosis, ventilation status and to set the mechanical ventilator. Her total cal-

cium levels were either kept above 8-9, or her ionized calcium kept above 1.2 mg/dL using calcium gluconate. On day 15, two units of the packed red blood cells were administered as her hemoglobin levels fell below 7g/dL.

On day 17, we tapered the sedatives as her ABGs, and clinical findings were improved. The next day she was extubated.

She was discharged on her 27<sup>th</sup> day of admission, after assuring normal lab test results and clinical findings (Table 1). However, 25 days after discharge, she was admitted with the diagnosis of mild tracheal stenosis and undergone tracheal dilation (Broncoplasty) with rigid bronchoscopy and successfully discharged again. The lab tests and clinical findings were normal in her 24-months follow-up visits and the patient did not have any complication.

## Discussion

ALP poisoning is regarded as fatal; however, it is commonly used in developing countries. In some countries, such as India and Iran, it is still being used to protect grain against pests at home. Some factors are known to improve the prognosis of ALP intoxication, which include younger age, ALP gas aspiration, drinking of ALP solution instead of tablet ingestion, vomiting following ingestion, and/or low-dose ingestion. On the other hand, some factors can worsen the prognosis of its intoxication, including hyperglycemia on admission, ingestion of fresh tablet, cardiac abnormality, especially VT, severe metabolic acidosis, cardiogenic shock and other types of shocks, electrolyte disturbances, other end-organ damages, and a need for mechanical ventilation [2, 4-7]. By induction of multi-organ failure, ALP kills its victims. Furthermore, the myocardium is regarded as one of the most critical targets of ALP. Cardiac toxicity induced by ALP includes all kinds of arrhythmias e.g. VT, ECG abnormalities, HF, and myocarditis, of which VT is considered as a factor indicating poor prognosis and death.

In this study, we controlled the patient's VT using electric shock, amiodarone, and lidocaine, in addition to previously prescribed continuous prophylactic MgSO<sub>4</sub> [10, 11]. We also encountered the cardiogenic shock using norepinephrine, HDI, antioxidants, sodium bicarbonate, and normal saline. However, myocardial ischemia, recurrent VTs, and adverse cardiac effects were protected via tight control of magnesium and other electrolytes [10, 11].

Overall, our case report could indicate the effectiveness of the simultaneous usage of electric shock, amiodarone,

lidocaine, and MgSO<sub>4</sub>. Additionally, it was found that prescribing MgSO<sub>4</sub> and tight control of serum electrolytes and acid-base disturbances are beneficial for the management of ALP poisoned patients. However, more clinical studies are needed to prove or decline these suggestions.

To conclude, it is only recommended that these drug interventions should be prescribed based on patients' clinical conditions, in addition to the treatment of patients' dysrhythmias with standard protocols.

## Ethical Considerations

### Compliance with ethical guidelines

All ethical principles were considered in this article. All procedures performed in this study were in accordance with the ethical standards of the institutional and or national research committee and with the 1964 Helsinki declaration and its later amendments.

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### Author's contributions

All authors contributed to all process of the article preparation. Also, all authors reviewed and revised the manuscript several times.

### Conflict of interest

The authors declare no conflict of interest

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