

## CASE REPORT

# Inhalational Toxicity of Aluminum Phosphide as an Ongoing Concern; a Report of Two Cases

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Received: August 2019; Accepted: October 2019; Published online: 17 November 2019

**Abstract:** Acute aluminium phosphide (ALP) poisoning is an extremely lethal poisoning. Ingestion is usually suicidal in intent, uncommonly accidental and rarely homicidal. Unfortunately, the absence of a specific antidote results in very high mortality and the key to successful treatment is in rapid decontamination and institution of resuscitative measures. Phosphine gas is highly toxic, and fatality is expected even several hours after continuous exposure. However, intensive supportive treatments may be lifesaving in some cases. Here, two cases of accidental inhalation intoxication with ALP are reported. One patient was discharged and another suffered cardiac arrest during treatment.

**Keywords:** Aluminum phosphide; inhalation; inhalation exposure; gas poisoning

**Cite this article as:** Shafahi A, Mostafazadeh B, Dadpour B. Inhalational Toxicity of Aluminum Phosphide as an Ongoing Concern; a Report of Two Cases. Arch Acad Emerg Med. 2019; 7 (1): e69.

## 1. Introduction

Aluminium Phosphide (ALP) poisoning as a suicidal attempt or accidental poisoning has become notorious in Iran over the past decades. Phosphine gas is a colorless, flammable and highly toxic substance, which is released when Phosphide comes in contact with water (moisture) or acid, with an odor of garlic or decaying fish (1-3). By inhibition of cytochrome oxidase c and inducing oxidative stress, phosphine causes severe mitochondrial dysfunction, tissue hypoxia, and finally multiple organ failure (2, 4).

Common clinical manifestations include gastrointestinal symptoms, progressive metabolic acidosis, refractory hypotension, and dysrhythmia (4). Most reports of ALP poisoning are following ingestion of ALP. Unfortunately, phosphine gas inhalation also may lead to life threatening toxicity, even in large areas (4-6).

To date, no specific antidote has been defined for this rapidly fatal poisoning and supportive measures are mainly recommended. Immediate diagnosis in addition to continued in-

tensive monitoring and supportive measures may be lifesaving. We aimed at reporting this incident from a public health emergency viewpoint as this type of (ALP) poisoning may re-occur (although is relatively uncommon), at least in countries where ALP is still used by general population.

## 2. Cases presentation:

A 60-year-old woman (case one), with her 29-year-old daughter (case two) were referred to a regional hospital and then transferred to clinical toxicology department of Imam Reza Hospital, Mashhad, Iran, with upper abdominal discomfort, nausea, and with primary suspicion to food poisoning.

### 2.1. Case one

Case one suffered from shortness of breath, weakness, dizziness, restlessness, and nausea on admission. Vital signs at arrival included: SBP / DBP = 100/40 mmHg, RR = 20 / minute, O<sub>2</sub> Saturation= 93% on room air, and PR = 90 / minute. The patient had no fever and was alert and able to answer questions. There was no history of any drug addiction. She had a history of hypertension and diabetes. QTC prolongation was the most important finding of her Electrocardiogram (ECG). Hydration, oxygen therapy, cardiac monitoring and other supportive measures were performed. Red Blood cells and

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**Table 1:** Laboratory findings of case one

Laboratory parameters	First day	Second day
Blood Sugar (mg/dl)	251	241
Creatinine (mg/dl)	1.1	1.2
Urea (mg/dl)	31	43
Sodium (mg/dl)	140	141
Potassium (mg/dl)	3.2	3.2
Calcium, Total (mg/dl)	8.9	
Alanine Aminotransferase (ALT) (units/L)	46	37
Aspartate aminotransferase (AST) (units/L)	89	61
Alkaline Phosphatase (Alp) (units/L)	239	168
Creatine phosphokinase (units/L)	124	145
Cardiac troponin	89.30	-
Magnesium (mg/dl)	2.4	-
Serum Cholinesterase	7356	-
RBC Cholinesterase	5.2	-
<b>Complete Blood Count</b>		
Red Blood Cell $\times 10^6$	4.56	4.20
Hemoglobin	15.1	14.1
Mean Corpuscular Volume (MCV)	98.0	91.2
Mean Corpuscular Hemoglobin (MCH)	33.1	33.6
Mean corpuscular Hemoglobin Concentration (MCHC)	33.8	36.8
White blood cell $\times 3$	7.4	6.0
Neutrophil (%)	87.4	60.6
Lymphocyte (%)	9.1	33.0
Platelets $\times 10^3$	183	185

**Table 2:** Venous blood gas analysis of the first patient on admission and 2, 5, and 8 hours after presenting to emergency department

Parameters	On admission	2 hours	5 hours	8 hours
pH	7.31	7.37	7.41	7.41
Pco <sub>2</sub>	18.2	23	25.6	36.9
Hco <sub>3</sub>	9.2	13.1	16	23.5
Base excess	-14.4	-	-	-0.1

Serum cholinesterase levels were in normal range and serum test for the presence of acetaminophen, ethanol, methanol, and other toxic substance were negative. Mild Metabolic acidosis was evident in the first venous blood gas (VBG). Results of other laboratory tests are shown in Table 1. Cardiology consult and echocardiography were performed and left ventricle ejection fraction (LVEF) was 35%.

With suspicion to ALP poisoning, based on clinical and para-clinical findings, magnesium sulfate, bicarbonate, N-acetyl cysteine (NAC) were initiated. NAC was discontinued because of symptoms of hypersensitivity reactions. Insulin was prescribed based on the routine protocol because of high Blood Sugar. ECG was repeated and prothrombin time was also reported high, which became normal in recheck. VBG was repeated several times and improved with supportive acidosis treatment.

## 2.2. Case two

Vital signs of daughter on arrival included: Systolic Blood Pressure (SBP)/ Diastolic Blood Pressure (DBP) = 120/80

mmHg, Respiratory Rate (R.R) = 12 / minute, Pulse Rate (P.R) = 65 / minute. The patient had a seizure attack on admission and after a short time had a cardiac arrest. She was immediately intubated and cardiopulmonary resuscitation (CPR) was performed. Unfortunately, resuscitation efforts were unsuccessful and she died after an hour. The course of events was so rapid that there was no time for any laboratory test.

## 2.3. Cause of poisoning

After taking a more accurate history it was revealed that the patient's neighbor had sprayed a pesticide in his home two days earlier and had left the house. Following investigations, it was determined that the mentioned poison was rice tablet (aluminum phosphide) which was obtained from an illegal center and was placed in the neighboring house in large numbers. The mother and daughter had been inhaling the poison odor for the past two days and had been at home all the time until they had progressive symptoms leading them to refer to hospital. History, clinical signs, and results of laboratory tests were in concordance with inhalation poisoning



with aluminum phosphide. On the fourth day of hospitalization after partial recovery, case one was discharged with personal consent against medical advice.

### 3. Discussion

Cardiac arrest due to cardiac toxicity of ALP inhalation led to death of case two in this report. Phosphine gas toxicity following unintentional inhalation is not a common report in medical literature, although suicidal ingestion of aluminum phosphide tablets has been reported frequently in our country (2, 7, 8).

The National Institute for Occupational Safety and Health (NIOSH) has established a limit for occupational exposure to phosphine gas at 0.3 ppm and has deemed it "immediately dangerous to life or health" at 50 ppm or more." (9). A study on different animals regarding degree of lethal exposure it has been reported that 200 ppm-hours leads to roughly 50% mortality (10). However, another study on male rats reported 11 ppm for 4 hours resulting in 50% lethality (9).

Phosphine gas concentration in this family's home was certainly sufficient for major toxicity following several hours of exposure. An accurate estimation of fatality rates of phosphine gas inhalation is not possible. However, it is documented that phosphine is placed in the highest category of toxicity based on the Environmental Protection Agency (EPA) (9, 11, 12).

In the current report, after 48 hours of inhalational exposure, death occurred. Unfortunately, since there is no accepted definitive treatment for this lethal poisoning, supportive measures are the only treatment option at present. Several maneuvers are suggested for possibly improving the outcome, including administration of N-acetylcysteine, magnesium sulfate, pralidoxime, and hyperinsulinemia with hypoglycemia (13). Although none of them are sufficiently evidence advocated at the present time (6). However, intensive supportive treatments may be lifesaving in some cases, like in the current report.

### 4. Conclusion:

Phosphine gas is highly toxic, and fatality is expected even several hours after continuous exposure. A high level of vigilance and a more restrictive policy is needed for keeping the general population from having access to these pesticides and consequently preventing the high rate of mortality following this preventable poisoning in our community.

### 5. Appendix

#### 5.1. Acknowledgements

The authors express their appreciation to department of Clinical Toxicology, Imam Reza Hospital, Mashhad University of Medical Sciences, Mashhad, Iran.

city of Medical Sciences, Mashhad, Iran.

#### 5.2. Authors contribution

All authors devised the work, the main conceptual ideas, proof outline and interpretation of the data. Also, all authors discussed the cases and commented on the manuscript.

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#### 5.3. Conflict of interest

Hereby, the authors declare that there is no conflict of interest regarding the present work.

#### 5.4. Funding

All the expenses of this research were paid by the researchers.

#### 5.5. Ethical consideration

All the authors met the criteria of authorship based on the recommendations of the international committee of medical journal editors.

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