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CASE REPORT

# Successful Treatment of Aluminium Phosphide Poisoning: A Case Report

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**ABSTRACT**

Aluminium phosphide (AlP) is used to kill rodents in grain storage. It produces phosphine gas which is toxic for mitochondria. The exact mechanism of action is unclear, but phosphine is thought to produce toxicity by blocking cytochrome-c oxidase, which inhibits oxidative phosphorylation and result in cell death. This poisoning has a high mortality, and survival is unlikely if more than 1.5 g is ingested. Shock is the commonest and most important clinical feature and cause of death in phosphine poisoning. AlP poisoning if not treated causes death within 24 hours, presumably due to cardiogenic shock. The high mortality is due to the rapid onset of shock, metabolic acidosis, cardiac arrhythmias and adult respiratory distress syndrome. Here, a case of 27–year-old woman with intentional ingestion 2 AlP tablets (6 g) is reported. She was admitted to Afzalipour Hospital in Kerman with symptoms and signs of shock. Because of refractory hypotension to crystalloid and vasopressor, we started glucagon. After 72 hours, there was no symptom and sign of shock, vital signs became stable and drugs were tapered. She was discharged to psychiatry ward 6 days after initial admission with full recovery. In Conclusion, early administration of glucagon to AlP poisoning patients in refractory shock may be helpful but this needs to be confirmed by further studies.

**Keywords:** *Aluminium phosphide, Phosphine, Glucagon, Shock*

Aluminium phosphide (AlP) is used throughout the world to protect stored grains from rodents and other pests. The exact mechanism of action is unclear, but phosphine is thought to produce toxicity by blocking cytochrome-c oxidase, which inhibits oxidative phosphorylation and eventually results in cell death [1]. Phosphine gas is rapidly formed when AlP comes in contact with dilute acids or with water [2]. In oral ingestion, the phosphine gas released isabsorbed by the gastrointestinal tract by simple diffusion and is mainly excreted by the kidneys and lungs. The reason for human poisoning is usually suicide [3]. Shock is the commonest and most important clinical feature and cause of death in phosphine poisoning [4]. ALP poisoning, if not treated, causes death within 24 hours, presumably due to cardiogenic shock [5]. Using glucagon, we report successful treatment of a case of intentional AlP poisoning in Kerman, the Center of Kerman Province Southeast of Iran. For this purpose, informed consent was obtained from the patient.

## CASE REPORT

A 27–year- old woman was admitted to our hospital with complaining of vomiting, chest discomfort, and malaise for 6 hours after ingestion of 2 ALP tablets (6 g) as powder form in capsules for the suicidal attempt. On admission, the patient was agitated. The initial vital signs were: Blood Pressure: 70/40 mmHg, Pulse Rate: 110 beats/min with filly form pulses, Temperature: 36.4˚c axillary, and oxygen saturation percent undetectable with pulse oximetry. On physical examination, she had cool, dry and clammy skin. High anion gap metabolic acidosis was detected in arterial blood gas: PH: 7.02, $PCO_2$: 44.9 mmHg, $HCO_3$: 11.4

**Table 1.** Serial blood gas and Blood pressure of the reported case

| Laboratory tests | Time after ingestion | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 1 h | 4 h | 8 h | 24 h | 36 h | 48 h | 72 h | 6 days |
| Blood pressure (mmHg) | 60/pulse | 65/pulse | 70/pulse | 100/70 | 100/80 | 110/80 | 120/80 | 120/80 |
| PH | 7.02 | 7.28 | 7.33 | 7.23 | 7.30 | 7.47 | 7.52 | 7.43 |
| $HCO_3$ (mmol/L) | 11.4 | 6.8 | 9.2 | 13 | 11.8 | 29.9 | 27.50 | 23.6 |
| BE (mmol/L) | -18.50 | -17.50 | -10.3 | -11.50 | -13.6 | +7.3 | +6.3 | +0.7 |
| Na (meq/L) | 142 | 140 | 144 | 146 | 144 | 140 | 141 | 140 |
| K (meq/L) | 4.8 | 4.5 | 4.4 | 4 | 3.8 | 4 | 3.9 | 4.2 |

mmol/L, BE: -18.5 mmol/L, Cl: 98 mmol/L, Na: 142 meq/L. Initially, all others laboratory data were normal.

She underwent emergency treatment as follows:

***1) Gastrointestinal decontamination:*** including nasogastric tube (NG Tube) insertion and aspiration of gastric content following pushing four vials of sodium bicarbonate (each vial contains 55 meq $NaHCO_3$) to stomach, then gastric lavage with potassium permanganate and ultimately pushing four vials of NaHco3 into the stomach at the end of lavage.

***2) Correction of metabolic acidosis*** with: $NaHCO_3$, 55 meq every 15 min and serial control of VBG, Na, K. (Table 1).

***3) Management of shock*** with: IV infusion of sodium chloride 0.9%, dopamine and epinephrine.

***4) Prevention of cardiac dysrhythmia*** with magnesium sulfate and calcium gluconate both with the dose of 1 g at the beginning followed by 1 g every 6 h. Approximately 12 h after hospitalization, despite using crystalloids and vasopressors, the patient was hypotensive, and glucagon was started with loading dose of 5 mg, then 1 mg every 5-10 min to increase blood pressure up to 100/60 mmHg and then maintenance dose of 4 mg in an hour. After 72 h, vital signs became stable and intravenous fluid and drugs were tapered. She was discharged to psychiatry ward 6 days after initial admission. In this time, she had normal vital sign and laboratory data without any complain. A follow-up telephone conversation 2 months after discharge revealed that the patient was asymptomatic with no evidence of toxicity or sequel.

## DISCUSSION

Aluminium Phosphide (AlP) is a highly effective insecticide and rodenticide. Although, its using has been banned in Iran, it is still used to protect rice (hence the local name “rice tablet”) and stored grains from rodents and other household pests [6]. AlP is a solid fumigant and can be formulated in the form of tablets (green, brown or gray), pellets, granules or as a dust. As shown in Fig 1, It is available as dark grey tablets of 3 g (each tablet releasing 1 g of phosphine), consisting of 56% AlP and ammonium 44% carbamate.

AlP poisoning has a high mortality (30–100%), and survival is unlikely if more than 1.5 g is ingested [7]. The toxicity of AlP is attributed to the liberation of phosphine gas.

$AlP + 3H_2O \rightarrow Al(OH)_3 + PH_3$ (phoshine)

Phosphine has inhibitory effects on mitochondrial cytochrome c oxidase, which leads to the generation of superoxide radicals and cellular peroxides. Cellular injury occurs through lipid peroxidation and other oxidant mechanisms. The major lethal consequence of AlP, profound circulatory collapse, is secondary to these toxins generated, which lead to direct effects on cardiac myocytes, fluid loss, and adrenal gland damage [8]. Cardiotoxicity of AlP poisoning has been shown as an increased left ventricular dimension, left ventricle hypokinesia, akinesia, low ejection fractions, increased systemic venous pressure, normal pulmonary artery wedge pressure and ECG abnormalities [9]. The high mortality is due to the rapid onset of shock, metabolic acidosis, cardiac arrhythmias and adult respiratory distress syndrome [10]. Shock has been found most important clinical feature and cause of death in phosphine poisoning. Most of the patients had low blood pressure refractory to inotropic support [11]. In this case, the patient had the refractory shock 12 hours after hospitalization despite crystalloid and vasopressor therapy with dopamine and epinephrine. The exact cause of shock is not clear. Several factors such as myocardial damage, peripheral vasodilatation, fluid loss due to vomiting, and pulmonary edema appears to be operative [12]. The use of high doses of glucagon may benefit in the treatment of aluminum phosphide poisoning [13]. In this case, we started glucagon after the first 12 h hospitalization, and the patient replied 72 h

**Fig 1.** Aluminium phosphide tablets

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after treatment. Blood pressure is returned to the normal range. In this patient with refractory hypotension, glucagon may be the preferred drug due to its positive inotropic effects. Intravenous glucagon can be used by giving 2-5 mg initially followed by 4-6 mg in an hour until blood pressure became stable. Then tapering is started. Glucagon has dose-dependent positive inotropic and chronotropic qualities. Glucagon stimulates adenyl cyclase via G proteins, resulting in increased intracellular cyclic AMP and calcium concentration which in turn leads to stimulation of muscle contraction [14]. With a single-dose method of 2 or 5 mg glucagon intravenously, the inotropic action of the drug produced immediate increased myocardial contractility with the significant increase in cardiac output and enhanced cardiac performance, and lowering of pulmonary arterial pressure and pulmonary vascular resistance. No primary peripheral vascular effect was evident, and the increased systemic pressure and lowered systemic resistance appear to be secondary to the central action of the drug. Repeated booster doses rather than continuous infusion may be the method of choice to maintain an increased cardiac output [15].

In conclusion, ALlP poisoning has a high mortality rate and survival is unlikely if more than 1.5 g is ingested, but there is no specific antidote for AlP poisoning. However, in addition to crystalloid and vasopressor, early administration of glucagon to AlP-poisoned patients in refractory shock may be helpful.

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