
CASE REPORT

Suicide by ingestion of aluminium phosphide: a case report

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This is the first Cuban report of a suicide carried out by lethal intake of aluminum phosphide, a pesticide used to control rodents and insects. We review the main toxic effects, placing emphasis on hypoglycemia, which is seldom mentioned, and electrocardiographic abnormalities suggestive of subendocardial infarction. [Emergencias 2009;21:228-231]

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CONFLICT OF INTEREST:

None

Introduction

Aluminum phosphide (AP) is a solid pesticide commonly used for preserving grain. In Iran it is known as the rice tablet. It is cheap and highly toxic¹. After exposure to moisture it releases phosphine gas (PH₃), the active pesticide component, which is rapidly absorbed by inhalation, ingestion or contact (Figure 1). In the case of oral intake, the phosphine gas released is absorbed by the gastrointestinal tract by simple diffusion and is mainly excreted by the kidneys and lungs². Phosphine, like cyanide, inhibits mitochondrial cytochrome c oxidase and impedes cellular oxygen utilization; cellular superoxide and peroxide radicals are generated, with subsequent cellular insult by lipid peroxidation³. Some experimental results suggest that glutathione, melatonin, vitamin C and carotenes play an important role in reducing the effects caused by oxidative phosphine⁴.

AP is available as tablets or pills (Phostoxin® Bhostoxin® Celphos® Quickphos® Phosphume® Phostek®). The tablets are green, brown or gray, and each tablet contains 56% AP and 44% aluminium carbonate, releasing 1 g of phosphine. The lethal dose for a person of 70 Kg is 150-500 mg^{1,2,5,6}. The reason for human poisoning is usually

suicide, occasionally accidental and rarely homicidal². Thus, in the United Kingdom 93 cases were described between January 1997 and June 2003 according to the National Poisons Information Service, and most cases were accidental⁷. But in Iran AP is the main substance used for suicide¹. The prevalence of cases is higher in rural populations.

We report the first case of suicidal AP poisoning described in Cuba.

Case report

A man aged 17 years of rural origin intentionally ingested half a tablet of AP (Figure 2) at the pig farm where he worked; shortly afterwards he presented vomiting. He was taken to the emergency service of his health area medical centre where gastric lavage was performed and activated charcoal was administered. Hypotension motivated the administration of sodium chloride 0.9% (1,500 ml). He presented further vomiting and diarrhea, and was transferred to our hospital emergency room, somnolent and with hypotension (80/40 mmHg) that persisted despite receiving further saline solution (1500 ml) and infused



Figure 1. Tablets of aluminum phosphide. A) Just placed. B) Reaction to phosphine release. C) Phosphine completely released.

dopamine (5 mcg/Kg/min). Toxicologically, urine was negative for benzodiazepines, barbiturates, phenothiazines and carbamazepine.

He was admitted to the intensive care unit (ICU) about 6 hours after the toxic ingestion. The state of consciousness was normal, and the main manifestation was hypotension which was treated with 12 hours administration of more sodium chloride and gelofusine (1 L), without response (blood pressure: 60/40 mmHg) and therefore increased dose of dopamine (7 mcg/kg/min) and hydrocortisone (200 mg IV). We also observed hypoglycemia (1.5 mmol/l) and, in arterial blood gases, metabolic acidosis (pH 7.12, pCO₂ 13.3 mmHg, pO₂ 195 mmHg, HCO₃ 4.3 mmol/l, EB -24.6 mmol/l, SO₂ 99.3%) which required hyper-

tonic glucose and sodium bicarbonate. The rest of the blood test was normal except for leucocytosis (15.6 x 10⁹/l). Chest radiograph was also normal. Initial ECG was normal, but later showed ST segment elevation in DII, DIII, AVF, V2 and V3 (Figure 3). Other drugs used were ranitidine (50 mg IV C/12 h) and vitamin C (1 g IV for 24 h).

After the first 12 hours in the ICU, the rate of diuresis began to decrease (from 1 ml/kg/h to 0.2 ml/kg/h) and serum creatinine increased (154 mmol/l); dopamine dose was increased (10 mcg/kg/min) and dobutamine added (10 mcg/kg/min). The second 12h period was characterized by hypoglycemia (2.2 mmol/l), arterial hypotension and metabolic acidosis, without further changes in the ECG and respiratory manifestations. Metabolic acidosis was corrected several times with bicarbonate without the need for hemodialysis.

At 28 hours after UCI admission, the patient presented irreversible cardiac arrest and died. The post mortem macroscopic examination showed generalized visceral congestion and hemorrhagic lesions in the lung, kidney and spleen. The heart showed subendocardial infarction. Microscopic study was not performed.

Discussion

The spectrum and severity of signs and symptoms of AP poisoning depend on the dose, the route of absorption and the time lapse between exposure and initiation of treatment. The onset of symptoms is, in most cases, instantaneous². In our case, vomiting began immediately after ingestion.

Other early symptoms are gastrointestinal and include nausea, abdominal pain, burning epigastrium, thirst and diarrhea that may respond to the release of phosphine in the stomach immediately after the intake of AP. Jaundice develops late. In AP intoxication, blood glucose levels may be normal, high or low. Both hypo and hyperglycemia have been explained by a variety of biochemical changes that stimulate or inhibit enzymes and



Figure 2. Bottle of aluminum phosphide (Quickphos®).

hormones that catalyze and regulate glucose metabolism⁸. However, hypoglycemia is infrequent.

Cardiovascular involvement is common and is manifested by hypotension, shock, bradycardia or tachycardia, congestive heart failure with toxic myocarditis and electrocardiographic abnormalities (changes in ST segment and T wave, rhythm disturbances and conduction). Our case showed, after an initial normal ECG, an apparent elevation of the ST segment compatible with subendocardial infarction, confirmed in the post mortem visual examination. These findings are infrequent^{6,9,10}. It is likely that several factors influence the occurrence of myocardial infarction during the course of poisoning by AP, such as hypoxia, shock and toxic myocardial injury. Respiratory manifestations appear after 24 hours in the form of cough, dyspnea, cyanosis and adult respiratory distress syndrome (ARDS). These events were never present in our case.

Renal involvement is rare and is considered a sign of poor prognosis³. Oliguria with elevated serum creatinine were present in this patient, probably in the context of renal hypoperfusion.

Neurological manifestations are rare; they include headache, vertigo, convulsions, confusion, irritability, drowsiness, coma, ataxia, and paresthesia. Death usually occurs within the first 24 hours due to acute cardiotoxicity, ranging in time from exposure between 1 and 48 hours⁶. However, death may occur on the fourth day when ARDS appears. Other causes include gastrointestinal bleeding, metabolic disorders and liver failure. Mortality described for all varieties of AP poisoning varies between 37 and 100%².

Visual post-mortem examination usually shows, as in our case, congestion of all the vital organs. In the microscopic study, the liver shows central venous congestion, degeneration, hemorrhage, sinusoidal dilatation, cholestasis, centrilobular necrosis, hyperplasia of Kupffer cells and infiltration by mononuclear cells. Microscopic changes of the lung reveal alveolar thickening, edema, capillary dilation, alveolar collapse and bleeding. In the kidney, these changes are degeneration, infiltration and tubular dilatation. Changes in the brain include congestion and coagulative necrosis, while in the stomach there is congestion and bleeding¹¹.

Diagnosis with a high level of certainty of AP ingestion can be made by testing a sample of gastric aspirate with paper impregnated with silver nitrate (AgNO_3). In one study, this test was positive in 100% of AP intake and negative in all controls. However, when the test was investigated

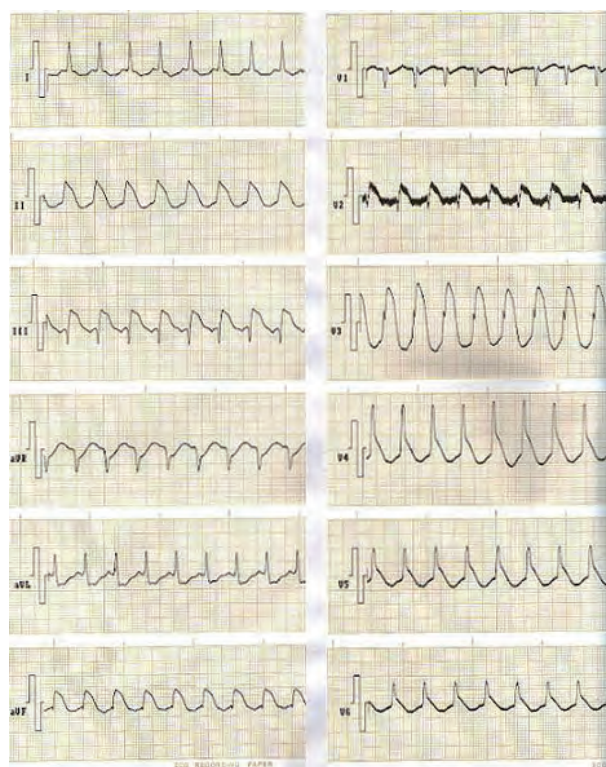


Figure 3. Electrocardiogram showing ST segment elevation in DII, DIII, AVF and V₂.

to detect phosphine in the breath of patients with poisoning by AP ingestion, the results were positive in only 50% of the cases⁴. This test was not available in our emergency department, so the diagnosis was presumptive.

The immediate release of phosphine gas when AP makes contact with aqueous solutions leads to rapid absorption by the lungs when inhaled or gastrointestinal tract when swallowed. Its gaseous form and extremely high toxicity mean that this agent is considered a potential terrorist chemical weapon². Phosphine gas has a characteristic smell of rotten fish or garlic, which should alert the emergency team to the possibility of exposure to AP/phosphine. However, even at toxicologically relevant concentrations of phosphine, the smell may be absent⁷. This product may also be a risk to those involved in the care of the patient⁷.

Among the measures employed to decrease the absorption of phosphine by the digestive system is hourly gastric lavage with oxidizing agents (eg, potassium permanganate 1:10.000), until the gastric contents is negative for phosphine, followed by washing with sodium bicarbonate. Activated charcoal administered by mouth or gastric tube followed by a cathartic agent can prevent absorption of the toxin.

As hypotension is a cardinal manifestation, replacement with intravenous fluids is crucial (4-6 L in the first 4 hours, 50% saline). Also, infusion of dopamine, hydrocortisone (200 mg/4-6 h) and continuous administration of oxygen are recommended. In severe cases, impaired adrenal function has been found; hence the indication for steroid administration.

Another important event is metabolic acidosis, so correction with sodium bicarbonate and ventilator assistance may be needed. This is probably due to lactic acidosis caused by blockage of oxidative phosphorylation. In addition to supportive measures, other therapies such as n-acetylcysteine, glutathione, melatonin, vitamin C, beta carotene, and magnesium, which have demonstrated antioxidant properties in animals, may have some future use¹²⁻¹⁴. In rats exposed to AP, N-acetylcysteine increased survival and reduced the myocardial oxidative insult¹².

Other possible therapeutic agents include trimetazidine, which shifts cardiac energy metabolism from fatty acid oxidation to glucose oxidation, reducing the consumption of oxygen¹⁵. The use of intravenous magnesium sulfate has been suggested to reverse cardiovascular complications, because magnesium is a physiological antagonist of calcium and some cases show hypomagnesemia. However, routine use of magnesium sulphate has been questioned because hypermagnesemia has been described in some cases of AP poisoning³. Since death may occur suddenly and survival after AP poisoning is rare because there is no effective antidote, prevention of this type of poisoning should be a priority.

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Suicidio con fosforo de aluminio: presentación de un caso

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Se describe el primer caso publicado en Cuba de envenenamiento letal por ingestión intencional de fosforo de aluminio, un pesticida usado contra los roedores e insectos. Se revisan los principales efectos tóxicos. Entre ellos, destaca la hipoglicemia, manifestación infrecuentemente descrita y los cambios en el electrocardiograma sugerentes de infarto subendocardico. [Emergencias 2009;21:228-231]

Palabras clave: Fosforo de aluminio. Suicidio. Intoxicación aguda. Infarto de miocardio.