Fatal myocardial damage due to zinc phosphide intentional ingestion

Francesco Marino,1 Massimo Salvetti,1 Abramo Bazza,1 Mara Rossi,2 Maria Lorenza Muiesan1
1Department of Clinical and Experimental Sciences, University of Brescia; 2Department of Internal Medicine, Spedali Civili General Hospital, Brescia, Italy

Abstract

We present a case of fatal myocardial damage caused by zinc phosphide ingestion. It is a highly toxic poison that causes life-threatening complications (cardiac and respiratory acute failure above all) by its active metabolite phosphine. Phosphine toxicity’s case reports from Europe (and Italy), United States and western countries are rare. A 69-year-old man drank a great amount of alcohol and unspecified amount of diluted zinc phosphide and was admitted to emergency department with a mild metabolic acidosis and acute respiratory failure. After gastro-intestinal decontamination, a transient improvement of his clinical conditions was observed. In the emergency medicine unit a sudden onset of severe bradycardia and hypotension appeared, electrocardiogram showed an increase in QRS duration with ST-elevation in many leads; 2 min later a third-degree atrio-ventricular block was evident. Bradycardia went into asystole and the patient had cardiac arrest. Despite all resuscitative maneuvers, 6 h after zinc phosphide ingestion the patient died. No antidote or specific therapy or management of this potentially life-threatening poison are actually available, but only supportive and resuscitative measures.

Case Report

A 69-year-old man, with type 2 diabetes, ischemic heart disease, a bipolar disorder, chronic alcohol abuse and previous suicide attempts, drank a great amount of alcoholics (2 liters of wine) and unspecified amount of diluted zinc phosphide powder after an argument with his wife. After 1 h he was admitted to the emergency department (ED).

At admission he was lethargic, confused, with a Glasgow Coma Scale score of 15/15; physical examination showed hypotension (blood pressure (BP) 80/40 mmHg), a normal heart rate (60 bpm), oxygen saturation 98% with O2-therapy at 3 L/min., cardiac, thoracic and abdominal physical examination were normal. The first electrocardiogram showed sinus rhythm, no ST- T alterations and a first degree atrio-ventricular block (PR 0.21 s).

Arterial blood gas analysis (without O2 therapy) revealed a mild metabolic acidosis and acute respiratory failure (pH 7.3, pCO2 33, pO2 51 mmHg HCO3-17 meq/L, P/F=242); serum lactate was 49 mmol/L (normal test values <1.2 mmol/L); calculated anion gap was high (25 meq/L).

It was also found a slight elevation of hepatic enzymes (aspartate aminotransferase 107 IU/L, alanine aminotransferase 81 IU/L). Blood alcohol concentration was 253 mg/dL. Red and white blood cells, platelet count and troponin I were all within the normal range. Creatinine was 1 mg/dL (modification of diet in renal disease-estimated glomerulur filtration rate of 79 mL/min/1.73 m2).

Routine blood toxicologic panel was negative for all other investigated substances and drugs.

After a first phone contact with the reference Poison Control Center in Pavia, Italy, the patient underwent a gastric lavage, administration of activated charcoal and gastro-intestinal decontamination by magnesium sulphate. Crystalloids and esomепazole intravenously (IV) were also administered.

Esophagogastroduodenoscopy was not performed in ED because of the patient’s lack of cooperation and was postponed. At admission to the EMU, 2 h later, the patient’s haemodynamic and respiratory parameters improved: hypotension resolved (BP 120/60 mmHg), heart rate was 60 bpm and oxygen saturation 98% with O2-therapy at 3 L/min.

The patient was less confused and lethargic; physical examination was still normal, with no abnormalities in cardiovascular or respiratory function. After one hour a sudden onset of severe bradycardia (37 bpm) and hypotension (BP 70/45 mmHg) was recorded at the monitor, followed by a rapid worsening of clinical conditions (loss of consciousness, bradypnea). A complete 12-leads ECG showed an increase in QRS duration with QT-elevation in V1-V6, III and aVF; ST-depression in V4-V6, I and aVL leads; two minutes later a third-degree atrio-ventricular block was evident (Figures 1 and 2). In few seconds bradycardia went into asystole and the patient had cardiac arrest.

Cardiopulmonary resuscitation’s maneuvers started immediately and were repeated for 30 minutes, epinephrine IV was administered and defibrillation was performed after short appearance of ventricular fibrillation.

The Poison Control Center was contacted.

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The amount of absorbed phosphine and its deadly effects on the patient’s heart.

Phosphine’s toxicity on heart and lungs has been described in other few case reports, showing signs and symptoms of myocardial damage and severe respiratory failure, resulting in a fatal outcome despite an intensive management. Despite this, the acute management is currently just supportive and mortality rate can reach high levels.

**Discussion**

As we said above, phosphides’ fatal poisonings have been rarely observed in Italy and Western Countries; however these substances are easily and widely available for different uses and there is little knowledge about their toxicity by occupational, accidental or intentional contact or ingestion.

Acute oral lethal doses for humans in literature and technical sheets range widely between 50 and 500 mg/kg.

In a prospective analysis of 20 patients with acute zinc phosphide poisoning, clinical signs and symptoms, including vomiting, restlessness and anxiety, palpitations and sweating, dyspnea, tachypnea, metabolic acidosis, hypotension, jaundice with hepatomegaly, pulmonary edema and shock were reported following a mean ingestion of 7.5 g (5-20 g). Five of the 20 patients died due to intractable shock, unresponsive to resuscitative measures.

In our case the main phosphine’s target was heart: probably there were involved a direct myocardial tissue damage and hypoperfusion, accelerated by the patient’s pre-existent condition of coronary artery disease and the initial hypotensive state. Furthermore, the alcoholic acute intoxication, the mild metabolic acidosis and the acute respiratory failure may have promoted the onset of the irreversible and fatal myocardial damage. It cannot be excluded that a more complete and prolonged gastro-intestinal decontamination, by an early esophagastroduodenoscopy, could have reduced the amount of absorbed phosphine and its deadly effect on the patient’s heart.

Phosphine’s toxicity on heart and lungs has been described in other few case reports, showing signs and symptoms of myocardial damage and severe respiratory failure, resulting in a fatal outcome despite an intensive management.

Acute poisoning’s clinical features have been so well described, even if it’s only recently that the mechanisms of toxicity have been more clearly understood. Despite of this, the acute management is currently just supportive and mortality rate can reach high levels.

**Conclusions**

Zinc phosphide is a highly toxic poison that causes life-threatening complications by its active metabolite phosphine (2PH3). No antidote or specific therapy or management of this potentially life-threatening poisoning are available.

Supportive and resuscitative measures should be implemented as required by clinical findings and changes.

In conclusion, despite prompt and supportive treatment, zinc phosphide poisoning-induced cardiac, respiratory and metabolic damages are associated with a high risk of death. Mortality rate can reach very high levels (range 37-100%).

**References**