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Case Report

Fatal Suicidal Ingestion of Aluminum Phosphide in an adult Syrian female- A Clinical Case study

Sahar Y. Issa[1]*, Essam M. Hafez[2], Maha Khaled Al-Mazroua[3] and Mohammed G Saad[4]

[1]*Department of Forensic Medicine and clinical Toxicology, Faculty of Medicine - Alexandria University, Egypt*

[2]*Department of Forensic Medicine and clinical Toxicology, Faculty of Medicine - Minia University, Egypt*

[3]*Head of Dammam Regional Poison Control Center - Eastern Region, Kingdom of Saudi Arabia*

[4]*Intensive Care Unit, King Fahad Specialist Hospital, Dammam, Saudi Arabia*

***Corresponding author**

Sahar Y Issa, Department of Forensic Medicine and clinical Toxicology. Faculty of Medicine - Alexandria University, Egypt, Tel: 00966550748326; E-mail: sahar_issa71@yahoo.com

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Abstract

Aluminum phosphide is a powerful insecticide and rodenticide. It is commercially used for crop protection either during storage, or transportation. Nevertheless, it is considered as a highly toxic substance. Its detrimental effects may range from nausea and headache to renal failure and death. It is, therefore, important to ensure their cautious handling to avoid poisoning episodes. Its poisoning has a high mortality and recent years have seen a boost in the number of poisoning cases and deaths caused by suicidal ingestion. Diagnosis is typically reached based on the patient's history, clinical presentation and laboratory findings.

ABBREVIATIONS

AP: Aluminum Phosphide; **ED:** Emergency Department; **GCS:** Glasgow Coma Scale; **ECG:** Electrocardiogram; **VT:** Ventricular Tachycardia; **CT:** Computed Tomography; **BUN:** Blood Urea Nitrogen; **INR:** International Normalized Ratio.

INTRODUCTION

Aluminum phosphide (AP) is pesticide frequently used in agricultural areas and is considered to be one of the leading agents involved in suicides in Asia. When acid hydrolysis of the metal phosphide occurs, cytotoxic phosphine gas is generated [1]. Phosphine is a potent cellular oxidant and an inhibitor of cytochrome (C) oxidase in the electron transport chain. This leads to disperse cellular toxicity, severe metabolic acidosis, adult respiratory distress syndrome and death [2]. Due to the absorption of trace amount ofphosphide into body, delayed effects are observed on heart, liverand kidney; delayed deaths result from cardiotoxicity and, central nervous system intoxication. Till present there is no known antidote for AP toxicity [3].

CASE PRESENTATION

A 27 year-old Syrian female presented to the emergency department (ED) nearly one hour after intentionally ingesting unknown amount of grey pesticide pellets. Early symptoms entailed repeated vomiting and abdominal pain. There was no past medical history and accompanying family member denied use of illicit drugs, ethanol, or tobacco. Vitals upon arrival and after 45 minutes from arrival were given in Table 1. Clinical examination was unremarkable except for sweating, and cold clammy skin. 45 minutes later, she became confused, hypotensive, tachycardia, and tachypneic.Laboratory, radiological & Electrocardiogram (ECG) Findings were demonstrated in Table 2.

A repeated ECG showed ventricular tachycardia (VT). She was intubated and mechanically ventilated. Magnesium sulfate was rapidly started as a 1 mg/kg intravenous bolus dose followed by one 1 g/h. Magnesium sulfate failed to convert VT. Consequently, amiodarone was started as a 150 mg of intravenous bolus, followed by a continuous infusion at a rate of 1 mg/ min, where it failed as well to terminate VT. Twenty minutes after amiodarone bolus infusion, the patient's blood pressure declined dramatically. Four attempts at electrocardioversion (200 J, biphasic) failed to terminate VT. Progressive hypotension, acidemia, and respiratory failure ensued ending inasystole and death. The family brought in the offending agent which was identified as an aluminum phosphide pesticide pellets.

Table 1: Vital signs & O_2 saturation at admission to ED & 45 minutes later.

Parameter studied	At admission	45 minutes from admission
Blood pressure	110/85 mm Hg	75/45 mm Hg
Heart rate	110 b/m	172 b/m
Respiratory rate	20 /min	31 /min
Temperature	36.9 °C	36.8 °C
O_2 saturation	96 %	80 %

Table 2: Laboratory, radiological & ECG Findings.

Parameter studied	Result	Reference Interval
Arterial blood gases		
pH	7.1	7.35 –7.45
PCO2 in mmHg	25	35 – 45
PO2 in mmHg	84	80 –100
Bicarbonate in mmol/l	14	24 – 31
Lactate in mg/dl	16	3.6 – 9.0
Anion gap in mmol/l	26	3 – 11
Electrolytes		
Sodium in mmol/l	140	136 – 145
Potassium in mmol/l	3.6	3.7 – 5.2
Chloride in mmol/l	102	98 – 108
Bleeding/Coagulation profile		
PTT in seconds	34	26 - 36
PT in seconds	11.2	10.4 – 12.6
INR	1.1	0.9 – 1.2
Renal Profile		
Blood Urea Nitrogen (BUN) in mg/dl	21	15 - 50
Creatinine mg/dl	1.4	0.2 – 1.1
Liver Profile		
Albumin in g/dl	3.7	3.5 – 5.2
Total protein in g/dl	6.9	6.4 – 8.2
ALT in IU/l	54	0 – 65
AST in IU/l	31	0 – 37
Total Bilirubin in mg/dl	0.5	0.2 – 1.2
Cardiac Panel		
LDH in IU/l	587	98 – 192
CK in IU/l	785	38 – 397
Serum		
Glucose in mg/dl	136	75 – 105
Iron in mcg/dl	49	50 – 175
Total iron binding capacity in mcg/dl	198	250 – 450
Measured Osmolality in mOsm/kg	311	280 – 300
Blood and urine Gas Chromatography head space findings		
Blood ethanol level in mg/dl	< 10 mg %	Negative
Urine ethanol level in mg/dl	< 10 mg %	Negative
Blood methanol level in mg/dl	Negative	Negative
Urine methanol level in mg/dl	Negative	Negative
Serum acetone level in mg/dl	2	0 – 1.9
Urine acetone level in mg/dl	21	3 – 15
Serum and urine drug screen enzymatic immunoassay		
Drugs of abuse (Amphetamine, Barbiturates, Benzodiazepines, Cannabis, Cocaine, Tramadol, Synthetic Cannabinoids & opiates)	Negative	Machine Cut-off
Radiological & ECG findings		
Chest X-ray	Bilateral pulmonary oedema	
CT brain	Normal	
ECG on arrival to ED	Sinus tachycardia.	
Second ECG	Ventricular Tachycardia.	

METHODOLOGY

A rapid, sensitive and reliable test for detection of phosphine in gastric content and, other biological samples is by using mixed indicator paper strip impregnated with dimethylyellow (0.05%), cresol red (0.1%) and mercuric chloride (1%) in methanol. The appearance of red coloron strip indicates the presence of phosphine. It is a highly sensitive, rapid and easy method [4].

Aluminum phosphide is used widely as a cheap and effective grain fumigant and rodenticide in most of the developing countries [5]. The reasons behind its extensive use include high potency against a broad spectrum of insect species, does not affect seed viability, being cost effective and leaves little residue on food grains. Yet it elicits extreme toxic effects to humans for which no suitable antidote is available [6]. Once ingested, AP is decomposed into highly toxic phosphine gas by the action of dilute hydrochloric acid content of the stomach. Phosphine acts as a respiratory poison. Even 20:100,000 part of phosphine in air is reported to fatal [7].

Phosphine gas is rapidly absorbed throughout the gastrointestinal tract, reaches the blood stream, a part of it is carried to the liver by portal vein. It is also rapidly absorbed through lungs. It blocks the enzyme cytochrome C oxidase as a result of which mitochondrial oxidative phosphorylation is inhibited causing, in turn, the cells to die rapidly [6]. Mitochondrial cytochrome C oxidase inhibition may also lead to pulmonary and cardiac toxicity [7].

After massive exposures (more than 20 mg/kg), most of the phosphine gas is excreted unchanged in expired air, and the residual amount will be metabolized and excreted in urine. Even 20:100,000 part of phosphine in air is considered to be fatal, thus prime focus on the safety of the health care staff due to possible off-gassing of lethal phosphine should be kept in mind. Nearly 16% of phosphine interacted with hemoglobin is oxidized and hence recovered as phosphite and phosphate but the remainder could not be accounted for. Such residual quantity that is oxidized to phosphite and hypophosphite ions will be excreted in urine. Hydrolysis of metal phosphides on the skin could lead to the evolution of gaseous phosphine which could be absorbed by inhalation [8,9].

Aluminum phosphide causes widespread organ damage due to cellular hypoxia by inhibition of enzyme cytochrome C

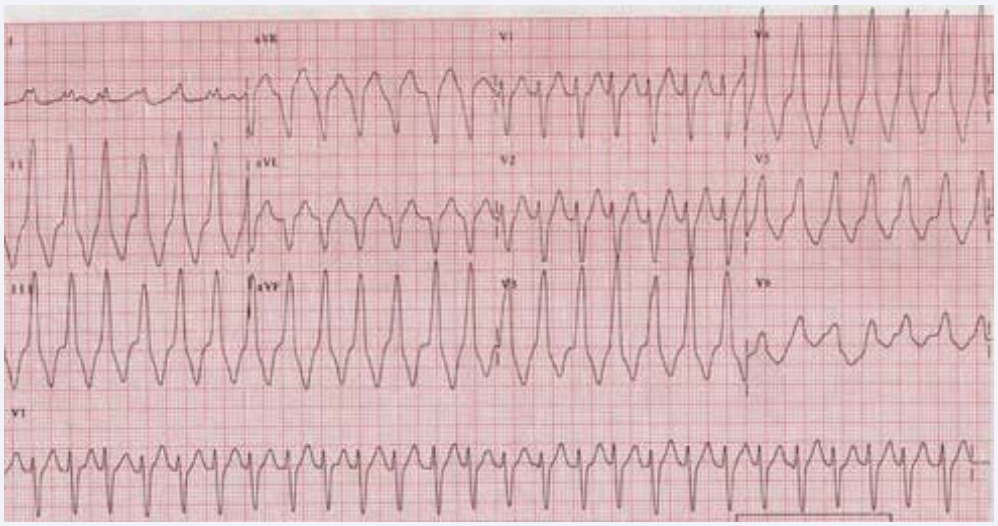

Figure 1 Ventricular tachycardia in a 37 years old female with Aluminum phosphate poisoning.

oxidase inside the mitochondria. Ingestion of AP leads to a high superoxide dismutase activity and low catalase levels that result in formation of a high quantum of free radicals and accelerate lipid peroxidation. The latter, in turn, results in damage to cellular membrane, disruption of ionic barrier, nucleic acid damage and finally, cell death [10].

Cytotoxic phosphine gas produced due to acid hydrolysis of metal phosphide affects heart, lungs, kidneys and gastrointestinal tract [11]. Poisoning with metal phosphides causes nausea, restlessness, abdominal pain, palpitation, pulmonary edema, cyanosis, hypotension, shock and cardiac arrhythmias. Other rare effects include hepatitis, acute tubular necrosis, disseminated intravascular coagulation and respiratory alkalosis [12]. The inhalation of phosphine gas causes diarrhea, pulmonary edema, cold and clammy sweats, tremors, convulsions, delirium, coma and death from respiratory and cardiac arrest [13].

Cardiac toxicity in AP poisoning results from noncompetitive inhibition of cytochrome C in myocardial mitochondria, inhibition of incorporation of amino acids into myocardial proteins, altered permeability of membranes to electrolytes, and generation of superoxide radicals and cellular peroxidases [14]. The last mechanism leads to cellular injury because of lipid peroxidation and other oxidative mechanisms [13]. Clinical cardiac manifestations include toxic myocarditis, refractory heart failure, brad arrhythmias, atrial and ventricular ectopy, and tachyarrhythmia including VT [15].

In episodes of metal phosphide poisoning, the treatment depends on route of exposure. If the victim has ingested metal phosphide, slurry of activated charcoal may be administered (1 g charcoal per kg body weight). Milk, fats or saline emetics should be avoided orally. Proper supportive care should be initiated, metabolic acidosis is to be treated by administering sodium bicarbonate and shock must be treated with suitable vasopressors. Activated charcoal, sorbitol suspension or sodium bicarbonate solution should be administered orally. Intravenous administration of magnesium sulfate, sodium bicarbonate and calcium gluconate is also an alternative. Specific therapy with intravenous magnesium sulfate is recommended. No known specific antidote is however, available for metal phosphide poisoning till date [16].

CONCLUSION

Although AP ingestion is a common cause of suicidal death in many parts of Asia, it is rarely encountered in Saudi Arabia. Clinicians need to be aware of its rapid and fatal toxicity. Management is primarily supportive in addition to a prime focus on the safety of the health care staff due to possible off-gassing of lethal phosphine.

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