

TOXIC DEATH: SUICIDE BY ALUMINIUM PHOSPHIDE (PHOSTOXIN®)**Badr Adouani^{1*}, Abderhmane Elwali² and Yassir Bousliman³**¹Pharmacology and Toxicology Laboratory, Faculty of Medicine and Pharmacy, Hassan II University of Casablanca, Morocco.²Department of Anaesthesiology and Critical Care, Faculty of Medicine and Pharmacy of Rabat.³Pharmacology and Toxicology Laboratory, Faculty of Medicine and Pharmacy, Mohammed V University of Rabat, Morocco.***Corresponding Author: Badr Adouani**

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ABSTRACT

Following aluminium phosphide poisoning, suicide attempts are a significant public health concern. They are liable for very high lethality. We present the case of a patient who died after ingesting an aluminium phosphide pill (Phostoxin®) and experiencing multiorgan failure. In the absence of a specific antidote for this intoxication, the only way to maintain control is through prevention. Efforts must be made to limit over-the-counter sales to cope with this scourge.

KEYWORDS: Suicide attempt, Aluminium Phosphide, poisoning.**INTRODUCTION**

Suicide attempts connected to aluminium phosphide poisoning (Phostoxin®) are a serious public health concern in Morocco and underdeveloped nations.^[1,2] Poisoning has a high mortality rate, ranging from 40 to 91 % in the first 24 hours.^[3] It is a true medical emergency that requires prompt and appropriate care. Despite advancements in toxicodynamics and therapies, understanding of this product's toxicokinetics remains very restricted^[2], and no particular antidote is now available.^[4] Given Phostoxinproven®'s efficacy against rats and the possibility of resistance to conventional rodenticides, we predict that the number of poisonings will increase alarmingly as the drug's usage is expanded.

OBSERVATION

We present the case of a 40-year-old married lady with no notable pathological history who said she had eaten a rat poison pill (Phostoxin®) with the purpose of committing suicide. The patient sought consultation at the regional hospital for stomach discomfort one hour after eating the toxin, had gastric lavage with saline, and was subsequently sent to our hospital three hours later. The patient was awake, eupneic, and afebrile at arrival, with discoloured conjunctivae. The measured blood pressure and heart rate were 110/70 mmHg and 96 beats per minute, respectively. The conducted ECG demonstrated atrial rhythm abnormalities (complete arrhythmia owing to atrial fibrillation) and anterolateral ST-segment elevation repolarization abnormalities. With the exception of hyperleukocytosis, the biological examination upon admission was normal (16.10³). After

three hours, the patient presented with altered mental status and shock, with a blood pressure of 60/30 mmHg under filling and a SPO₂ of 89% under oxygen treatment. The biological samples serving as controls exhibited the following results: hyperleukocytosis: 22.6.10³; thrombocytopenia: 31. 10³µl; cardiac enzyme ancestry: CK-MB: 9.8 ng/ml; troponin-I (hsTnI): 0.359 ng/ml; AST transaminases: 204 IU/l; ALT: 256 IU/l; Gamma-GT: 182 IU/l; serum creatinine: 2.42 mg/dl. In the absence of an antidote, therapy consisted of symptomatic measures alone. Filling with 0.9% saline, the vasoactive medications noradrenaline and dobutamine, then adrenaline, mechanical breathing, and magnesium sulphate (3 g) for 20 minutes, followed by 1 g/h for 3 hours. The patient's condition deteriorated fatally due to multiorgan failure.

DISCUSSION

Aluminum phosphide is a fumigant insecticide that has been used for decades in grain storage and protection.^[5] The most common form is the tablet, which typically weighs 3 grams. When phosphides come into contact with water or stomach fluids, a situation that occurs after eating, they emit phosphine (PH₃). At 0.3 ppm, this gas, which has an allium or rotting fish odour, is hazardous.^[6] Acute intoxication may result from consumption during a suicide attempt or from inhaling PH₃ gas in the environment.^[7] It is in charge of cellular hypoxia as well as oxidative phosphorylation.^[5] The cause of mortality in our instance was mostly cardiotoxicity, which caused abrupt circulatory collapse. Several researchers^[8-9] have described fatalities as a result of shock, gastrointestinal

haemorrhage, or liver failure.^[7] The mortality rate remains high.

The primary objective of therapy is to manage the state of shock^[10,11], but the most common issue is resistant hypotension, which does not respond to substantial crystalloid administration and has limited effectiveness with vasoactive agents such as adrenaline or dobutamine.^[12] Mg sulphate is used as a membrane stabiliser in the therapy of arrhythmias detected during the first 24 hours in the absence of an antidote.^[2] Despite its significance, only a few studies have tried to evaluate its effectiveness in considerably enhancing survival rates.^[13,14]

Phostoxin® poisoning is therefore very dangerous and poses a significant challenge to medical staff. There are no official national data on the actual number of people who suffer from this sort of poisoning, and national publications are irregular. According to a retrospective analysis conducted by Morocco's Poison Control Center on instances of acute pesticide poisoning collected between 1992 and 2007, Phostoxin® was responsible for 129 cases of poisoning, or 5.7%, and they were the most lethal.^[1] The occurrence in Morocco will become more concerning in the coming years since the use of aluminium phosphide on dates in storage will expand, in addition to the disinfection of cereal grains meant for planting or consumption.

CONCLUSION

Phostoxin® poisoning is a serious public health issue; survival is uncommon, and death occurs quickly in the absence of an antidote. Prevention is the best approach to fighting. Efforts must be focused on the implementation of rigorous controls vis-à-vis access to this substance; its sale in public must be restricted, and an approved issuing authority must be formed to offer advice and instruction to users.

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