Aluminum Phosphide Poisoning; a Case of Survival

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Abstract

Background: Aluminum phosphide is a commonly used as a pesticide in Egypt and other agricultural countries to protect grains from pests. Phosphine gas (PH3) is a highly toxic gas, which is released when the tablet is exposed to humidity, causing cardiogenic shock and severe metabolic acidosis.

Case presentation: A 22-year-old female patient with history of ingestion of 1 tablet of Aluminum phosphide presented with recurrent vomiting, severe hypotension and metabolic acidosis.

Discussion: Although Aluminum phosphide is considered a highly toxic substance and its rate of death is very high, this case survived even after development of severe toxic manifestations like hypotension and severe metabolic acidosis.

Conclusion: Aluminium Phosphide is a highly effective insecticide and rodenticide. However, it is highly toxic with high mortality rate if ingested. It produces severe metabolic acidosis and cardiogenic shock with no available antidote, so management may be the only supportive treatment

Keywords: Aluminum Phosphide; Phosphine Gas; Poisoning; Survival

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INTRODUCTION

Acute poisoning is a global problem which increased over the past few years in developing countries and has become as one of the major causes of morbidity and mortality in these countries (1). Two types of pesticides can be used to save grains and rice from rodents and pests; one of them is an herbal product with no or mild toxicity but the other which is highly toxic and commonly used is aluminum phosphide (2).

Phosphine gas is a highly toxic gas, which is released when the tablet is exposed to humidity. However, the exact mechanism of phosphine toxicity is still unclear. It is thought to block cytochrome-c oxidase, thus inhibiting oxidative phosphorylation leading to cell death (3).

Aluminum phosphide is one of the major causes of suicidal poisoning in many countries (4). However, poisoning may occur occasionally or accidental and rarely homicidal (5).

It was established that the fatal oral dose of aluminum phosphide is 0.5 g to 0.75 g in a 70-kg adult (6). Management of Aluminum phosphide toxicity is mainly supportive due to the fact that there is no specific antidote, so mortality with Aluminum phosphide poisoning is very high, ranging from 37% to 100% (7). Despite high mortality rates, this case survived and was discharged in 24 hours, making it as an important unusual case.

CASE REPORT

A 22-year-old female patient from a rural area in Fayoum governorate presented to our national toxicology center by severe recurrent vomiting after suicidal ingestion of one tablet of aluminum phosphide (ALP). On detailed history, she said that she ingested one tablet 3-4 hours before admission and she took part of the tablet out shortly after ingestion. On admission, she was conscious and alert, suffering from epigastric pain, vomiting and thirst. On examination, her pulse was 110 bpm regular, blood pressure 90/60, O2 saturation 100% with mild peripheral cyanosis, temperature normal and CVP was average. ABG showed severe metabolic acidosis (PH 7.00, pco2 32, Hco3 7), other routine labs (complete blood picture, random blood glucose, liver and kidney function and coagulation profile) were within normal ranges. Rapid decontamination was done via Gastric lavage and 5 ampoules of sodium bicarbonate were taken orally during lavage and then activated charcoal was given in a dose of 1 gm per kg. To correct hypotension, one liter of saline was given and sodium bicarbonate was given to correct metabolic acidosis in a dose of 2 meq/kg IV, then she was admitted to ICU. ICU management began with continuous cardiac monitoring and tight blood pressure observation. ABG was repeated for follow-up showing slight improvement. However, Sodium bicarbonate given continued infusion (25 mEq/h intravenously). In a few hours, blood pressure dropped to 70/40 and central venous
pressure CVP was slightly affected, therefore noradrenalin was infused in the rate of 2.3 µg/kg/min, and saline (500 cc) was given regularly every 6 hours. ECG showed sinus tachycardia. After twelve hours of this supportive management, blood pressure improved and metabolic acidosis was corrected gradually. After another twelve hours, the patient was discharged due to complete improvement.

**DISCUSSION**

Although Aluminum phosphide is considered a highly toxic substance and its rate of death is very high, this case survived even after development of severe toxic manifestations like hypotension and severe metabolic acidosis. In our national toxicology institution in Cairo, Egypt, we manage large number of patients with aluminum phosphide and zinc phosphide; many of cases usually die from cardiogenic shock in spite of supportive treatment. The *diagnosis* of AIP in this case was based on the history, the clinical symptoms and the availability of ALP tab container with the patient family. In this case, although she ingested one tablet (3 g) and toxic manifestations appeared, she responded to supportive management and survived. I explained that this response to treatment may be because she took part of the tablet out shortly after ingestion and also due to frequent vomiting. In addition, early giving of activated charcoal and gastric lavage with sodium bicarb have played a great role in the management. Furthermore, early supportive management was a very important step that helped in survival. The stable condition on admission (no coma, O₂ saturation 100%, no severe hypotension and normal routine labs), the fully conscious state and oxygen saturation above 90% without need to mechanical ventilation were good indicators for survival according to Sulaj et al. (8) and Farzaneh et al. (9). Sulaj et al. (8) reported that the dose of AIP ingested, the interval between ingestion and the beginning of treatment, and the depth of coma were the predictors of mortality in patients poisoned with AIP tablets. Farzaneh et al. (9) reached that HCO₃⁻ <12.9 mEq/L, UOP <1725 ml/day, SBP <92.5 mmHg and GCS <14.5 are able to predict mortality (9), although this case has only 2 of these 4 parameters. Early decontamination, admission and supportive care may have helped this case to survive. In addition, Louriz et al. concluded that the prognostic factors for AIP cases were APACHE II (P=0.01), low Glasgow coma scale (P=0.022), shock (P=0.0003), electrocardiogram abnormalities (P=0.015), acute renal failure (P=0.026), low prothrombin rate (P=0.020), hyperleukocytosis (P=0.004), use of vasoactive drugs (P<0.001), and use of mechanical ventilation (P=0.003) (10).

Torabi (3) managed a successful case of aluminum phosphide poisoning with metabolic acidosis and shock. His case survival may be due to aggressive early management of shock and metabolic acidosis in addition to early start of glucacon after the first 12 h hospitalization. Mathai and Bhanu (11) concluded that aluminum phosphide poisoning is severe with nearly 60% mortality rate and they showed that different variables at admission as hypotension at admission needed vasoactive drugs, need for mechanical ventilation, severe metabolic acidosis with low PH and bicarbonate, low serum creatinine and low APACHE II scores can protect patients at higher mortality risk from aluminum phosphide poisoning. Sanaei-Zadeh (12) sent an editorial letter to the previously mentioned journal that we should look and try to expect survival not mortality as done by Mathai and Bhanu (11), and he put many criteria for this, such as early and proper management, and if the patient took the tablet out of their mouth, which occurred in the current case.

**CONCLUSION**

Aluminium Phosphide is a highly effective insecticide and rodenticide. However, it is highly toxic with high mortality rate if ingested. It produces severe metabolic acidosis and cardiogenic shock with no available antidote, so management may be the only supportive treatment. Early decontamination and interventions may be helpful. Restricted use and awareness programs to farmers may be beneficial in prevention of toxicity.

**REFERENCES**