A case report of aluminium phosphide poisoning

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Abstract

Aluminium phosphide (ALP) is highly toxic pesticide which is commonly used in Thailand for grain preservation. ALP has interested with a number of cases due to rising use for agriculture. Its easy purchase in the markets has increased its misuse for suicide. A 39-year-old Thai male with history of ingestion of around 10 tablets of ALP presented with severe repeated vomiting. He developed cardiac arrest with pulseless electrical activity (PEA). Despite aggressive resuscitation, he submitted to death estimate seven hours after ingestion. Due to no known specific antidote, management remains primarily supportive care. A better outcome can be achieved with early arrival, prompt diagnosis, decrease the exposure of poison, aggressive resuscitation and intensive monitoring.

Keywords: **aluminium phosphide, Phosphine gas, suicide**

Introduction

Aluminum phosphide (ALP) has been extensively used on account of its ideal properties like leaving little residue on food grains and ex-terminating insects with no impact on seed viability [1]. However, its widespread use has contributed to a marked increase in the related suicidal [2] with high-risk mortality [3]. Phosphine gas (PH_3) is rapidly formed when ALP comes in contact with dilute acids or with water [4] which is the toxic principle of ALP poisoning [5-7]. ALP-related fatality is attributed to cardiac failure caused by inhibition of cytochrome c oxidase, decrement of adenosine triphosphate (ATP) production and cardiomyocyte impairment [8]. Several experimental and clinical investigations have suggested able to counteract ALP-related toxicity and thus improve the prognosis of ALP-poisoned patients. The level of evidence is still very low [9].

Case study

A 39-year-old male patient presented to our emergency department by severe recurrent vomiting after suicidal ingestion of around 10 tablets of aluminum phosphide (ALP). On present history, he said that he ingested tablets 1 hour before arrival. On admission, he was conscious and agitation, suffering from epigastric pain, vomiting and thirst. Physical examination, his pulse was 126 bpm regular, blood pressure 86/50, O2 saturation 93%, normal temperature. ABG showed severe metabolic acidosis (PH 7.02, pco2 31, Hco3 12), other routine labs (complete blood picture, random blood glucose, liver and kidney functions

and coagulation profile) were within normal ranges. Rapid decontamination was done for removed part of tablets via Gastric lavage tube and then activated charcoal was given in a dose of 1 gm per kg. To correct hypotension, 4 litters of saline was given and sodium bicarbonate was given to correct metabolic acidosis in a dose of 2 meq/kg IV, then he was transferred immediately to an isolated room and an intravenous infusion of dopamine, adrenaline and hyperinsulinemia euglycemia and intravenous fat emulsion (HIE) started. The patient's condition gradually deteriorated and apnea developed, which needed resuscitation and ventilator support. Despite all possible effort the patient developed cardiac arrest and could not survive for more than seven hours.

Discussion

"Wheat pill" or Aluminum Phosphide (ALP) poisoning is rampant in the northern Indian subcontinent and many other parts of Asia, especially South Asia [10]. It is also known as "rice pill" in Thailand handed exterminating insects on food grains [11]. The most common form of ingestion reported is suicidal, followed by accidental ingestion or occupational exposure [10]. Aluminum Phosphide pill is composed of about 44% inert ingredients to prevent decomposition of the tablet, while ALP is about 56% of the pill. Chemical reaction between aluminum phosphide (ALP) and water (H2O) liberates the phosphine gas (PH3) which is highly toxic [10]. Exposure to 1400 mg/m or 1000 ppm is usually fatal [10, 12].

$$AlP + 3H2O \longrightarrow AL(OH)3 + PH3$$

Subsequent to ingestion and upon contacting water/acid in the gastrointestinal (GI) tract, ALP produces PH3 up to 70% which is then absorbed [9, 13]. PH3 causes toxicities similar to that induced by metal phosphides to inhibiting the production of cell enzymes and proteins [14]. PH3 inhibits cytochrome c oxidase in the cardiac cell as it negatively affects mitochondria and myocardial proteins leading to cardiomyocyte impairment related fatality is attributed to cardiac failure [8].
Approach used special antidotes against ALP in human studies. Magnesium sulfate, Melatonin, Coconut oil, N-acetylcysteine (NAC), Vitamin E, Liothyronine were suggested as a potentially beneficial approach against ALP-related toxicity and thus improve the prognosis of ALP-poisoned patients. By contrast the level of evidence is still very low [9].

Conclusion

The study case report stresses on the need that the emergency physicians need to be extremely alert and adequately prepared while handling such patients with no available antidote, so management may be the only supportive treatment. Spontaneous combustion with the release of phosphine from ALP poisoned patients can affect not just the patient, but also pose a health hazard to emergency physicians and medical staff. Restricted use and awareness programs to farmers may be beneficial in prevention of toxicity.

References

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