

CASE REPORT

Aluminum Phosphide Poisoning and Blast in Gastric Tube; A Case Report

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Abstract

Background: Suicide by poisoning is the second most common cause of death by suicide (27.9%) as per The National Crime Records Bureau (NCRB) India in 2015. Aluminum phosphide (ALP) is the leading agricultural poison used to commit suicide as where its availability is not properly regulated. ALP is a highly toxic solid fumigant pesticide, rodenticide and insecticide.

Case presentation: A 40-year-old woman was brought by the police to the emergency section. Her extremities were cold and hypotonic, and her skin was pale. Patient's vitals were unstable with pulse rate 112/minute irregularities, blood pressure 68/54 mm of Hg, respiratory rate 22/minute shallow and body temperature 30.1 °C and characteristically strong garlicky odor on her breath. During the suction of gastric contents, spontaneous ignition of gas with flames and white fumes with sound like a blast was observed by resident doctors and para-clinical staff. Gastric aspirate along with Ryle's tube were immediately sent to the forensic department where the Silver Nitrate test was performed and it was found strongly positive for phosphine gas.

Discussion: Aluminum phosphide readily reacts with water and hydrochloric acid in the stomach to produce phosphine (hydrogen phosphide, PH₃) and a small amount of diphosphine.

Conclusion: The present case report stresses on the need that the emergency physicians need to be highly alert and adequately prepared while handling such patients. Spontaneous ignition with the release of phosphine from ALP poisoned patients can not only affect the patient, but also pose a health hazard to emergency physicians and medical staff.

Keywords: Aluminium Phosphide; Pesticide; Phosphine; Spontaneous Ignition; Suicide

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INTRODUCTION

Suicide by poisoning is the second most common cause of death by suicide (27.9%) as per The National Crime Records Bureau (NCRB) India in 2015(1). Aluminum phosphide (ALP) is the leading agricultural poison used to commit suicide as where its availability is not properly regulated (2). ALP is a highly toxic solid fumigant pesticide, rodenticide and insecticide. It is commonly used as an outdoor and indoor pesticide in developing countries like India, Pakistan, Srilanka and Iran because it is a cheap, effective, free from toxic residue and dose not alter the viability of seeds(3-7). In India, it is marketed as a tablet in the name of Alphos, Celphos, Quickphos, Phosphotek, Phosphume, etc., and available as dark brown or grayish green tablets of 3 g each and also available as pellets, granules and as powder, sealed in tens and twenties in airtight aluminium containers (8).

Each tablet is composed of pure ALP (active ingredient 56%) and ammonium carbamate / carbonate / urea (inert ingredient 44%) which releases CO₂ and NH₃ gases

preventing the self-ignition of phosphine gas. On coming in contact with moisture, each (3g) tablet of aluminium phosphide liberates 1g of phosphine gas, which is the toxic principle of ALP poisoning.⁷ The fatal dose of ALP is around 0.5 g and acute poisoning with ALP occurs either due to intentional ingestion of an ALP tablet for committing suicide or accidental inhalation of PH₃ gas. Death has been reported when people were travelling in fumigated boxcars containing grains fumigated with aluminum phosphide (7).

Phosphine gas is highly flammable and very toxic in nature (9, 10). Phosphine on coming in contact with atmospheric air may ignite spontaneously and burn with white fumes causing health hazards such as burns in poisoned patient as well as in health care professionals (11, 12). The purpose of the present case study is to spread awareness among emergency physicians and staff regarding rare but dangerous complications of ALP intoxication which liberate phosphine.

CASE REPORT

A 40-year-old woman was brought by the police to the

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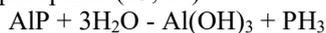
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emergency section of the J. N. Medical College Hospital Aligarh from a town about 20 km away from the city Aligarh with alleged history of ingestion of some unknown poisonous substance. On arrival, she was very drowsy and not responding to verbal stimuli (EMV-8). Her extremities were cold and hypotonic, and her skin was pale. Patient's vitals were unstable with pulse rate 112/minute irregularities, blood pressure 68/54 mm of Hg, respiratory rate 22/minute shallow and body temperature 30.1 °C and characteristically strong garlicky odor on her breath. The ECG showed sinus tachycardia, pulse oximetry showed an arterial O₂ saturation of 90% and ABG (arterial blood gas) analysis indicated metabolic acidosis with blood pH of 7.1. The patient was attended immediately and treatment started with high-flow oxygen through face mask and IV line was secured. In order to remove residual ALP tablets from the stomach, a Ryle's tube was inserted into the stomach and suction was applied using 50 ml disposable syringe. During the suction of gastric contents, spontaneous ignition of gas with flames and white fumes with sound like a blast was observed by resident doctors and para-clinical staff. Gastric aspirate along with Ryle's tube were immediately sent to the forensic department where the Silver Nitrate test was performed and it was found strongly positive for phosphine gas.

The patient was transferred immediately to an isolated room and an intravenous infusion of magnesium sulfate and calcium gluconate started. The patient's condition gradually deteriorated and apnea developed, which needed resuscitation and ventilator support. Despite all possible efforts, the patient developed cardiac arrest and could not survive for more than two hours.

DISCUSSION

Aluminum phosphide promptly responds to water and hydrochloric acid in the stomach to create phosphine (hydrogen phosphide, PH₃) and a modest volume of diphosphine (13, 14).



Phosphine gas is taken in quickly via the gastric mucosa, passes into the blood circulation system and spans to the cell. At the cell level, it prompts non-aggressive restraint of the cytochrome oxidase in mitochondria, hindering the electron transmission chain and oxidative phosphorylation, delivering a vitality emergency in the cells that results in cell death (15).

Further inhibition of catalase and the induction of superoxide dismutase leads to free radical formation, lipid peroxidation and protein denaturation of cell membrane, finally causing hypoxic injury to the cells.¹⁴ PH₃ further inhibits myocardial cellular metabolism and necrosis of the cardiac tissue, resulting in the release of reactive oxygen intermediates, which leads to refractory cardiac muscle depression and this causes high mortality rate (16, 17).

Sudden internal ignition and thermal injury following ALP poisoning are unusual complications that may complicate the patient's condition and may be hazardous to the medical personnel attending the patient (18-20).

The pure phosphine gas itself is odorless, but a commercially available preparation of ALP tablets has a noticeable odor

similar to 'garlic' or 'rotten fish' because of the presence of trace impurities such as the diphosphine (P₂H₄) (11, 13). It is generally believed to be the presence of traces of diphosphine which increases the risk of spontaneous ignition of phosphine/air mixtures at room temperature.

Phosphine and diphosphine are highly flammable gases, which may ignite spontaneously when its concentration exceeds its lower explosive limit of 1.8% volume by volume (v/v) in air (20-25). In case an air/phosphine blend, in which the phosphine accumulation surpasses this level, sparkles in an enclosed space, at that point an explosion-like circumstance will be developed. Some sources report the temperature for self-ignition of phosphine gas to be 38 °C (11, 20).

At the point when burning, phosphine delivers a thick white haze of "phosphorus pentoxide," an extreme respiratory tract aggravation.

ALP is incompatible with oxidizing agents, which means it induces adverse reactions (22). Potassium permanganate is recommended in ALP poisoning to convert phosphine to phosphate (26), but it is an oxidizing agent and when in contact with organic matter, it is reduced to manganese dioxide and the very corrosive potassium hydroxide (20, 26, 27). This reaction is also exothermic and may have contributed in increasing injury as well as the occurrence of ignition events. However, it is suggested that negative pressure during gastric suctioning may contribute to the ignition (27, 28).

CONCLUSION

The present case report stresses on the need that the emergency physicians need to be highly alert and adequately prepared while handling such patients. Spontaneous ignition with the release of phosphine from ALP poisoned patients can not only affect the patient, but also pose a health hazard to emergency physicians and medical staff.

Forensic pathologist should take into the consideration that the signs of burn in the face and neck of the deceased may not be misleading at autopsy.

Conflict of interest:

The authors certify that they have no affiliations with or involved with institution at any time receiving payment or services from a third party (government, commercial, private foundation, etc.) for any aspect of the submitted work (including but not limited to grants, data monitoring board, study design, manuscript preparation, statistical analysis, etc.). Further that there are no patents, whether planned, pending or issued, broadly relevant to the work discussed in this manuscript.

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