

A case report of aluminium phosphide poisoning

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SUMMARY

Aluminium phosphide (ALP) is highly toxic and poisoning can result in high mortality rates. A 26-year-old female who allegedly ingested a toxic dose of ALP presented with vomiting and diarrhoea. She developed cardiac arrest with refractory pulseless ventricular tachycardia. Despite aggressive resuscitation, she succumbed to death seven hours following ingestion. In cases like this, a better outcome can be achieved with early arrival, prompt diagnosis, aggressive resuscitation and intensive monitoring.

KEY WORDS:

Poisoning, Aluminium phosphide

INTRODUCTION

Aluminium phosphide (ALP) is a widely available fumigant and pesticide. When ingested, it is highly toxic and can result in refractory hypotension, Acute Respiratory Distress Syndrome (ARDS) and fatal arrhythmias. Currently, there is no antidote for ALP poisoning. Treatment is supportive.

CASE REPORT

A 26-year-old lady was brought to a private hospital after allegedly ingesting two tablets of 3g aluminium phosphide. Her boyfriend presented a packet of the tablets (Figure 1). Each tablet contained 56% ALP, which releases 1g of phosphine gas following ingestion. She took the tablets at 0600 hrs following an argument with her boyfriend. Subsequently, the patient vomited and passed stools 10 times. At the private hospital, gastric lavage was performed. Due to her unstable haemodynamic parameters (Pulse rate 146 bpm, BP 60/40 mmHg), she was initially resuscitated with intravenous fluids. There was improvement with fluid resuscitation, hence inotropes was not given at that time. Transfer to a tertiary government centre was made due to inadequate resources.

Upon arrival at the receiving emergency department, the patient was drowsy, pale, cold and clammy. While transferring the patient to the hospital bed, she became unresponsive and pulseless. Cardiopulmonary Resuscitation was initiated. Cardiac monitor showed a shockable rhythm (Ventricular Tachycardia). The patient was defibrillated three times and was given intravenous amiodarone as recommended by Advanced Cardiovascular Life Support protocols. The arrhythmia persisted despite intervention.

Intravenous magnesium sulphate, calcium gluconate and sodium bicarbonate were also administered in response to severe metabolic acidosis. The patient's trachea was intubated during the resuscitation.

Return of spontaneous circulation was achieved after 30 minutes. The patient developed seizures post resuscitation, which were aborted with midazolam. A central venous catheter was inserted and the patient was commenced on sodium bicarbonate infusion. Bedside ultrasound showed poor cardiac contractility. Four inotropes (noradrenaline, adrenaline, dobutamine and dopamine) were administered because of persistent hypotension.

The patient suffered a second cardiac arrest after 30 minutes. Resuscitation was terminated and the patient was pronounced dead seven hours following ingestion of the ALP tablets. Post mortem showed ALP still present as a gastric content. The gut lining was oedematous and lung autopsy revealed bilateral pleural effusion.

DISCUSSION

ALP is a common and effective agricultural fumicide. While ALP poisoning is frequently seen in India and Iran, there is as yet, no published case of ALP poisoning in Malaysia. ALP toxicity is due to the release of lethal phosphine gas following exposure to atmospheric moisture and hydrochloric acid in the stomach. This gas causes cell hypoxia due to inhibition of mitochondrial oxidative phosphorylation. ALP has a low fatal dose, ranging from 0.15 to 0.5 mg, and the mortality rate is high (37 – 100%).¹

The clinical manifestations of ALP poisoning include gastrointestinal symptoms such as abdominal pain, vomiting and loose stools. Its effect on the cardiovascular system causes profound circulatory collapse, congestive heart failure and fatal arrhythmias. The patient can develop arrhythmias and ischaemic changes due to myocyte depression. Electrocardiogram variations include ST changes, QRS prolongation, heart blocks and ventricular fibrillation. Drowsiness, delirium and coma may result from cerebral anoxia. Other complications reported were pulmonary oedema, hepatitis, disseminated intravascular coagulation, gastrointestinal haemorrhage and renal failure.²

The diagnosis of ALP poisoning can be made by silver nitrate test on gastric lavage content. Currently, there is no antidote for ALP poisoning, therefore supportive care remains the mainstay of treatment. A good outcome can be achieved with

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Fig. 1: Aluminium Phosphide Packet.

early arrival, prompt diagnosis, toxin removal, aggressive resuscitation and intensive monitoring. As learnt from this case, physicians treating such patients should not be misled by the small amount of ingested substance or the transient improvement in haemodynamic status.

The initial approach is to stabilise the patient by ensuring adequate oxygenation, ventilation and circulation. A high inspired oxygen concentration together with tracheal intubation may be needed. Intravenous access must be established, and crystalloids administered to restore circulatory volume. Vasopressors will be required.³ Noradrenaline or phenylephrine is preferable as dopamine and dobutamine have a higher propensity for developing arrhythmias.

Reducing exposure to the substance is imperative. In accidental or occupational exposure, victims should be evacuated to an open space with fresh air, and immediate transfer to the nearest healthcare facility arranged. Healthcare providers must wear personal protective equipment when attending to the patient.

Phosphine gas can be absorbed cutaneously, thus the patient's skin and eyes should be decontaminated with plain water. If the substance is ingested, gastric lavage under airway protection should be done within one to two hours. Unfortunately, friction from the orogastric tube used for the lavage can aggravate thermal burns.⁴ In view of the fact that there is no available antidote and this patient's post mortem revealed presence of ALP in the gastrointestinal tract even after seven hours, effective gastric lavage should be considered an important treatment modality.

Diluted Potassium permanganate (1: 10 000) can be administered to oxidise phosphine into non-toxic phosphate. Nonetheless, this oxidation process can result in thermal burns; hence the use of potassium permanganate is discouraged.⁴ Phosphine gas is rapidly absorbed through the gastrointestinal tract, therefore activated charcoal should be given within 1 hour. The use of charcoal may be ineffective after ALP releases phosphine gas as the molecular size is small and it is in gaseous form. Coconut oil has been used in Iran and India with positive outcome but confirmatory evidence is lacking. Magnesium sulphate helps to scavenge oxygen radicals and reduce cellular toxicity from the phosphine gas.³ Enhancing elimination can be achieved by judicious intravenous fluid administration and diuresis. However, this is contraindicated once myocyte damage leading to heart failure has occurred.

Most deaths in ALP poisoning are due to cardiovascular failure. ARDS can develop and patients may need invasive ventilation support.

CONCLUSION

ALP is a very lethal poison with no specific antidote. Early detection, elimination of toxin and intensive supportive care are the mainstay of treatment. Coconut oil, magnesium sulphate and potassium permanganate are some of the other treatment modalities in ALP poisoning.

REFERENCES

1. Gupta S, Ahlawat SK. Aluminum phosphide poisoning-a review. *J Toxicol Clin Toxicol* 1995; 33(1): 19-24
2. Singh S, Singh D, Wig N, Jit I, Sharma BK. Aluminium phosphide ingestion--a clinico-pathologic study. *J Toxicol Clin Toxicol* 1996; 34(6): 703-6.
3. Agrawal V, Bansal A, Singh R, Kumawat B, Mahajan P. Aluminium phosphide poisoning: Possible role of supportive measures in the absence of specific antidote. *Indian J Crit Care Med* 2015; 19(2): 109-12
4. Mirakbari SM. Proposal for a new mechanism of action for aluminium phosphide (ALP) for causing local injuries in ALP poisoning: Should treatment strategies be modified? *Hum Exp Toxicol* 2015 Nov 30. pii: 0960327115619221. [Epub ahead of print]