International Journal Dental and Medical Sciences Research
Volume 3, Issue 6, Nov-Dec 2021 pp 332-333 www.ijdmsrjournal.com ISSN: 2582-6018

A case report of aluminium phosphide poisoning

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Submitted: 01-12-2021 Revised: 11-12-2021 Accepted: 14-12-2021

SUMMARY
Aluminium phosphide (ALP) is highly toxic and poisoning can result in high mortality
rates. A 23-year-old male who allegedly ingested a medium dose of ALP presented with vomiting and nausea. He developed tachycardia and hypotension with acute kidney injury. He had improved with resuscitation with IV fluids and Inotrops supports. In cases like this a better outcome can be achieved with early arrival, prompt diagnosis, aggressive resuscitation and intensive monitoring.

KEY WORDS:Poisoning, Aluminum phosphide.

I. 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 ALPpoisoning. Treatment is supportive.

II. CASE REPORT

A 23-year-old male was taken to a local hospital after allegedly ingesting two tablets of 39 aluminium phosphide. His relatives presented a packet of the tablets (Figure 1). Each tablet contained 56% ALP, which releases 1g of phosphine gas following ingestion. He took the tablets at 6 (six) hrs prior to admission to the hospital. Subsequently, the patient had nausea and episodes of vomitingq. At the local hospital, gastric lavage was performed. In hospital, his vital parameters were- (Pulse rate 101 bpm, BP 100/60 mmHg). Intravenous fluids were administered and he was referred to tertiary care centre for further management.

Upon arrival at the receiving emergency department, the patient was conscious and oriented , pale, had cold and clammy extremities. Patient had tachypnea and tachycardia with hypotension with metabolic acidosis. Patient was shifted to intensive care and treated with intravenous fluids , inotrops (Noradrinaline)support. In view of metabolic acidemia, sodium bicarbonate infusion was administered.

Bedside ultrasound showed good cardiac contractility. Patient responded well to the treatment administered. Patient had hypoxia , HRCT thorax was normal, he was given 2L O2 via nasal prongs.. On second day patient responded well with supportive care. ABG was repeated which showed resolution of acidemia.

III. DISCUSSION

ALP is a common and effective agricultural fumicide. While ALP poisoning is frequently seen in India and Iraq.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%).

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

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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 edema , hepatitis, disseminated intravascular coagulation, gastrointestinal haemorrhage and renal failure."

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.

Fig 1: Aluminium Phosphide Packet

A good outcome can be achieved with 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 hemodynamic 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 with Vasopressor will be required. Noradrinaline or phenylephrine is preferable as dopamine and debutamine have a higher propensity for developing

Reducing exposure to the substance is imperative. In the 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 cutaneuosly, thus the patient's skin and eyes should bedecontaminated with plain water. If the substance is ingested, gastric lavage under airway protection should be done within one to two Unfortunately, friction from the orogastric tube used for lavage can aggravate thermal burns. In view of the fact that there is no available antidote and 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 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 fluld 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.

IV. 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, KUNAWAT B, Mahajan P.Aluminium phosphide poisoning.posible role of supportive messures in the absence of specific antidote. Indian J Crit care made 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.pil:0960327115619221.[Epub ahead of print]