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Aluminium Phosphide Poisoning- Isolated Severe Myocardial Injury.

KEYWORDS: aluminium phosphide Poisoning, Cardiogenic shock

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ABSTRACT *ALP poisoning is a major cause of suicidal deaths in rural areas of India. Accidental exposure to the chemical is also common and is equally hazardous. Major cause of high mortality is delay in the primary treatment. In this case the patient suffered from cardiogenic shock and severe metabolic acidosis. The key to her survival was early and aggressive management of cardiogenic shock. Magnesium sulphate was used due to its membrane stabilizing action on the myocardium.*

INTRODUCTION

Aluminium phosphide (ALP) poisoning is a common occurrence in accidental and suicidal cases, predominantly in rural India, which is mainly attributable to poor regulation regarding the accessibility of this gravely toxic rodenticide.(1, 2). Aluminium phosphide on contact with moisture forms PHOSPHINE (PH3) gas, which leads to poisoning on inhalation, ingestion and dermal contact. The LD50 dose of Aluminium phosphide is 10 mg/kg of body weight. In India, most of the patients who come withAlphos(trade name for Aluminium Phosphide) poisoning succumb to its toxicity because of the considerable time gap between the ingestion of the poison and the initiation of proper treatment (3, 4). This has led to widely prevalent scepticism among physicians while managing cases of Alphos poisoning.

CASE REPORT.

We describe a lethal poisoning in a healthy woman caused by deliberate ingestion of aluminium phosphide (ALP), a pesticide used to kill rodents and insects. .

26 year old lady presented 4 hours after ingestion of a 3 gm of Alphos -aluminium phosphide (ALP),followed by multiple episodes of vomiting, and severe epigastric pain . On examination she was conscious , restless and irritable. Her heart rate was 140 per minute,tachypnoeic with a respiratory rate of 34/mt,blood pressure 70/50 mmHg with cold peripheries.Oxygen saturation was 86% on room air.

Arterial blood gases (ABG) on room air showed PO2 89.7 mmhg, PCO2 24.1 mmhg, pH 7.25, bicarbonate 10.3mmhg.

Routine blood work which included CBC/LFT/RFT were normal with a Hb of 10.6 gm%, total WBC count of 6410cells /cu mm(N51.8, L38.8), platelet count- 3.08lacs/cu mm, RBS-132, Serum transaminase -SGOT-56 u/L , SGPT 30 u/L, Sodium 140mmol/L, Potassium 3.7mmol/L , serum Creatinine- 0.8mg/dl, UREA- 28mg/dl.

ECGshowed Sinus tachycardia (fig 1A). 2D echocardiograph showed global hypokinesia with Ejection fractionF of 29 %.(fig 1 B)

She was given gastric lavage with normal saline,5 litres of oxygen via mask, Intravenous pantoprazole 40 mg stat and once a day, and 50 mmol sodium bicarbonate. Infusions of N- acetylcysteine (300mg/kg over 24 h) and magnesium sulphate 1 gm iv TID for 3 days, ionotropes were commenced as she remained hypotensive despite fluid resuscitation (dopamine and noradrenaline 5 microgrm/ kg/ minute). Metabolic acidosis was treated with 50 mmol/h sodium bicarbonate . on day 2 her blood pressure improved to 96/60 mmhg , dopamine and noradrenaline were tapered and stopped but was started on injection dobutamine (5 micro gram / kg/mt) in view of global hypokinesia and the infusion was continued for for 3 days (5 micrograms /kg/mt), gradually tapered and stopped.

She improved symptomatically, her repeat 2 D echo showed significant improvement in ejection fraction to 54 %.(fig. 1C)

However, her serial LFT/ CBC/ RFT were unremarkable.

Figure 1 A- ECG showing sinus tachycardia.

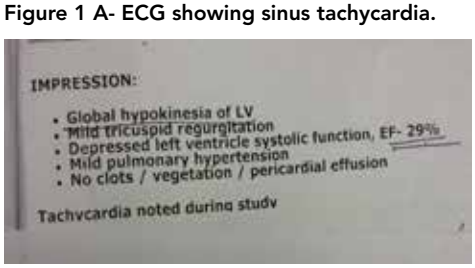

Figure 1-B. 2 D echocardiograph.

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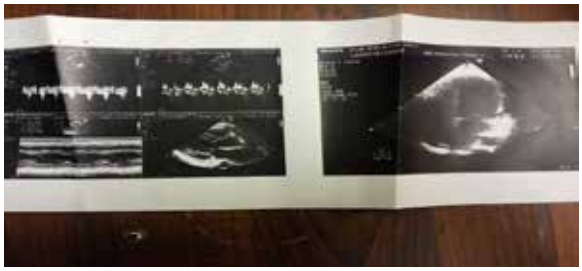

Figure 1-c- . Repeat 2D echocardiograph wih EF of 54%

DISCUSSION

This patient presented with the usual initial symptoms after ingestion of Alphos i.e. epigastric pain and vomiting, followed by the development of hypotension, which is the cardinal feature. Shock was suggested by feeble peripheral pulse, cold clammy skin and low blood pressure. Other associated symptoms which were present were restlessness and tachypnea . (1, 2, 3)

ECG changes seen in ALP poisoning cases include spectrum of atrial fibrillation, supraventricular tachycardia, premature ventricular contractions and ST- T changes. Of these, the ST-T changes with T wave inversion were by far the commonest . These changes were attributed to focal myocardial necrosis and changes in action membrane potential as a result of the alteration in the permeability of Na+, Mg++ &Ca++ ions.

However , in our case we documented sinus tachycardia.

Magnesium Sulphate is administered based on the documented evidence of its membrane stabilizing action. However, the rational use of Magnesium Sulphate had to be guided by serum Magnesium levels, as there have been reports of the occurrence of hypermagnesaemia.(5, 6) Metabolic acidosis resulted, probably due to lactic acidosis which was caused by the blocking of oxidation phosphorylation, which is similar to the effect of cyanide. In animal studies, phosphine has been reported to inhibit ADP uncoupler and ion stimulated respiration. It was found to be strong inhibitor of mitochondrial respiration in the active state. This inhibition could not be reversed by uncouplers, which suggested that it is due to the direct effect on electron transport which is an import mitochondria. Spectral and dichroisim studies revealed an interaction of phosphine with the heme moiety of cytochrome oxidase (cytochrome- C). A study demonstrated that cytochrome oxidase-c activity in the platelets of 26 patients with Aluminium phosphide poisoning was found to be inhibited to more than 50% ($p<.001$) as compared to healthy controls as well as to those in shock due to other causes.

Aluminium phosphide has no specific antidote and so favourable outcome correlated best with the severity of vomiting and the promptness of the initiation of treatment after toxicity. Unfavourable outcome was strongly correlated to the degree of hypotension and acidosis.(3, 4, 5)

In conclusion, the main guiding principles of management are early aggressive lavage with KMnO4 and treatment of hypotension and shock. Other appropriate supportive measures which are tailored to requirements of the patient complete the management of Aluminium poisoning.

CONCLUSION.

The skepticism about the prognosis of cases of ALP poisoning with complications like renal, hepatic and cardiac involvements has been long known in this part of the world where this agricultural poison still remains one of the most common suicidal agent. The above case report shows how a patient developed isolated cardiac involvement due to ALP poisoning and recovered completely with supportive management and close monitoring. This was the result of continuous intensive treatment and monitoring for the complications. The case also highlights the importance of colloids in management of shock due to ALP poisoning.

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