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CASE REPORT

The Successful Treatment Of Aluminium Phosphide Poisoning With Limited Resources

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ABSTRACT

Celphos (trade name for aluminium phosphide) poisoning is a major cause of morbidity and mortality in northwest and central India. The outcome is poor, largely due to delay in appropriate management and skepticism amongst physicians regarding the outcome. Things are further complicated by limited resources in tier 3 cities where most of the cases present initially. In this case, the favourable outcome was largely attributable to episodes of vomiting and aggressive gastric lavage done by an unacknowledged person, who first came in contact with the patient. Still, the patient presented with typical signs and symptoms of celphos poisoning and was managed well with saline lavage, IV Fluids, inj. Magnesium sulphate, inj. Hydrocortisone and broad spectrum antibiotics. In the absence of any specific antidote, management of celphos poisoning hinges on early aggressive gastric lavage and appropriate supportive measures dictated by the presenting sign and symptoms of the patient. The role of Magnesium Sulphate is not clearly documented, but it is used widely based on the membrane stabilizing action and hypo-magnesemia documented in some Aluminium Phosphide Poisoning cases.

Key Message: The favourable outcome of Celphos poisoning correlates best with the prompt removal of poison from the body and good supportive treatment.

Key Words: Celphos, aluminium phosphide poisoning, phosphine, magnesium sulphate, management of celphos poisoning.

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Introduction

Aluminium phosphide (ALP) poisoning is a common occurrence in accidental and suicidal cases, predominantly in rural northwest and central India, which is mainly attributable to poor regulation regarding the accessibility of this gravely toxic rodenticide [1],[2] It is uncommon in other parts of India as well as in rest of the world except in Iran and Jordan [3],[5] Aluminium phosphide on contact with moisture forms PHOSPHINE(PH3) gas which leads to poisoning on inhalation, ingestion and dermal contact [2]. The LD50 dose of ALP is 10 mg/kg of body weight. In India, most of the patients who come with Celphos (trade name for Aluminium Phospide) poisoning succumb to its toxicity because of the considerable time gap between the ingestion of the poison and the initiation of proper treatment. This has led to widely prevalent skepticism among physicians while managing cases of Celphos poisoning.

We are presenting here, a case that was managed in a small centre with limited resources .

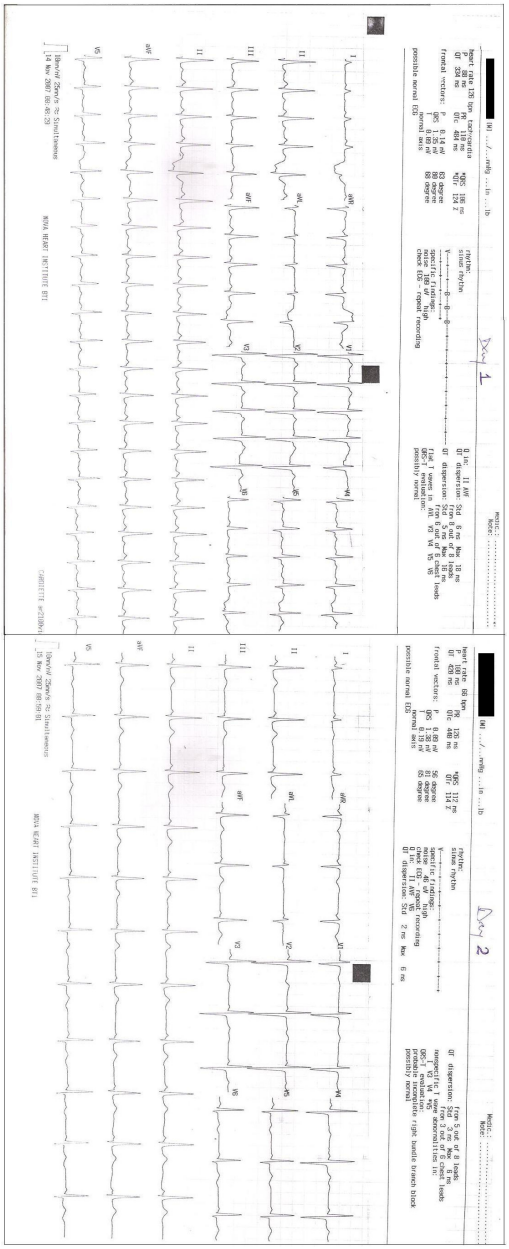
Case Report

A patient presented to the Casualty of Nagpal Hospital, Bhatinda, with history of

ingestion of five 3 gm tablets of celphos 7 hrs ago, with a heart rate of 128/min, SPO2- 78% (by pulse oximetry), respiratory rate -38/min,temperature 98.6 F, blood pressure- unrecordable, absent peripheral pulses and cold clammy extremities. The patient was irritable but was oriented. The patient was immediately shifted to the ICU where he was treated with Oxygenation, Inj. hydrocortisone 200mg I.V. stat, Inj MgSO4 1g I.V. stat and 0.5g I.M. in each buttock stat. IV fluids, Hemacel and RL were started. Dopamine infusion was started @ 10 mcg/kg/min. His samples were sent for investigations.

Following this primary resuscitation, a detailed history was elicited, which revealed that of the five tablets, three were vomited 5 minutes after ingestion and the other two were also thrown out after 45 minutes. He received treatment in his village, which consisted of KMnO4 lavage and enema, Inj. Dexamethasone 8 mg and IV fluids.

ABG revealed severe metabolic acidosis with PH-7.09, PCO2-24.4, PO2-306.5, Na-134.3, Ca-4.58 and HCO3 -7.3. 100ml of Soda-bicarbonate was given. One hour later, his systolic blood pressure had increased to 80 mm Hg. After 2hrs, his systolic blood pressure was noted to be 90 mm Hg and after 4 hrs, the pulse was recorded to be 98/min, blood pressure was 100/60 mm Hg and SPO2 was 94%. Gastric lavage was done with normal saline till the lavage fluid was negative for rotten fish smell (approx 12 hrs). Normal saline was used as no other solution for lavage was available at our center at that time (KMnO4 was unavailable in the hospital pharmacy and Bhatinda was deep asleep). Inj. MgSO4 1g in 100ml NS I.V was repeated every hour for three consecutive hours and then 8th hourly and Inj hydrocortisone 200mg iv 6th hourly and Inj Calcium Gluconate 1 amp iv 6th hourly were given for the first 48 hours. Inj Forticlav (Amoxicillin + Clavulanic Acid) 1.2 gm iv 8th hourly and Inj metrogyl 100ml iv 8th hourly were also given. During the MgSO4 therapy urine output, DTR and respiratory effort were monitored closely. Meanwhile, other investigations were noted as Hb- 10.8, blood urea- 34, S.creatinine- 1.5, S.bilirubin- 0.8, Total S.proteins- 6.9, S.albumin -4.5, S.globulin -2.4, SGOT/SGPT- 60/52 and S.alkaline PO4 - 198. ECG showed tachycardia on the 1st day and on the 2nd day, it revealed T inversion in aVL, V5 and V6. X ray Chest was normal. Tapering off of the infusion dopamine was started after stabilization of haemodynamics and was discontinued 24 hrs after admission. The patient continued to improve physiologically and biochemically over the next five days and was discharged in a stable condition after 5 days of stay [Table/Fig 1].



(Table/Fig 1)

Discussion

This patient presented with the usual initial symptoms after ingestion of ALP i.e epigastric pain and vomiting, followed by the development of hypotension, which is the cardinal feature. Shock was suggested by absent peripheral pulse, cold clammy skin and unrecordable blood pressure. Other associated symptoms which were present were restlessness, tachypnea and altered sensorium [2],[6],[7],[8],[9],[10].

ECG changes seen in ALP poisoning cases included 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 (which were seen in this patient). 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 [11],[12]. Magnesium Sulphate is administered, based on the documented evidence of its membrane stabilizing action. However, the rational use of Magnesium Sulpahte had to be guided by serum Magnesium levels, as there have been reports of the occurrence of hypermagnesaemia [11],[12],[13].

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 (14). 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 important electrochemical link between respiration and phosphorylation in the 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 ALP 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 [15],[16],[17]

ALP 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 [18].

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 ALP poisoning.

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