CASE REPORT

AN UNUSUAL SURVIVAL IN A CASE OF CELPHOS POISONING

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ABSTRACT

Celphos poisoning is a major cause of suicidal deaths in rural areas of developing countries like India. Accidental exposure to the chemical is also common and is equally hazardous. Major cause of high mortality is delay in receiving the primary treatment. In this case the patient suffered from altered sensorium, severe metabolic acidosis, shock, atrial fibrillation and later ventilator associated pneumonia and yet survived. The key to his survival was early lavage and use of colloids in management of shock which was till then not responding to high doses of vasopressors. Magnesium sulphate was used due to its membrane stabilizing action on the myocardium. Physicians managing cases of this lethal poisoning must leave their scepticism about the outcome and hence manage them aggressively.

Keywords: Celphos Poisoning, Survival, suicide, shock

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] It is uncommon in other parts of India as well as in rest of the world except in Iran and Jordan. 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 with Celphos (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 Celphos poisoning.

CASE REPORT

We present here a case of celphos poisoning who suffered almost all the grave complications of celphos poisoning despite getting treatment at a relatively early period after ingestion and yet survived.

This patient presented with an alleged history of ingestion of two 5gm tablets of celphos which were not exposed to air previously. The gap between ingestion of poison and presentation to the hospital was about 2 hrs at presentation he had a heart rate of 120 per minute with a blood pressure of 70/30mmof mercury, feeble peripheral pulses, cold clammy skin, respiratory rate of 26/min and an SpO2 of 95%.he was conscious but irritable and confused. Arterial blood gas showed severe metabolic acidosis (pH7.06, HCO3-13.8), Na+ 141.7, K+-5.13, iCa-1.192, pCO2-49.8.

Patient was immediately shifted to Intensive care unit where lavage was performed with normal saline, and intravenous fluids along with vasopressors were started from two separate intravenous accesses. During all this the SpO2 fell to 70% and the cardiac rhythm became irregularly irregular with absent P waves on ECG. The patient also developed cyanosis

And urgent endotracheal intubation was done. Inj hydrocortisone 400 mg i.v. stat was given and inj. MgSO4 1gm in one unit of i.v. fluid was given. An urgent 2-D echocardiogram was performed which revealed no regional wall motion abnormality.

Even after intensive use of vasopressors (nor adrenaline and dopamine) the shock did not respond. At this time colloid infusion at the rate of 100 ml per hour was started. The blood pressure improved after about 6 hrs and vasopressors were gradually tapered over the next 24 hours with continuing hydrocortisone injections (100 mg 8th hourly), magnesium sulphate (1gm every 8th hourly) and colloid transfusion.

Routine investigations showed a total WBC count of 13500/cu mm(N75, L22), Hb-14.5g/dl, plt count-3.63lacs/cu mm, RBS-148, Serum transaminase elevated (SGOT/SGPT-220u/L/184u/L), Sodium

Na+/ Potassium K+(141mmol/L, 3.7mmol/L) with renal impairment Creatinine-(3.04mg/dl), UREA-(56mg/dl).

On the third day of stay the chest x-ray of the patient showed a large pneumonitic patch in the upper zone of right lung. The antibiotics were changed to Piperacillin-tazobactam, amikacin and metrogyl and intensive chest physiotherapy was given.

The patient improved gradually over next 7 days of ICU stay. Atrial fibrillation reverted spontaneously on day 8, chest x-rays showed serial improvement and extubation was done on 12th day and shifted to general ward for further management. He was discharged with stable vitals on day 15.

DISCUSSION

This patient presented with the usual initial symptoms after ingestion of Celphos 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, tachypnea and altered sensorium. [1, 2, 3]

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. 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 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 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 celphos 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 with all the above complications along with Ventilator Associated Pneumonia was saved from death. 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 celphos poisoning.

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