SURVIVAL OF A CASE OF CELPHOS POISONING WITH DERANGED LIVER FUNCTION AND ACUTE RENAL FAILURE

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HOW TO CITE THIS ARTICLE: Samota MK, Bedwal H, Beniwal P, et al. Survival of a case of celphos poisoning with deranged liver function and acute renal failure. J. Evid. Based Med. Healthc. 2017; 4(91), 5521-5522. DOI: 10.18410/jebmh/2017/1104

PRESENTATION OF CASE

A 29-year-old female was admitted in ICU of Jhalawar Medical College and Hospital, Jhalawar (Rajasthan). Two hours after alleged ingestion of a fresh full packet of powder (10 gm) of celphos poison (aluminium phosphide).

She presented with cold and clammy extremities with HR of 120 per minute and she was in shock with blood pressure 70/60 mm of Hg, feeble peripheral pulse, RR of 26/minute and SpO2 of 95%. She was conscious, but irritable and confused. ABG showed metabolic acidosis (pH 7.02, HCO3 12.4, Na 140, K 5.4, PCO3=50).

DIFFERENTIAL DIAGNOSIS

Agricultural poisoning, e.g. organophosphates and organochloride can be differentiated from celphos by its clinical presentation like salivation, lacrimation, urination, runny nose, impaired and blurry vision, muscle weakness, muscle twitching with bilateral meiotic pupil confusion, agitation and coma.

Zinc phosphide (rat killer poison) is important differential diagnosis for celphos poisoning. It can be differentiated by celphos by its colour, odour and clinical symptoms. Both Aluminium Phosphide (AIP) and zinc phosphide poisoning is a common occurrence in accidental and suicidal cases, predominantly in rural India.[1,2] Celphos poisoning is a major cause of suicidal deaths in rural areas of developing countries like India.

ALP is a solid type of fumigant, which is available in tablet and powder form. In one study, it was found to be the most common cause of acute poisoning in India.[3]

As the lethal dose of aluminium phosphide is 150-500 mg for an adult, even a single tablet of 3 g can cause mortality.[4]

It is colourless and odourless, however, on exposure to air, it gives a foul odour (garlicky or decaying fish) due to the presence of substituted phosphine and diphosphines.[5]

Financial or Other, Competing Interest: None.
Submission 09-11-2017, Peer Review 16-11-2017,
Acceptance 23-11-2017, Published 27-11-2017.
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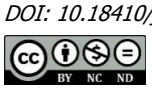
DOI: 10.18410/jebmh/2017/1104



This odour also helps in the diagnosis of ALP poisoning. ALP after exposure to moisture or acid in the stomach releases the implicating phosphine (PH3) gas.

CLINICAL DIAGNOSIS

A diagnosis of ALP was made by history given by her relatives and packet of celphos brought by her relatives. There was garlic-like smell coming from her mouth. Diagnosis can be made easily by silver nitrate impregnated paper test on gastric content or on breath.

Gas chromatography with a nitrogen phosphorus detector is the most specific and sensitive test and it can be used for analysis of airtight samples.

PATHOLOGICAL DISCUSSION

Exact mechanism of toxicity is still not fully understood, but it is speculated that it acts by blocking the cytochrome c oxidase in the mitochondria, thus disrupting the Oxygen (O2) transport in Electron Transport Chain (ETC) eventually leading to cell death.[6] Phosphine primarily inhibits cytochrome c oxidase or complex IV of the ETC, thus leading to the inability of mitochondria to utilise O2 and causing cellular hypoxia.[7] There occurs generation of Reactive Oxygen Species (ROS), mainly Superoxide (O2) and Hydrogen Peroxide (H2O2) causing cellular oxidative stress. These ROS are highly damaging to biological macromolecules ultimately leading to cell death. Glutathione is the strongest protective antioxidant.[8] Glutathione not only protect against oxidative cell damage, but enhances cell survival as well. Magnesium ions help in scavenging-free radicals by raising glutathione level, also magnesium is an antiarrhythmic agent.

DISCUSSION OF MANAGEMENT

Gastric lavage with mixture of 50% soda bicarbonate and 50% coconut oil was done immediately and repeated after 15 minutes and bolus IV saline, IV infusion of soda bicarbonate was started to correct metabolic acidosis and injection of 2 g of magnesium sulphate followed by 1 g every 6 hourly was given.

For next 48 hours, vitals and other parameters were taken at regular interval, relevant investigations were done in time and supportive treatment was given accordingly.

At the time of admission, routine investigations, CBC, LFT, RFT, serum electrolytes, serum calcium and serum magnesium were done.

On day 2, she developed septicaemia with raised WBC count of 15,360/µL and increase in blood urea (68 mg/dL) and serum creatinine (2.6 mg/dL), SGOT 19 6 units/L, SGPT 110 units/L, while serum K+ 5.8 mEq/L, S. Na+ 146 mEq/L associated with fall in systolic BP below 70 mm of Hg and inotropes dopamine and dobutamine infusion were started and tapered according to BP, thereafter next day RFT and LFT were further deranged and hyperkalaemia and septic shock was present with decreased 24 hours urinary output. On day 3, patient developed GTC seizures, which was managed with IV lorazepam and phenytoin.

Patient was sent to dialysis unit and haemodialysis was done on day 7. Next 4 days, patient was maintaining BP on inotropes, and on day 11, patient had normal BP without inotropes. Meanwhile, patient become anaemic and one unit packed cell volume was transfused.

On day 12, patient was shifted to ward for further observations and was given supportive treatment.

FINAL DIAGNOSIS

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 was the cardinal feature. Patient was in shock suggested by feeble peripheral pulse, cold clammy skin and low blood pressure (systolic BP 70 mm of Hg). Other symptoms present were restlessness, palpitation and altered sensorium (1, 2 and 3). Seizures are known to occur in ALP poisoning as seen in our patient also because of the suppression of acetylcholine esterase by phosphine, which increases acetylcholine neurotransmission and in extreme cases can cause excitotoxicity and seizures.[9] Acute cardiovascular collapse is the most common presentation seen in 60% to 100% of cases and also in our patient.[10] This is secondary to the direct effect of PH3 on cardiac myocytes, fluid loss and adrenal gland damage.[11] The patient was given supportive treatment as there is no specific antidote available for ALP. Early use of soda bicarbonate and magnesium have helped in saving the patient. Magnesium ions help in scavenging free radicals by raising glutathione level, also magnesium is an antiarrhythmic agent. Magnesium sulphate is administered based on the documented evidence of its membrane stabilising 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.[12,13] Metabolic acidosis resulted probably due to lactic acidosis, which was caused by the blocking of oxidation phosphorylation, 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.[14] Main guiding principles of management are early aggressive lavage with mixture of 50% soda bicarbonate and 50% coconut oil and treatment of hypotension and shock. Other appropriate supportive measures according to requirements of the patient to complete the management of aluminium poisoning. Survival of cases of celphos poisoning with complications like renal, hepatic and cardiac involvements is unusual. This patient could be saved because of early initiation of aggressive proper treatment, close monitoring of vitals, use of vasopressors, correction of renal and hepatic functions, dialysis and timely therapy with magnesium and soda bicarbonate infusion.

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