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SURVIVAL OF A CASE OF CELPHOS POISONING WITH DERANGED LIVER FUNCTION AND ACUTE RENAL INJURY WITH DEEP VEIN THROMBOSIS

Internal Medicine

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KEYWORDS

PRESENTATION OF THE CASE

A 40 year old male was admitted in intensive care unit of Jhalawar Medical College and Hospital ,Rajasthan three hours after alleged fresh ingestion of 1 tablet (3 gram) of celphos (aluminum phosphide).

He presented with cold clammy extremities with heart rate of 110 bpm and was in shock with systolic BP 70mm hg and diastolic BP 50 mm Hg, feeble peripheral pulse ,respiratory rate 26 per minute and SpO2 92% on room air.The patient was conscious ,irritable and confused.ABG showed metabolic acidosis with pH 7.4 bicarbonate 19mMol/ litre, Na 137 mEq/litre K 4.2 mEq/litre.ECG changes depicted sinus tachycardia and ST depression with T wave inversions in anteroseptal leads.2D ECHO revealed no regional wall motion anomaly.

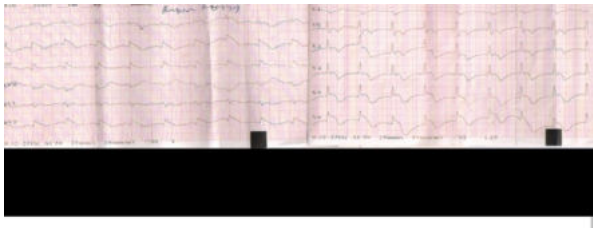

Figure 1

Electrocardiogram suggestive of ST-T wave changes in the form of ST segment elevation in inferolateral leads and ST depression with T-wave inversion in anteroseptal leads

DIFFERENTIAL DIAGNOSIS

Agricultural poisoning like organophosphorus and organochlorines can be differentiated from celphos by its clinical presentation like salivation, lacrimation , urination, running nose ,impaired and blurry vision, miotic pupils ,muscle weakness, muscle twitching, agitation ,confusion and coma.

Zinc phosphide (rat killer poison) is important in differential diagnosis for celphos poisoning.It can be differentiated from celphos by its colour ,odour,and clinical symptoms .Both zinc phosphide and aluminum phosphide(ALP) is a common occurrence of accidental and suicidal poisoning predominantly in rural India[1,2]. Celphos poisoning is a major cause of suicidal death in rural areas of developing countries like India[3].

ALP is a solid type of fumigant , which is available in tablet and powder form. The lethal dose of alumunium phosphide is 150-300 mg for an adult so even a single tablet of 3gram can cause mortality[4].

It is colourless and odourless, however on exposure to air it gives foul odour (garlicky or decaying fish) due to presence of substituted phosphine and diphosphine[5] and this odour helps in diagnosis of ALP poisoning.ALP after exposure to moisture in the stomach releases the implicating phosphine PH3 gas.

CLINICAL DIAGNOSIS

Diagnosis of ALP Poisoning was made by the typical history of garlic like smell coming from mouth as narrated by the patient's relatives. Diagnosis can be easily made by silver impregnated paper test on gastric content or on breath . Gas chromatography with nitrogenous phosphorus detector is most specific and sensitive test for analysis of airtight samples.

PATHOLOGICAL DISCUSSION

Exact mechanism of injury is still not understood ,but it is speculated that ALP acts by blocking cytochrome c oxidase 1 in the mitochondria thus disrupting the oxygen transport in electron transport chain eventually leading to death[6] .Phosphine primarily inhibits cytochrome c oxidase or complex 4 of ETC thus leading to inability of mitochondria to utilize oxygen thus resulting in cellular hypoxia[7]. Generation of reactive oxygen species (ROS) like superoxide ,hydrogen peroxide causes oxidative stress and damage to biological macromolecule ultimately leading to cellular death. Glutathione is the most potent and protective antioxidant[8] not only protecting against oxidative damage but also enhancing cell survival. Magnesium molecule is also a potent free radical scavenger by increasing glutathione levels and also acts as an antiarrythmic agent[9].

DISCUSSION OF MANAGEMENT

Gastric lavage with mixture of 50% sodium bicarbonate was done immediately and repeated after 15 minutes and bolus IV saline ,IV infusion of sodium bicarbonate was started to correct metabolic acidosis and injection of 2 gram magnesium sulphate followed by 1 gram every 6 hourly.At the time of admission, routine investigations like complete blood count,renal function test,liver function test , serum electrolytes, calcium and magnesium were sent and vitals, ECG and other parameters were taken at regular intervals and supportive treatment like ionotropes (nor adrenaline and dopamine)support was started accordingly.On investigation it was found that the patient was in shock with fall of systolic BP to 70mm of Hg , sepsis with deranged liver and renal parameters.Blood urea was 89mg/dl and serum creatinine 4.9mg/dl which after 48 hours rose to 183mg/dl and 5.9mg/dl respectively and there was development of acute renal injury and with hyperkalemia (potassium K 6.3 mmol/litre), sodium (Na147mmol/litre) , septic shock and decreased urine output.Liver parameters were also disturbed with SGOT 2951IU/L and SGPT 1357IU/L which also showed a rising trend within 48 hours with values reaching 5687IU/L and 2547IU/L respectively of SGOT and SGPT.The patient was put on higher antibiotics(Piperacillin-Tazobactam and Metronidazole), ionotrope support, ventilator support, blood transfusion and a central line was secured.Ionotropes were titrated according to BP.Colloid infusion of 100ml per day was also given for first 3 days. For the next 7 days he was sent to dialysis unit and sequentially hemodialysis were done. Over the course his liver and renal parameters came down gradually to normal levels and there was spontaneous reversion of ECG ST-T changes. Within 15 days of ICU admission ,he developed swelling of his left lower limb ,for which D-dimer (5040ng/ml) was done and USG colour Doppler revealed edema with thrombus in his left CFV ,SFV, popliteal vein and proximal GSV .Subcutaneous enoxaparin was started for 5 days with overlapping rivoroxaban tablet(Direct oral anticoagulant) and prophylactic treatment was continued .With further treatment patient recovered, maintained BP without ionotropes and hence was shifted to general ward after30 days of ICU admission for further

observation.On day 36 he was discharged with stable vitals and psychiatric counselling.

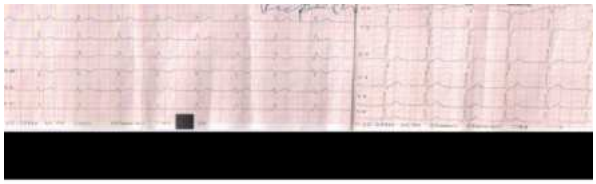

Figure 2

Electrocardiogram showing resolution of changes

FINAL DIAGNOSIS

The patient presented with usual initial symptoms after ingestion of celphos like epigastric pain, vomiting followed by hypotension which was the cardinal feature. Patient was in shock suggested by feeble pulse ,cold clammy skin and low blood pressure. Other symptoms presented were restlessness ,palpitation and altered sensorium. Acute cardiovascular collapse is the most common presentation in 60-70 % of cases and also in our patient.[10] This is secondary to direct effect of PH3 on cardiac myocytes, fluid losses, and adrenal gland damage[11]. The patient was given supportive treatment as there is no specific antidote available for ALP. Early use of sodium bicarbonate and magnesium have helped in saving the patient .Magnesium ions helps in scavenging free radicals by raising glutathione levels, also magnesium is an antiarrythmic agent[12]. Metabolic acidosis resulted probably due to lactic acidosis, which is caused by blocking oxidative phosphorylation[13]. Unfavorable outcome was strongly correlated to the degree of hypotension and acidosis[14] .Main guiding principle of management are early aggressive lavage with sodium bicarbonate and treatment of hypotension and shock. Other appropriate supportive measures according to requirement of patient, like timely hemodialysis complete the management of ALP poisoning. Survival of cases of celphos poisoning with complications like cardiac, renal ,hepatic and vascular(Deep vein thrombosis) is unusual. The 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, hemodialysis and timely therapy with magnesium and sodium bicarbonate infusion.

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