

Role of Chelating Agent in Acute Copper Sulphate Poisoning: A Case Report

Alka Chandra^{1, *}, Dheeraj Narain Agarwal¹, Anil Vivek², Umesh Yadav³

¹Department of Anaesthesiology and Critical Care, Hindurao Hospital, New Delhi, India

²Department of Anesthesia, Hindurao Hospital, New Delhi, India

³Department of Medicine, Hindurao Hospital, New Delhi, India

Abstract

Copper sulphate poisoning was one of the commonest mode of poisoning in certain parts of India, but its suicidal ingestion has dramatically decreased over the years. We present a case of copper sulphate poisoning successfully treated by general supportive measures and D-penicillamine.

Keywords

Copper Sulphate, Poisoning, D-Penicillamine

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1. Introduction

Acute copper sulphate poisoning is rare in developed countries. It occurs more frequently in developing countries. Acute toxicity usually results from oral ingestion and there are several local and systemic effects. Specific management can be difficult as there is little evidence regarding the efficacy of chelating agents in acute toxicity [1]. D-penicillamine is a structurally distinct metabolite of penicillin, an orally bioavailable monothiol chelator, which was used in our patient with promising results [2]. However early deaths are the consequence of shock while late mortality is related to renal and hepatic failure [3].

2. Case Report

A 18 year old male, who was apparently healthy presented with history of haemetemesis and altered sensorium two hour before admission to emergency department. There was no history of fever, drug usage trauma or alcohol ingestion. The blood pressure was 80/50, heart rate 106beats /min respiratory rate 20 beats minutes and oxygen saturation 94%.

The systemic examination revealed no abnormality, intravenous catheter was inserted and blood sample taken for laboratory test. A ryle's tube was inserted and gastric lavage done. The patient had a sudden episode of generalised tonic clonic convulsion, which was controlled by intravenous diazepam 10mg. Computerised tomographic scan of head revealed no abnormality. Electroencephalography could not be done due to lack of facilities. Blood sugar, electrolytes and arterial blood gas analysis showed no deviation from normal values. The patient was shifted to intensive care unit for further management. When the patient gained consciousness; he gave the history of consuming copper sulphate approximately 20 gms.

Gastric lavage was done with activated charcoal and tablet D-penicillamine 500 mg was started at 6 hrly interval. Injection phenytoin 100 mg was given 8 hourly, however there was no further episode of convulsions. Proton pump inhibitor was started through continuous infusion due to severe gastritis and abdominal pain, however there was no organomegaly. A broad spectrum antibiotic was started Meropenam 1gm intravenously 8 hourly.

* Corresponding author

E-mail address: dralkadelhi@yahoo.co.in (A. Chandra)

The investigations were within normal limits on the 1st day but the serum bilirubin level increased to 6 mg/dl with more of unconjugated fraction, liver enzymes level were normal. The methemoglobin level was also within normal limits as measured by CO-oximeter available in our ICU. The bilirubin levels came back to normal on the 5th day. The condition of the patient also improved gradually. The patient was shifted to the ward from where he was discharged on the 7th day on D-penicillamine and proton pump inhibitor for further 3 days.

3. Discussion

Copper sulphate was chiefly used for agricultural purposes as a pesticide and in a leather industry. It has a nauseous and metallic taste^[4]

It is consumed mainly with suicidal intentions. Accidental poisoning have been reported from children as well^[5]. The immediate symptom following ingestion of copper sulphate universally is gastrointestinal in the form of nausea, vomiting and crampy abdominal pain. Vomiting is characteristically greenish blue. In severe cases haematemesis and malena occur^[6]

Copper is rapidly taken up by erythrocyte and result in oxidative damage and many result in haemolysis of RBC. This may account for the delayed secondary episode of haemolysis, that occur in some copper sulphate poisoning patients^[4,7]. Intravascular haemolysis appears to be the chief factor responsible for renal lesion in these patients. Histological lesion observed in the kidney varied from those of mild shock to well established acute tubular necrosis^[8]

Copper ions can oxidize haem ion to form methemoglobin which loses its oxygen carrying capacity, chemical cyanosis and chocolate brown blood may be seen^[9]. Jaundice appears after 24-48 hr in more severe poisonings, which may be hemolytic or hepatocellular. It may be associated with tender hepatomegaly^[10]. The central nervous system depression may occur ranging from lethargy to coma or seizure which are likely epiphenomenon related to other organ involvement. If the history is not clear serum and blood copper estimation on a sample collected early in the course may be of help^[11]

Serum concentration normally range from 10.5 to 23 micromoles/litre but it is not mandatory to get serum level done if the diagnosis is obvious by history and clinical examinations.^[12] Most patients with copper poisoning are initially treated with intramuscular British anti-Lewisite (BAL). BAL proves useful in patients with renal failure. When tolerated, D-penicillamine therapy should be started simultaneously or shortly after the initiation of therapy with BAL.^[11] The D-penicillamine copper complex undergoes

rapid renal clearance in patient with competent kidneys. The use of D-penicillamine is not properly studied in the patient with acute copper salt poisoning but case study and animal models suggest that copper elimination is enhanced.^[13]

In our case the patient presented with history of vomiting which was suggestive of haematemesis and in the hospital the patient had a generalized tonic clonic convulsion. Once the convulsions were controlled the patient gave the history of ingestion of copper sulphate he was then managed with activated charcoal lavage. The patient had severe abdominal pain but no episode of vomiting in the intensive care unit so endoscopy was not done, but infusion of pantoprazole started. D-penicillamine was started as a chelating agent as it was readily available and the patient dramatically responded to the therapy.

4. Conclusion

Lavage with activated charcoal and use of chelating agent such as D-penicillamine which is readily available improves the morbidity and mortality of copper poisoned victims.

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