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Case Report

Acute poisoning with copper sulphate in a young lady

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ABSTRACT

Ingestion of copper sulphate is an uncommon form of poisoning which is usually reported in Indian subcontinent. This poisoning can lead to life threatening serious complications like acute intravascular hemolysis, renal failure, rhabdomyolysis and methemoglobinemia. Due to its rarity, physicians may face difficulties with management. Here we report a case of acute ingestion of copper sulphate in a 30-year-old lady who presented with intravascular hemolysis and acute renal impairment. She was managed with gastric lavage, intravenous fluid, blood transfusion, broad spectrum antibiotics, D-penicillamine and proton pump inhibitor. She recovered fully after two weeks. With effective supportive measures and chelating agents, the morbidity and mortality can be reduced in severely poisoned patients.

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1. Introduction

Copper sulphate, also known as 'blue vitriol', is a chemical substance which is used mainly in the agricultural field as pesticide and in the leather industry. It is a powerful corrosive agent. Lethal dose of copper sulphate is 10-20 gram (Ellenhorn, MJ, 1997). It is easily accessible and found over the counter. Poisoning with copper sulphate is uncommon. Few cases were reported in Bangladesh (Chowdhury *et al.*, 2011). Mode of poisoning is usually suicidal, though accidental poisoning was reported in children. Copper sulphate poisoning may lead to intravascular hemolysis, hepatic and renal injury. Here we report a case of severe acute copper sulphate poisoning to create awareness among the physicians regarding its management.

2. Case report

A 30-year-old lady was admitted in Enam Medical College and Hospital, Savar, Dhaka with history of ingestion of unknown amount of copper sulphate 2 days back. She had been managed initially with activated charcoal and intravenous fluid in a local clinic without any improvement. She was complaining of diffuse abdominal pain and pain in the throat along with vomiting for several episodes. She also complained of passage of black colored scanty urine and black stool for 1 day. On examination, she was moderately anemic and mildly icteric. Her heart rate was 105 beats per minute, respiratory rate 18 breaths per minute, blood pressure 90/60 mm of Hg, temperature 37.3°C. Her abdomen was soft but diffusely tender without any organomegaly. Lungs were clear. Her investigation reports revealed Hb 8 g/dL, WBC 45000 $\mu\text{mol/L}$, serum bilirubin 3.2 mg/dL, SGPT 53U/L, prothrombin time 14 sec, serum creatinine progressively raised from 93 $\mu\text{mol/L}$ to 124 $\mu\text{mol/L}$, serum electrolytes were normal. Urine routine microscopic examination revealed albumin 3+, free hemoglobin was positive, urinary total protein was 3.3g/24hrs. Chest radiography and ultrasonography of abdomen were normal. The electrocardiogram had features of sinus tachycardia. Endoscopy of upper gastrointestinal tract showed gastric erosions.

Patient was managed with intravenous fluid, omeprazole infusion, broad spectrum antibiotic (ceftriaxone 1 gm two times daily) and 2 units of red cell concentrates transfusion. Initially D-penicillamine 1000 mg 6 hourly was given along with intramuscular injection of dimercaprol. But D-penicillamine was withdrawn as there was progressive renal impairment. Dimercaprol was administered 4 hourly and was continued for 2 days, then every 6 hours for another 2 days followed by every 12 hours for more 6 days. The diagnosis of methemoglobinemia was excluded by clinically observing the features like chocolate colored cyanosis and poor peripheral oxygen saturation which were absent in this patient. We could not confirm it biochemically as this assay was not available in this hospital. With chelation and adequate fluid replacement, she had a good clinical recovery with subsidence of jaundice and straw color urine with increased urinary output. Her WBC count came down to 12000 $\mu\text{mol/L}$. She was discharged on the 11th day and was followed up after 2 weeks. Her complete blood count and serum creatinine came back to normal range.

3. Discussion

Copper is one of the important trace elements in human body which acts as a cofactor in several enzyme reactions. Total body content of copper is 150 mg. It is found in serum in two forms. About 93% is tightly bound to ceruloplasmin and 7% is loosely bound to albumin (Ellenhorn, MJ, 1997). It is copper-albumin complex which is responsible for the toxicological active portion (Barceloux, DG, 1999).

After ingestion of copper, immediate dilution with water or milk is preferred as these agents may dislodge the adherent solid particles and dilute caustic products. Emesis should be avoided to avoid the re-exposure of the esophagus to the corrosive (Friedman and Lovejoy, 1984). Activated charcoal can be considered if the patient comes within 1 hour of ingestion though it has no proven benefit. Our patient was given activated charcoal in the local clinic within 2 hours of ingestion of copper sulphate.

Endoscopy of upper gastrointestinal tract is considered early if there is suspicion of corrosive injury. Several studies revealed that endoscopy did not cause any complications; rather this procedure helped clinician to grade the extent of injury and predict the prognosis (Di Costanzo *et al* 1980, Dilawari *et al* 1984). In this case, endoscopy was done as the patient had melaena. The procedure revealed gastric erosions.

Poisoning with copper sulphate affects mainly erythrocytes, liver and kidneys (Friedman and Lovejoy, 1984). Patients may present with features of intravascular hemolysis and methemoglobinemia which occur due to oxidative damage to the erythrocyte membranes. After ingestion of copper sulphate, vomiting usually occurs along with abdominal pain. Hematemesis and melaena occur in severe cases. In a study in India involving 19 patients with copper sulphate poisoning requiring hemodialysis, 37% developed gastrointestinal bleeding and significant hypotension developed in 5% (Agarwal *et al*, 1993). In a retrospective analysis of 35 patients presenting with copper sulphate poisoning over a period of 10 years in India showed that major complications included hemolysis (68.57%), renal failure (51.43%), acute hepatitis (45.71%) and upper gastrointestinal bleed (40%) (Naha *et al*, 2012). In our case, patient developed features of intravascular hemolysis suggested by presence of mild jaundice and passage of black colored urine which was positive for free hemoglobin.

Methemoglobinemia can occur in 3.4% to 42% cases (Ellenhorn, 1997). It is usually suspected by blue or chocolate colored cyanosis and confirmed by serum methemoglobin level. Patients with this condition should be

treated with methylene blue infusion 1-2 mg/kg/dose (0.1 to 0.2 ml/kg of 1% solution) intravenously over 5 minutes. The dose may be repeated if cyanosis does not disappear within one hour (Di Costanzo *et al* 1980). In this case, patient did not have any features of methemoglobinemia though it was not biochemically proven due to lack of facility of measuring serum methemoglobin in the hospital.

Jaundice may develop in severe poisoning within 24-48 hrs. Type of jaundice may be prehepatic or hemolytic and hepatocellular. Patient in this case had jaundice with mild elevation of SGPT, bilirubin and normal prothrombin time. With conservative management, her jaundice subsided gradually. In one series, jaundice was seen in 58% patients and 5% patients died of hepatic encephalopathy (Agarwal *et al*, 1993).

Renal involvement usually occurs on 3rd or 4th day after poisoning. It is a combination of direct toxic effects on the proximal tubules, reduced renal perfusion secondary to hypovolemia and intravascular hemolysis. Renal recovery may be incomplete (Oon *et al*, 2006). About 20-40% patients may develop renal failure (Dash, 1989). Urinary presentations are commonly oliguria, anuria, albuminuria, hemoglobinuria and hematuria. Our patient had hemoglobinuria with progressive increase of creatinine which became normal with conservative therapy.

Cardiovascular system, skeletal muscles, central nervous system and endocrine systems are rarely affected (Levy *et al*, 2003). Hypotension and shock are responsible for early death, whereas late death occurs due to renal and hepatic causes (Oldenquist and Salem, 1999). Seizure and arrhythmias were reported in few cases (Saravu *et al*, 2007, Fernando, 2007).

Multi-organ dysfunction and sepsis can aggravate the clinical condition of the patients. Sepsis may be due to transmucosal invasion (Nelson, 2002). Translocation of intestinal bacteria may be the source of respiratory infection (Hantson *et al*, 1996). Infection should be controlled with broad spectrum antibiotics. In this case, patient was given broad spectrum antibiotic with subsequent reduction of leucocyte count.

Chelation therapy is used to remove excess copper from the body. D-penicillamine, intramuscular dimercaprol and edetate calcium disodium are used as chelating agents. Among these, dimercaprol is more appropriate in renal impairment as it forms a complex with copper and undergoes primarily biliary excretion (Nelson, 2002). It should be given at 3 to 5 mg/kg/dose deep intramuscularly every four hours for two days, every four to six hours for next two days, then every four to 12h for up to seven additional days (Hantson *et al*, 1996, Takeda, 2000). D-penicillamine should be avoided in renal failure as it may further deteriorate renal function. In this case, D-penicillamine was stopped earlier as there was deterioration of renal function. Dimercaprol was given for 10 days and patient fully recovered.

4. Conclusion

Copper sulphate poisoning can lead to serious complications which may cause death. Physicians should be aware about the fact that early management with supportive therapy, chelation and methylene blue in methemoglobinemia would reduce the mortality to a great extent.

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