Case Report

A Fatal Case of Copper Sulfate Poisoning

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ABSTRACT
Copper sulfate or 'blue vitriol' is commonly used as a herbicide and fungicide. It is cheap and easily available, and can also be easily prepared in the laboratory. We report a fatal case of suicidal poisoning by copper sulfate by a pregnant adult female. Although medical treatment was instituted, the fatality ensued. Histopathological examination revealed hepatosplenomegaly and evidence of septic abortion. Chemical analysis of viscera confirmed copper sulfate poisoning. This case is being reported on account of its rarity, the victim being a pregnant woman.

Key Words: Copper sulfate; Blue vitriol; Suicide; Poisoning

Introduction
Copper sulfate is a chemical compound with formula $CuSO_4$. It is widely used as herbicide, fungicide, as a colouring agent in paints, and as a common reagent in leather industry.[1] Copper sulfate can also be easily prepared in the laboratory by the action of sulfuric acid on copper containing salts. It occurs as blue coloured crystals, the colour being due to water of hydration. When heated in an open flame, the crystals of copper sulfate get dehydrated and turn grayish-white.[2]

Of late, cases of copper sulfate poisoning are relatively rare in medical literature, and very few cases have been reported of the effects in a pregnant victim. Copper sulfate can be absorbed though skin, gastrointestinal tract and lungs causing both systemic and local toxicity.[1] Medically, copper sulfate was used in the past as an emetic but was discontinued owing to its toxicity. It is listed as an obsolete antidote for phosphorus poisoning by the World Health Organization.[3,4]

Here, a case is presented where an adult pregnant female consumed some poison and died in hospital while undergoing treatment. After postmortem examination of the body, histopathological examination of organs, and chemical analysis of viscera, the cause of death was revealed to be copper sulfate poisoning.

The Case: A 30-year-old female was admitted to the hospital with history of consumption of some poison a few hours ago. She manifested nausea, vomiting, abdominal pain and diarrhoea. She died within 48 hours of hospitalization before any positive diagnosis could be made, and an autopsy was ordered. Due to some technical problems from the side of investigating authorities and relatives, autopsy could be performed only after 2 days.

At autopsy, the conjunctivae appeared yellowish, among other non-specific findings. On internal examination, brain and lungs were noted to be oedematous, while the spleen was enlarged. The stomach and small intestines contained brownish coloured liquid with a peculiar odour. Uterus weighed 100 gm and showed clotted blood and tissues within its cavity. All internal organs showed mild putrefactive colour changes.

Chemical analysis of routine viscera was positive for copper sulfate. Histopathology showed congestive

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hepatosplenomegaly, interstitial haemorrhages in kidney, and evidence of septic abortion with retained products of conception in the uterus.

The cause of death was opined as complications of copper sulfate poisoning.

Discussion

Copper sulfate is an irritant metallic poison. Humans can receive toxic exposure to copper sulfate through ingestion, by eye or skin contact, as well as by inhaling powders and dusts.[4] Skin contact may result in itching or eczema, while eye contact can cause conjunctivitis, inflammation of the eyelid, ulceration, and clouding of the cornea. Upon ingestion, because of its irritating effect on the gastrointestinal tract, vomiting is automatically triggered and poison may be forced out.[5]

However, if it is retained in the stomach, the symptoms can be severe. After 1–12 grams of copper sulfate are swallowed, there may be metallic taste in the mouth, burning pain in the chest, nausea, diarrhoea, vomiting, headache and discontinued urination. These symptoms may appear within 10 minutes to 1 hour after ingestion.[1,6] Later, hepatosplenic failure may ensue which manifests as yellowing of the skin and sclera.[5] In severe cases, injury to the brain and kidneys may also occur. Major systemic symptoms include delirium, stupor, coma, convulsions, hypotension, shock, respiratory failure, pallor and jaundice. In complicated cases, methemoglobinaemia, rhabdomyolysis, hepatotoxicity, intravascular haemolysis and renal failure have been reported.[6-9]

Copper inhibits the function of glucose-6-phosphate dehydrogenase in the red blood cells, an enzyme necessary for protection of red blood cells against the haemolytic effects of oxidizing substances. In addition, copper may interact with oxygen species (e.g., superoxide anions and hydrogen peroxide) and catalyze the production of reactive toxic hydroxyl radicals. Lethal dose is about 10-20 g.[6]

Copper sulfate being a corrosive substance, results in caustic burns of the oesophagus, and superficial and deep ulcers in the stomach and the small intestine. Changes of acute gastritis, haemorrhages in the intestinal mucosa, necrosis of the intestinal mucosa and perforation have been reported.[10,11]

Treatment of copper sulfate poisoning is supportive, including blood products, intravascular fluids and vasopressors. Gastric lavage and activated charcoal are not recommended because copper sulfate is a corrosive agent and may cause mucosal damage and perforation. The treatment of choice is chelation with dimercaprol and penicillamine. Haemodialysis is inefficient in removing copper from the body. Dialysis is recommended only in cases of acute renal failure.[1,6,12]

The incidence of copper sulfate poisoning may vary depending on its local use and availability. Its incidence is reported to be 34% and 65% of the total poisoning cases in two studies from Agra and New Delhi.[13,14] In another autopsy series, copper sulfate ingestion was responsible for 22% of poisoning deaths from 1972 to 1977. However, it declined to 3.85 and 3.33% between 1977-1982 and 1982-1987 respectively.[15,16]

Copper sulfate has a peculiar blue colour because of which it can be easily identified; thus, incidence of homicides as well as accidental ingestion in adults is rare.[8] On the other hand, accidental poisoning is common in children who are easily fascinated by its appearance.[17] Voluntary consumption of copper sulfate is rare, but reported.[6,18,19] Suicidal episodes are uncommon owing to painful clinical features developing before death. A person may desist from consuming copper sulfate for suicide due to fear of undergoing painful symptoms before death when other painless modes of death are available. On the other hand, low cost and easy availability of copper sulfate may motivate a potential suicidee for its use.

In the present case, the victim belonged to an agricultural family who had easy access to copper sulfate owing to its use as herbicide and fungicide. During a period of stress, the victim consumed it with the intention of commiting suicide. Although she was rushed to hospital, death ensued in spite of medical treatment. Congestive hepatosplenomegaly with jaundice, and interstitial haemorrages in kidneys suggest the possible mode of death to be hepatorenal failure. Septic abortion followed the irritant effect of poison on the uterus. Diagnosis was confirmed after chemical analysis of viscera.

REFERENCES

1. Sharma A. Acute copper sulphate poisoning: A case report. Indian J Forensic Med Toxicol 2010; 4(2): 4-5.
2. Copper (II) sulphate. Available online at: http://en.wikipedia.org/wiki/Copper(II)_sulfate.

3. Holtzmann NA, Haslam RH. Elevation of serum copper following copper sulfate as an emetic. Pediatrics 1968; 42 (1): 189-193.
4. Pillay VV. Modern Medical Toxicology. 2nd edn, 2001. New Delhi: Jaypee Medical Publishers. p74.
5. Nelson LS. Copper. In: Goldfrank LR, Flomenbaum NE, Lewin NA (editors). Goldfrank's Toxicologic Emergencies. 7th edn, 2002. New York: McGraw-Hill. p1262-1271.
6. Saravu K, Jose J, Bhat M, Jimmy B, Shastry B. Acute ingestion of copper sulphate: A review on its clinical manifestations and management. Indian J Crit Care Med 2007; 11(2): 74-80.
7. Jantsch W, Kulig K, Rumack BH. Massive copper sulfate ingestion resulting in hepatotoxicity. Clin Toxicol 1985; 22: 585-588.
8. Hassa S, Shaikh MU, Ali N, Riaz M. Copper sulphate toxicity in a young male complicated by methemoglobinemia, rhabdomyolysis and renal failure. J Coll Phys Surg Pakistan 2010; 20(7): 490-491.
9. Mortazavi F, Javid AJ. Acute renal failure due to copper sulphate poisoning; a case report. Iran J Paediatr 2009; 19(1): 75-78.
10. Deodhar LP, Deshpande CK. Acute copper sulphate poisoning. J Postgrad Med 1968; 14: 38-41.
11. Chugh KS, Sharma BK, Singhal PC. Acute renal failure following copper sulphate intoxication. Postgrad Med J 1977; 53: 18-23.
12. Rao RB, Hoffman RS. Caustics and batteries. In: Goldfrank LR, Flomenbaum NE, Lewin NA (editors). Goldfrank's Toxicologic Emergencies. 7th edn, 2002. New York: McGraw-Hill. p1326.
13. Chuttani HK, Gupta PS, Gulati S, Gupta DN. Acute copper sulphate poisoning. Amer J Med 1965; 39(5): 849-854.
14. Wahal PK, Lahiri B, Mathur KS, Kehar U, Wahi PN. Acute copper sulphate poisoning. J Assoc Physicians India 1963; 11: 93-103.
15. Singh D, Jit I, Tyagi S. Changing trends in acute poisoning in Chandigarh zone: A 25 year autopsy experience from a tertiary care hospital in northern India. Am J Forensic Med Pathol 1999; 20: 203-210
16. Singh D, Dewan I, Pandey AN, Tyagi S. Spectrum of unnatural fatalities in the Chandigarh zone of north-west India - a 25 year autopsy study from a tertiary care hospital. J Clin Forensic Med 2003; 10: 145-152.
17. Sharma NL, Singh RN, Natu NK. Accidental poisoning in infancy and childhood. J Indian Med Assoc 1967; 48: 20-25.
18. Ahasan HA, Chowdhury MA, Azhar MA, Rafiqueddin AK. Copper sulphate poisoning. Trop Doctor 1994; 24(2): 52-53.
19. Franchitto N. Gandia-Mailly P, Georges B, et al. Acute copper sulphate poisoning: A case report and literature review. Resuscitation, 2008; 78 (1): 92-96.