

CASE REPORT

ACUTE COPPER SULFATE POISONING: A CASE REPORT

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Abstract:

Copper Sulfate known as a powerful oxidizing agent and it can lead to widespread cellular damage, depending upon the dose ingested. The systemic effects of poisoning are seen primarily on red blood cells, gastrointestinal system, kidneys and cardiovascular system. The ingestion of poison can be lethal in severe cases. We reported the case of a 16-year-old girl who presented to the emergency department after ingestion of an unknown amount of copper sulfate 3 days earlier. On admission to the hospital, she had upper abdominal pain, vomiting and red colored urine with a normal serum copper level. She underwent symptomatic treatment and was monitored for 3 days. The outcome was favorable, and she had no signs and symptoms of organ failure.

Keywords: Copper sulfate, Poisoning, Intravascular haemolysis, Penicillamine

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Introduction:

Copper Sulfate, CuSO_4 , mainly used as a fungicide, bactericide which is an inorganic compound, a potent oxidant. It is also used to kill animals like snails for agriculture.¹ Although copper (Cu) is an essential mineral, it is very harmful in excess.²

Copper (II) sulfate is a large, bright blue crystal containing five water molecules ($\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$) and is also named blue vitriol or blue stone which is a salt containing copper²⁺ as the metal ion, created by treating cupric oxide with sulfuric acid. Copper sulfate was used in burn wound debridement until cases of systemic copper poisoning were reported³. Copper damages the cell membranes of the tissue by making them swollen and causes cell death.^[4] It causes haemolysis by affecting red blood cells; causes rhabdomyolysis by damaging myocytes and acute hepatitis by destroying hepatocytes. These are tissues

commonly affected.⁵ After absorption in plasma copper occur in plasma as ceruloplasmin excreted largely in faeces. If it exceeds its biological half life which is 13-33 days, it gets deposited mainly in the liver⁶. Copper Sulfate Poisoning (CSP) is rare, but it has significant entity because of its higher risk of mortality even with smaller doses of ingestion. Features of toxicity can manifest even with a dosage of 1 g and dose of 10-20 g could even be lethal.⁷

Case report:

A young 16 year old female student admitted to PMCH medicine department with history suicidal attempt by taking copper sulfate. Stomach wash was given on the day of ingestion in another hospital. Although she was alert, but presented more than 72 hours after ingestion, with yellowish discoloration of sclera, mucous membrane along with vomiting and epigastric pain.

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On arrival in hospital she was conscious, oriented and stable vital parameters. Her vital signs included a pulse rate of 80/min, blood pressure of 110/70 mmHg, SpO₂ of 97%. Jaundice was present on general examination and there was also epigastric tenderness. Although tender, there were no signs of peritonitis on abdominal examination. She had normal neurological function. Her past medical history includes GTCE, reflex epilepsy. For which she has been taking Levetiracetam. Patient developed red-colored urine (haemoglobinuria) (Figure:1).



Figure 1: Red color urine on the day of admission (3rd day of ingestion)

Routine investigations were done. Arterial blood gas (ABG) on admission revealed metabolic alkalosis with a pH of 7.51, PaO₂108, PaCO₂ 29.9, HCO₃⁻25 mmol/L. Haemoglobin reduced to 5.60 g/dL, MCV 88 fl; for which 2 units of Packed Red Blood Cell (PRBC) were given on the next day. CPK was 438 U/L and Bilirubin was also raised to 6.70 mg/dL. LDH was high as 2001 U/L. S. Ammonia, B. Urea, S. lipase, S. creatinine, S. Electrolytes, Prothrombin time, ALP, ALT were normal. Serum copper was 82.80 microgram/dL. Other haematological & biochemical investigations were normal. ECG and Echocardiography revealed no abnormality.

The treatment started with maintaining hydration of the patient with intravenous saline and antiemetic drugs. Oral loading dose of D-penicillamine 4 tablet stat orally (1 gm) then maintenance dose (250gm, thrice daily) was given as a copper chelator. Methylene blue

2 TSF, thrice daily was prescribed to the patient. Intravenous infusion of omeprazole was started suspecting gastric erosions. LDH and liver function tests were done on every alternate day to monitor the patients. The patient gradually improved with the given treatment and yellow color urine on 4th day of admission (Figure:2). Just the day before discharge all parameters were in normal range and those which previously abnormal ones are as follows: Haemoglobin 9.80 g/dL, MCV 85.8, Bilirubin 0.90 mg/dL; CPK 140 U/L, LDH 509 U/L.



Figure :2: yellow color urine on 4th day of admission

Discussion:

Poisoning by copper sulfate is a rare but often fatal, mainly related to suicide attempts. The route of administration of this substance is usually oral. Our case represents a rare and novel poisoning and it is the first case reported in Popular medical college hospital, Dhaka in which digestive system discomfort and features of haemolysis were the only sign and symptoms presented, contrary to our expectations.

There are few case reports where patient with copper sulfate poisoning presented with multi-system involvement. Such as hepatic and gastrointestinal effects, acute renal failure, cardiovascular events, methaemoglobinaemia, rhabdomyolysis.⁸

A case report was published in March 2009 a case of accidental copper Sulfate poisoning developed renal failure, intravascular haemolysis where the patient was treated with dimercaprol, penicillamine and peritoneal dialysis⁹.

Copper plays a vital role in protecting cells against oxidative stress as it acts as a cofactor for oxidative enzymes including peroxidase, catalase, glucose-6 phosphate dehydrogenase, glutathione reductase and cytochrome oxidase.¹⁰ In the serum, copper exists in two forms; bound to ceruloplasmin (93%) and bound to albumin (7%).¹¹ Liver is the main organ that stores copper. The main mechanism of excretion is via fecal route, whereas only about 4% is excreted via urine.^[12] Features of toxicity occur when there is ingestion of more than 1g of copper sulfate.¹³ It is associated with higher mortality when the dose is more than 10g and it is considered to be lethal.¹⁴

There are some mechanisms by which copper sulfate causes damage at cellular level. In this article we are discussing only the mechanism by which rhabdomyolysis and hemolysis occurs after acute intoxication. Copper sulfate-induced rhabdomyolysis occurs by Na⁺/K⁺-ATPase pump inhibition and subsequent increase in myocyte permeability in the skeletal muscle.¹⁵

Copper sulfate degrades red cell membranes and denatures their hemoglobin content thus it can lead to hemolysis by accumulating in red blood cells.¹⁶ It may disrupt the activity of various cellular enzymes such as erythrocyte glucose 6-phosphate dehydrogenase (G6PD)¹⁷, glutathione reductase, and catalase¹⁸

Conclusion:

Though acute copper poisoning is very rare, it has a high mortality rate. The management is mainly supportive.

Conflict of Interest:

The authors stated that there is no conflict of interest in this study.

Funding:

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Consent for publication:

Informed written consent was taken from the parents of the patient to publish details relevant to the disease and management.

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Authors' contributions:

All authors were involved in the management of the patient.

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