

Fatal Ingestion of Copper Sulfate

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Objective

Copper sulfate is a soluble copper salt with fungicide, algicide, bactericide, herbicide, molluscicide, and insecticide properties.

It is a strong irritant to skin and mucous membranes, and can be corrosive. Oral application can lead to gastrointestinal as well as systemic effects.

Despite its known toxic properties, copper sulfate is widely marketed as a chemical agent for private use, i.e. in algicides for swimming pools, in antiparasitic agents for aquarium fishes, as component of experimental kits for children (home-grown crystals), and other applications.

In the past, it was medically used as an emetic and to treat malaria, but is now considered to be too toxic for these indications.

We report on a case of fatal ingestion of a large amount of copper sulfate.

Case series

Between June 1996 and June 2016, a total of 83 exposures to copper sulfate were reported to the Poisons Information Centre Erfurt; hereof 45 (54.2 %) exposures to children and 37 (44.6 %) exposures to adults, in one case more than one age group was affected. Of all exposures, 60 cases (72.3 %) were accidental ingestions and 10 cases (12 %) were suicide attempts, whereas misuse was reported in 6 cases (7.2 %). Most cases (84 %) resulted in no (32 cases) or minor (38 cases) gastrointestinal symptoms, in 8 cases (9.6 %) moderate to severe symptoms occurred including one fatality, and in 5 cases symptoms were unknown.

Most inquiries to the Poisons Information Centre Erfurt were concerning accidental ingestions of copper sulfate mainly by children, yet misuse is regularly observed as well: Only recently, a young woman (22 years of age) ingested one teaspoon of copper sulfate crystals in an attempt to lose weight - which she stated to have read on the internet as a "good way to slim down".

Case report

A 50-year-old male ingested 150 g (> 2 g/kg) of copper sulfate in a suicide attempt. He only reported to hospital after 7 days (!) and at this point already showed severe symptoms like prolonged nausea and vomiting (not haemorrhagic), metabolic acidosis, acute renal failure (GFR 20), elevated transaminase levels, and hyperkalaemia.

The following day, gastroscopy was performed and showed distinct necrosis of gastric mucosa without perforation or bleeding. Serum copper levels were 15 $\mu\text{mol/L}$ (day 7) and 12.3 $\mu\text{mol/L}$ (day 8), respectively [normal range 11.8 to 23.6 $\mu\text{mol/L}$]. In the course of the next 3 days, the patient developed haemolysis, organ failure, and respiratory insufficiency.

Treatment with D-penicillamine was attempted, and the patient's condition temporarily improved. However, he died on the 21st day after ingestion due to multiple organ failure.

Toxicity of copper sulfate

Copper ions, i.e. from copper salts such as copper sulfate, can be absorbed by the human body and are then rapidly bound to carriers and distributed to the liver and other tissues. Ingestion of small amounts mainly causes gastrointestinal symptoms (vomiting, gastrointestinal bleeding, diarrhoea etc.), whereas large doses can result in haemolysis, coagulopathy, methaemoglobinaemia, metabolic acidosis, acute organ failure (liver, kidney), hypotension, tachycardia, seizures, coma, and shock, and can ultimately lead to multiple organ failure and death.

The acute lethal dose of ingested copper sulfate is suggested to be 0.15 to 0.3 mg/kg. (1)



Fig. 1 - Copper (II) sulfate (anhydrous)

Management of copper sulfate poisoning

In acute overdose, gastrointestinal decontamination can be considered within one to two hours after ingestion, but is usually of minor interest due to rapid onset of emesis. In patients without gastrointestinal symptoms, nasogastric aspiration could be useful. Activated charcoal does not effectively bind to copper ions and is therefore not efficient. Symptomatic patients should be treated with antiemetics and receive supportive care, including fluid and electrolyte correction. Additionally, gastrointestinal endoscopy is recommended in order to evaluate the corrosive effects of copper sulfate to the mucosal surface. (1)

Chelation Therapy

Normalization of vital signs is prior to the consideration of chelation therapy. In cases of severe poisoning, chelation therapy should be initiated. However, the efficacy of D-penicillamine and British anti-Lewisite (BAL), respectively, following acute copper salt poisoning is limited. Despite early and appropriate administration, multiple organ failure and death still occur.

Use of D-penicillamine, the antidote primarily used in Germany, is not without side effects and can itself result in organ toxicity. Moreover, it is contraindicated in patients with renal insufficiency, and hence administration may not be possible in severely poisoned patients. (1)

Conclusion

Copper is an essential trace dietary mineral due to its role in several enzymatic processes. Yet, ingestion of large amounts of copper salts (10 to 100 grams) can result in severe symptoms and even death. Excess copper in the blood is primarily taken up and stored by the liver, which explains the elevated transaminase levels and severe symptoms despite "normal" serum copper levels in the aforementioned case. Moreover, as it can only remove free copper from the blood, delayed administration of D-penicillamine could not prevent the fatal outcome. (2)

References

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- (2) Naha K, Saravu K, Shastry BA. Blue vitriol poisoning: a 10-year experience in a tertiary care hospital. Clin Toxicol (Phila). 2012 Mar; 50(3):197-201.