Case Report

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Toxin Tides: Navigating the Waves of Copper Sulphate Poisoning

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Abstract

Copper sulfate, a prevalent fungicide utilized in agriculture, poses substantial hazards due to its toxicity. This manuscript explores a case study of a 56-year-old male who presented with symptoms of copper sulfate poisoning, including respiratory distress, metabolic acidosis, and cardiovascular instability. Despite immediate medical intervention, including the patient succumbed to ventricular fibrillation. The discussion delineates the diverse manifestations and management strategies for copper sulfate poisoning, emphasizing the importance of prompt recognition and treatment. With fatal outcomes attributed to conditions like methemoglobinemia and organ failure, early initiation of supportive care and chelation therapy are paramount. While uncommon in emergency settings, this case underscores the significance of vigilance in recognizing and managing copper sulfate toxicity to mitigate adverse outcomes. The study contributes to the existing literature by elucidating clinical presentations, treatment modalities, and prognostic factors associated with copper sulfate poisoning.

Keywords: Copper sulfate poisoning; Toxicology; Methemoglobinemia.

INTRODUCTION

Copper sulfate, commonly referred to as blue vitriol, serves as a widely used fungicide for the control of bacterial and fungal diseases in crops such as vegetables, fruits, and grains. Available in both powder and liquid forms, it stands as an economical and easily accessible solution for agricultural needs. However, despite its effectiveness in safeguarding crops, copper Sulfate poses significant risks due to its toxicity.[1]

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Its widespread availability and low cost unfortunately make it easy for individuals seeking to cause harm to themselves. Even minute quantities of copper sulfate can lead to adverse effects, emphasizing the importance of proper handling and storage to prevent accidental exposure or ingestion.[2] The majority of reported cases involving copper sulfate poisoning typically arise from deliberate self-harm incidents.[3]

Copper sulfate ingestion is a rare form of poisoning whether it is accidental or intentional. It is essential for emergency physicians to identify the symptoms, complications and also early chelation.

CASE

A 56-years-old male with alleged history of consumption of copper Sulfate nearly 250gm five hours prior to arrival to Emergency Department with decreased responsiveness since past hour. On primary survey his airway was patent, in breathing he was tachypneic with respiratory rate of 25cpm and hypoxic with saturation of 85% on room air,

thus he was connected to a non-rebreather mask with oxygen 15 litres and saturation improved to 95%. On auscultation bilateral basal crepitations were also present. His Arterial Blood Gas test showed elevated anion gap metabolic acidosis with compensation. In circulation he was tachycardic with a feeble pulse of 132 beats per min and hypotensive with a blood pressure of 70/40 mm Hg. Despite fluid resuscitation with one litre of normal saline the blood pressure didn't pick up. Thus he was started on Noradrenaline infusion at rate of 0.2 mcg/kg/min. His ECG showed sinus tachycardia with tall tented T wave in V3 to V6 with a flattened P wave in limb leads (fig. 1). A provisional diagnosis of hyperkalemia was made and Inj. Calcium glaciate 10% 10ml iv over 10min, Inj regular Insulin 10 U in 25%D and 10mg of nebulisation Salbutamol was administered and repeat ECG was taken after 30min which showed settling of T waves and appearance of P waves. On disability he had a GCS score of E2 V3 M5. Bilateral pupils were 4mm and reacting to light, random blood sugar was 140 mg/dl. On exposure there was dry mucosa. Investigations done in emergency department are shown in Table 1. On secondary survey he had blue discoloration of teeth, tongue and gums. He was a known case of hypertension on medication Tab. Telmisartan 20 mg, last meat at 5 am.

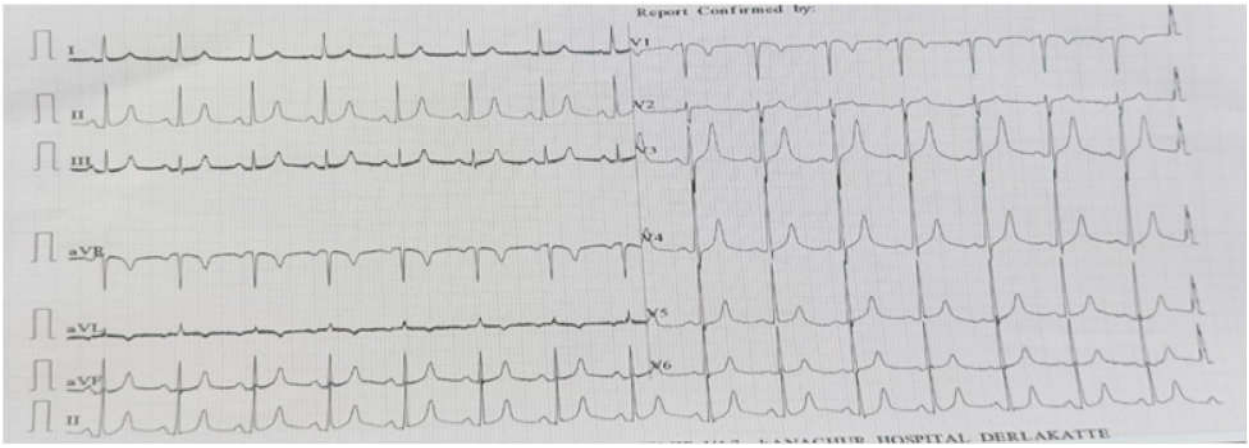

Fig. 1: ECG showing sinus rhythm with tall T waves in precordial leads

Table 1: Lab investigations sent from ED

Hb	15.6 gm%
WBC	27160 cells /cumn
Platelets	4.7 lakh /cumn
Neutrophils	91 %
pH	7.54
Pco2	23.1 mm Hg
PaO2	178 mm Hg
SaO2	100%
HCO3	19.8 mmol/L
Lactate	4.6 mmol/L
Urea	44.80 mg/dl
Uric acid	6 mg/dl
Serum creatinine	3.1 mg/dl
Sodium	149 mEq /L
Potassium	6.30 mEq /L
Chloride	115 mEq /L
AST	420 U/L
ALT	160 U/L
Albumin	5.80 gm/dl
Globulin	4.80 gm/dl
Total protein	10.60 gm/dl
ALP	138 IU/L

He was given a stomach wash with potassium permanganate in our ED. 100ml of milk along with 10ml of milk of magnesia was also instilled in Ryles tube to prevent absorption of copper Sulfate. He was started on Tab Vitamin C 500mg and Tab Zinc 50mg. He was stated on fluid Ns at 100ml per hour NS. He was shifted to critical care for chelating and monitoring. He was planned for chelating. But patient developed ventricular fibrillation after 2 hours of arrival to hospital and was declared dead after failed resuscitation.

DISCUSSION

Copper sulfate can penetrate the skin, gastrointestinal tract, and lungs, leading to both systemic and local toxicity. Skin contact may cause itching or eczema, while exposure to the eyes can result in conjunctivitis, inflammation of the eyelids, ulceration, and clouding of the cornea. If ingested, vomiting is induced automatically, helping to expel the poison.[3]

The fatal dosage of copper sulfate ranges from 10 to 20 grams, but symptoms can manifest with

as little as 1 gram. Initial signs include a metallic taste in the mouth, chest pain, nausea, vomiting, headache, decreased urination, and diarrhea. Higher doses lead to severe systemic symptoms such as delirium, stupor, coma, convulsions, low blood pressure, shock, respiratory failure, pallor, and jaundice. Mortality is attributed to conditions like methemoglobinemia, rhabdomyolysis, liver damage, intravascular hemolysis, and kidney failure.[2] A retrospective analysis of 35 cases of copper sulfate poisoning in India between 2001 and 2010 revealed a mortality rate of 22.9%.[1]

The treatment for acute copper sulfate poisoning involves both supportive and specific measures. Supportive care is tailored to the patient's condition and may include oral administration of activated charcoal for decontamination. Gastric lavage and inducing vomiting are not recommended due to the corrosive nature of copper sulfate, which can increase the risk of perforation. Early gastroscopy within the first 24 hours is advised to assess the extent of corrosive damage.[4] Maintenance of intravascular volume is crucial. The preferred treatment involves chelation therapy using dimercaprol and penicillamine. Penicillamine is typically administered at a dosage of 1-1.5 grams per day in 2 to 4 divided doses, while dimercaprol is given at a dosage of 3-5 mg/kg/dose every four hours over two days. Dialysis is recommended only for cases of acute renal failure. Methylene blue is the recommended treatment for methemoglobinemia, with a dosage of 1-2 mg/kg, which may be repeated if cyanosis persists.[5]

CONCLUSION

Copper sulfate, despite its corrosive nature and high toxicity even in small doses, is not commonly encountered in emergency rooms as a suicidal agent. Its primary impact typically affects the renal system, with methemoglobinemia being a common occurrence among patients. While copper sulfate is not typically considered a dialyzable toxin, early initiation of hemodialysis may contribute to improved outcomes in cases of poisoning.

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