

A CASE REPORT ON ACUTE COPPER SULFATE POISONING IN TERTIARY CARE TEACHING HOSPITAL

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ABSTRACT

Copper sulfate is an inorganic compound with metallic taste and its crystals appears to be blue color, poisonous for human consumption. Poisoning cases of copper sulfate are very rare throughout the world with exceptional cases in India. A case report of 24 year old male patient presented to medicine ward with the acute clinical manifestations of acute copper poisoning. He received supportive treatment along with chelation therapy after 8 days of successful treatment he was discharged from hospital and we conclude that early treatment approaches can save life of the patient, which is further clearly explained in the text and the marketed preparations of copper sulfate should not be freely available in India.

KEYWORDS: Copper sulfate, Clinical Manifestations, Poisoning.

INTRODUCTION

Copper sulfate is an inorganic compound with metallic taste and its crystals appears to be blue color.^[1] Molecular formula of copper sulfate is $\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$.^[2] It is used in paint, leather industries as well as for various other purposes like as a pesticide in agricultural field.^[3] Copper sulfate is poisonous for human consumption.^[4] Copper is an essential trace element found in humans involved in various complex enzymatic reactions.^[5] Copper sulfate is highly acidic in nature; concentrated solution of copper sulfate has pH of 4, enteral administration of copper sulfate leads to erosion of gastro intestinal track mucosa due to its strong oxidizing properties.^[6] Acute poisoning of copper sulfate is studied in animal models aside of it corrosive nature, decreases or completely inhibit the activity in excess of various enzymes like glutathione reductase and glucose 6 phosphate dehydrogenase by binding to their sulfhydryl group as well as causes cell damage and cell death in most of the cases.^[7]

Accidental exposed cases of copper sulfate are reported throughout the world with rare poisonous cases, exceptionally in India intentional suicidal attempts are being reported and most of the patients present to emergency departments with history of orally ingested copper sulfate^[8]. We report a case of a young boy having alleged history of copper sulfate with intention to commit suicide.

CASE REPORT

A 24 year old male was presented to emergency department after 4 hours of with alleged history of consumption bluish crystalline substance, as history narrated by patient's attender he tried to commit suicide by consuming copper sulfate; he had multiple episodes of vomiting, hematemesis and several episodes of loose stools with melena. On examination he was hypotensive with systolic blood pressure as 90 mmHg and diastolic blood pressure as 50 mmHg, respiratory rate was 27 per minute, heart rate was 117 beats per min, bilateral air entry was positive with no added sounds, oxygen saturation was 85%. Patient underwent immediate gastric lavage, general condition of patient was sick he was in distress because of burning type of abdominal pain, he was afebrile, abdomen was soft with diffuse tenderness and his glass glow coma scale was found to be 9. Patient was found to be anemic as per CBC report which was due to hemolysis of RBCs it was confirmed by peripheral blood film he was suffering from hemolytic anemia, his WBC count was increased to 17100/microL. Neutrophils were 90% lymphocytes were 07% monocytes were 02% and basophils were 0%, random blood sugar was 139 mg/dL, liver function test were elevated SGOT was found 72 IU/L and SGPT was 83 IU/L and ALP was 250 IU/L. Chest X-rays were normal. ECG revealed decreased sinus rhythm. Blood copper levels were quite high they were $>50 \mu\text{mol/L}$, blood pH was Patient was under hypovolemic shock for which he received intravenous normal saline and norepinephrine infusion, dose of norepinephrine was tapered according to blood pressure, aside of symptomatic treatment he received chelation therapy of calcium disodium EDTA with dose of 1 gram after every 12 hour in 250 ml of 5% solution of dextrose in a time period of 1 hour and intra venous L-aspartate and L-ornithin with 5mg dose in 5% of dextrose thrice a day, ondansetron 4mg was given thrice a day, continuous infusion of pantoprazole at dose of 80 mg was given at rate of 8ml/hour. He received 2 units of packed red blood cells, piperacillin along with tazobactam was given as a prophylactic antibiotic. On day 40 he conditions were improved he was shifted to medicine ward, endoscopy was also done it revealed ulceration of pyloric antrum and rest other endoscopic

findings were normal. Patient was sent for psychiatric consultation; he was discharged after 8 days and was followed up in outpatient department and now patient is living normal life.

Highlights of Treatment received by patient

- Inj. Norepinephrine @ 6ml/hr
- Inj piperacillin tazobactam 4.5gm I.V TDS
- Inj Pantoprazole 80mg I.V.Stat than 80 mg in 500ml Normal Saline @20 drops/min
- Inj Vitamin K 10 mg I.V. OD
- Inj. Tranexamic acid 500mg I.V TDS
- Syp. Sucralfate 20ml P.O TDS
- Inj. Ondansetron 8mg I.V. TDS
- IV Fluids 2 units PRBC, Normal Saline QID, Ringer Lactate Solution BD
- Inj. L ornithine and L Aspartate 8mg OD in 5% solution of dextrose.

Table 1 Showing serum copper concentration and liver enzymes.

Lab investigation	Day1	Day2	Day3
S. copper	>50 $\mu\text{mol/L}$	34 $\mu\text{mol/L}$	21 $\mu\text{mol/L}$
SGOT	72 IU/L	55 IU/L	34 IU/L
SGPT	83 IU/L	60 IU/L	290 IU/L
ALP	250 IU/L	200 IU/L	150 IU/L

DISCUSSION

Copper sulfate poisoning management includes symptomatic approach along with its chelation therapy, usual lethal dose of copper sulfate is >1 gm, if copper sulfate is ingested between 10-20gm leads to severe poisoning.^[9] Clinical manifestations of acute copper sulfate poisoning include erosion of GI mucosa followed by hemolysis of red blood cells, elevated levels of methemoglobin, acute hepatitis, interstitial nephritis later on muscle damage and in severe case acute renal failure.^[10]

Mechanisms involving GI manifestation are irritation of the membranes and necrolysis of mucosa, leading to ulceration.^[11] On absorption of copper it leads to generation of free radicals, superoxide molecules which causes oxidation of lipid membranes of cells leading to rupturing of RBCs and necrolysis of hepatocytes and muscle damage.^[12] In our patient hemolysis occurred after 24 hours of ingestion and it was managed with transfusion of PRBCs. Due to vomiting and severe dehydration patient went into hypovolemic shock. Acute renal failure and rhabdomyolysis are common in case of severe poisoning cases and these were not observed in this case due to decreased absorption of copper sulfate. Initially

treatment was started with vitals stabilization, gastric lavage, chelation therapy with calcium disodium EDTA and maintenance of fluid and electrolyte balance.

CONCLUSION

Early aggressive treatment can prevent the further organ involvement, which includes supportive measures gastric lavage along with chelation therapy. As per societal aspects are concern marketed preparations of copper sulfate should not be freely available so they are not consumed for suicidal purposes in Indian society.

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