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COPPER SULPHATE (NEELA TOTA) POISONING- AN UNCOMMON MODE OF POISONING : A CASE REPORT.

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ABSTRACT Ingestion of copper sulphate is an uncommon mode of poisoning in the Indian subcontinent.Cases being mainly suicidal in nature. The clinical course of the copper sulphate intoxicated patient is often complex involving intravascular hemolysis , jaundice, and renal failure. The treatment is mainly supportive. In severe cases methemoglonemia needs treatment . Mortality is quite high in severe cases. A comprehensive review of the clinical presentation and management of copper sulphate poisoning is done.

KEYWORDS :

INTRODUCTION

Copper sulphate also known as “Blue Vitriol” and “ Neela Tota” in local language is used in agriculture as pesticide , leather industry, precipitator in heavy metal poisoning. Total body content of copper is 150mg. Average half life of copper in healthy individual is 26 days. Copper toxicity causes erosive gastropathy, intravascular haemolysis, methaemoglobinaemia, hepatitis, acute kidney injury and rhabdomyolysis.

Copper sulphate being a corrosive acid , results in caustic burns of the oesophagus, superficial and deep ulcers in the stomach and the small intestine.

CASE PRESENTATION

A 21 year old male was admitted in GMCH Aurangabad following ingestion of approximately 10 grams of copper sulphate 5 hours before admission in a alleged suicidal attempt. On admission he complained of epigastric and right hypochondriac pain and vomiting. There was no bleeding manifestation. His urine output was reduced and dark in colour. He did not complain of shortness of breath at rest or exertion.

On examination he was febrile ,icteric and cyanosed(chocolate cyanosis). Patient was conscious, rational, oriented. Pulse rate was 106/min and blood pressure was 124/80 mmHg. Patient had induced fasciculations in muscles. Pupils were normal size reacting to light.Right hypochondrial region was tender with firm and tender liver that was palpable 2 cm below costal margin. Rest examination had no abnormal finding.

There was significant drop in haemoglobon level from 15.5 to 11.3 mg/dl within 6 days which would have been due to copper sulphate induced hemolysis.Methhemoglobinemia was confirmed biochemically as levels were 1 %. The peripheral saturation was 89%(with pulse oximetry) when the central saturation was 67% (on ABG). Patient was not treated for methhemoglobinemia. Hepatitis was evident from raised enzyme levels which had declining trend.

Chelation therapy was started with penicillamine at a dose of 500 mg 6 hourly. The patient made a good clinical recovery.Icterus of the patient was declining over the week.Patient was discharged 7 days after admission. He had no further complaints and his liver function tests returned to normal levels after 28 days when patient was assessed again .

INVESTIGATION CHART

	Day 1	Day 2	Day 3	Day 5	Day 7	Day 28
Hemoglobin[mg/dl]	15.5			11.3	11.3	12.4
PCV	45			33	33	42
TLC	13500			9700	4900	5600
Platelet[lacs/mm3]	2.5			1.79	2.10	2.3
Sr. Bilirubin[mg/dl]	8.1	7.8	5.6	4.2	3.0	0.9
SGOT[IU/dl]	53	78	97	46	41	24
SGPT[IU/dl]	60	92	115	87	53	28
Sr. Alkaline Phosphatase[IU/dl]	116	120	132	122	47	42
Sr. Protein[gm%]	7.8	6.3		6.0	6.6	7
Sr Sodium[meq/l]	136	138		138	139	136
Sr.Potassium[meq/l]	4.7	4.6		4.2	3.9	3.8
Sr.Creatinine[mg%]	1.0	1.0		0.8	0.6	0.6
Sr. Urea[mg%]	45	27		24	22	26
Methhemoglobin	1 %					

DISCUSSION

Ingestion of more than 1 g of copper sulphate results in manifestation of symptoms of toxicity [2]. Mortality in cases of severe poisoning is high and the lethal dose of ingested copper sulphate is between 10-20 g [3].

The clinical manifestations of copper sulphate poisoning include; erosive gastropathy, intravascular haemolysis, methaemo globinaemia, hepatitis, acute kidney injury and rhabdomyolysis. Arrhythmias and seizures are also reported probably secondary to other organ system involvement [1,4].Common gastrointestinal manifestations of copper poisoning are predominantly due to corrosive injury. Haematemesis and melaena are observed with severe overdose. Liver gets damaged early due to copper deposition in liver after absorption from the portal circulation. Acute liver failure following tissue necrosis can occur due to direct copper toxicity [2,5].

Two major haematological manifestations of copper sulphate poisoning are intravascular haemolysis and methaemoglobinaemia [1]. Intravascular haemolysis can start as early as within the first 24 hours since ingestion and is due to the direct oxidative damage to erythrocyte membranes. The Cu2+ ion oxidizes the Fe2+ ion in haemoglobin to Fe3+ resulting in its conversion to methaemoglobin causing cyanosis[2].

AKI happens in 40-60% of cases [6-9] and mechanisms of kidney damage include; pre-renal failure due to dehydration (vomiting, diarrhea, reduced fluid intake), haemoglobinuria, sepsis, rhabdomyolysis(causing fasciculations), direct copper toxicity on proximal tubules and secondary effects of multi organ dysfunction. The recovery of renal function following copper sulphate ingestion is observed to be slow and incomplete.

The management of copper sulphate poisoning centre's on four key principles-

- Reducing absorption
- Close observation for complications
- Supportive therapy
- Chelation therapy

After ingestion, the contact damage to mucosa can be minimized by drinking large quantities of milk and water [1]. Dilution reduces the direct mucosal injury. Emesis must be avoided due to the corrosive activity of compound. Some authors recommend the use of activated charcoal to reduce absorption, but it is of unproven benefit [1].

All complications mentioned above must be monitored for from the first 24 hours onwards (daily full blood counts, serum electrolytes, liver and renal function tests). If the patient had vomited, aspiration pneumonia or a chemical pneumonitis should be anticipated and a baseline chest roentgenogram advised. Evidence for hemolysis must be looked for with daily blood pictures and reticulocyte counts.

Anaemia from haemolysis or bleeding must be corrected with transfusion with red cell concentrates. Methaemoglobinaemia is treated with methylene blue (IV 1-2 mg/kg/dose and repeated if cyanosis persists beyond one hour)[11]. Alternatives to methylene blue when contraindicated would be hyperbaric oxygen and ascorbic acid[11].

Renal failure must be looked by measuring serum creatinine and urine output. Though dialysis is ineffective in clearing copper from the body, it will be an essential requirement to sustain life in event of AKI [1]. Peritoneal dialysis is an alternative when hemodialysis is contraindicated.

Chelation therapy aims at removing ingested copper from the body. Penicillamine is a commonly used chelating agent at a dose of 1-1.5 g/d in 2-4 divided doses [1].Intramuscular administration of dimercaprol or BAL 3-5 mg/kg/dose with four hourly administration in first two days and tailed off over a total of 7-11 days) is recommended when penicillamine is contraindicated[12]. Edetate Calcium disodium is another option when the use of penicillamine is deemed unsafe [13].Despite being rare, intoxication with copper sulphate can be fatal. Therefore it is important to concentrate on prevention of copper sulphate ingestion by measures such as

- Stopping over the counter sale of copper sulphate
- Restriction of purchase, distribution and sale to authorised agents only

CONCLUSION

Ingestion of copper sulphate is a rare form of poisoning. However, given the complications and high mortality rates, it's essential that clinicians are familiar with the management of such patients. The effects of poisoning can have deleterious effects on the upper digestive tract, kidneys, liver and blood (intravascular haemolysis, methaemoglobinaemia). We further recommend that restricting the availability of the pulverized powdered form of the compound in the open market might be an effective measure in preventing deliberate self harm by ingestion of copper sulphate.

CONSENT

Written informed consent was obtained from the patients for publication of this case report. Copies of the written consent are available for review by the Editor-in-Chief of this journal.

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