ORIGINAL ARTICLE

Burden and outcome of acute copper sulphate poisoning in a teaching hospital

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Abstract

Copper sulphate poisoning is the most common poisoning next to organophosphorus poisoning with significant mortality. Gastrointestinal, liver, renal and haematological toxicities are common. A prospective observational study was conducted in department of medicine, KMCH, Khulna during the period of September, 2005 to March, 2007 to observe the presentation and outcome of copper sulphate poisoning. Forty patients with acute copper sulphate poisoning of different age group of both sexes were studied. Patients were evaluated through detailed history, clinical features, laboratory investigations and they were monitored closely at regular interval during hospital stay. Copper sulphate poisoning was found mostly in rural people (85%) and maximum (62.5%) patient were in the young age group of 16-25 yr. All patients had taken it as a suicidal attempt. Almost all patients presented with gastrointestinal symptoms-nausea (100%), vomiting (100%), and abdominal pain (90%). Subsequently hepatic toxicity was manifested by jaundice (32%), yellowish discoloration of urine (35%) which developed usually on the 2nd or 3rd day. Renal toxicities were manifested by oliguria (25%), haematuria (32.5%), albuminuria (30%) and renal dysfunction (30%). 35% of the patient were found to have anemia with Hb below 60%. Eight patients (20%) became unconscious at the terminal stage and died.

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Introduction

Copper is an essential trace element in human.It is vital for the function of certain enzymes, such as cytochrome c oxidase and superoxide desmutase which protect against oxydative stress. In Bangladesh, epidemiological study Farmers and fishermen in southern Bangladesh use copper sulphate to treat tinea pedis and other skin disease.6

Copper sulphate causes toxicity in human body mainly through oral route and usually by consumption of contaminated foodstuffs or beverages, including drinking water or deliberate ingestion of high quantities of copper salt or accidental entry into the body through the skin, eye contact, as well as inhalation.2,7,8 There is also a case report on intravenous copper sulphate poisoning as a suicidal attempt.9 It is used commonly as a suicidal agent in the Indian subcontinent.10,11 Accidental poisoning also occur mainly in pediatric age group. It is reported that the lowest dose of copper sulphate that has been toxic when ingested by human is 11 mg / kg.12 Deliberate ingestion of copper sulphate causes metallic taste, burning sensation in throat, nausea, vomiting, abdominal pain, diarrhoea, melaena, jaundice, oliguria, convulsion, rapid pulse, hypotension and shock.

Gastro intestial tract toxicity occurs due to local irritation, erosion, congestion of vessels in sub mucosa with ulceration. Hepatic copper accumulation causes hepatocellular damage. There may be jaundice and hepatomegaly, increase transaminase level and possibly prolongation of prothrombin time.3,13 Jaundice usually occur on the second or third day.9 Jaundice may be cholestatic, hemolytic, hepatocellular or mixed.14

Renal complication are observed frequently 3-4 days post poisoning and presenting features included dark reddish coloured urine and oliguria. Albuminuria, haematuria may be present. Blood urea and creatinine are increased. Haemolytic anaemia, a common complication of copper sulphate poisoning is caused either by direct cell membrane damage or indirectly as a result of the inactivation of enzyme (including glutathione reductase) which protect shown that suicidal trend such as copper sulphate poisoning is more prevalent in rural region and Copper sulphate poisoning is the most common poisoning next to organophosphorus poisoning with significant mortality.15 But there is scarcity of data regarding the toxicity of copper sulphate after oral ingestion in this area.

Materials and Methods

It was a prospective observational study conducted on patients with acute copper sulphate poisoning, admitted in department of medicine, KMCH, Khulna during the period from September, 2005 to March, 2007. A protocol was prepared for the study and approved by ethical committee.

Total 50 patients were admitted during study period of which 40 patients of different age group of both sex were included in the study. The criteria for selection of cases were- poisoning history, clinical findings and laboratory data of routine urine examination (albumin, RBC.), liver function test (SGPT and serum billirubin) and renal function test. Patients with lack of data regarding clinical and laboratory investigation were excluded. Some cases absconded and were excluded automatically.

Selected patients were evaluated subsequently through detailed history, clinical examination and laboratory investigations. All patients were treated conventionally. Patients were monitored closely at regular interval during hospital stay. The data was analyzed by SPSS and presented in tabulated form.

Results

Total 40 patients were included in this study; male and female were 17 and 23 respectively with age ranges from 13 to 55 years. Poisoning most commonly observed in the age group of 16-25 years (62.5%), less common in the old age group of 46-55 years (5%) (Table-1).

Table I

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Incidence of copper sulphate poisoning in different age group (N=40)

Age (year)	Number	Percentage
0-15	3	7.5
16-25	25	62.5
26-35	6	15.0
36-45	4	10.0
46-55	2	5.0
Total	40	100.0

Table II
Sex & community distribution

Sex	No. (%)	Urban	Rural
Male	17(47.5)	02	15
Female	23(57.5)	04	19
Total (%)	40 (100)	06(15)	34(85)

Copper sulphate poisoning was more common in the females (57.5%) than males (42.5%). 85% of the patient from rural area, 15% from urban area (table-II).

Table III
Clinical features of poisoning.

Clinical features	No. of patients	Percentage
Nausea	40	100.0
Vomiting	40	100.0
Abdominal pain	36	90.0
Diarrhoea	15	37.5
Hematemesis / melena	4	10.0
Jaundice	13	32.5
Pallor/Anemia	15	37.5
Urinary suppression/Oliguria	10	25.0
Altered colour of urine	14	35.0
Respiratory distress	10	25.0
Paralysis of limbs	2	5.0
Shock	8	20.0
Unconsciousness	8	20.0

Almost all patients presented with gastrointestinal symptoms like nausea (100%), vomiting (100%), and abdominal pain (90%). Other gastrointestinal symptoms that were common in early days of poisoning- diarrhoea (37.5%), hematemesis/ melena (10%) (Table-III).
Anemia (pallor) also developed on the subsequent day (37.5%). Anemia was a common feature with hemoglobin level below 60% in 35% of patients.

Table IV
Urine analysis

Urine anal sis		No. of patients	Percentage
Haematuria	Present	12	30.0
	Absent	28	60.0
Albuminuria	Present	13	32.5
	Absent	27	67.5

Table-IV shows haematuria and albuminuria in 30% and 32% of the patients respectively.

Table V

Liver & renal function tests of the patients (N-=40)

	Name of Exam		No. of patients	Percen-tage
Liver function test	S.Bilirubin	Increased	15	37.5
		Normal	25	62.5
	SGPT	Increased	16	40.0
		Normal	24	60.0
	Blood urea	Increased	13	32.5
Renal function test		Normal	27	67.5
	S. creatinine	Increased	12	30.0

Hepatic toxicity developed later (usually on 2nd or 3rd day) manifested as jaundice (32%), altered color of urine (35%) (Table-III). Liver function tests showed elevated serum bilirubin in 37.5% and raised SGPT level was in 40% of the patients (Table-V). Renal function test was done at 3rd day of copper sulphate ingestion. It reveals elevated blood urea and serum creatinine level in 32.5% and 30% of the patients respectively.
All the patients were treated conventionally. 90% patients were forced to vomit by artificial means (induced vomiting). 100% patient received gastric lavage by plain water or 1% potassium ferrocyanide.
Copper chelating agent we given either Penicillamine (72.5%) or Dimercaprol (27.5%). The choice of chelating agent was on the basis of severity of the disease and economic condition of the patients. Eight patients (20%) became unconscious at the terminal stage and ultimately died from copper sulphate poisoning and rest of the patients (80%) recovered.

Discussion
Acute copper sulphate poisoning is a common suicidal agent in the Indian subcontinents.11,12 We have studied 40 cases of acute copper sulphate poisoning patient. In this study almost all patients taken copper sulphate as a suicidal agent (100%) which is consistent with the data of Klein WJ et al.11 They reported that copper sulphate is commonly used as a suicidal agent in Indian subcontinents.
The amount of copper sulphate ingested by the victims ranges from 5 gm to 50 gm, although most of the copper sulphate toxicity develop with ingestion of at least 1 gm.13 In a review study of 123 cases, Ahasan et al observed an "unpredictable" outcome in those consuming less than 50 gm while 100 gm was "invariably fatal". In contrast Akintonwa et al15 claimed 10-20 gm of copper sulphate to be a "definitely fatal" dose.
This study showed that copper sulphate poisoning was most common in females (57.5%) than males (42.5%). Age of the victims ranged from 13 to 55 years and the incidence of poisoning varies in different age groups with the highest incidence in the age group of 16 to 35 years (62.5% of the patients), which are similar to the previous study conducted by Mital VP et al.16
Following ingestion of copper sulphate, gastrointestinal symptoms appear first and almost always present. In the current study, nausea (100%), vomiting (100%), abdominal pain (90%), diarrhoea (37.5%), hematemesis /melena (10%) were the observed gastrointestinal symptoms, which are consistent with the data of previous studies.18,19
Hepatic functions usually impaired at the 2nd and 3rd day of ingestion.17, 19 In the previous study by Wahal et al14, jaundice was detectable in 36% of the patients. Jaundice may be hepatocellular or mixed. Clinical jaundice developed on the 2nd or 3rd day in 32.5% patients (13 of total 40). We did liver function tests on 3rd day of ingestion and biochemically SGPT & serum bilirubin were elevated in 40% and 37% patients respectively.

In some study it is found that there may be persistent elevation of SGPT and serum bilirubin without evidence of haemolysis.18 So jaundice is hepatocellular in origin due to direct cellular toxicity.

In our study renal toxicity was manifested by urinary suppression (less than 500 ml/day) in 25%, altered colour urine in 35% of cases (it may be due to haematuria and/or haemoglobinuria). Albuminuria and haematuria was present in 32.5% and 30% of the patient respectively. In previous studies, researchers found all this features in patients with copper sulphate poisoning.21,22 Renal lesion usually occurs due to direct tubular necrosis, hypotension or due to tubular block by haemolytic product.

Total eight patient developed anemia and hemoglobin level was found below 60%, possibly due to haemolysis or blood loss. In the two previous studies fatality rate were 24.2 % and 18.75%.3,13 In our study, fatality rate was 20% which is similar to the previous studies.

The limitations of our study was failure to ascertain the actual toxic dose of copper sulphate as the victims could not mention the exact dose ingested and most of them mentioned the dose of copper sulphate bought by them in terms of taka. Here we mention the dose in terms of grams (in converted scale). We failed to show the percentage of copper sulfate poisoning in relation to total poisoning cases and total admission in this hospital.

Conclusion

Copper sulphate is mostly a suicidal agent of rural people with significant mortality. It is more common in female of young age group. Oral copper sulphate ingestion has significant effects on gastrointestinal, hepatic renal and haematological system. Gastrointestinal symptoms like nausea, vomiting, diarrhea, abdominal pain develop almost invariably. Hepatic toxicities develope later usually on 2nd and 3rd day with appearance of jaundice and elevated level of bilirubin and SGPT. Renal toxicities manifest by urinary suppression, haematuria, albuminuria and renal dysfunction.

References

1. Haded LM, Winchester JF. Clinical Management of poisoning and drug overdose. 2nd ed. Philadelphia: WB Saunders, 1990; 1030-1031.
2. U.S Environmental Protection Agency. guidance for reregistration of pesticide products containing copper sulphate. Fact sheet no. 100. 1986
3. Chuttani HK, Gupta PS, Galati S, Gupta DN. Acute copper sulphate poisoning. Am J Med 1965; 39:849-854.
4. Walsh FM, Crosson FJ, Bayley M. Acute copper intoxication; pathophysiology and therapy with a case report. Am J Dis Child 1977; 131:149-151.
5. Eldad A, Simon GA. The phosphorus burn-a preliminary comparative experimental study of various form of treatment. Burn 1991; 17:198-200.
6. Ahasan HAMN, Chowdhury MAJ, Ajhar MA, Rafique Uddin AKM. Copper sulphate poisoning Trop Doct 1994; 24: 52-3.
7. Turnlund JR, KeyesWR, Anderson Hl, Acrod LL. Copper absorption and retention in young men at three levels of dietary copper by use of the stable isotope 65 Cu. Am J Clin Nutr1989; 49:870-878.
8. Moreno M, Aguilar C, Arola L, Mas A. Respiratory Toxicity of Copper: Environ Health Perspect 1994; 102 (suppl 3):339-340.
9. Oldenquis G, Salem M. Parenteral copper sulphate poisoning causing acute renal failure. Nephlor Dial Transplant1999; 14: 441-443.
10. Reddy KS. The Essential of Forensic Medicine and Toxicology 5th ed. K.Suguna;1995: 436.
11. Klein WJ, Metz EN, Price AR. Acute copper intoxication: a herald of hemodialysis. Arch Intern Med 1972;129:578-582.
12. EXTOXNET, Extention Toxicology Network,copper sulphate 1994:1-8.
13. Agarwal BN, Bray SH, Berez P, Plotzker R, Lavobitz E. Ineffectiveness of haemodialysis in copper sulphate poisoning. Nephron 1975; 15: 74-75.
14. Wahal PK, Mittal VP, Bansal OP. Renal complication in acute copper sulphate poisoning. The Indian Practitioner 1965; 18: 807-12.
15. Akintonwa A, Mebadeje AFB, Odutola TA. Fatal poisonings by copper sulphate ingested from "spiritual water". Vet Hum Toxicol 1989; 31: 453- 4.
16. Mital VP, Wahal PK, Bansal OP. A study of erythrocytic glutathione in acute copper sulphate poisoning. Indian J Pathol Bacteriol 1996; 9: 155-62.
17. Chaowdhury AK, Ghosh S, Pal D. Acute copper sulphate poisoning. J Indian Med Assoc1961; 36 : 330-336.
18. Sing MM, Sing GS. Biochemical changes in blood incase of acute copper sulphate poisoning. J Indian Med Assoc 1968; 50 : 549-54.
19. Thirumalaikolundu subramanian P, Chandramohan M, Jhonson ES. Copper sulphate poisoning. J Indian Med assoc 1984; 82: 6-8.
20. Hantson P, Lievens M, Mahieu P. Accidental ingestion of a zinc and copper sulphate preparation. Clin Toxicol 1996; 34: 725-30.
21. Mehta A, Patney NL, Bhati DPS, Singh SP. Copper sulphate poisoning -its impact on kidneys. J Indian Med Assoc 1985; 83: 108-10
22. Holtzman NA, Elliott Da, Heller RH. Copper intoxication. Report of a case with observation on ceruloplasmin. N EngI J Med 1966; 275: 347-52