INTRODUCTION
Copper sulphate is a compound prepared by the action of sulphuric acid on copper II. Copper sulphate is widely used as fungicide, herbicide and for photography. In a human being, it can lead to anemia. Medical literature is lacking regarding accidental or suicidal poisoning cases of copper sulphate in Pakistan. We present a case of accidental ingestion of copper sulphate resulting in severe acute toxicity, which was successfully managed by intensive supportive measures and Dimercaprol (BAL).

CASE REPORT
A 22-year-old male with no significant past medical history presented in the emergency department with multiple episodes of severe vomiting as a consequence of accidental ingestion of a cup of copper sulphate (powdered form) 3 hours before presentation. Vomiting was non-projectile, initially blue in colour and then turned coffee-ground in appearance. It was accompanied with severe abdominal pain and odynophagia.

On examination, the patient was conscious but anxious and appeared dehydrated. His blood pressure was 168/112 mmHg, heart rate 98 beats/minute, respiratory rate 20 breaths/minute and oxygen saturation by pulse oximeter was 99%. On general physical examination, he was cyanosed on the extremities. Abdomen was soft and tender in epigastrium with audible gut sounds. Other systemic examination was unremarkable.

Gastric lavage was done and the patient was started on intravenous (IV) infusion of 0.9% normal saline at 150 cc/hour. Blood samples were sent for initial investigations which revealed leucocytosis with a total leucocyte count of 24.9x10^9/L with predominantly neutrophils. Hemoglobin was 17.6 gm/dl, platelets 377x10^9/L. Other laboratory investigations were within normal limits.

The patient was initially started on IV antiemetics and Penicillamine 1 g daily. Methemoglobin level at the time of admission was 0.0% which increased to 27% on the next day. Injection methylene blue was started immediately at a dose of 1 mg/kg to treat methemoglobinemia. Methemoglobin levels continued to rise and reached 34% after 30 hours of admission. At this time, hematologist was consulted and an advice for exchange transfusions with packed red cells along with vitamin C supplements was given.

Despite IV methylene blue, multiple exchange transfusions, and fluid reconstitution with sodium bicarbonate, methemoglobin level continued to rise reaching 43% on the 3rd day. The patient also developed acute renal insufficiency with serum creatinine level of 4.3 mg/dl. Kidney functions declined further and the patient was started on hemodialysis on daily basis when the creatinine level reached 5.3 mg/dl on the 5th day of admission. Liver dysfunction developed with total bilirubin of 2.9 mg/dl, alanine aminotransferase 85 IU/L, alkaline phosphat of 30 IU/L and gamma glutamyl transferase of 12 IU/L. Patient also developed rhabdomyolysis with CPK levels of 10420 IU/L.

The oxygen saturation progressively deteriorated due to volume overload and chest X-ray findings were compatible with adult respiratory distress syndrome. Subsequently

ABSTRACT
Copper sulphate is a compound prepared by the action of sulphuric acid on copper II. Copper sulphate is widely used as fungicide, herbicide and for photography. In a human being, it can lead to anemia. Medical literature is lacking regarding accidental or suicidal poisoning cases of copper sulphate in Pakistan. We present a case of accidental ingestion of copper sulphate resulting in severe acute toxicity, which was successfully managed by intensive supportive measures and Dimercaprol (BAL).

Key words: Copper sulphate. Methemoglobinemia. Rhabdomyolysis.

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the patient was intubated and shifted to Intensive Care Unit. Methemoglobin levels did decline from the baseline but were still persistently high i.e. 2.8% therefore, Dimercaprol (BAL) given intramuscularly at a dose of 3 mg/kg 4 hourly for the first 2 days was started. This was followed by 6 hourly intervals of BAL for 1 day and finally 12 hourly for the next 10 days. After 10 days of intensive management, the clinical and laboratory parameters improved and finally the patient was extubated. Liver and renal functions also improved considerably. Hemodialysis was stopped since the patient developed diuretic phase of acute renal insufficiency. Rhabdomyolysis was resolved with a fall in CPK to 2128 IU/L. He was finally discharged after 2 weeks of hospitalization and intensive treatment. Methemoglobinemia was controlled and its level was 0.2 but he was advised to continue Dimercaprol for another 3 days.

In subsequent follow-up, he was asymptomatic and clinically stable with an excellent performance status.

**DISCUSSION**

Copper is one of the essential trace metals in human which is vital for the function of enzymes such as cytochrome c. Commercially it is used in insecticides, white-washing and manufacturing of leather. Copper sulphate is an oxidizing agent and acts as an irritant to body mucous membranes.2 Acute toxicity results in acute renal insufficiency, hepatic dysfunction, with deranged coagulation thrombocytopenia, intravascular hemolysis, methemoglobinemia, gastrointestinal symptoms which include nausea, vomiting, abdominal pain and malena, hypotension, rhabdomyolysis, cardiovascular collapse and shock.4,5 However, copper sulphate toxicity is not uncommon in India. Case studies from Agra and New Delhi reported the incidence of copper sulphate toxicity in 1960’s to be 34% and 65% respectively.6 Early death was related to hypovolemic shock while late deaths were attributed to renal or hepatic failure.

Most cases of copper sulphate poisoning occur with ingesting at least 1 gram of copper,7 which is similar in this case as well. Severity of symptoms is monitored by serum or blood copper levels. However, the main predictor was methemoglobin levels which correlated with the prognosis. Treatment is mainly supportive and early measures include administration of chelating agents to prevent fatal outcomes of copper sulphate toxicity. Methemoglobinemia can be treated with methylene blue. Among the chelating agents British anti-Lewisite (BAL), D-penicillamine, 2,3-dimercapto-1-propane sulphonate (DMPS), ethylene diamine tetra acetate (EDTA) and edetate calcium disodium have been used.8-10 In addition to chelating agents blood products, intravenous fluids, vasopressors and dialysis is also required as evident in this patient. Hemodialysis also is ineffective in removing copper from the body since it binds to serum and tissue proteins. Chelated copper can be removed from serum by diuresis and dialysis.2 This was also practiced during the management of the present case.

In short, a case of copper sulphate poisoning with rhabdomyolysis and acute renal failure can be successfully treated with administration of dimercaprol, blood products and hemodialysis.

**REFERENCES**