Case Report

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Acute Renal Failure due to Copper Sulfate Poisoning; a Case Report

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Abstract

Background: Copper sulfate is a blue and odorless salt with various industrial, chemical, agricultural and medicinal applications. Copper sulfate poisoning is rare in children.

Case Presentation: A 23-month old boy accidentally ingested a solution of copper sulfate, used as a disinfectant agent in animal husbandry. He was referred to Children's Hospital of Tabriz because of frequent vomiting and lethargy. The major systemic complications were intravascular hemolysis, anemia and acute renal failure. The patient was successfully treated with multiple packed cell transfusions, dimercaprol, penicillamine and peritoneal dialysis. Plasma copper level 15 days after ingestion was 216 µg/dl.

Conclusion: Copper sulfate is a highly toxic agent that, when ingested, can cause local and systemic damage including coma, shock, severe intravascular hemolysis, hepatotoxicity and acute renal failure with high mortality.

Key Words: Copper sulfate; Poisoning; Renal failure; Dimercaprol; Toxic agents

Introduction

Copper is an essential trace element in humans. Copper compounds are widely used in electrical industry, metallurgy, photography, painting, leather manufacture and water purification. Among the medicinal applications of the copper is its utilization in certain types of dental cement and intrauterine contraceptive devices (IUCD). Copper sulfate, one of the most available salts of copper, is a blue and odorless salt that is employed in various products such as fungicides, algacides, herbicides and insecticides[1,2]. Copper sulfate is also found in chemistry laboratories as wettable powders and fluid concentrates. It can be distributed in air as dust. Copper sulfate can be absorbed through the gastrointestinal tract, lungs and skin causing both systemic and local toxicity. In the past, copper sulfate was being used as an emetic. There are some case reports of

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death due to copper sulfate containing emetics, from China[3]. Nowadays its use as an emetic is contraindicated.

Most case reports of copper sulfate poisoning, as a suicide attempt or accidental exposure, have involved adults[2-7]. In a literature review we found few reports in children[1,8,9]. This article presents a 23-month old boy who accidentally ingested a solution of copper sulfate, used as a disinfectant and fungicide agent in animal husbandry.

Case Presentation

A previously healthy 23-month-old boy was referred to Children's Hospital of Tabriz/Iran, 3 hours after accidental ingestion of 10^{cc} solution containing copper sulfate. Before admission, he vomited frequently with bloody contents at last episode. He had received gastric irrigation by normal saline before referral to this center. At admission he was lethargic. His blood pressure was 80/60 mmHg and heart rate was 140/min. Other physical examinations were normal.

Laboratory findings at arrival were as follows: hemoglubine (Hgb) 12.5 g/dl, reticulocyte 2%, serum sodium 127 meq/lit, serum potassium 4.9 meq/lit, 21700/mm^3 leukocytes in peripheral blood (WBC) and serum creatinine 0.7 mg/dl. Treatment with dimercaprol (British 3-5 Anti Lewisite or BAL) was started 4 mg/kg/q6hr. Within hours he developed pallor and excreted brownish red urine. The laboratory tests 24 hours post ingestion revealed: Hb 5.3 g/dl, WBC 28000/mm^3, reticulocyte 10%, SGOT 189, SGPT 12, total bilirubin 4.8 mg/dl, and direct bilirubin 0.2 mg/dl. Serum albumin, protrombin time (PT) and partial thrombin time (PTT) were normal. Urine analysis revealed hemoglobinuria.

Regarding the severe hemolysis and hemoglobinuria, we increased the volume of fluid therapy to 1.5 times of maintenance and sodium-bicarbonate was added to fluids for prevention of tubular damage. He received three transfusions of packed red cells (10 ml/kg/dose) during the first and second day of admission. On the second day, D-penicillamine 25 mg/kg/8hr was added to the treatment regimen. On third day, urine output decreased to less than 0.5 ml/kg/hr, serum creatinine increased to 3.3 mg/dl and periorbital edema appeared.

Regarding the development of oliguric acute renal failure, dimercaprol was discontinued. On sixth day, serum creatinine level was 8 mg/dl and serum urea was 250 mg/dl. So, peritoneal dialysis was initiated. After remaining oliguric for one week urine output gradually increased and we stopped peritoneal dialysis on tenth day. The plasma level of copper, measured 15 days after ingestion was 216 µg/dl (normal 70-140 µg/dl). He was discharged on 20th day in good condition. At discharge Hgb level was 11.5 g/dl and serum creatinine 0.6 mg/dl. Treatment with D-penicillamin was continued for two months after discharge and then stopped when the plasma copper level was 140 µg/dl.

At follow-up visits 2 and 4 months after discontinuation of D-penicillamin, he was normal in physical examination without any symptoms and all laboratory tests including plasma level of copper, hemoglobin, serum electrolytes, serum creatinine, liver function tests and urinalysis were in normal range.

Discussion

Following the ingestion of copper sulfate, gastrointestinal symptoms are usually the first to appear and include metallic taste, nausea, vomiting, epigastric pain, diarrhea and gastrointestinal hemorrhage[2]. These symptoms may appear within 10 minutes to one hour after ingestion. The irritating effect of copper sulfate causing vomiting may reduce the amount of copper sulfate absorption. Symptoms related to systemic toxicity include delirium, stupor, coma, convulsion, hypotension, shock, respiratory failure, pallor and jaundice[2,10]. Also, Methemoglobinemia, rhabdomyolysis and hepatotoxicity have been reported[10,11]. In two studies from India and Bangladesh, hepatotoxicity was one of the main complications of copper sulfate

poisoning[10,12], but we didn't find any hepatocellular toxicity in our patient. Intravascular hemolysis and acute renal failure are the cardinal consequences of copper toxicity in most reported cases [2,3,4,10,11,12] as occurred in our patient. Copper inhibits the function of glucose-6-phosphate dehydrogenase in the red blood cells. This enzyme is necessary for protection of red blood cells against the hemolytic effects of oxidizing substances. Copper ion not only inhibits this enzyme, but also is a strong oxidizing agent. Hemoglobinuria due to intravascular hemolysis and a direct action of copper on the kidneys often leads to tubular necrosis and acute renal failure. Acute copper sulfate poisoning may result in irreversible chronic tubulointerstitial nephritis[7]. Also, acute interstitial nephritis has been reported due to chronic exposure to copper containing IUCD in a woman with copper allergy[13].

Blundell et al reported a case with severe hemolysis and coma whose plasma copper sulfate level was normal on several occasions throughout the admission[1]. Hantson et al[4] reported a case with cardiovascular collapse and acute renal failure whose plasma level of copper 90 minutes after ingestion, was 209µg/dl (normal 70-140 µg/dl). In our case plasma level of copper was 216 µg/dl even 15 days after ingestion. So plasma copper sulfate level does not correlate with severity of poisoning. Whole blood copper measurement may predict the severity of toxicity more accurately, because copper rapidly accumulates in red blood cells. Copper is also transported to the liver and binds with proteins. The subsequent release of copper protein complex into the serum leads in a secondary rise in plasma copper levels[2].

The mortality rate of copper sulfate poisoning is high in comparison with other forms of poisoning. Its mortality rate in Bangladesh has been reported as high as 24.9%[12]. There are some reports of early death within hours after copper sulfate poisoning[6,8]. Unlike these reports, our patient had a good outcome, maybe because of rapid initiation of chelators. Early death is usually the consequence of shock and circulatory collapse, while late death is related to hepatic or renal failure.

Treatment of copper sulfate poisoning is supportive including blood products, intravascular fluids and vasopressors. Gastric lavage and activated charcoal is not recommended because copper sulfate is a corrosive agent and may cause mucosal damage and perforation. The treatment of choice is chelation with dimercaprol and penicillamin[2,4]. Hemodialysis is inefficient in removing copper from the body[2]. Dialysis is recommended only in cases of acute renal failure. Takeda et al. proposed a combined treatment with chelating agents and blood purification with hemoperfusion and hemodiafiltration[11].

Conclusion

Copper sulfate is a highly toxic agent that when ingested can cause local and systemic damage, including coma, shock, severe hemolysis, hepatotoxicity and acute renal failure, with high mortality. This compound should be kept away from access of children.

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