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CASE REPORT

Acute copper sulphate poisoning: A case report and literature review☆

Nicolas Franchitto[a,b,*], Peggy Gandia-Mailly[c], Bernard Georges[d], Anne Galinier[e], Norbert Telmon[b], Jean Louis Ducassé[a], Daniel Rougé[b]

[a] *Service d'Aide Médicale d'Urgence, Centre Hospitalier Universitaire Purpan, 330 avenue de Grande Bretagne, TSA 70034, 31059 Toulouse Cedex 9, France*
[b] *Service de Médecine Légale, Centre Hospitalier Universitaire Rangueil, 1 avenue du Professeur Jean Poulhès, TSA 50032, 31059 Toulouse Cedex 9, France*
[c] *Laboratoire de Pharmacocinétique et de Toxicologie Clinique, Institut Fédératif de Biologie, Centre Hospitalier Universitaire Purpan, 330 avenue de Grande Bretagne, TSA 70034, 31059 Toulouse Cedex 9, France*
[d] *Service de Réanimation Polyvalente, Centre Hospitalier Universitaire Rangueil, 1 avenue du Professeur Jean Poulhès, TSA 50032, 31059 Toulouse Cedex 9, France*
[e] *Laboratoire de Biochimie de la Nutrition, Centre Hospitalier Universitaire Rangueil, 1 avenue du Professeur Jean Poulhès, TSA 50032, 31059 Toulouse Cedex 9, France*

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Summary Voluntary copper poisoning is a rare mode of suicide. We report a case of copper sulphate poisoning in a patient presenting delusions with mystic demands for purification. The initial gastrointestinal symptoms were followed by intravascular haemolysis and renal failure. The course was favourable after symptomatic treatment and specific copper chelation therapy. However, the pathogenesis is not fully understood and with the present state of knowledge, no one treatment can be said to be superior to another. The authors discuss the various treatments of this rare poisoning through a review of the available literature.

Introduction

Copper is an essential trace element in humans and is involved in complex enzymatic reactions. Like all trace elements, it is not only deficiency, but also excessive intake, that can lead to life-threatening complications. Acute copper poisoning is well described in animals.[1,2] In man, it is most often chronic and accidental, being occupation-related. Sources of exposure are generally found in the copper industry and the metal industry in general, in

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* Corresponding author at: Service de Médecine Légale, Centre Hospitalier Universitaire Rangueil, Avenue Jean Poulhès, TSA 50032, F-31059 Toulouse Cedex 9, France. Tel.: +33 5 61 32 29 54; fax: +33 5 61 32 21 77.
E-mail address: franchitto.n@chu-toulouse.fr (N. Franchitto).

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Table 1 Hematologic laboratory values at admission

Hematologic laboratory values	Day 1
Hemoglobin (g/dl)	16.9
Hematocrit (%)	49.9
White cell count per μl	36,800
Platelet count per μl	416,000
Prothrombin time (%)	88
Activated partial thromboplastin time (s)	33

Table 2 Blood chemical findings at admission

Blood chemical findings	Day 1
Sodium (mmol/l)	132
Potassium (mmol/l)	2.7
Chloride (mmol/l)	101
Bicarbonates (mmol/l)	22
Total protein (g/l)	84
Calcium (mmol/l)	2.16
Urea nitrogen (mmol/l)	11
Creatinine (μmol/l)	126
Glucose (mmol/l)	16.9
Aspartate aminotransferase (IU/ml)	46
Alanine aminotransferase (IU/ml)	5
Alkaline phosphatase (IU/ml)	170
Lactate dehydrogenase (IU/ml)	440
Creatine phosphokinase (IU/ml)	166
Amylase (IU/ml)	24
Lipase (IU/ml)	57
Myoglobin (μg/l)	61
Haptoglobin (g/l)	1.11
Serum copper (μmol/l)	140
CRP (mg/l)	147.2
PaO_2 (mm Hg)	146
$PaCO_2$ (mm Hg)	29
pH	7.48

the wood industry, via disposal of household waste by incineration, in agriculture and, in particular, during the manufacture of fertilizers, fungicides and insecticides.[3] Cases of acute poisoning are rare, generally voluntary with suicidal intent, and are infrequent in the world with the exception of India.[4]

We report a case of poisoning by ingestion of copper sulphate in a young man who was in daily contact with the substance in the course of his occupation, and we discuss the various treatments available through a review of the literature.

Case report

A 29-year-old man, a wine grower, was admitted to the emergency department of our university hospital with a 1-day history of vomiting and diarrhoea with fever. Acute gastroenteritis had initially been diagnosed and treated symptomatically. The patient was accompanied by his parents, who suspected possible voluntary poisoning with homemade rodent poison associated with ingestion of an undetermined amount of copper sulphate salts. They reported a previous suicide attempt 2 years earlier and a journey to India for 'philosophical initiation'. The patient, who worked in the family vineyards, was subsequently seen by a psychologist. However, no repeated suicide attempts were made and he was not under any medication. His medical history was otherwise unremarkable.

At admission to the emergency department, the patient's Glasgow Coma Scale score was 11, his temperature was 38.5 °C, respiratory rate 28 min^{-1}, pulse rate 110 min^{-1}, and blood pressure 105/71 mm Hg. It was not possible to question the patient because he was clearly psychotic. Abdominal palpation showed epigastric tenderness without guarding or tenseness. Clinical examination revealed coarse rales at the base of the right lung, suggesting possible aspiration pneumonitis. There was no bleeding of the gums or nose, or external signs of haemorrhage.

The patient was admitted to the intensive care unit (ICU). Although he was not initially hypoxaemic, oxygen at 3 l min^{-1} was started because of tachypnoea and the clinical suspicion of aspiration pneumonia. Blood gas analysis and laboratory findings on admission are summarised in Tables 1 and 2. Toxicological screening revealed no other poisons (tricyclic antidepressants, paracetamol, benzodiazepines, carbamates, barbiturates). Intravenous saline 0.9% (3 l over 24 h) was started and potassium chloride 6 g was given over 6 h. Prophylactic antibiotic therapy was started with amoxicillin–clavulanic acid (2 g t.d.s.) and levofloxacin (500 mg b.d.) and omeprazole (40 mg daily) was given to protect the gastrointestinal mucosa from the toxic effect of the copper. Serum potassium was measured 3 h after the start of rehydration. The delusional syndrome was treated with loxapine (50 mg/day) and risperidone (4 mg/day). Copper chelation therapy was started with intravenous edetate calcium disodium, 1 g every 12 h in 250 ml of 5% dextrose over a 1 h period twice daily.[5–7]

Intravascular haemolysis occurred the day after admission, which was day 3 after poisoning. The patient had macroscopic haematuria, oliguria, and tachycardia, but no other signs of hypovolaemic shock. The laboratory findings are summarised in Tables 3 and 4.

Gastroduodenal endoscopy revealed a perforating ulcer of the pyloric antrum with signs of recent stage IIC haemorrhage and diffuse bleeding from the duodenal mucosa. There was no greenish colouring of the gastrointestinal mucosa. Renal ultrasound and laryngeal endoscopy were normal. Parenteral copper chelation therapy was continued for 3 days. If renal insufficiency had worsened, the dose of edetate calcium disodium would have been decreased or the number of injections per day reduced from two to a single injection.[8] However, as the clinical gastrointestinal manifestations regressed, edetate calcium disodium was replaced by oral dimercaprol, available in our university hospital, at a dose of 3 mg/kg four times daily for 5 days. Four packed red cell transfusions were given to correct the anaemia. Haemodialysis was not required as diuresis was well maintained.

The patient was in the ICU for 9 days and was then transferred to the psychiatric department, where he was treated for 1 month. Laboratory findings became normal at 13 days. Schizophrenia was also diagnosed. The delusional syndrome included delusions of being controlled and the predominant