

MORPHOLOGICAL CHANGES IN COPPER SULFATE POISONING - A CASE OF SUICIDE

Atanas Hristov*, Teodora Kiryakova, Alexandar Alexandrov

Department of Forensic Medicine and Deontology - Medical University – Sofia

*Corresponding author:

Atanas Christov, MD, PhD

Medical University, Sofia-1431, Bulgaria

Department of Forensic Medicine and Deontology

1 Georgi Sofiiskiblvd.

Tel: +35929230749

Fax: +35929230412

E-mail: iamnasko@yahoo.com

Abstract

We present is a case of a young 23-year-old woman found in her home in a comatose state, who was taken to the nearest medical center and died there despite the emergency resuscitation measures. The autopsy revealed a characteristic bluish discoloration of the gastric and necrotic changes in the duodenal mucosa. The conducted toxic-chemical studies proved that this was a case of copper sulfate poisoning. This was confirmed by the subsequent inspection at the home of the deceased, where a suicide note was found, written by the deceased, where in addition to the reasons for her decision, she specified the way - intake of copper sulfate.

Key words: copper sulfate; "bluestone" poisoning; toxicology

Introduction: In modern forensic practice acute heavy metal poisoning (an accident or suicide) is very rare. For the period 1990-2020 for the regions of Sofia city and Sofia district in the Department of Forensic Medicine and Deontology at the Medical Faculty, Medical University-Sofia there was only one case (current) of acute lethal poisoning with copper sulfate diagnosed. Copper sulfate (blue stone) is a solution used in agriculture to control parasites and fungi, having a contact effect [1]. Copper sulfate poisonings are most often accidents (by accidental inhalation of vapors containing copper sulfate or by incorrectly drinking liquid with a copper sulfate dissolved in it). Cases of suicide or attempted suicide are extremely rare [2, 3, 4]. The symptom complex and morphological changes in acute copper sulfate poisoning taken orally are characteristic and, if known, clinicians can quickly and easily put the correct diagnosis [5].

Clinical symptoms: After oral administration (most commonly in forensic cases) of a toxic or lethal amount of CuSO₄, dry mouth, metallic taste and severe burning pain in the pharynx, esophagus and stomach are felt (this is due to the fact that copper sulfate has a strong corrosive and necrotic effect on the lining of the digestive tract), which is often accompanied by nausea and vomiting, which leads to aggravation of the corrosive-necrotic changes in the lining of the esophagus and oral cavity, manifested by increased pain. Very soon after ingestion of the poisonous substance, its hemolytic action is manifested with the appearance of methaemoglobin in the blood, blood plasma and urine, followed by acute anemia, acute renal failure, hypotension and hypovolemic shock [6, 7]. The victim may experience fever - up to 39°C and above, profuse sweating, severe muscle pain (especially in the limbs) and severe fatigue. In addition, acute poisoning is usually accompanied by severe general cerebral symptoms, such as increasing tension headaches, convulsions and rapid development of deep coma. In acute poisoning, which even with timely resuscitation, often results in death [8].

Toxicological effects: Oral administration of one gram of copper sulfate in humans immediately causes gastrointestinal, liver and kidney damage, clinically manifested by severe stomach pain, vomiting, diarrhea, hemolysis with methaemoglobinemia, hepatic necrosis, hematuria, hypotension, tachycardia, proteinuria, convulsions and death (US AF, 1990). Gastrointestinal and toxic liver damage also occur when ingesting a glass of water containing 2.2 mg-7.8 mg Cu / 1 [9, 10].

Pathomorphology: Bluish or bluish-green coloration, edema, erosions and hemorrhages of the mucous membrane of the oral cavity, esophagus, stomach and the initial sections of the small intestine; hepatic and renal degenerative changes to necrosis; postmortem spots with a purple color and a slightly brownish tinge; morphological cadaveric finding of rapidly occurring death with dark red liquid blood, with a slight brownish tinge [11, 12, 13].

Case presentation: We observed a case of a 23-year-old woman who was admitted to the regional medical center in the town of Novi Iskar in a comatose state and hemodynamic collapse, with unknown genesis. Despite emergency resuscitation, the young woman died and was sent for a forensic examination.

Materials and methods: A forensic examination of the case was performed, including full forensic autopsy, histological examination of internal organs and chemical analysis of intra-organ parts and their contents.

Results: The autopsy of the deceased revealed: bluish-green coloration of the mucous membrane of the oral cavity, esophagus, stomach and the initial sections of the small intestine, as on the gastric mucosa (Fig. 1), duodenum (Fig. 2). The initial part of the small intestine had also many superficial and deep erosions with hemorrhages along their edges (most pronounced in the area of the duodenum and the initial part of the jejunum); postmortem lividity with a purple color and a slightly brownish tinge; morphological cadaveric finding of rapidly occurring death with dark red liquid blood with a slight brownish tinge. During the autopsy, a preliminary test of the gastric contents was performed with litmus paper, which showed a weak acid reaction.



Fig 1



Fig. 2

Microscopic examination revealed: stomach and duodenum (Fig. 3, 4, 5, and 6) - severe exfoliation of the epithelium; extensive linear hemorrhages, ulcerations and necrosis of the gastric and intestinal epithelium, in some places reaching the lamina muscularis, the most severe changes are in the area of the duodenum, where in many places the mucosa is completely obliterated and only glandular shadows are visible. Liver (Fig. 5) - degenerative changes in the

structure of liver cells, blood filling of vessels and sinusoids, and in some places they gape empty.

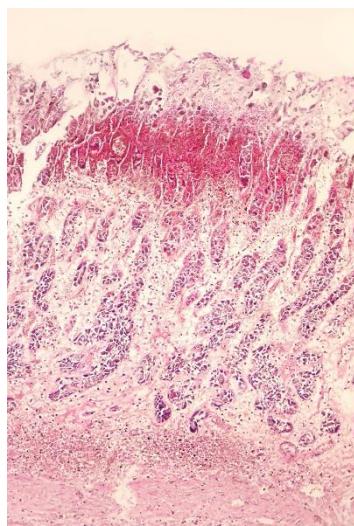


Fig. 3

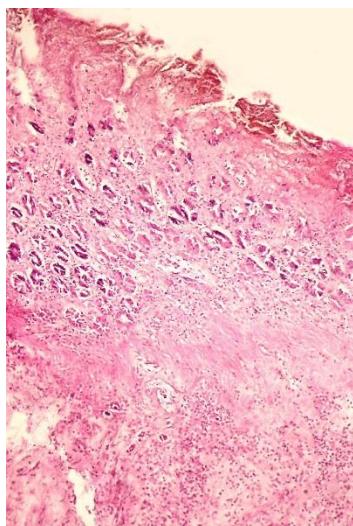


Fig. 4

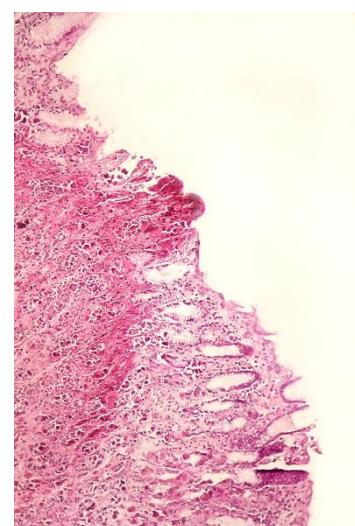


Fig. 5

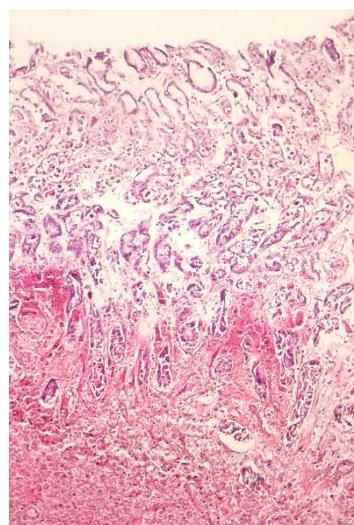


Fig. 6

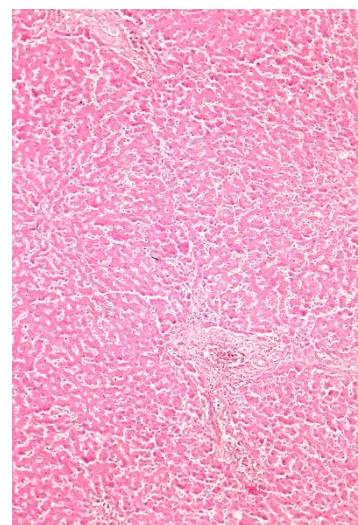


Fig. 7

The toxicological examination of the blood samples taken at the autopsy and intra-organ parts (stomach, small intestine and contents, liver, kidney) revealed the presence of copper ions (by color reactions), as well as the presence of 46% methemoglobin.

Discussion: Based on the characteristic morphological autopsy finding (bluish-greenish staining of the mucous membrane of the oral cavity, esophagus, stomach and initial sections of the small intestine, accompanied by edema, erosions, hemorrhages and necrosis of the same; degenerative changes; with a purple color and a slightly brownish tinge; morphological finding of rapid death with the presence of dark liquid blood with a slight brownish tinge) and the results of chemical studies, gave reason to believe that it was acute poisoning with the compound copper sulfate (CuSO_4), which was confirmed both by the clinical symptoms observed when the woman was found (comatose state with hemodynamic collapse) and by the subsequently found suicide note.

REFERENCES:

1. Aaseth, J. and T. Norseth. 1986. In: L. Friberg, G.F. Nordberg and V.B. Vouk, Eds. Handbook on the Toxicology of Metals, 2nd. ed., Vol. II. Specific Metals, Elsevier, Amsterdam, pp. 233-254.
2. Ahsan HAMN, Chowdhury MAJ, Azhar MA, Rafiqueuddin AKM. Copper sulphate poisoning. *Trop Doct* 1994; 24: 52-3.
3. Akintonwa A, Mabadeje AFB, Odutola TA. Fatal poisonings by copper sulfate ingested from "spiritual water". *Vet Hum Toxicol* 1989; 31: 453-4.
4. Askergren, A. and M. Mellgren. 1975. Changes in the nasal mucosa after exposure to copper salt dust. *Scand. J. Work Environ. Health* 1: 45-49. (Cited in ATSDR, 1990; Stokinger, 1981)
5. Chuttani HK, Gupta PS, Gulati S, Gupta DN. Acute copper sulfate poisoning. *Am J Med* 1965; 39: 849-54.
6. Goyer, R.A. 1991. Toxic effects of metals. In: M.O. Amdur, J. Doull and C.D. Klaasen, Eds., Casarett and Doull's Toxicology, 4th ed. Pergamon Press, New York, NY, p. 653-655.
7. Hantson P, Lievens M, Mahieu P. Accidental ingestion of a zinc and copper sulfate preparation. *Clin Toxicol* 1996; 34: 725-30.
8. Isolauri J, Markkula H, Auvinen O. Copper sulfate corrosion and necrosis of the esophagus and stomach. *Acta Chir Scand* 1986; 152: 701-2.
9. Jantsch W, Kulig K, Rumack BH. Massive copper sulfate ingestion resulting in hepatotoxicity. *Clin Toxicol* 1984/85; 22: 585-8.
10. Mueller-Hoecker, J., U. Meyer, B. Wiebecke, et al. 1988. Copper storage disease of the liver and chronic dietary copper intoxication in two further German infants mimicking Indian childhood cirrhosis. *Pathol. Red. Pract.* 183: 39-45. (Cited in ATSDR, 1990)
11. Stein RS, Jenkins D, Korns ME. Death after use of cupric sulfate as emetic. *JAMA* 1976; 235: 801.
12. Wahal PK, Mehrotra MP, Kishore B, Patney NL, Mital VP, Hazra DK, Raizada MN, Tiwari SR. Study of whole blood, red cell and plasma copper levels in acute copper sulphate poisoning and their relationship with complications and prognosis. *J Assoc Physicians India* 1976; 24: 153-8.
13. Walsh FM, Crosson FJ, Bayley M, McReynolds J, Pearson BJ. Acute copper intoxication. Pathophysiology and therapy with a case report. *Am J Dis Child* 1977; 131: 149-51.