

Copper sulphate induced hemolytic anemia and thrombocytopenia

M Mathew, G Goel, A Kurien, C Manohar

Citation

M Mathew, G Goel, A Kurien, C Manohar. *Copper sulphate induced hemolytic anemia and thrombocytopenia*. The Internet Journal of Hematology. 2008 Volume 5 Number 2.

Abstract

Copper sulphate (CuSO₄) is used for whitewashing, agricultural and leather industry in India. Even a very small quantity is toxic. Toxicity is generally as a result of accidental ingestion or suicide attempts and leads to intravascular hemolysis, methemoglobinemia, renal failure, hepatotoxicity, rhabdomyolysis and often death. Mortality is quite high in severe cases. We present a case of a 32 year old male who was hospitalized after copper sulphate ingestion and was diagnosed to have copper sulphate induced hemolytic anemia and thrombocytopenia along with acute renal failure.

INTRODUCTION

Copper is an essential trace metal in humans. It is a cofactor for several enzyme reactions in the human body.¹ Copper sulphate poisoning is sporadically reported in western countries but it is very common in India.² Various aspects of CuSO₄ poisoning have been documented in literature, especially its association with hemolysis and methemoglobinemia. However, its association with thrombocytopenia has not been documented. In this article, we present a case of CuSO₄ poisoning with a unique finding of thrombocytopenia along with hemolytic anemia and acute renal failure.

CASE REPORT

A 32 year old male with history of copper sulphate ingestion presented with tachycardia, low blood pressure and dark stools and urine. He also complained of pain in right hypochondrium, breathlessness and features of acute renal failure. He slowly progressed to coma, but subsequently recovered with treatment.

The complete blood picture of the patient revealed a normocytic normochromic anemia with features of hemolysis (presence of spherocytes, nucleated RBCs and polychromatophils), an increase in WBC count with a marked shift to left and a decrease in platelet count (86,000/cu mm). The coagulation profile (BT/CT/PT) was within normal limits.

The renal function was abnormal with a creatinine of 7.9

mg% and blood urea of 253 mg%. The liver function tests were within normal range.

The urine examination showed presence of 2+ protein, 2-4 WBCs and 1-2RBCs/hpf. The stool examination was positive for occult blood.

All the investigations along with the clinical history led to a diagnosis of copper sulphate induced hemolytic anemia with thrombocytopenia and acute renal failure.

DISCUSSION

Consumption of even more than one gram of copper containing substance can lead to signs and symptoms of copper toxicity.¹ Copper salts are good oxidizing agents and can be easily absorbed by skin and mucosa and lead to immediate irritation and erosion of mucous membranes and acute toxicity.^{3,4} Clinical features of CuSO₄ toxicity are the consequence of multiple mechanisms like inhibition of glycolysis, oxidation of NADPH and inhibition of G6PD.⁵ Free reduced copper in the cell binds to sulfhydryl groups and inhibits the action of certain enzymes like glucose-6-phosphate dehydrogenase and glutathione reductase. It also reacts with reactive oxygen species and catalyzes the production of reactive toxic hydroxyl radicals leading to cellular damage and cell death.⁴

Intravascular hemolysis is seen 12-24hrs after CuSO₄ ingestion. The inorganic copper accumulates in the erythrocytes and leads to increase in membrane permeability and osmotic fragility either by direct damage to erythrocyte

membranes or by oxidative damage.¹⁴ Hemolysis is followed by the appearance of methemoglobinemia. Copper oxidizes the heme iron to a ferric state, the methemoglobin, which has a decreased oxygen carrying capacity. The clinical appearance of cyanosis depends on the amount of methemoglobin formed.⁴⁶ The patient in our report presented with thrombocytopenia along with features of hemolysis. This finding has not been documented so far in the literature.

The renal complications are generally observed after 48hrs. Renal failure occurs due to the direct toxic effect of copper on proximal tubular epithelium, decreased renal perfusion secondary to hypovolemia and intravascular hemolysis.¹⁴

The cardiovascular system, central nervous system, skeletal muscles and endocrine system are rarely directly affected by copper sulphate poisoning.¹ The mainstay of treatment for copper poisoning is supportive care but a few authors have found chelating therapy and hemodialysis to be effective.³⁴

CONCLUSION

To conclude, copper sulphate toxicity mainly presents with hemolysis and renal failure. But in this case, an unusual finding of thrombocytopenia was observed, which has not been documented in literature..

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Author Information

Mary Mathew, MD

Department of Pathology, KMC

Garima Goel, MD

Department of Pathology, KMC

Annamma Kurien, MD

Department of Pathology, KMC

Chetan Manohar, MD

Department of Pathology, KMC