International Journal of Research in Medical Sciences
Manivannan PR et al. Int J Res Med Sci. 2026 Sep;14(9):4101-4104
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20263135

Case Report

Severe multiorgan toxicity following low-dose copper sulfate poisoning in a patient with Psoriasis vulgaris on methotrexate: a case report

Prince Rueban Manivannan[1], Vishaalpalaniswamy Ramaswamy[2], Sakthikumar Soundarrajan[3]*

[1]Institute of Medical Gastroenterology, Madras Medical College, Chennai, Tamil Nadu, India
[2]Madras Medical College, Chennai, Tamil Nadu, India
[3]Department of Medicine, Dharmapuri Medical College, Dharmapuri, Tamil Nadu, India

Received: 01 July 2026
Accepted: 14 August 2026

***Correspondence:**
Dr. Sakthikumar Soundarrajan,
E-mail: sakthikumarmmc@gmail.com

ABSTRACT

Acute copper sulfate poisoning usually causes severe toxicity only after large ingestions. We report a man in his early 50s with psoriasis vulgaris receiving methotrexate who developed severe intravascular hemolysis, methemoglobinemia, acute hepatotoxicity, and acute kidney injury after ingesting only 1 g of copper sulfate. Serial peripheral blood smears, biochemical parameters, and clinical progression confirmed delayed multi-organ toxicity. He recovered following aggressive supportive care, forced alkaline diuresis, and withdrawal of methotrexate, without requiring dialysis. This case suggests that psoriasis-associated copper dysregulation and methotrexate-induced oxidative stress may lower the threshold for severe copper sulfate toxicity.

Keywords: Copper sulfate poisoning, Psoriasis vulgaris, Intravascular hemolysis, Multi organ failure

INTRODUCTION

Acute copper sulfate intoxication represents a challenging toxicological emergency in critical care, capable of driving rapid, life-threatening multi-organ failure through intense systemic oxidative stress. While toxic heavy metal exposures can occur through chronic environmental or occupational pathways, acute oral ingestions trigger immediate gastrointestinal mucosal injury followed by severe systemic sequelae which includes severe intravascular hemolysis, methemoglobinemia, acute hepatotoxicity, and secondary acute kidney injury.[1,2]

Recent clinical research demonstrates that patients with pre-existing inflammatory conditions, such as psoriasis vulgaris, live in a chronic state of altered microelement metabolism characterized by significantly elevated baseline serum copper levels and higher copper-to-zinc ratios that correlate directly with disease severity.[3] This may thereby increase the susceptibility of patient to copper sulphate poisoning even at sub lethal doses. We here report a male in his early fifties, known case of psoriasis vulgaris on methotrexate therapy, brought to our ED with alleged copper sulphate poisoning.

CASE REPORT

A male sculptor in his early 50s, was brought to the emergency department following the alleged intentional ingestion of approximately 1g of copper sulphate crystals. He is a known case of type 2 diabetes mellitus (T2DM) and Psoriasis vulgaris on methotrexate therapy.

On initial evaluation, the patient complained of severe abdominal pain, loose stools, vomiting, headache and chest pain. On physical examination, the patient was conscious and oriented. Vital signs were stable, and there was no evidence of pallor, icterus, or cyanosis. Extensive psoriatic plaques were noted on his upper extremities (as shown in Figure 1). Baseline hematological investigations

and a peripheral smear on Day 1 were entirely within normal limits. However, on Day 3 the patient's condition started to deteriorate.

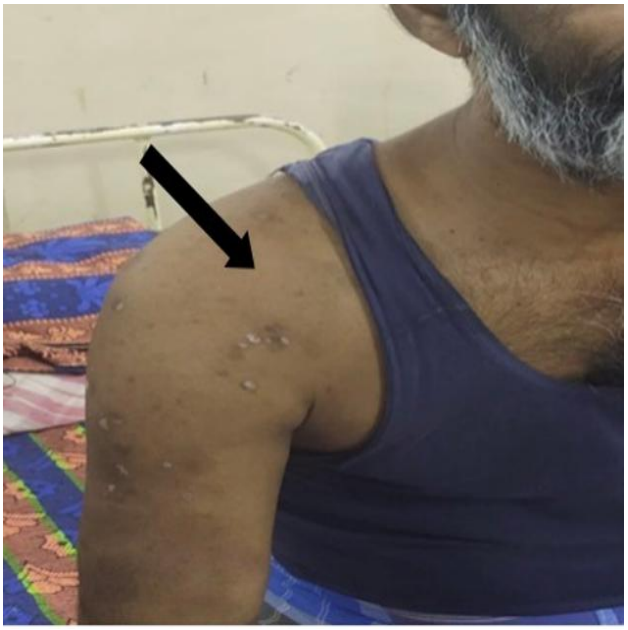

Figure 1: The patient showing extensive psoriatic plaques on upper extremities of the patient (arrow).

The patient developed a distinct icteric hue, visible cyanosis, and passed cola-colored urine (as shown in Figure 2). Serial lab parameters tracked over 12 days revealed a sharp drop in hemoglobin and hematocrit alongside deteriorating renal indices (Table 1).

Further biochemical investigations on Day 3 revealed a highly elevated serum lactate dehydrogenase (LDH) at 992 U/l and elevated serum creatine phosphokinase (CPK) at 387 U/l. Routine urinalysis confirmed 6–8 RBCs/hpf along with heavy hemoglobinuria. Sequential peripheral blood smears were done and recorded. Day 1 demonstrated normocytic normochromic red blood cells with neutrophilia and toxic granules. By Day 5, the smear demonstrated classic micro-spherocytes and fragmented red blood cells, confirming severe intravascular hemolysis. (as shown in Figure 3). Based on the chronological progression of the peripheral blood smears and serial biochemical parameters, a provisional diagnosis of Acute copper sulphate poisoning complicated by severe intravascular hemolysis, methemoglobinemia, secondary acute kidney injury (AKI), and acute hepatotoxicity, presenting in a background of psoriasis vulgaris on maintenance methotrexate therapy was made.

As part of initial treatment, the patient was aggressively hydrated, and methotrexate was immediately withheld. Gastric lavage via Ryle's tube and induced emesis were strictly avoided due to the corrosive nature of the compound. To treat the hemolysis and subsequent acute tubular necrosis, the patient was initiated on forced alkaline diuresis (FAD) to clear the toxic heme pigments. FAD was maintained continuously until the urine color completely normalized from its dark cola appearance back to a healthy straw color. Hemodialysis was planned but avoided as renal parameters stabilized under FAD. To counteract liver toxicity, oral liver supportives were initiated: Tablet Ursodeoxycholic Acid (UDCA) 300 mg thrice daily, Tablet Rifaximin 400 mg thrice daily, and Syrup Lactulose. The patient's clinical status and lab indices progressively recovered, and he was safely discharged on Day 12.

Figure 2: Clinical specimen of cola coloured urine passed by the patient.

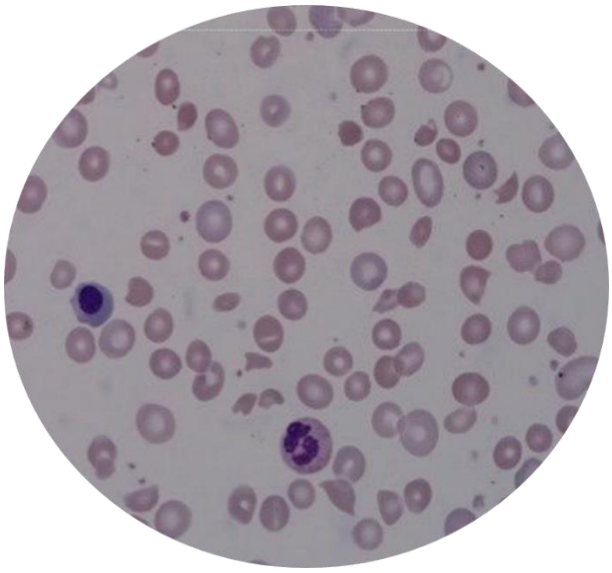

Figure 3: Peripheral smear done on day 5 showing- RBC: Micro spherocytes, Fragmented RBC, Nucleated RBC, Polychromatophilis, WBC: Reactive lymphocytes+ Relative neutrophilia, Suggestive of intravascular hemolysis.

Table 1: Serial lab parameters across admission

Lab parameters	Day 1	Day 3	Day 5	Day 7	Day 9	Day 11	Day 12
TC (10^9 /l)	10.7	9.3	7.1	7.3	8.7	12.3	—
HB (g/dl)	14.4	8.7	7.2	7.0	6.6	6.9	—
HCT (%)	42.7	30.8	21.3	21.7	21.3	23.1	—
Platelets (10^5/ ml)	3.96	2.64	2.38	2.27	2.09	3.17	—
Urea (mg/dl)	28	56	45	39	48	29	28
Creatinine (mg/dl)	0.9	1.2	1.2	1.0	1.4	0.8	0.9

DISCUSSION

Acute copper sulfate poisoning is a severe toxicological emergency characterized by rapid multi-organ dysfunction caused by extreme oxidative stress.[1] The lethal dose of ingested copper sulfate is generally reported to be between 10 and 20 grams.[1,2] Following ingestion, free copper ions rapidly absorb into the systemic circulation, where they directly damage cellular structures and inhibit protective antioxidant enzymes, particularly glucose-6-phosphate dehydrogenase (G6PD) and glutathione reductase.[2]

Our case is novel because the patient developed toxicity after ingesting only one gram. The unexpectedly severe clinical course observed in our patient raises the possibility that pre-existing alterations in copper metabolism associated with psoriasis, together with methotrexate-related oxidative stress, may have amplified tissue injury. Although causality cannot be established from a single observation, this hypothesis is biologically plausible and warrants further investigation.

Psoriatic patients exhibit significantly higher baseline serum copper and ceruloplasmin levels compared to healthy controls.[3,4] Because psoriasis naturally upsets systemic copper homeostasis and maintains patients in a chronic state of heightened inflammation, the threshold for exogenous copper toxicity is dangerously lowered.[3]

Furthermore, MTX is independently known to induce severe hepatotoxicity and nephrotoxicity through the generation of reactive oxygen species (ROS), lipid peroxidation, and the depletion of cellular antioxidants.[5,6] When exogenous copper is introduced to this environment, it binds with MTX to form a highly cytotoxic methotrexate-copper complex. This synergistic interaction disrupts the mitochondrial processes in liver, promoting acute cellular injury and death far beyond what either agent would cause independently.[6]

Beyond the immediate gastrointestinal mucosal erosion, copper sulfate poisoning precipitates several profound systemic complications. Hematologically, massive intravascular hemolysis typically occurs within the first 12 to 24 hours due to the direct oxidative rupture of erythrocyte membranes.[7] Concurrently, copper ions oxidize the ferrous iron within hemoglobin into the ferric state, resulting in severe methemoglobinemia, cyanosis, and systemic hypoxia.[8] Hepatic injury manifests rapidly as centrilobular necrosis and jaundice, while acute kidney injury (AKI) develops due to direct heavy-metal toxicity on the proximal tubules, heme-cast precipitation from hemoglobinuria, and pre-renal hypovolemia from severe gastrointestinal fluid loss.[9] Rare systemic sequelae, such as severe neurological impairment, persistent hyperglycemia, and rhabdomyolysis, have also been documented in severe acute intoxications.[10]

Puri et al described a young female who developed intravascular hemolysis and acute renal failure following an intentional ingestion, which was successfully managed with oral penicillamine chelation.[9] Perestrelo et al documented a case of progressive AKI and liver cytolysis resulting from chronic environmental exposure.[1] More recently, a 2026 report detailed extreme complications-including seizures and neuro-glycemic impairment-following a massive 50-gram ingestion of copper sulfate.[10]

In contrast to these cases, which either involved chronic accumulation or massive single-dose ingestions, our patient experienced life-threatening hemolysis and hepatorenal failure from just 1 gram. Reporting this case is for us as physicians to understand that the standard toxicological thresholds for copper sulfate are to be kept in check for psoriatic patients on MTX, such patients must be kept on high clinical suspicion and prophylactic therapy must be started as soon as possible.[3,10]

Recent advances in the management of heavy metal and MTX-induced toxicities heavily emphasize aggressive, early intervention. While traditional protocols rely on hydration, avoidance of emetics, and chelators like penicillamine or dimercaprol, modern critical care approaches for severe, complex hemolysis now advocate for the early deployment of therapeutic plasma exchange (TPE) and continuous renal replacement therapy (CRRT).[8,9] Furthermore, recent experimental pharmacology emphasizes that targeted antioxidant therapies-such as infliximab or hesperidin - can significantly ameliorate MTX-induced hepatorenal apoptosis and mitochondrial dysfunction.[5,6] These findings suggest that incorporating antioxidant and biological support may be useful in patients with toxicological emergencies.

CONCLUSION

This case highlights a critical toxicological exception to established lethal dose thresholds for acute copper sulfate

poisoning. While the ingestion of 1 gram is typically considered sub-lethal, the presence of underlying psoriasis vulgaris and concurrent methotrexate therapy created a unique vulnerability. This combination of pre-existing systemic copper dysregulation and synergistic oxidative stress precipitated rapid multi-organ failure, characterized by explosive intravascular hemolysis, methemoglobinemia, acute hepatotoxicity, and secondary acute kidney injury.

Ultimately, this case serves as a vital clinical reminder for emergency physicians and toxicologists to maintain a high index of suspicion, anticipate delayed severe deterioration, and initiate early, aggressive interventions like forced alkaline diuresis alongside the immediate suspension of methotrexate to prevent irreversible end-organ damage.

ACKNOWLEDGEMENTS

The authors would like to sincerely thank the Department of General Medicine and the Department of Pathology for their support in the clinical evaluation, laboratory investigations, and management of this patient. The authors also acknowledge the healthcare professionals involved in the patient's care for their dedicated assistance. Finally, authors would like to express gratitude to the patient and his family for providing informed consent for the publication of this case and the accompanying clinical images.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Manivannan PR, Ramaswamy V, Soundarrajan S. Severe multiorgan toxicity following low-dose copper sulfate poisoning in a patient with Psoriasis vulgaris on methotrexate: a case report. Int J Res Med Sci 2026;14:4101-4.