

# A Case Report and Insights from the Successful Treatment of a Patient with Acute Copper Sulfate Pentahydrate Poisoning

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**How to cite this paper:** Li, C.T., Zhang, X.X., Qian, Z. and Long, A.Y. (2025) A Case Report and Insights from the Successful Treatment of a Patient with Acute Copper Sulfate Pentahydrate Poisoning. *Case Reports in Clinical Medicine*, **14**, 541-548. <https://doi.org/10.4236/crcm.2025.149069>

**Received:** August 1, 2025

**Accepted:** September 15, 2025

**Published:** September 18, 2025

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## Abstract

This article summarizes the key emergency nursing interventions for a case of acute copper sulfate pentahydrate poisoning, which included: Early comprehensive care for copper ion elimination, complication prevention and recognition, dynamic nutritional support psychological care and continuity of care. Through intensive monitoring and meticulous nursing, the patient achieved stable vital signs and was eventually discharged with significant clinical improvement.

## Keywords

Copper Sulfate, Emergency Medical Services, Nursing Care, Case Study

## 1. Background

Copper sulfate pentahydrate, an inorganic compound commonly known as blue vitriol or chalcantite, appears as blue triclinic crystals. Acute copper sulfate poisoning can irritate the gastric mucosa, leading to gastrointestinal symptoms. Additionally, it may cause hemolytic anemia as well as hepatic and renal impairment [1]. While toxic in excessive amounts, copper sulfate pentahydrate exhibits beneficial pharmacological effects when used in appropriate doses. It is used as an emetic agent in controlled medical settings [2]. It demonstrates choleric properties by stimulating bile secretion. It acts on the chemoreceptor trigger zone to induce vomiting. It enhances hepatobiliary function through direct stimulation. However, ingestion exceeding approximately 10.0 g of copper sulfate may prove fatal. Currently, reported cases of oral copper sulfate poisoning remain relatively scarce in the literature, with most instances involving either accidental ingestion or iatrogenic overdose during therapeutic emesis induction [3]. The primary toxic

mechanism involves:  $\text{Cu}^{2+}$  ions induce irreversible structural changes in cellular proteins, disruption of redox enzyme systems, impairment of electron transport chains, failure of nutrient transport mechanisms, loss of muscular contractility, structural disintegration of cellular components, and complete loss of cellular function. Early intervention with the following modalities is critical: Plasma Exchange: 1) rapid removal of copper-protein complexes, effective reduction of circulating toxic metabolites. 2) Continuous Renal Replacement Therapy (CRRT): sustained clearance of free copper ions, maintenance of electrolyte balance during detoxification. 3) Clinical Outcomes: significant reduction of copper burden, prophylaxis against multi-organ dysfunction [4] [5].

Domestic literature demonstrates a notable paucity of case reports on acute copper sulfate pentahydrate poisoning, resulting in limited eferable nursing protocols. Our emergency department treated a representative case in February 2024, with the following clinical course.

## 2. Clinical Data

### 2.1. General Information

Patient information: male, 15 years old, admitted on February 21, 2024. The patient and family reported that approximately one hour prior to admission, after a verbal argument with family members, the patient intentionally ingested approximately 1.5 grams of copper sulfate. Ten minutes later, the patient experienced nausea and vomited once, with the vomitus being blue watery fluid, accompanied by chest tightness. No dizziness, headache, blurred vision, abdominal pain, or abdominal distension was reported. Emergency services (120) were called, and the patient was sent to our hospital for treatment. The patient has no history of similar episodes in the past, and no history of hypertension or diabetes.

### 2.2. Physical Examination

T 37.2°C, HR 116 bpm, RR 24/min, BP 118/72 mmHg. General Status: Conscious with normal mental status, no jaundice observed in skin/mucous membranes. Neurological Examination Pupils: Bilateral light reflexes intact. Extremities: No cyanosis in lips or fingertips. Motor function: Symmetrical muscle strength/tone. Pathological signs: Negative Babinski sign bilaterally. Meningeal signs: Absent. Cardiopulmonary Assessment. Lungs: Clear percussion notes, normal breath sounds, absence of rales or murmurs. Abdominal Examination: Abdomen: Flat, soft; Bowel sounds: 5/min. Tenderness: No rebound/guarding, negative Murphy's sign, no McBurney's point tenderness.

### 2.3. Laboratory Findings

Coagulation/Hematology D-dimer: 0.33 mg/L (normal range: 0 - 0.5 mg/L) NT-pro BNP 15 ng/L (normal range: <125 ng/L). Cardiac Markers Point-of-care troponin Normal. Electrolytes Potassium 3.33 mmol/L (L), Sodium 141.9 mmol/L, Chloride: 102 mmol/L, Ionized Calcium 1.22 mmol/L (L). Arterial Blood Gas

Analysis: pH 7.399, p O<sub>2</sub> 91.8 mmHg, p CO<sub>2</sub> 32.8 mmHg, Oxygenation Index 278.2%, Base Excess 3.6 mmol/L, HCO<sub>3</sub><sup>-</sup> 19.8 mmol/L, Lactate 3.26 mmol/L. Complete Blood Count (CBC) Results: White Blood Cells (WBC) 13.95 × 10<sup>9</sup>/L, Neutrophils % (NEUT %) 78.20%, Lymphocytes % (LYMPH %) 14.60%, Neutrophils (NEUT) 10.91 × 10<sup>9</sup>/L, Monocytes (MONO) 0.85 × 10<sup>9</sup>/L, RBC Distribution Width-SD (RDW-SD) 37.5 fL. Emergency Biochemical Profile Results.

Protein Metabolism Markers: Total Protein (TP) 85.7 g/L, Globulin (GLOB): 37.4 g/L, Albumin/Globulin Ratio (A/G): 1.29. Electrolytes & Acid-Base Balance: Potassium (K<sup>+</sup>) 3.19 mmol/L, Sodium (Na<sup>+</sup>): 136.7 mmol/L, Carbon Dioxide (CO<sub>2</sub>): 21.0 mmol/L (L). Special Tests: Uric Acid (UA): 727 μmol/L. Ceruloplasmin: 0.17 g/L (Normal range: 0.15 - 0.30). Other Examinations: Urinalysis Normal Infection Panel Negative, Function: Within normal limits.

## 2.4. Therapeutic Course and Clinical Outcomes

Based on the patient's medical history, physical examination findings, and auxiliary investigations, a diagnosis of acute copper sulfate pentahydrate poisoning was established. Therapeutic Protocol: Plasma Exchange Performed: February 22, 2024 (01:55 - 02:55), conducted after informed consent from family members. Procedure duration: 60 minutes. Intraoperative Monitoring. Vital signs: Heart rate 118 bpm, blood pressure 126/74 mmHg, oxygen saturation: 96%. Subjective reports: No discomfort. Administered 2000 ml of Type O Rh(D) positive frozen plasma and 500 ml of hydroxyethyl starch sodium chloride injection as prescribed. The transfusion proceeded smoothly without adverse reactions such as chills or fever and was completed safely. On February 22, 2024, at 05:42, bedside CRRT (Continuous Renal Replacement Therapy) was initiated due to clinical necessity. The treatment involved priming and rinsing the filter and circuit with 4000 ml of replacement fluid prepared with 3.8 ml of heparin, followed by anticoagulation therapy using citrate at a rate of 25 ml/h. The puncture site shows no signs of bleeding, exudate, hematoma, or bruising. The patient's current vital signs are as follows: SpO<sub>2</sub> 98%, HR 93 bpm, RR 19 breaths/min, BP 133/77 mmHg. As per medical orders, Arterial Blood Gas (ABG) analysis is being monitored dynamically, and the patient's vital signs and clinical condition are under close observation. At 10:00 on February 24, 2024, the Transmembrane Pressure (TMP) and filter pressure gradually increased, triggering frequent alarms. Suspecting intraluminal clotting, the CRRT treatment was discontinued after blood return. The total ultrafiltration volume was 0 ml. The double-lumen blood purification catheter was locked with diluted heparin. During the treatment, the patient remained in stable condition with no reported discomfort. Post-procedure vital signs were as follows: SpO<sub>2</sub> 94%, HR 62 bpm, RR 14 breaths/min, and BP 120/59 mmHg.

On February 25, 2024, post-rounds evaluation revealed normal liver and kidney function, and the femoral venous catheter was removed. As per physician orders, the patient continued to receive cardiac monitoring, oxygen therapy, anti-inflammatory treatment, and other supportive care before being transferred to the emer-

gency observation ward. After further monitoring with no reported discomfort, the patient showed significant improvement and was discharged in stable condition.

### **3. Nursing Care**

#### **3.1. Establish Intravenous Access & Maintain Effective Circulation**

Upon admission to the emergency resuscitation room, two venous access lines were promptly established. The preferred sites were relatively large, superficial veins that allowed for easy cannulation and rapid volume resuscitation. In special circumstances, such as severe trauma or vascular injury, central venous catheterization with appropriate infusion equipment was considered for vascular access establishment. During the puncture procedure, maintain aseptic technique to avoid complications such as infection [6]. Administer 100 ml of 0.9% sodium chloride intravenously to maintain vascular access, provide oxygen via nasal cannula at 2 - 3 ml/min, and perform continuous electrocardiographic monitoring and a routine electrocardiogram. The patient experienced vomiting, so the patient was assisted in turning the head to one side with the shoulders elevated to prevent reflux and aspiration, maintaining a patent airway. Arterial and venous blood samples were collected to assess liver and kidney function, electrolyte balance, and arterial blood gases.

#### **3.2. Comprehensive Copper Ion Removal Therapy**

##### **3.2.1. Plasma Exchange Combined with CRRT for Copper Reduction**

Early initiation of plasma exchange combined with CRRT is the cornerstone of acute copper sulfate poisoning management. This combined modality: Dual detoxification mechanism: Plasma exchange removes copper-protein complexes, and CRRT clears free copper ions. Hematologic stabilization: Replaces copper-damaged erythrocytes with functional RBCs. Organ protection: Prevents copper deposition in liver/renal/CNS tissues [7].

During treatment [8]: 1) Continuous monitoring of vital signs (temperature, pulse, etc.) and ECG surveillance for poisoning patients to promptly identify and manage any abnormalities. 2) Renal function monitoring with strict replacement fluid verification protocols; adjust the CRRT replacement fluid formula based on blood test results to ensure immediate preparation and use. 3) Secure catheter fixation to maintain patent tubing, monitor the insertion site for bleeding/leakage, and keep the dressing clean and dry. 4) Activity restrictions for patients with long-term catheterization: minimize movement of the punctured limb to prevent dislodgement, and provide routine oral care and basic hygiene. 5) Complication monitoring & prevention: Observe for hypotension, bleeding, or allergic reactions during CRRT; limit family visits to reduce infection risks.

##### **3.2.2. Adequate Chelation Therapy for Enhanced Copper Elimination**

Reducing copper ion absorption and promoting copper excretion are critical methods for treating acute copper sulfate poisoning. Following poisoning, patients should

receive a full course of sodium dimer cap to propane sulfonate (DMPS) at therapeutic doses to enhance copper elimination, typically administered over 3 - 4 treatment cycles [9]. DMPS has three main routes of administration: intravenous injection, oral, and intramuscular injection. The dosage regimens vary significantly: Intravenous injection: 5 - 10 mg/kg/dose, 2 - 3 times per week, Oral: 100 - 300 mg/dose, 2 - 3 times daily, Intramuscular injection: 5 mg/kg/dose. This route is less commonly used. Monitor patients for adverse reactions and therapeutic efficacy following administration. Sodium dimer cap to propane sulfonate may rarely induce allergic reactions and is contraindicated in patients with allergies or a history of hypersensitivity to sulfhydryl compounds. Additionally, strict control of the infusion rate is required, as excessively rapid administration can cause nausea, tachycardia, dizziness, lip numbness, or rashes [10]. If copper sulfate enters the eyes and causes possible discomfort such as eye pain or redness, immediately rinse the eyes and surrounding skin thoroughly with clean water or saline. Follow medical advice and use anti-inflammatory medications such as levofloxacin hydrochloride eye drops or lomefloxacin eye drops [11].

### 3.3. Close Monitoring of Urine Output

Acute renal failure is the leading cause of death in copper sulfate poisoning. Copper ions have an affinity for hydroxyl groups on cell membranes, leading to protein binding and subsequent obstruction of renal tubules by hemoglobin, resulting in Acute Kidney Injury (AKI) in poisoned patients [12].

Patients with copper sulfate poisoning must undergo continuous 24-hour monitoring of urine color and quality to detect early signs of AKI [13].

1) Urine Output Monitoring: Strictly observe the patient's urine volume (total output and frequency). Decreased urine output may indicate renal dysfunction; measure and record daily urine volume using a graduated measuring cup. Promptly report to the physician if persistent oliguria (low urine output) or anuria (no urine output) occurs. 2) Urine Characteristics Observation: Urine properties help assess the severity of renal impairment. Normal urine should be clear and yellow. Abnormal findings include darkened color, hematuria (blood in urine), or proteinuria (excess protein in urine) [14] [15].

### 3.4. Thorough Gastric Lavage to Promptly Remove Toxins

Gastric lavage should be performed under the guidance of medical professionals when patients exhibit severe symptoms or drug overdose. Prompt gastric lavage can effectively and rapidly remove harmful substances from the stomach. For such poisoned patients, immediately administer gastric lavage with 1% potassium ferrocyanide or sodium thiosulfate solution for detoxification, followed by oral administration of high-protein foods such as milk and egg white to compensate for the gastric protein damaged by copper ions, protect the patient's gastric mucosa, and reduce toxin absorption. The patient should be placed in an appropriate position with the head turned to one side. During gastric lavage, closely monitor the

color, volume, and characteristics of the lavage fluid for any signs of bleeding. Continuously observe the patient's consciousness, pupillary responses, vital signs, and abdominal signs. Abdominal distension may indicate excessive accumulation of irrigation fluid or poor drainage, while worsening abdominal pain may suggest potential gastric perforation [16].

### **3.5. Dynamic Nutritional Support Nursing**

Due to early gastrointestinal symptoms such as nausea and vomiting, the patient should be instructed to fast for 24 hours after gastric lavage to protect gastrointestinal function and ensure lavage effectiveness [17]. If no significant gastrointestinal symptoms occur, the patient may consume small amounts of warm water 4 - 6 hours post-lavage, followed by liquid or soft foods (e.g., rice water, noodles). On day 1, opt for low-residue soft foods (e.g., steamed buns, wontons); a normal diet can be resumed within 2 - 3 days to enhance nutrition. Emphasize frequent small meals to avoid overburdening the gastrointestinal tract [18] [19].

### **3.6. Skin Care**

Maintain aseptic techniques and instruct the patient to remain on bed rest. During shift handovers, monitor daily for scleral icterus and skin jaundice. Ensure skin hygiene by guiding family members to perform warm-water sponge baths for special body areas [20].

### **3.7. Psychological Care**

Most cases of acute copper sulfate poisoning are suicide-related. This patient, a ninth-grade student, attempted suicide by ingesting poison after a family argument. Being in an unfamiliar hospital environment may cause fear and agitation. Nurses should establish effective communication with both the patient and family members. Parents should regularly listen to their child's thoughts, spend quality time together, understand the child's psychological state, and promptly identify negative emotions [21].

### **3.8. Post-Discharge Continuing Care**

Continuing care involves implementing carefully designed nursing interventions after discharge to ensure comprehensive and continuous support throughout the treatment process. For this patient: 1) Schedule regular follow-up (once during the first week after discharge, then once a month, and then once every six months.); 2) Recommend a high-protein diet (eggs, soybeans, milk) while avoiding hard or irritating foods; 3) Encourage moderate exercise; 4) This patient has a strong interest in studying chemical drugs and conducting experiments, with particular knowledge about copper sulfate toxicity. Health education for parents should emphasize that the patient purchased copper sulfate independently online. Family members should monitor the patient's emotional state, understand psychological characteristics, and provide timely counseling [22] [23].

## 4. Discussion

Acute copper sulfate poisoning is relatively rare but progresses rapidly. This case did not develop severe complications like hemolytic anemia or kidney damage, as early plasma exchange therapy prevented multi-organ injury. For such patients, nurses should ensure early recognition and diagnosis to avoid delayed treatment. Implement immediate interventions (IV access, oxygen, gastric lavage, catharsis, liver/stomach protection) to reduce complications and mortality. Future practice should incorporate evidence-based approaches to refine nursing protocols.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

## References

- [1] Chuttani, H.K., Gupta, P.S., Gulati, S. and Gupta, D.N. (1965) Acute Copper Sulfate Poisoning. *The American Journal of Medicine*, **39**, 849-854. [https://doi.org/10.1016/0002-9343\(65\)90105-1](https://doi.org/10.1016/0002-9343(65)90105-1)
- [2] Franchitto, N., Gandia-Mailly, P., Georges, B., Galinier, A., Telmon, N., Ducassé, J.L., *et al.* (2008) Acute Copper Sulphate Poisoning: A Case Report and Literature Review. *Resuscitation*, **78**, 92-96. <https://doi.org/10.1016/j.resuscitation.2008.02.017>
- [3] Meena, M.C. and Bansal, M.K. (2014) Acute Copper Sulfate Poisoning: Case Report and Review of Literature. *Jasia Pacific Journal of Medical Toxicology*, **3**, 130-133.
- [4] Hernandez, G.N., Francis, A.J. and Hamid, P. (2024) Enhancing Survival in Septic Shock: A Systematic Review and Meta-Analysis of the Efficacy of Plasma Exchange Therapy. *Cureus*, **16**, e60947. <https://doi.org/10.7759/cureus.60947>
- [5] Inoue, T., Hanafusa, N., Kawaguchi, Y., Unagami, K., Hoshino, J., Ishida, H., *et al.* (2024) Safety and Efficacy of Selective Plasma Exchange vs. Conventional Plasma Exchange in Pretransplant Desensitization of Abo-Incompatible Kidney Transplantation: TH-PO816. *Journal of the American Society of Nephrology*, **35**, 10.1681. <https://doi.org/10.1681/asn.2024dxzvgfx6>
- [6] Pearce, L. (2024) Intravenous Infusions: What Do I Need to Know to Qualify? *Nursing Standard*, **39**, 38-39. <https://doi.org/10.7748/ns.39.12.38.s19>
- [7] Ba, J.H., Wu, B.Q., Wang, Y.H. and Shi, Y.F. (2019) Therapeutic Plasma Exchange and Continuous Renal Replacement Therapy for Severe Hyperthyroidism and Multi-Organ Failure: A Case Report. *World Journal of Clinical Cases*, **7**, 500-507.
- [8] Shen, L., Yang, J., Jin, X., Hou, L., Shang, S. and Zhang, Y. (2019) Based on Delphi Method and Analytic Hierarchy Process to Construct the Evaluation Index System of Nursing Simulation Teaching Quality. *Nurse Education Today*, **79**, 67-73. <https://doi.org/10.1016/j.nedt.2018.09.021>
- [9] Jones, M.M., Weaver, A.D. and Basinger, M.A. (1981) Characteristics of Chelate Antidotes for Acute Cu(II) Intoxication. *Journal of Inorganic and Nuclear Chemistry*, **43**, 2175-2181. [https://doi.org/10.1016/0022-1902\(81\)80576-3](https://doi.org/10.1016/0022-1902(81)80576-3)
- [10] Boosalis, M., McCall, J., Solem, L., Ahrenholz, D. and McClain, C. (1986) Serum Copper and Ceruloplasmin Levels and Urinary Copper Excretion in Thermal Injury. *The American Journal of Clinical Nutrition*, **44**, 899-906. <https://doi.org/10.1093/ajcn/44.6.899>
- [11] Foley, W., Middleton, W., Lawson, T., Erickson, S., Quiroz, F. and Macrander, S.

- (1989) Color Doppler Ultrasound Imaging of Lower-Extremity Venous Disease. *American Journal of Roentgenology*, **152**, 371-376. <https://doi.org/10.2214/ajr.152.2.371>
- [12] Oldenquist, G. (1999) Parenteral Copper Sulfate Poisoning Causing Acute Renal Failure. *Nephrology Dialysis Transplantation*, **14**, 441-443. <https://doi.org/10.1093/ndt/14.2.441>
- [13] Gunay, N., Yildirim, C., Karcioğlu, O., Gunay, N.E., Yılmaz, M., Usalan, C., *et al.* (2006) A Series of Patients in the Emergency Department Diagnosed with Copper Poisoning: Recognition Equals Treatment. *The Tohoku Journal of Experimental Medicine*, **209**, 243-248. <https://doi.org/10.1620/tjem.209.243>
- [14] Saravu, K., Jose, J., Bhat, M.N., Jimmy, B. and Shastry, B.A. (2007) Acute Ingestion of Copper Sulphate: A Review on Its Clinical Manifestations and Management. *Indian Journal of Critical Care Medicine*, **11**, 74-80. <https://doi.org/10.4103/0972-5229.33389>
- [15] Moussiegt, A., Ferreira, L., Aboab, J. and Silva, D. (2020) She Has the Blues: An Unusual Case of Copper Sulphate Intoxication. *European Journal of Case Reports in Internal*, **7**, Article ID: 001394.
- [16] Helal, N.E., Lashin, H.I., Nagy, A.A., Shama, M.A., Mostafa, T.A.H. and Wahdan, A.A. (2022) Potential Role of Paraffin Oil Gastric Lavage in Acute Aluminum Phosphide Poisoning: A Randomized Controlled Trial. *Environmental Science and Pollution Research*, **29**, 33844-33855. <https://doi.org/10.1007/s11356-021-17778-8>
- [17] Goodacre, S., Sampson, F.C., Sutton, A.J., Mason, S. and Morris, F. (2005) Variation in the Diagnostic Performance of D-Dimer for Suspected Deep Vein Thrombosis. *QJM: An International Journal of Medicine*, **98**, 513-527. <https://doi.org/10.1093/qjmed/hci085>
- [18] Kratochvíl, M., Klučka, J., Klabusayová, E., Musilová, T., Vafek, V., Skříšiovská, T., *et al.* (2022) Nutrition in Pediatric Intensive Care: A Narrative Review. *Children*, **9**, Article 1031. <https://doi.org/10.3390/children9071031>
- [19] Cook, F., Rodriguez, J.M. and McCaul, L.K. (2022) Malnutrition, Nutrition Support and Dietary Intervention: The Role of the Dietitian Supporting Patients with Head and Neck Cancer. *British Dental Journal*, **233**, 757-764. <https://doi.org/10.1038/s41415-022-5107-8>
- [20] Schwanitz, H.J., Riehl, U., Schlesinger, T., Bock, M., Skudlik, C. and Wulforth, B. (2003) Skin Care Management: Educational Aspects. *International Archives of Occupational and Environmental Health*, **76**, 374-381. <https://doi.org/10.1007/s00420-002-0428-z>
- [21] Koutsouleris, N., Hauser, T.U., Skvortsova, V. and De Choudhury, M. (2022) From Promise to Practice: Towards the Realisation of AI-Informed Mental Health Care. *The Lancet Digital Health*, **4**, e829-e840. [https://doi.org/10.1016/s2589-7500\(22\)00153-4](https://doi.org/10.1016/s2589-7500(22)00153-4)
- [22] Proctor, S.L., Wainwright, J.L. and Herschman, P.L. (2017) Patient Adherence to Multi-Component Continuing Care Discharge Plans. *Journal of Substance Abuse Treatment*, **80**, 52-58. <https://doi.org/10.1016/j.jsat.2017.07.003>
- [23] Goodman, H. (2016) Discharging Patients from Acute Care Hospitals. *Nursing Standard*, **30**, 49-60. <https://doi.org/10.7748/ns.30.24.49.s47>