# Copper Sulfate Drowning: A Case Report

**xiuqi zhu** ( zhuxiuqi2013@163.com )
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital
**Shengchao Wang**
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital
**honjuan Zheng**
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital
**Xuqing Ji**
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital
**Ji Li**
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital
**Jingmei He**
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital
**Li Zhu**
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital
**Xiao Xu**
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital
**Kun Chen**
Jinhua Hospital of Zhejiang University: Jinhua Municipal Central Hospital

**Case report**

**Keywords:** Copper poisoning, Drowning, Skin lesion, Conjunctival injury, Plasmapheresis

**Posted Date:** June 23rd, 2021

**DOI:** https://doi.org/10.21203/rs.3.rs-599040/v1

## Abstract

Cases of copper sulfate poisoning are rarely reported with a higher mortality rate. Herein, we reported a middle-aged man who had a copper sulfate drowning accident at work, resulting in airway damage, respiratory failure, intravascular hemolysis, hemolytic anemia, impaired liver function, and skin and conjunctival damage. After the rapid and effective treatments, including plasma exchange, irrigation under tracheoscopy, blood transfusion, liver and kidney protection and chelation therapy, the patient finally recovered and was discharged from hospital.


## Background

Copper is a component of some enzymes and acts as a cofactor involved in a variety of enzyme reactions. In medicine, copper sulfate has been used as an emetic, an antifungal agent and a deworming agent. Copper sulphate ingestion is a rare mode of poisoning except in India and Pakistan, where the cases are mainly suicidal in nature[1]. Due to its high fatality rate, it is necessarily to quickly recognize it.

In this article, we present a case of poisoning by drowning of copper sulfate a middle-aged man who was fell into a copper sulphate pool at work accidentally.

## Case Presentation

A health 50-year-old male fell into a copper sulfate pool at work in recycling plant with copper sulfate. The patient presented with nausea, bluish vomiting, dysphagia and abdominal discomfort immediately. He was conscious. Ten minutes later, the patient was sent to the emergency department of the local hospital. At that time, the patient was with blood pressure (BP) 137/85 mmHg, pulse (P) 98 per minute, respiratory rate (RR) 20 per minute, body temperature 36.7℃,percutaneous oxygen saturation ($SPO_2$) 98%. Multiple abrasions were visible on his face and back. The patient had wheezing, epigastric tenderness, corneal injury, but he had good visual acuity at first test. His laboratory tests were almost negative, except for elevated serum levels of white blood cells, lactic acid and troponin I (Table 1). The patient had received the initial treatment of milk gavage and dimercaprol for chelation of copper, dexamethasone, renal and hepatic support therapy, omeprazole and intravenous fluid infusion at the local hospital. Considered the emetic effect of copper, the patient was not given the treatment of gastric lavage. An hour after the accident, he was intubated for early definite airway preservation. Subsequently, he was given the tracheoscopy. The green sticky substance could be observed in the airway, bronchial mucosa was congested and edema and the mucosa were damaged lightly (Fig. 1).

Table 1
The results of the blood examination of the patient

| Blood chemical findings | local hospital(1h after poisoning) | local hospital (20h after poisoning) | emergency department in our hospital |
|---|---|---|---|
| WBC($\times10^{12}$/L) | 12 | 24.3 | 25.84 |
| Neu (%) | 44.3 | 92.0 | 93.8 |
| HB (mg/L) | 152 | 140 | 128 |
| RBC($\times10^{12}$/L) | 4.90 | 4.38 | 3.98 |
| PLT($\times10^{12}$/L) | 324 | 186 | 178 |
| TBIL(μmol/L ) | 9.6 | 54.1 | 76.1 |
| DBL(μmol/L) | 6.2 | 16.7 | 61 |
| ALT (U/L) | 13 | 6 | 14 |
| TP (g/L) | 70.6 | 56.4 | 58.6 |
| ALB (g/L) | 47.5 | 37.6 | 32.0 |
| GLB (g/L) | 23.1 | 18.8 | 26.6 |
| SCr (μmol/L) | 90 | 106 | 75.0 |
| pH | 7.350 | 7.260 | 7.30 |
| Lac (mmol/L) | 3.4 | 1.3 | 1.60 |
| $PCO_2$ (mmHg) | 38.5 | 39.7 | 38.6 |
| $PO_2$ (mmHg) | 77.0 | 137.0 | 61.4 |
| BE (mmol/L) | -4.2 | -9.1 | -6.7 |
| $PaO_2/FiO_2$ | 256.7 | 456.7 | 204.7 |
| $K^+$ (mmol/L) | 3.45 | 4.04 | 3.40 |
| $Na^+$ (mmol/L) | 149 | 152 | 143 |
| CL (mmol/L) | 107 | 110 | 126 |
| AST (U/L) | / | 69 | 83 |
| TropI (ng/mL) | / | 2.792 | 2.32 |

On the second day, the patient was transferred to our hospital. On admission, the patient was tubed and sedated with $SPO_2$ 100% (the oxygen concentration was 35%), T 37.9℃, P 95 per minute, RR 16 per minute, BP 133/84 mmHg. The conjunctiva was red and the cornea was damaged lightly. He had skin lesions on his face and back (Fig. 2). Chest radiograph showed pneumonia and electrocardiograph (ECG) was negative (Table 1, 2). On the second day of hospitalization, he was treated with plasmapheresis for three times (Fig. 3). Besides, the specific therapy with a chelate succinate (dimercaprol) in a dose of 375mg every 12 hours was started immediately and lasted for 10 days. Symptomatic treatment included mechanical ventilation, cefotaxime anti-infection therapy defames thane frantic-stress, renal and hepatic support therapy, intravenous fluid infusion, and omeprazole which was given to protect the gastrointestinal mucosa. Because the patient had, After the ophthalmological consultation, we gave calf's blood deproteinized extract ophthalmic gel in order to attenuate the corneal injury.

Table 2
Blood examination at hospitalization test results

| Date | 3.13 | 3.14 | 3.15 | 3.16 | 3.17 | 3.18 | 3.19 | 3.26 | 4.2 | 4.9 | 4.18 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Hospital stay | 1day | 2days | 3days | 4days | 5days | 6days | 7days | 14days | 21days | 28days | 37days |
| WBC(×1012/L) | 25.84 | 24.63 | 19.49 | 20.21 | 17.19 | 12.70 | 8.15 | 19.53 | 6.25 | 9.02 | 12.16 |
| Neu (%) | 93.8 | 93.1 | 90.7 | 88.4 | 86.3 | 81.1 | 85.8 | 95.4 | 80.1 | 81.0 | 78.6 |
| HB (mg/L) | 128 | 114 | 69 | 62 | 58 | 86 | 81 | 79 | 77 | 79 | 86 |
| RBC(×1012/L) | 3.98 | 3.61 | 2.08 | 1.85 | 1.77 | 2.66 | 2.58 | 2.50 | 2.49 | 2.58 | 2.86 |
| CRP(mg/L) | 174 | 119.3 | 85 | 53.4 | 29 | 104 | ▯200 | 192.9 | 115.7 | 31.9 | 14.7 |
| PCT(ng/mL) | 1.586 | 0.825 | 0.333 | 0.238 | 0.217 | 0.213 | 2.591 | 4.226 | 0.535 | 0.195 | 0.109 |
| HCT (%) | 40.2 | 37.2 | 21.5 | 18.7 | 17.2 | 24.7 | 24.4 | 23.9 | 24.0 | 24.7 | 26.4 |
| PLT(×1012/L) | 178 | 188 | 165 | 136 | 118 | 104 | 82 | 145 | 272 | 458 | 297 |
| TBIL(μmol/L) | 76.1 | 89.0 | 116.5 | 101.2 | 97.4 | 99.3 | 143.7 | 94.3 | 46.1 | 24.0 | 15.7 |
| DBL(μmol/L) | 0 | 21.8 | 11.9 | 18.3 | 23.2 | 36.3 | 85 | 41.9 | 3.8 | 0 | 0 |
| ALT (U/L) | 14.0 | 9.5 | 15 | 17.0 | 19.0 | 31.0 | 26.0 | 41.0 | 66.0 | 54.0 | 48 |
| TP (g/L) | 58.6 | 49.9 | 50.6 | 48.6 | 48.8 | 49.0 | 43.6 | 47.9 | 71.6 | 75.0 | 70.6 |
| ALB (g/L) | 32.0 | 32.1 | 27.1 | 25.8 | 25.8 | 26.2 | 22.5 | 20.7 | 32.8 | 34.1 | 35 |
| GLB (g/L) | 26.6 | 17.8 | 23.5 | 22.8 | 23.0 | 22.8 | 11.1 | 27.2 | 38.8 | 30.9 | 35.6 |
| SCr(μmol/L) | 75.0 | 86.0 | 76.6 | 63.4 | 66.8 | 59.0 | 58.2 | 95.3 | 104.7 | 81.4 | 71.2 |
| pH | 7.3 | 7.32 | 7.39 | 7.46 | 7.48 | 7.48 | 7.53 | 7.49 | 7.501 | 7.424 | 7.460 |
| Lac(mmol/L) | 1.60 | 0.90 | 1.40 | 1.60 | 2.50 | 2.40 | 2.00 | 1.90 | 1.30 | 0.6 | 1.10 |
| PCO2 (mmHg) | 38.60 | 44.00 | 46.00 | 43.80 | 39.10 | 38.1 | 34.10 | 37.00 | 33.10 | 40.40 | 33.60 |
| PO2 (mmHg) | 61.40 | 81.70 | 102.00 | 127.00 | 62.00 | 55.80 | 55.70 | 58.80 | 72.60 | 105.0 | 102.3 |
| BE(mmol/L) | -6.90 | -3.30 | 2.20 | 6.60 | 5.00 | 4.30 | 6.00 | 4.70 | 2.90 | 2.00 | 0.2 |
| PaO2/FiO2 | 204.7 | 233.4 | 185.5 | 254.0 | 137.8 | 93.0 | 55.7 | 117.6 | 242.0 | 350.0 | 341.0 |
| K(mmol/L) | 3.40 | 3.60 | 3.40 | 3.2 | 3.80 | 3.90 | 3.70 | 4.20 | 4.20 | 3.70 | 3.60 |
| Na(mmol/L) | 143.0 | 143.0 | 151.0 | 150.0 | 149.0 | 148.0 | 138.0 | 133.0 | 133.0 | 138.0 | 137.0 |
| CL(mmol/L) | 126.0 | 123.0 | 120.0 | 117.0 | 117.0 | 103.0 | 108.0 | 104.0 | 105.0 | 103.0 | 105.0 |
| AST(U/L) | 83.0 | 65.0 | 40.0 | 31.0 | 35.0 | 45.0 | 26.0 | 21.0 | 48.0 | 33.0 | 27.0 |
| TropI (ng/mL) | 2.320 | 1.080 | 0.613 | 0.266 | 0.215 | 0.146 | 0.094 | 0.882 | 0.043 | < 0.012 | < 0.012 |
| CU (μmol/L) | 44.9 | / | / | / | 16.4 | / | / | / | / | 43.6 | / |
| CP (g/L) | 0.25 | / | / | / | 0.19 | / | / | / | / | / | / |

After admission, troponin I decreased gradually. However, it suddenly increased to 11.1 ng/mL on the 11th day of admission. The patient did not complain of chest pain, and the repeated ECG did not show signs of myocardial infarction. On the next day, this level dropped again rapidly. (Fig. 4)

The third day after admission (4 days after poisoning), the hemoglobin decreased significantly, we believed that the patient developed into intravascular hemolysis. Reductive glutathione infusion was given to stabilize erythrocyte membrane, blood

transfusion improved the hemolytic anemia. The levels of blood transfusion and hemoglobin were shown in **Table 3**.

The patient has been feverish since admission, Chest computed tomography (CT) showed a severe infection in the right lung (Fig. 5). The use of cefotaxime is to fight infection and inflammation. However, the patient still had fever. In parallel, the decline in CRP, PCT and leukocyte was not obvious decrees changed the antibiotics to piperacillin and tazobactam. The 8th day following admission, blood culture indicated *Escherichia coli* (*E. coli*). Chest CT revealed pneumonia and pneumothorax., and a thorax puncture was performed to drain gas and purulent fluid. Subsequently, the antibiotics was changed to meropenem.

The 19th day after admission (20 days after poisoning), the peak body temperature of the patients decreased, PCT decreased from 17.109 ng/ mL to 0.535ng/ mL, WBC decreased from $25.84*10^9$ to $6.25*10^9$, CRP decreased from 174 mL /L to 115.7mg/L, and oxygenation index increased from 55.7 to 242.Re-examination of chest CT showed improved absorption of pulmonary inflammation., the patient was successfully extubated and then switched to high-flow nasal cannula oxygen therapy. One day later, the patient was changed to nasal catheter oxygen inhalation with oxygenation index = 263.3. Chest CT re-examination showed that the lesion of the right lung was significantly improved (Fig. 5).

After 5 weeks of treatment, the broken skin has healed after iodine volt disinfection and left scars on his face and back (Fig. 2). Re-examination of chest CT displayed markedly improved infection. In addition, the patient still had conjunctival redness and visible scarring of the cornea, but had good visual acuity. Finally, the patient was discharged in good clinical condition.

## Discussion

Copper is one of essential trace elements in the human body, but excessive copper is harmful for health. Copper sulfate poisoning is rare and little reported that and is mainly associated with suicide.

Ingestion of a copper substance (> dose of 1g) results in signs and symptoms of copper poisoning.[3] Physically, copper is absorbed from the intestine and is stored in the liver lysosome in the form of ceruloplasmin. Once the lysosome is destroyed and liver cells die, copper is released to blood circulation. There have also been reports of lung lesions caused by spraying vineyards with a mixture of copper sulfate and quicklime[4]. There once reported that copper sulphate had been inserted into the rectum in order to be pregnant[5]. But there have never been reports of copper sulfate drowning.

The initial manifestations of copper sulfate poisoning include nausea, vomiting, abdominal pain and other nonspecific clinical symptoms, followed by severe complications such as digestive tract mucosal injury, intravascular hemolysis, hemolytic anemia, methemoglobinemia, renal insufficiency, cerebral arterial thrombosis and even die[6].

Copper sulphate is an oxidizing agent that as the stimulating effects on mucosa membranes. The patient developed digestive tract symptoms, which consist of a metallic taste in the mouth, nausea, vomiting of blue-green stomach contents, diarrhea, upper abdominal pain, and gastrointestinal nausea, vomiting. After poisoning, the patient does not develop serious gastrointestinal complications, such as gastrorrhagia or perforation, under the protection of gastric mucosal protectant.

Since most of the absorbed copper is deposited in the liver after circulating through the portal vein, liver damage is often seen early in patients with copper sulfate poisoning[7] due to cell necrosis and obstruction[8]. However, the rise of total bilirubin approximately occurred on the 20th hour after poisoning, and reached the peak on the 7th day. After liver protection treatment, the liver enzyme of the patient was not very high, but the indexes of liver function completely returned to normal about four weeks later.

Renal insufficiency usually occurs 3 to 4 days after copper sulfate poisoning. and the causes of renal insufficiency were as follows[9]: (1) the direct toxic effects of copper sulfate; (2) cohemolytic anemia and rhabdomyolysis; (3) hypotension due to hypovolemia. The toxic effects of copper sulfate on muscles lead to rhabdomyolysis[10], which leads to renal insufficiency. Patients may benefit from alkalizing the urine or starting continuous renal replacement therapy (CRRT)as early as possible when patients have progressed to renal insufficiency. Fortunately, this patient did not develop renal damage and rhabdomyolysis. We wondered if this was related to the different routes of copper sulfate into the body, because the vast majority of cases reported in

the past were poisoning by oral copper sulfate, and our patient was drowning by copper sulfate. The specific reasons deserve further study.

Copper sulfate is a potent oxidant. In response to copper ions, oxygen-containing hemoglobin is oxidized from ferrous to iron, resulting in methemoglobinemia and reduced oxygen-binding capacity[8]. Copper sulfate can decrease the stability of erythrocyte membrane, which will lead to in vascular hemolysis in patients, generally occurring 12-24h after poisoning. At high concentrations, copper ions bind to the sulfhydryl group of erythrocyte membrane, reducing glutathione in erythrocyte and reducing the activity of glucose-6-phosphate dehydrogenase[2]. On the third day after poisoning, the patient experienced a significant decrease in hemoglobin, and we considered the occurrence of intravascular hemolysis. We treated the patient by using reduced glutathione and a large amount of blood transfusions (We had a total of six transfusions, 17 U of red blood cells). The hemoglobin was stable for 17 days after the poisoning. Although jaundice can be secondary to hepatic necrosis and hemolysis[8], The absence of jaundice may be related to the lack of severe liver impairment. This patient did not experience another decrease in hemoglobin after treatment with reduced glutathione and blood transfusion, but delayed hemolytic episodes have also been reported in the literature. This released copper is taken up by the erythrocytes and may account for the delayed secondary episode of haemolysis[11].

The skin may also be one of the routes through which copper (metals and/or aside salts) enters the body[12]. The patient accidentally fell into a copper sulphate pool and suffered skin damage, which is reeking Sun Park reported a case of exposing to a hot copper exposing patient had a deep dermal exposing cerebral infarct, which may have been due to hypercoagulability and anemia, caused by severe intravascular hemolysis[6]. But the skin damage in this case was mainly burns from the explosion. The case we report is a rare case of skin damage caused by direct exposure to copper sulfate. This patient suffered from skin lesions on the right face and back. Since there was no previous case or experience, we only gave iodophor dressing change. Although the patient's skin healed, it left a scar.

The most serious complication in this patient was pulmonary lesions caused by copper sulfate, resulting in respiratory failure. The patient's lungs developed rapidly and ferociously. At the local hospital, the patient showed no symptom of respiratory failure, such as decreased oxygenation, but the emergency physician administered an endotracheal intubation because of the rapid progression of the copper sulfate disease, which was confirmed by subsequent treatment. On the 8th day after the poisoning, the oxygenation could not be maintained and the patient needed pure oxygen ventilation on the ventilator. After antibiotic therapy (initially meropenem, later imipenem and cystatin), tracheoscopy sputum aspiration and irrigation, the tracheobronchial intubation was successfully removed 20 days after poisoning without reintubation, although the right lung was still severely inflamed. The mechanism underlying pulmonary toxicity induced by copper consists of oxidative effects resulting from the generation of reactive oxygen species (ROS)[13]. In addition, copper blocks antioxidant activities in epithelial cells by inhibiting the activity of catalysts and glutathione reductase sand and increasing the activity of glutathione peroxidase[14]. Three stages-intra-alveolar desquamation of macrophages, formation of predominantly histiocytic granulomas in the septa, and the healing of these lesions generally under the form of fibro-hyaline nodules very similar to those found in silicosis[4]. There have been few reports of lung lesions caused by copper sulfate, it has been reported that inhalation of copper sulfate could lead to pulmonary tuberculosis, known as “vineyard lung[4]”. Elisabetta Giudice[15]reported a dog exposed to copper sulfate powder leading to acute hypoxemic respiratory failure. In the case of the patient we reported, more copper sulfate choked into the airway during drowning, much more than the two cases previously reported. Therefore, we do not know whether this patient will suffer severe complications such as airway stenosis and lung cavity in the future. We told the patients that he needed long-term follow-up.

The patient suffered corneal injury due to direct contact between the copper sulfate and the cornea during the copper sulfate drowning.. After admission in our hospital, we had an ophthalmological consultation, and applied eye ointment to both eyes. The physical examination revealed scarring of the patient's cornea. Unfortunately, the patient refused toconduct a further eye examination.

The mortality rate from copper sulfate poisoning is high, and the leading cause of early death is shock.. Earl Schwartz[16] reported a case of refractory shock after copper sulfate poisoning. Although shock did not appear in this patient, it is still a matter of caution and concern. The cause of shock is still unknown. Some believe that early vomiting, diarrhea, and intravascular concern.

There may be other key mechanisms that we don't know about that merit further investigation. This deserves further investigation. Copper sulfate poisoning can also cause heart damage, the cause is unknown and may be related to pulmonary hypertension.

Our patient showed an increase in troponin 20 hours after poisoning, the peak value was 11.1ng/mL on day 11. but repeated ECG reexamination showed no abnormality, and there was no pulmonary hypertension. 22 days after poisoning, the patient's troponin was reduced to normal. Because there are no other symptoms of cardiac insufficiency, we did not give special treatment.

Symptomatic treatment is the common treatment of copper sulfate poisoning, including but not limited to dexamethasone frantic-stress, renal and hepatic support therapy, omeprazole which was given to protect the gastrointestinal mucosa,intravenous fluid infusion and special treatment of chelate succinate (dimercaprol). Chelation therapy was administered even though the concentration of copper ions remained within the normal range. Hemodialysis is not very helpful for the removal of copper[8]. But, early application of plasma replacement (plasmapheresis) is an effective way to block further tissue damage caused by cupric sulfate. The patient underwent a plasmapheresis immediately after admission this should be the basis for successful patient care. In addition, our repeated tracheoscopy rinsing reduced the amount of copper sulfate entering the lung, which provided a very important role for the successful removal of tracheal intubation.

# Conclusions

Copper sulfate poisoning is relatively rare, and copper sulfate poisoning from drowning is even rarer. In addition to the digestive tract symptoms, in vascular hemolysis, hemolytic anemia and other symptoms common to copper sulfate poisoning, the patient developed injuries to the eyes, skin, heart and lungs. Although he was better and discharged from hospital, the patient had respiratory failure (improved after treatment), and there were still corneal and skin scars at discharge. This case provides us with a new form of copper sulfate poisoning, which is rare but very dangerous.

# Declarations


### Acknowledgements

Not applicable.


### Authors' contributions

XQZ made contributions to the case analysis,and were major contributions in writing the manuscript.KC and XX made important contributions the conception and design of the research.JL,SCW and LZ were mainly responble for date collection and arrangged.JMH,XQJ and HJZ drafted the work.And all the authors read and approved the final manuscript.

### Authors' information

Not applicable.


### Funding

Not applicable.


### Availability of data and materials

Data sharing is not applicable to this article as no datasets were generated

or analyzed during the current study.

### Ethics approval and consent to participate

This study was approved by the ethics committee of Jinhua Hospital, Zhejiang University School of Medicine, (Zhejiang, China). Written informed consent was obtained from the patient. All clinical investigations were conducted in accordance with the

principles expressed in the Declaration of Helsinki.

### Consent for publication

Written consent for publication was obtained from the patient.

### Competing interests

The authors declare that they have no competing interests.

## References


1. Saravu K, Jose J, Bhat MN. Acute ingestion of copper sulphate: A review on its clinical manifestations and management. Indian J Crit Care Med Apr-Jun, 2007.
2. Du Y, Mou Y. The Role of Plasmapheresis in Treating Lethal Cupric Sulfate Poisoning. Am J Med Sci. 2019;357:338–42.
3. Oon S, Yap CH, Ihle BU. Acute copper toxicity following copper glycinate injection. Intern Med J. 2006;36:741–3.
4. Pimentel JC, Marques F. "Vineyard sprayer's lung": a new occupational disease. Thorax. 1969;24:678–88.
5. Moussiegt A, Ferreira L, Aboab J, et al. She Has The Blues: An Unusual Case of Copper Sulphate Intoxication. Eur J Case Rep Intern Med. 2020;7:001394.
6. Park KS, Kwon JH, Park SH, et al: Acute copper sulfate poisoning resulting from dermal absorption. Am J Ind Med, 2018.
7. Gamakaranage CS, Rodrigo C, Weerasinghe S, et al. Complications and management of acute copper sulphate poisoning; a case discussion. J Occup Med Toxicol. 2011;6:34.
8. Malik M, Mansur A. Copper sulphate poisoning and exchange transfusion. Saudi J Kidney Dis Transpl. 2011;22:1240–2.
9. Lubica C, Rudolf M, Jiri L. Acute Copper Sulphate Poisoning. J Coll Physicians Surg Pak. 2017;27:527–8.
10. Takeda T, Yukioka T, Shimazaki S. Cupric sulfate intoxication with rhabdomyolysis, treated with chelating agents and blood purification. Intern Med. 2000;39:253–5.
11. Franchitto N, Gandia-Mailly P, Georges B, et al. Acute copper sulphate poisoning: a case report and literature review. Resuscitation. 2008;78:92–6.
12. Bentur Y, Koren G, McGuigan M, et al. An unusual skin exposure to copper; clinical and pharmacokinetic evaluation. J Toxicol Clin Toxicol. 1988;26:371–80.
13. Cho YS, Moon JM, Jeong YH, et al. Successful extracorporeal life support in respiratory failure after copper sulphate ingestion. Natl Med J India. 2018;31:83–5.
14. Rahman I, Biswas SK, Kode A. Oxidant and antioxidant balance in the airways and airway diseases. Eur J Pharmacol. 2006;533:222–39.
15. Giudice E, Crino C, Lanzafame P, et al. Acute Hypoxemic Respiratory Failure With Hemoptysis in a Dog Exposed to Copper Sulfate Powder. Top Companion Anim Med. 2017;32:36–40.
16. Schwartz E, Schmidt E. Refractory shock secondary to copper sulfate ingestion. Ann Emerg Med. 1986;15:952–4.


## Figures

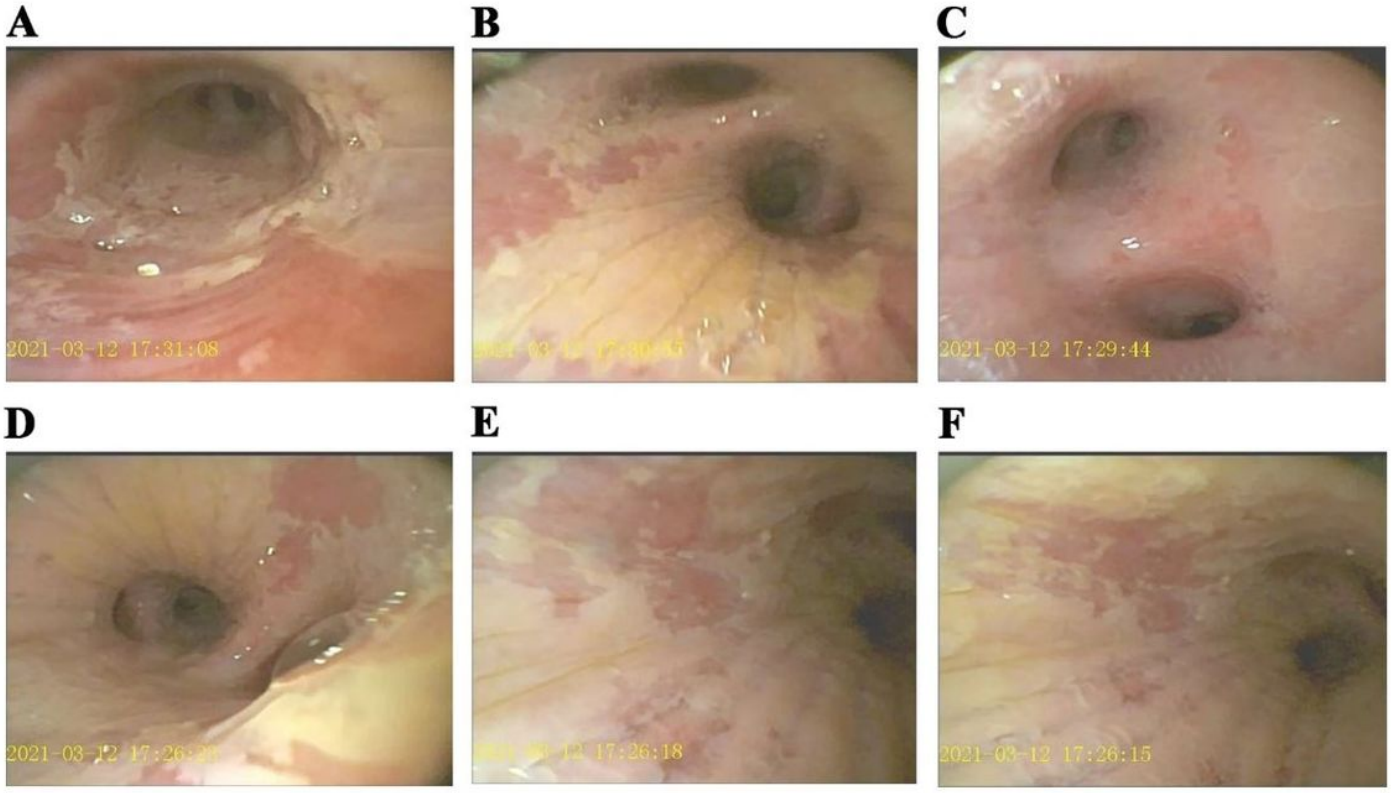


**Figure 1**

Tracheoscopy image of the patient

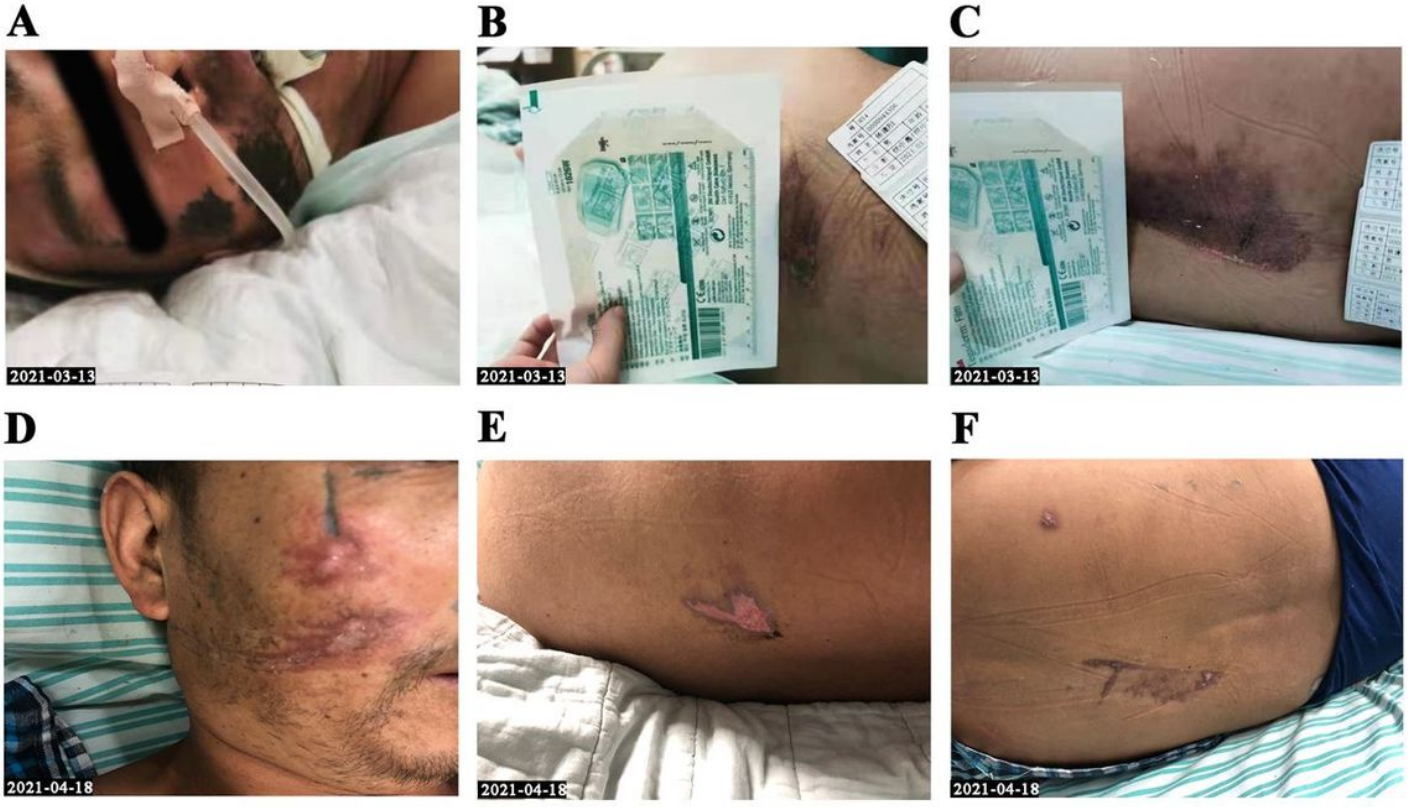

**Figure 2**

Skin lesion of patient

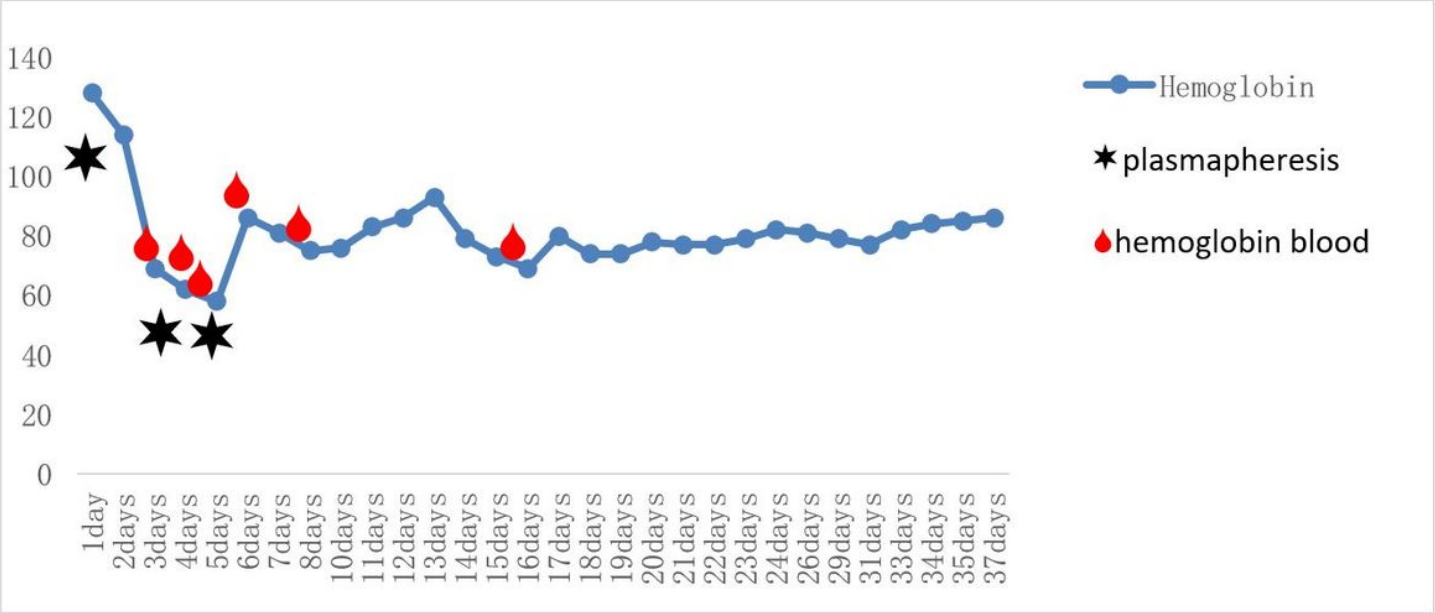


**Figure 3**

The detailed information of plasmapheresis method, hemoglobin and blood transfusion

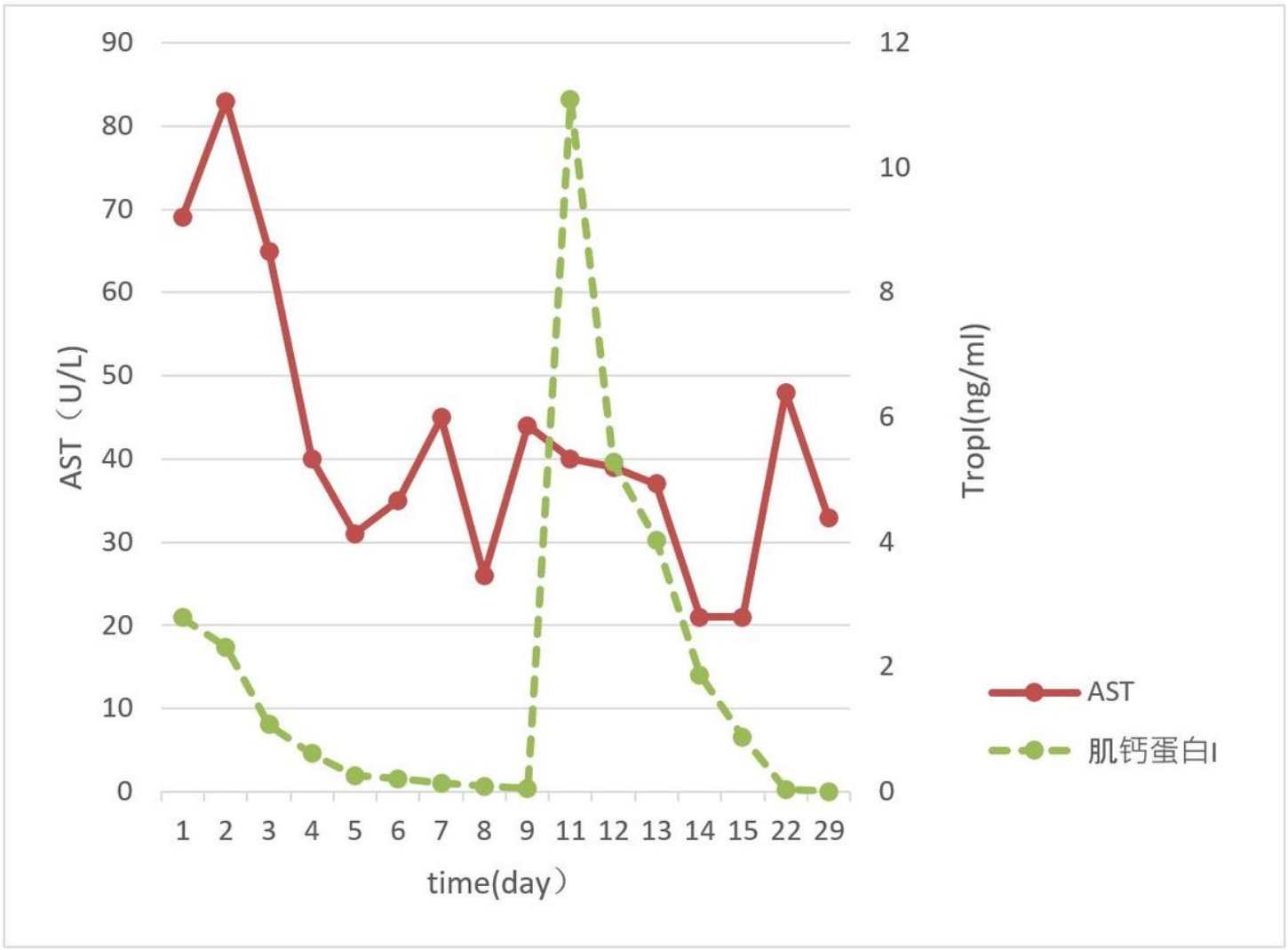


**Figure 4**

Details of AST and troponin I during hospitalization

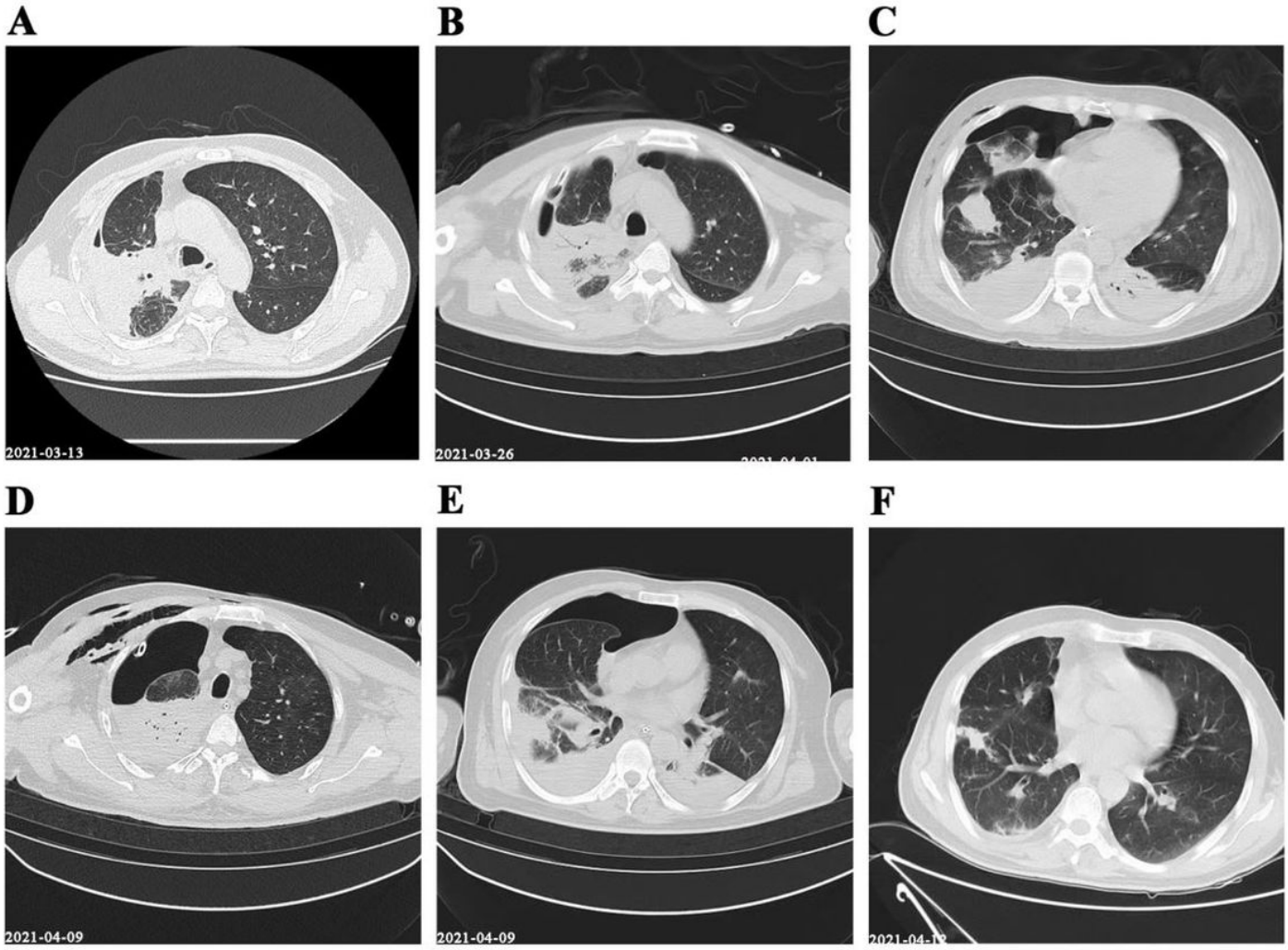


Figure 5

CT scan during hospitalization