

## A case of acute Copper Sulphate poisoning presenting with multiple complications

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### ABSTRACT

Copper sulphate (CUSO<sub>4</sub>) is a commonly available chemical in India. It is a fairly common cause of poisoning here. We report a case of a 23 year old female with CUSO<sub>4</sub> poisoning presenting with acute renal failure, pancreatitis, methemoglobinemia, hepatitis and severe hemolysis. However she responded to conservative management. The relevant literature about CUSO<sub>4</sub> poisoning and its pathophysiology are also discussed at length along with the treatment.

**Keywords :** poisoning, copper sulphate, pancreatitis, haemolysis

### INTRODUCTION

Copper sulphate (CuSO<sub>4</sub>) poisoning is quite common in India.<sup>1</sup> In some parts of the country, it is, in fact, the commonest poisoning identified.<sup>2</sup> Its widespread use in agriculture and industry makes it an easily available chemical. This compound, with powerful oxidizing potentials causes damage to multiple organs and systems.<sup>1</sup> It is often lethal if not promptly managed.

We present a case of CuSO<sub>4</sub> poisoning presenting with acute renal failure, pancreatitis, methemoglobinemia, hepatitis and severe hemolysis. Such constellation of findings in the same patient and also complete recovery only with conservative management is very rarely reported.

### CASE REPORT

A 23 year old woman presented with history of ingestion of about 8 grams of CuSO<sub>4</sub> powder with suicidal intentions two days back. Since she lived in a remote village, she came into first medical contact after about 18 hours; hence, gastric washing could not be done. However, as her condition continued to deteriorate, she was transferred to our tertiary care institution afterwards.

On examination, the patient was in visible distress with abdominal pain, decreased urine output (black in colour, Fig. 1), dyspnoea, central cyanosis (fig. 2) and severe nausea. Abdomen was mildly tender on superficial palpation only and peristaltic sounds were scanty. Her pulse was 130/min, blood pressure was 90/60 mm of Hg. Chest was clear on auscultation and consciousness was full.

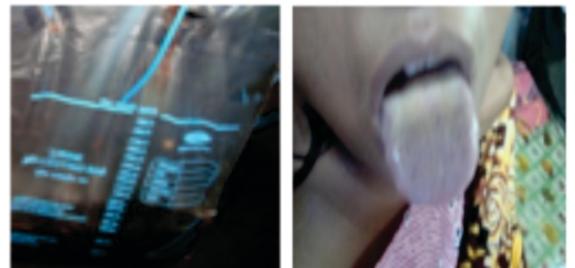


Fig.1

Fig.2

Laboratory examinations revealed hemoglobin of 8.9 gm/dl with total leukocyte count of 42600/cmm (N:89%). Platelet count was 20 000/cmm and ESR was 50 mm in first hour. Red cell indices were normal and reticulocyte count was 6%. Peripheral blood smear showed some fragmented cells and nucleated RBCs. Urine test showed blood 4+ in dipstix with 15-20 RBCs/HPF, signifying hemoglobinuria. Urea/creatinine was 140/2.2 mg/dl on Day 1,

which increased to 160/4.3 on Day 3. Also, the hemoglobin decreased to 6 gm/dl on Day 3 with reticulocyte count of 8%. The different blood parameters are temporally shown in table 1 below.

**Table 1:** Table showing the temporal profile of different blood tests in our patient

Parameter	Day 1	Day 3	Day 7	Day 10
Hemoglobin [gm/dl]	8.9	6	7	10.2
Urea [mg/dl]	140	160	99	52
Creatinine [mg/dl]	2.2	4.3	4	1.6
SGOT [IU/L]	51	58	106	77
SGPT [IU/L]	45	60	65	50
Urine blood	4+	4+	3+	1+
TLC [/cmm]	42600	38000	21600	16000

At first, the patient was treated with proton pump inhibitors for gastritis. But when the abdominal pain failed to resolve on Day 3, a serum amylase, lipase was done; 426/393 IU/L respectively (N: 60 IU/L for both). She was treated conservatively for pancreatitis. Ultrasonographic study of abdomen also showed an oedematous pancreas. CECT was not done due to the renal failure. The patient did not develop icterus clinically during the hospital stay. Peripheral saturation was 88%. Blood spectrometric analysis for methemoglobin could not be done due to cost; however as the central cyanosis resolved with blood transfusion and oxygen inhalation, it was thought to be due to methemoglobin only.

The patient was treated conservatively with blood transfusion, D-penicillamine and fluid support. Urine alkalisation was also done for the hemoglobinuria. Myoglobinuria was ruled out by serum CPK levels (126 IU/L; N: <150). After Day 5, the urine output started to increase and the colour of urine also became lighter. The symptoms of nausea or pain started to resolve.

Cyanosis disappeared completely by Day 8.

Thus, this patient had multiple severe complications of CuSO<sub>4</sub> toxicity. However, she responded to medical therapy alone and did not need invasive procedures like dialysis. The patient was discharged in clinically stable condition on Day 11.

## DISCUSSION

CuSO<sub>4</sub> is responsible for oxidant injury in various tissues. Also the copper ions can bind to and deactivate important tissue enzymes like glutathione reductase.<sup>1</sup> Hemolysis is a very common effect of this poisoning.<sup>3</sup> It can occur due either to RBC membrane damage or depletion of vital enzymes. Our patient showed hemolysis with massive hemoglobinuria, which stopped spontaneously after one week. But, sometimes, hemolysis can be very severe.<sup>4</sup> No definite treatment for this hemolysis is documented but methylene blue or steroids are sometimes reported to be beneficial.<sup>1,4</sup>

The earliest effect of CuSO<sub>4</sub> poisoning is on the gastrointestinal tract.<sup>4</sup> This often leads to shock and death in many cases. Pancreatitis is very rarely reported in this poisoning.<sup>4</sup> In fact, serum amylase levels may rise non-specifically in these cases and do not indicate pancreatitis. However, the rise of lipase in our patient was suggestive of pancreatitis, along with the ultrasonographic features. In a patient with CuSO<sub>4</sub> poisoning, severe abdominal pain may also indicate severe hemorrhagic gastroenteritis.<sup>4</sup>

Renal failure in CuSO<sub>4</sub> ingestion may be due to direct effect of copper on kidney tubules or secondary to hemolysis or dehydration.<sup>4</sup> Hemodialysis may be needed and recovery can be prolonged.<sup>5</sup> Acute kidney injury is reported in up to 60% of the cases in different case series.<sup>6</sup> Typically, hemoglobinuria is found, but

sometimes complete anuria can also occur. Sometimes myoglobinuria can occur in CuSO<sub>4</sub> poisoning. Thus in cases of dark coloured urine, a serum CPK test should also be done.<sup>1</sup>

Methemoglobinemia is a reported complication of this toxicity.<sup>1,6</sup> The oxidant copper ions are responsible for converting the ferrous form of haem to ferric form. If severe, this can be treated by methylene blue infusion or hyperbaric oxygen.<sup>6</sup>

Adrenal failure can occur sometimes due to toxic copper deposition.<sup>5</sup> So, in patient with CuSO<sub>4</sub> poisoning with refractory hypotension, iv steroid administration may be considered. But, hypotension may also occur due to hemorrhagic gastritis or severe hemolysis, thus, hemodynamic monitoring in these patients is of paramount importance.

Since CuSO<sub>4</sub> is not dialyzable, chelation therapy is the only mode of definitive therapy. In western world, DMPS is used.<sup>5</sup> However, since it is difficult to obtain DMPS in India, instead penicillamine is used.<sup>7</sup> The measurement of serum copper in CuSO<sub>4</sub> poisoning is debated.<sup>1,7</sup> While some authors advocate the serial measurement of serum copper to decide on the

chelation therapy, others do not support this line of action.<sup>7,8</sup> Also, in India, cost of tests is an important prohibitive factor.

A study from Bangladesh and India had reported jaundice in number of patients with CuSO<sub>4</sub> poisoning.<sup>9, 10</sup> However, jaundice is variable in CuSO<sub>4</sub> poisoning and is often due to hemolysis only. Our patient had elevations of liver enzymes, signifying some liver toxicity, but clinically there was no jaundice.

## CONCLUSION

CuSO<sub>4</sub> poisoning involves multiple organ. This case highlights the myriad effects of this chemical on the human body. While treating such a patient, a multidisciplinary team should be involved at an early stage. However, prevention is also needed and thus, the availability of this chemical should be restricted by law and surveillance.

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