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Clinical Features of Acute Copper Sulphate Poisoning

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Introduction

Copper Sulphate, $CuSO_4$, is an inorganic compound, a potent oxidant, with a long history of use especially as a fungicide, bactericide and also a killer for smaller animals like snails for agriculture. The heavy metal, Copper (Cu) is an essential mineral although in excess is very harmful [1,2]. Copper sulphate can be available as powder, crystals or liquids and can be exposed by skin contact, inhalation or by ingestion in the way of self harm or by accident. Copper causes damage to the cell membranes of the tissue cells making them swollen and causes cell death [2]. Red blood cells (causing haemolysis), myocytes (causing rhabdomyolysis) and hepatocytes (causing acute hepatitis) are the commonest tissues affected [3]. Once absorbed copper occur in plasma as ceruloplasmin (Cp) bound to protein and excreted largely in faeces. It has a biological half-life (BH) of 13-33 days. When in excess it may get deposited mainly in the liver [4].

Plasma level of copper and other reference ranges related to copper metabolism in human is as follows; this data is adapted from Medscape article on Copper by Joshua Sloan- https://emedicine.medscape.com/article/2087780-overview

Free serum copper: 1.6-2.4 µmol/L or 10-15 µg/dL

Total copper: 10-22 µmol/L or 63.7-140.12 µg/dL

Serum ceruloplasmin: 2.83-5.50 µmol/L or 18-35 µg/dL

24-hour urine copper 0.3-0.8 µmol or 20-50 µg

Liver copper 0.3-0.8 µmol/g of tissue or 20-50 µg/g of tissue

Copper Sulphate Poisoning (CSP) is rare, but a significant entity due to its higher risk of mortality even with smaller doses of ingestion. Features of toxicity can manifest even with a dosage of 1 g and dose of 10-20 g could even be lethal [5]. However clinical manifestations and complications likely depend on other factors like, time taken to seek medical attention, quality of medical care one would receive, especially at emergency care and patient's medical background. This article particularly concentrates on clinical manifestations and management of CSP in a more illustrated manner, to provide a guideline to the clinician.

Signs and Symptoms

There is multi-organ involvement with wide range of severity of each organ involved. Therefore clinical syndrome one would present will fall in to a large spectrum of possibilities.

1. Exposure related signs and symptoms

   Skin contact – irritation, allergy if the person is allergic to compound.

   Eye – irritation

   Inhalation – breathing difficulty

   Ingestion – nausea, vomiting and diarrhoea. Vomitus and diarrhoea can be greenish or bluish in colour [1].

2. Symptoms due to erosive gastropathy

   Corrosive injury to gastric mucosa – epigastric pain and tenderness, nausea, vomiting

   Mucosal erosions or ulcers causing upper gastro-intestinal bleeding – haematemesis and malaena

3. Intravascular haemolysis

   Oxidative damage to red cell membrane causes acute haemolysis – icterus, dark urine (Haemoglobinuria) and pallor.

4. Methaemoglobinaemia – cyanosis

   Absorbed Cu^{2+} ions oxidises Fe^{2+} to Fe^{3+} ions in Hb generating Methaemoglobin. These carry less Oxygen than normal Hb. Further, the hybrid Hb (ferri-ferro Hb)

shows higher affinity to oxygen, making it difficult to release at tissue level, making tissue hypoxia.

5. Hepatitis - Right hypochondriac and epigastric pain and tenderness, icterus, malaise, weakness, nausea and vomiting.

   Hepatitis is due to Hepatotoxicity caused by copper, which is directly delivered to liver after excessive portal absorption.

6. Pancreatitis (possibly) [3,5]
7. Acute kidney injury

   Pre-renal – dehydration (vomiting, diarrhoea and reduced fluid intake), bleeding (upper gastro-intestinal bleeding)

   Renal – Haemoglobinuria, Sepsis, Rhabdomyolysis and direct toxicity (to proximal tubules) by copper

8. Rhabdomyolysis
9. Cardiac involvement – arrhythmias
10. Nervous system involvement – seizures

Investigations

Investigations are carried out with the initial blood drawing in the emergency department and will be repeated at least daily or more frequently depending on patient's condition. Initial investigations one would request are FBC (full blood count), RBS (random blood sugar), BU (blood urea), S.Cr (serum creatinine), SE (serum electrolytes like, Na^+, K^+, Ca^{2+}), AST/ ALT (aspartate transaminase/alanine transaminase), Bilirubin levels, ALP (alkaline phosphatase), Serum Amylase, ECG (electrocardiography), Chest X-ray and Serum copper level (if available). Other baseline investigations may be indicated depending on the patient's clinical background. Clotting profile, blood picture, reticulocyte count, haptoglobin level (for IVH), serum proteins, serum lipase (for pancreatitis) and CRP (C-reactive protein – E.g.; for aspiration pneumonia) may also be needed initially. Lactate dehydrogenase (LDH) level (for haemolysis) and Creatinine Phosphokinase (CPK) level (for rhabdomyolysis) also be done subsequently.

Daily FBC, reticulocyte count and blood picture are needed to monitor IVH. Renal function tests and liver function tests also repeated to monitor the progression. CPK level and S.Cr level will help to monitor for Rhabdomyolysis [6-8].

Imaging studies include chest x-ray, ultrasonography of abdomen for liver and kidneys and computerized tomography (CT) of abdomen for acute pancreatitis. However, Contrast CT has the disadvantage of added renal insult when there in already multiple risks on kidneys.

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