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HAEMODIALYSIS IN ACUTE AMMONIUM DICHROMATE POISONING – A CASE REPORT

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ABSTRACT

Chromium compounds are used as oxidizing agents in leather industries. Acute dichromate poisoning is rare. Ingestion of potassium dichromate is often fatal with rapidly progressive renal and hepatic failure. We report a case of ammonium dichromate poisoning in a young male with a successful outcome. The patient had presented with abdominal pain and metabolic acidosis. On presentation there was no hepatic involvement. He was treated with prophylactic haemodialysis and the discussion is on the role of prophylactic haemodialysis in patients with chromium compounds poisoning.

Keywords: Haemodialysis, Acute Ammonium Dichromate Poisoning

INTRODUCTION

Chromium is a compound frequently encountered in leather and printing industries. Ingestion of these substances tends to have toxic effects on the liver. The reports of such ingestion are very sparse and hence this case highlights how our approach and the effect it had on the patient **[1]**.

CASE DISCUSSION

A 31-year-old male was brought with complaints of vomiting and abdominal pain

for one hour. Patient works in a printing press and gave history of consumption of about 15mg of ammonium dichromate mixed with alcohol about two hours ago. He gave history of 3 episodes of vomiting containing food particles, non-blood stained. Patient had a history of diffuse abdominal pain. Patient had no other symptoms. Patient is not a known case of Diabetes/ systemic Hypertension or any other co-morbidities. Patient also gave history of attempting suicide by cutting left wrist the previous day. On arrival patient was drowsy, responding to painful stimuli and afebrile. Patient was tachypnoeic. BP was 120/90mmHg, RR: 30/min, SpO2 was 96% at room air. Abdomen was diffusely tender and rest of the systems were normal.

Patient was admitted in Intensive care unit. ABG showed metabolic acidosis (pH: 7.23, PCo2: 38, Hco3: 15.3). Nephrology opinion was sought and patient was started on haemodialysis to prevent renal impairment. RFT was slightly deranged. In view of Chromium poisoning, he was given ascorbic acid as treatment. Liver supportive drugs were also given. LFT showed slight increased ALP, SGOT and GGT. S. Cr was 1.1 on arrival but went as high as 1.8 during the course of treatment. Patient received three cycles of haemodialysis and other supportive medication renal function returned to normal parameters by day 7. Patient was asymptomatic and discharged. He was followed up on day 7 and day 30. He continued to be asymptomatic and liver and kidney showed no signs of reduced function. LFTs were normal by Day 30.

DISCUSSION

Chromium is an essential nutrient for the human body as well as has multiple purposes in industry. Hexavalent form of Chromium is fatal even in small doses. Although poisoning is rare, similar case reports mention clinical features such as vomiting, abdominal pain and metabolic acidosis. These patients develop hepatic and renal failure. This is usually a late manifestation **[2]**.

Management aims at removal of the chromium as early as possible. Most cases are treated with ascorbic acid, other supportive measures and haemodialysis and liver transplantation based on severity of toxicity. In outpatient, we attempted to do haemodialysis even before systemic toxicity set in with an aim to clear out the chromium before it results in damage. Fortunately, the patient lived to tell the tale **[3]**.

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