Ammonium dichromate poisoning: A rare cause of acute kidney injury

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ABSTRACT

Ammonium dichromate is an inorganic compound frequently used in screen and color printing. Being a strong oxidizing agent, it causes oxygen free radical injury resulting in organ failure. We report a 25-year-old female who presented with acute kidney injury after consumption of ammonium dichromate. She was managed successfully with hemodialysis and supportive measures. This case is reported to highlight the toxicity of ammonium dichromate.

Key words: Acute kidney injury, ammonium dichromate poisoning

Introduction

Ammonium dichromate is an odorless, bright orange to red, crystalline powder used in pyrotechnics, fireworks, photography, and in dyes used for screen and color printing. Skin manifestations after industrial exposure of chromate salts are well-known, but little is known about systemic toxicity and its treatment.

Case Report

A 25-year-old female patient presented with history of vomiting and oliguria for 10 days. Patient had taken a handful of orange red crystals with suicidal intent 10 days earlier while working in a printing press [Figure 1]. She developed severe abdominal pain and vomiting immediately. She was taken to a local hospital where stomach wash was given and managed conservatively. Patient was discharged after 3 days. Patient continued to have abdominal pain, vomiting, and progressive oliguria.

On examination, patient was dehydrated with blood pressure of 130/80 mm of Hg and pulse of 98/min. Her investigations showed hemoglobin 7.2 g/dl, urea 650 mg/dl, creatinine 19 mg/dl, sodium 122 mEq/L, potassium 7.9 mEq/L, calcium 6.7 mg/dl, phosphorus 5.6 mg/dl, bilirubin 0.8 mg/dl, albumin 4.1 g/dl, total protein 7.2 g/dl, alanine transaminase 28 IU/l, aspartate transaminase 106 IU/l, alkaline phosphatase 66 IU/L, lactate dehydrogenase 124 IU/L, and creatine kinase 67 IU/L. Her urine analysis showed trace proteinuria, and few dark granular casts. Her arterial blood gas analysis showed severe metabolic acidosis with pH of 7.01, pCO_2 of 23 mm of Hg and bicarbonate of 6 mEq/L. Her electrocardiogram showed tall peaked T waves with prolongation of PR interval and QRS duration. Coagulation parameters were normal.

Patient was taken up for hemodialysis through right internal jugular catheter. She received 5 sessions of hemodialysis and other supportive measures. Chemical analysis of stomach contents was positive for ammonium ions. Patient later identified the empty bottle containing ammonium dichromate. Upper gastrointestinal endoscopy done 3 weeks after ingestion was normal. Patient urine output improved gradually and she was discharged with creatinine of 1.3 mg/dl. Her serum creatinine during the last follow-up was 1.1 mg/dl.

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Access this article online	
Quick Response Code:	Website: www.indianjnephrol.org
	DOI: 10.4103/0971-4065.133781

Figure 1: Ammonium dichromate crystals

Discussion

Hexavalent chromium salts have been recognized as occupational health hazards for more than two centuries. Chromium compounds can exist in a variety of valence states, of which chromium (III) appears to be the most stable and important form. Chromium (III) will form stable organic complexes with proteins, and amino acids.

Chromium salts are strong oxidants and cause free radical injury. Being a strong corrosive agent, dermal contact with chromium (VI) compounds can cause allergic dermatitis or sensitization. After ingestion, ammonium dichromate causes corrosive injury to the oral mucosa, esophagus, and stomach. The systemic manifestations include intravascular hemolysis, acute respiratory distress syndrome, toxic hepatitis, acute kidney injury, thrombocytopenia, and encephalopathy.[1] Lethal dose of chromium salt varies between 6 and 8 g in adults.[1] The primary cause of death due to acute chrome exposures is multi organ failure.[2]

Diagnosis mainly depends on history, clinical examination and direct identification of the compound. Chemical analysis of the stomach aspirate is often not useful as ammonium dichromate is highly dissociable compound. Various treatment modalities tried are exchange transfusion, ascorbic acid, N-acetylcysteine, chelation with dimercaprol and dialysis. Ascorbic acid has reducing property and in large doses accelerates the rate of reduction of chromium VI to chromium III leading to the formation of chromium protein complex, which is nontoxic and excreted in the urine. Though exact dose and duration of ascorbic acid is not known, large doses of ascorbic acid (1 g) has been successfully used to prevent renal failure. N-acetylcysteine has also been used successfully in few cases. Hemodialysis can effectively remove chromium (VI) though the reports on its efficacy in clinical settings are conflicting.[3]

There are very few case reports of ammonium dichromate poisoning in India.[2,4] Meert *et al.*, report the death of a child following a 1 g ingestion of ammonium dichromate.[3] Hassan reported a case of 24-year-old female who presented with multiorgan failure after ammonium dichromate ingestion. She was treated with hemodialysis and ascorbic acid (500 mg/day) after which she improved.[1]

The cause of renal failure in our patient is probably acute tubular necrosis secondary to both emesis related hypovolemia and direct tubular toxicity of chromium compound. Renal biopsy was not done as her urine output started improving during 2nd week of admission. Our patient presented 10 days after the ingestion with severe renal failure without features of any other organ involvement. Since she presented very late, we did not treat her with ascorbic acid and chelating agents.

Conclusion

Screen-printing and coloring work is becoming more and more popular in India and the availability of these compounds is much easier now. Hence, we can expect to encounter more such cases in future. The health care professionals should be aware of the manifestations of chromate poisoning and benefits of early management, especially with large doses of ascorbic acid.

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How to cite this article: Radhakrishnan H, Gopi M, Arumugam A. Ammonium dichromate poisoning: A rare cause of acute kidney injury. Indian J Nephrol 2014;24:380-1.

Source of Support: Nil, **Conflict of Interest:** None declared.