RESEARCH PAPER

Ammonium Dichromate Poisoning: A Case Report and Role of Dialysis and Ascorbic Acid in its Management

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ABSTRACT

Background: Ammonium dichromate is a highly toxic and carcinogenic compound utilized in printing, pyrotechnics, lithography and leather tanning industries, but toxic reports are rare. We report a 32-year-old male who deliberately ingested not less than 50 grams of ammonium dichromate mixed with water and alcohol. The efficacy of use of dialysis in such patients is unclear and hence we decided to estimate chromium levels in the dialysis discharge fluid and blood to quantify its efficacy. We discuss here the science of dialysis in such patients and the importance of ascorbic acid in managing ammonium dichromate poisoning.

Keywords: Ammonium dichromate poisoning, hexavalent chromium, oxidative stress, NAC, haemodialysis, metabolic acidosis, toxic ingestion

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Introduction:

Ammonium dichromate is an orange-red crystalline compound containing hexavalent chromium (Cr VI), used in photographic processing, printing, pyrotechnics and tanning industries. Its toxicity arises from its strong oxidizing potential and ability to generate reactive oxygen species (ROS) causing gastrointestinal corrosion, oxidative stress, and delayed organ failure. This case-report highlights the importance of early recognition, metabolic monitoring, and early aggressive management for favourable outcomes following ammonium dichromate poisoning.

Case Presentation:

A 32-year-old male, employed at a printing press and with a history of chronic alcohol consumption, was brought to the emergency department after deliberate ingestion of approximately 50 grams of ammonium dichromate dissolved in alcohol following a domestic altercation.

He initially presented to a local hospital within around 30 minutes with complaints of abdominal pain and recurrent vomiting. Gastric lavage was performed and referred to our care for further evaluation and treatment within an hour of consumption.

On presentation, patient was conscious, cooperative, BP 110/70 mmHg, Pulse 108 bpm, SaO2 98% in room air, afebrile, tachypneic with persistent epigastric tenderness. Abdominal examination revealed no guarding or rigidity. Neurological examination was unremarkable.

Initial blood work done within 2 hours, including renal and liver function tests, CBC, and electrolytes, were within normal limits. However, arterial blood gas (ABG) analysis revealed high anion gap metabolic acidosis with a pH 7.12 and HCO_3^- 12 mmol/L.

The patient was managed with IV fluids, IV ascorbic acid (1 gm stat followed by 1gm three times a day), IV N-acetylcysteine, and early hemodialysis for four hours daily for the next three days. Supportive care was

continued in the intensive care unit with continuous monitoring of renal, hepatic, and hematologic functions. Since there was an unanswered question of efficacy of haemodialysis in ammonium dichromate poisoning, serial assay of the dialysate discharge fluid was undertaken to estimate chromium loss during dialysis. A total of 120 liters of Dialysate was used in every session of dialysis and chromium excretion per session was then estimated in the discharge fluid (using inductively coupled plasma opitical emission spectrometry – ICP-OES) at the beginning, midway and then at the end of dialysis with measurement of Blood chromium levels by Atomic Absorption Spectrophotometry(AAS). (Table 1).

The patient's metabolic acidosis resolved over the next 48 hours with supportive care and dialysis. He remained clinically stable, with normal laboratory parameters and was discharged after five days of hospitalization. Follow up till 6 months was uneventful.

Discussion:

Chromium exists in various valence states and this state affects its absorption capability. In its natural form it exists as chromium (III), while the toxic form, chromium VI is anthropogenically produced as a result of activities like welding, combustion of coal and oil, cement works, waste inceration etc..

It is universally accepted that the lethal oral dose of dichromates and chromium trioxide ranges from 2.5 to 195 mg/kg[1]. For an adult male of 60 kgs this does not exceed 12 gms. Our patient had consumed around 50 gms and hence it was concerning and all steps were undertaken to neutralise and prevent complications related to the acute toxicity of ammonium dichromate.

Around 2-8% of Hexavalent chromium is readily absorbed through oral and inhalation routes. It is unstable, widely distributed into the body and gets converted to Chromium V, Chromium IV and then to a stable Chromium III by endogenous reducing agents like ascorbate and glutathione. It is primarily excreted in the urine though some amount of chromium is lost in faeces. However, the lethal documented dose for ammonium dichromate in the world literature significantly varies from 1 gm in a 2 year old child[2] to even 100 gms[3] being found not to contribute to death. In spite of consumption of any amount orally, all patients uniformly vomit almost immediately, thus effectively reducing the availability of ammonium dichromate for further absorption, making the safe-dose person- and intervention-specific. Though five case reports of ammonium dichromate is reported from India, only two children aged 2 years[3] and 8 years[4] with unknown quantity consumed died. In the 2 year old[3], brought in around 14 hours after suspected ingestion died within 16 hours of hospitalization without any dialysis or ascorbic acid treatment. While the details are unclear about the 8 year old[4], specific care potentially started only on day 3 after consumption of ammonium dichromate. Another 2 year old[5] brought within one hour of ingestion of an undetermined quantity treated promptly with oral ascorbic acid and IV N-acetyl cysteine (150 mg/kg) for one day alone recovered well without dialysis and discharged home on the third day. A 26 year old male[6] with a potential consumption of around 100 gms also survived well after being treated with IV ascorbic acid at 100mgs/ hr given for around 24 hours without need for dialysis and discharged by day 3.

The analysis of discharge fluid reveals that the loss of chromium during dialysis is concentration dependent, with maximum loss happening when the blood concentration was high and its loss decreasing with reducing blood concentration. However the total loss from the body does not exceed 2mg per session of 4 hours, though this seems to be much higher than 0.17 mgs per session of 3 hours as reported by Meert et al[2]. In the same case report[2] double volume exchange transfusion appeared better in reducing the serum levels by 3.77mgs but yet could not save the patient. Thus though dialysis does reduce the blood levels of chromium, it does not reduce it significantly to overcome its systemic effects nor does it do so within the time frame of initiation of the toxic effect as chromium gets widely distributed within the body including the RBCs, lungs, intestines, heart and kidneys, immediately after ingestion[2].

Hexavalent chromium toxicity results from intracellular ROS production, leading to mitochondrial injury, lipid peroxidation, DNA damage, and cellular apoptosis presenting with symptoms involving multiple systems of the body[2, 4]. Gastrointestinal symptoms usually appear early, while systemic complications like acute kidney injury related to either vomiting and or acute tubular toxicity, hepatic failure, intravascular haemolysis, Acute respiratory distress syndrome, results from direct toxicity and encephalopathy and death normally due to the multi organ failure. Metabolic acidosis is a critical early indicator of systemic toxicity, even in the absence

of abnormal renal function. Hence management involves conversion of toxic chromium (VI) to the non-toxic chromium (III) that subsequently gets excreted over a period of time sometimes even up to 24 weeks without systemic toxic effects[7].

In vitro studies have clearly shown the usefulness of ascorbic acid in plasma at 1000ppm in reducing chromium VI at concentrations of 5ppm to chromium III within 40 minutes[8] Thus the toxicity of ammonium dichromate is influenced by the timing of initiation of this treatment, the earlier the initiation, better the prognosis. This is effectively achieved by ascorbic acid – either oral or intravenous[9]. This management however will be useful only before the multi-organ damage begins, which could start as early as within few to 3 hours of ingestion[2], after which supportive care in the face of organ dysfunction potentially decides outcomes[10, 4]

Case reports of usefulness of haemodialysis in ammonium dichromate poisoning is reported[11]. However in this report both dialysis and ascorbic acid was and the patient potentially had a good prognosis in view of low toxin consumption as well. Hemodialysis under these circumstances corrects acid-base disturbances and assists in managing acute kidney injury. Thus dialysis is not the most appropriate method for managing acute ammonium dichromate toxicity, but is indicated for management of acute kidney injury till the kidney function improves.

Conclusion

This case report thus underscores the need for prompt diagnosis of ammonium dichromate poisoning, immediate decontamination to the extent possible, starting ascorbic acid at high dose around 100 -200 mgs/hour as soon as possible, with supportive care as required for favourable outcomes. In the absence of iv ascorbic acid, oral doses should be immediately initiated to reduce toxic Chromium VI to non-toxic Chromium III. Double volume exchange transfusions though better in reducing serum chromate levels than haemodialysis is not useful in reducing the toxic effects of chromium(VI) that starts within the first few hours post-consumption. Dialysis is indicated only when acute kidney injury manifests and is usually required till the return of renal function.

Timely diagnosis and immediate initiation of supportive and detoxification measures can significantly improve outcomes in ammonium dichromate poisoning. Clinicians must maintain a high index of suspicion and act swiftly, especially amongst people and their families working with easy access to ammonium dichromate.

Table 1 – the relative amount of chromium in dialysate discharge fluid and corresponding blood levels.

Sample day	discharge samples	microgm/L	Excretions per session (approx.) (micrograms)	Serum Levels (microgm/L)
D1	1	20.87		>75
	2	15.38		
	3	15.22	2093.11	
D2	1	14.46		>75
	2	11.67		
	3	9.3	1440.8	
D3				75
D4	1	5.41		15.22
	2	5.26		
	3	4.7	625.0	
D5	-	-		2.95
Total			4158.98	

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