



Acute Potassium Dichromate Poisoning Presenting With Myocarditis and Urethral Injuries: A Case Report

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Abstract

Acute potassium dichromate poisoning may be accidental or suicidal. The potassium dichromate is highly toxic and is lethal to humans in relatively smaller doses. There is no specific antidote to it and the treatment is symptomatic. Many experimental modalities of treatment have been tried with variable success. We report a case of 33 year old young man who upon ingestion of potassium dichromate, presented to us with myocarditis and severe urethral injuries along with multi-organ failure.

Keywords: Acute Potassium Dichromate Poisoning; Dichromate induced Myocardial injury; Dichromate induced Urethral Injury.

Introduction

Potassium dichromate is a cellular poison. Once ingested, it is rapidly absorbed and taken up by various tissues of the body where it generates reactive intermediates and oxygen free radicals. These metabolites damage various intracellular components in renal tubules, hepatocytes, RBCs and platelets which in turn results in hepatic

failure, renal failure, intravascular hemolysis & coagulopathy. Here we are reporting a case of 33 years old young man who had ingested more than 9gm of dichromate powder with suicidal intent. In our case, in addition to the renal and hepatic injury, the patient had developed myocarditis due to myocardial damage inflicted by this cellular

poison. Secondly, there was very severe urethral injury which again, although theoretically agreed to happen, hasn't been reported anywhere yet to the best of our knowledge.

Case Presentation: A young man of 33 years of age, employed in a screen printing enterprise, presented with complaints of not passing urine for more than 24 hours and inability to speak for 2 days. On taking history, patient's attendants told that he has consumed about two and half sachet of the dichromate powder 4 days back. He got the powder from his working place. After some family brawl, he consumed the powder in apparent effort to end his life in a rush of emotions. He developed pain abdomen, nausea and vomiting within minutes of consuming the powder and he told his family members about his ingestion of the powder and showed them the sachet (pic.1).



Pic. 1 showing one of the pouches of dichromate consumed by the victim

He was taken to some local clinic for treatment where he received gastric lavage (probably with normal saline) and given intravenous fluids along with some other medications in the form of injections not known to the attendants. Despite treatment, his vomiting didn't improve and he started having difficulty in speaking, so he was shifted to another hospital where he again received intravenous fluids and some injections. Here he developed jaundice, anuria, inability to speak (at presentation he could only move his lips but with no speech) and unstable blood pressure. So attendants got him referred to Govt. Medical College/Guru Nanak Dev Hospital, Amritsar.

On presentation, he was conscious, tried to communicate verbally but was not able to speak and followed commands sluggishly. He was having scleral icterus, abdomen mildly tender with non palpable bladder, bilateral crackles up to middle of chest on auscultation and heart sounds muffled with tachycardia but there was no bleeding manifestation yet. Pupils were dilated and non-reacting to light which probably was a manifestation of central action of chromates and his oro-pharyngeal mucosa was grossly inflamed and friable. His vitals were: PR - 110 beats/min, regular but feeble, BP - 88/50 mmHg, RR - 20/min. His hemoglobin was 12.9 gm/dl, total leukocyte count was 18,900/cmm, differential leukocyte count was P80 L10 E3 B2, Platelet count was 83,000/cmm. His blood sugar was 40 mg/dl, blood urea 245mg/dl, serum creatinine 17.5mg/dl, serum bilirubin (total) 4.1mg/dl, SGOT 356 IU/dl, SGPT 340 IU/dl, total serum proteins 5.7 and albumin 2.8, Prothrombin time index was 70%, serum Na^+ 139 and K^+ 5.7 meq/dl. ECG showed tachycardia with non-specific ST segment and T wave changes suggestive of acute myocardial injury. His CPK-MB levels came out to be 2569 IU/L and they further firmly pointed towards myocarditis. He had severe urethral injury. It was to the extent that even the pediatric sized Foley's catheter didn't pass due to inflamed and damaged urethral lining epithelia. A supra-pubic catheter had to be inserted as an emergent measure instead.

Patient developed shock within half hour of the admission. He was managed with high doses of hydrocortisone, crystalloids, antibiotics, pantoprazole, ionotropes and other supportive care but he rapidly deteriorated and succumbed to the widespread multi-systemic injury caused by the dichromate.

Discussion

Potassium dichromate, one of the many inorganic salts of chromium, is used as oxidizing agent in various laboratory and industrial applications like as reagent in analytical chemistry, as cleaning agent in glassware industry, leather tanning and wood treatment and in photography & screen printing. It is easily available and cheap chemical.

A 3.5gm sachet of dichromate powder costs just 5.0 rupees in India. Humans can be affected in the form of contact dermatitis or chronic ulcers from chronic exposure, or acutely because of accidental or suicidal ingestion, absorption from burnt/scalded skin and inhalation of chromate fumes.¹ Acute systemic injury following ingestion is usually lethal as the fatal dose is very small; 2-3gm of potassium dichromate powder usually leads to death.² Potassium dichromate, in its hexavalent form, is a cellular poison. Once ingested, it causes gastrointestinal injury first and then is rapidly absorbed and taken up by various tissues of the body where it generates reactive intermediates and oxygen free radicals that damage various intracellular components especially in renal tubules, hepatocytes, RBCs and platelets.³ This ultimately results in hepatic and renal failure and intravascular hemolysis & coagulopathy, the usual cause of death in dichromate poisoning. Acute Potassium dichromate poisoning is a recognized occupational hazard but its ingestion is uncommon which is usually fatal. Its ingestion with suicidal intent is even less common, but this is a toxin which may be more prevalent than previously thought because of its use in traditional medications and lowest reported lethal dose is as small as 0.1gm.⁴ Our patient consumed more than 9gm of the potassium dichromate powder which is nearly 3-4 times of the fatal dose which is 2-3gm. Hepatic and renal failure, intravascular hemolysis, coagulopathy and acidosis are usual cause of death but in our case it appears that renal failure and myocardial injury proved to be detrimental, other mentioned pathophysiologic mechanisms played lesser role. There is no specific antidote to the potassium dichromate poisoning but there are many treatment modalities suggested for the treatment. Hexavalent form of chromate is mainly responsible for its toxic effects whereas trivalent chromate less dangerous and more rapidly eliminated in urine, also its hexavalent form that crossed cell membranes and gets sequestered intracellularly whereas trivalent form not crosses cell membranes. Based on this observation oral ascorbic acid and gastric lavage with ascorbic acid has been tried in treatment of potassium dichromate poisoning as ascorbic acid fastens the conversion of hexavalent chromate to

trivalent chromate.⁵ Other treatment modalities like hemodialysis and charcoal hemoperfusion have been tried but not with much success.⁶ A case successfully treated with peritoneal dialysis has been reported.⁷ Liver transplantation has been successfully used in cases where hepatic failure is predominant feature of dichromate poisoning.^{8,9}

Conclusion

Potassium dichromate is a commonly used chemical agent in various industrial and lab applications. It is very poisonous chemical and any acute occupational or suicidal exposure usually proves fatal because of low lethal dose. So it is of paramount importance to institute treatment at the earliest, so as to attempt to eliminate its hexavalent form as early as possible because once it is sequestered intracellularly, it becomes impossible to remove this cellular toxin. It is important to educate the workers to reduce its occupational exposure and providing them appropriate protection. Similarly periodic assessment of psychosocial aspects of the persons working with this chemical is equally important to prevent suicidal attempts with this chemical.

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Conflicts of Interests

There are no conflicts of interests

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