Occupation-related chromium toxicity a rare cause of renal failure and rhabdomyolysis

Abstract

A 21-year-old gentleman, a worker in a mobile phone shop, was admitted with rhabdomyolysis, renal failure, and pulmonary edema requiring mechanical ventilation and hemodialysis. After extensive workup and ruling out other causes, heavy metal poisoning was considered. Investigations during the course of the hospital stay revealed chromium poisoning. With repeated hemodialysis, his parameters normalized and he was discharged home in a stable condition. Diagnosis of chromium toxicity needs high index of suspicion. A history of occupational exposure might offer a clue to diagnosis. With hemodialysis and supportive care, it is a potentially salvageable condition.

Key words: Chromium, renal failure, rhabdomyolysis

INTRODUCTION

Chromium, though a micronutrient naturally present, can lead to life threatening complications when ingested in high levels. It is a rare poison and is associated with life threatening complications involving various organ systems.

CASE DESCRIPTION

Mr. R, a 21-year-old gentleman from South India, who worked in a cellphone shop, was brought to the hospital with sudden onset breathlessness gradually worsening for the last 2 days. He also complained of generalized weakness and muscle pains. There was no premorbid illness or history of drug intake. On arrival to the hospital, he was found to have tachypnea, tachycardia, and was in hypotension. Chest examination revealed bilateral crepitations.

Cardiovascular system did not reveal any murmurs. Abdomen and central nervous system (CNS) examination did not reveal any abnormality. His oxygen saturation was low, and the arterial blood gas analysis (ABG) was done which revealed type I respiratory failure with severe metabolic acidosis and lactic acidosis [Table 1].

The patient was immediately intubated and given fluid resuscitation and shifted to the medical intensive care unit (ICU). His initial hemogram and biochemical investigations revealed leukocytosis, with acute renal failure and deranged liver function tests [Table 2].

Chest radiograph revealed bilateral lower zone haziness and electrocardiogram revealed sinus tachycardia. Cardiac enzymes and echocardiogram did not reveal any abnormality.

In the ICU, he was investigated for his oliguric renal failure. His creatinine phosphokinase (CPK) was high, which was suggestive of rhabdomyolysis. In view of the worsening renal function and oliguria with severe metabolic acidosis, he was dialysed after consultation with the nephrology team. He continued to require daily dialysis in the ICU. His ventilatory parameters improved gradually, and he was extubated on the 7th day of the admission and shifted to the ward on the 9th day.

He was worked up extensively for his renal failure and rhabdomyolysis, including a muscle biopsy which was inconclusive. After ruling out infectious and autoimmune causes, a heavy metal screen was requested.
The heavy metals were checked in the patients’ blood by inductively coupled plasma mass spectrometer (ICP-MS), which can accurately detect the concentration of heavy metals in the biological materials. The abovementioned test revealed high levels of chromium in the blood (338 microgram/L) (normal range <10 microgram/L). Hence, a diagnosis of chromium toxicity was made. The exact source of chromium was not clear from history. On further questioning, the patient mentioned that he works in a mobile phone shop and has been handling mobile phone batteries for more than 2 years checking the power by putting the wires on his mouth. He improved gradually in the ward and required a total of 13 dialysis. He was in the hospital for a duration of 27 days and was followed-up in the outpatient department [Table 3].

He was discharged home in a stable condition. On follow up, his repeat chromium levels were 5.8 microgram/L (normal range: <10 microgram/L) and renal function test were in the normal range. The patient was educated regarding the possible source of his exposure and the related health implications.

**DISCUSSION**

Chromium toxicity can occur as an occupational hazard among electroplating, tanning, and leather industry workers and welders.[3,4] The absorption could be due to ingestion, skin absorption, or inhalation. There are even case reports of transmucosal absorption, which can occur through nasal mucosa,[3] as well as dermal absorption leading to burn injuries.[4] Airborne exposure has also been reported among welders.[5] In addition, contamination of water sources and soil can lead to exposure.[5-7] There have been studies revealing that electronic equipment, such as mobile phones batteries, contain heavy metals such as chromium.[8] In our patient, the possible source could have been handling of mobile phone batteries and directly putting the electrodes in the mouth while checking for charge.

The dosage, rate, and route of exposure may determine the manifestations pertaining to the symptoms. The abovementioned toxicity can occur as acute poisoning or chronic poisoning. Acute poisoning occurs usually secondary to suicidal intent, accident, or due to spillage where there is exposure to large dose of toxin over a short period of time. Acute exposure should be suspected when patients present with sudden symptoms or rapid worsening. In our patient, we assumed that he had acute on chronic exposure in view of a rapid worsening.

The primary mechanism of toxicity is by oxidative stress.[8,9] There is increase in lipid peroxidation and decrease in plasma antioxidant capacity.[9] On long-term exposure, it can even cause DNA damage and mutation.[9,10]

Various organ systems including the kidneys and liver and bleeding manifestations leading to circulatory failure have been described with acute chromium poisoning.[11] Renal failure has been described in both acute as well as chronic exposure. There is preferential accumulation of chromium in the kidneys, as described in previous studies leading to tubular damage.[12-14] Our patient had muscle involvement in the form of rhabdomyolysis. Rhabdomyolysis in chromium poisoning is rarely reported.[15] In our patient, severe rhabdomyolysis could also have contributed to his renal failure in addition to direct renal toxicity. Our patient also had severe metabolic acidosis with hypotension and high lactate levels, suggesting tissue hypoxia and circulatory failure. His deranged liver function tests could have been due to ischemic hepatitis or due to direct liver injury caused by chromium itself.

Previous studies have revealed high mortality associated with severe toxicity.[11] The treatment is primarily supportive, enhancing elimination and preventing further exposure. Gastric decontamination and role of activated charcoal ingestion has not been studied in acute poisoning. It may be harmful due to the corrosive action of chromium.[3] Early administration of ascorbic acid has been tried in few cases; however, there is no definite evidence to prove benefit.[16] N-acetyl cysteine has been also tried due to its antioxidant
effects.[17] Plasmapheresis has been tried and was associated with good outcome.[19] Hemodialysis initiated early is associated with a good survival rate, as in our patient.[19,20] Though peritoneal dialysis can be tried, hemodialysis is more effective in chromium clearance.[21] Chromium levels were investigated in our patient during his hospital stay much later after multiple dialysis, in spite of which the levels were high. Probably his levels could have been much higher at arrival. With repeated hemodialysis, his renal failure improved and his chromium level reduced, which signifies that hemodialysis is an effective method to remove chromium.

**CONCLUSION**

In patients who present with unexplained rhabdomyolysis or renal failure, a toxin-mediated injury should be considered. Index of suspicion should be higher in patients who can have risk of occupational exposure or in those who come from an industrial area where there is assess to chromium. Though the abovementioned condition is associated with life threatening complications, prompt and supportive management with dialysis would lead to favorable outcome as in our patient.

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**Conflicts of interest**

There are no conflicts of interest.

**REFERENCES**

1. Fishbein L. Sources, transport and alterations of metal compounds: An overview. I. Arsenic, beryllium, cadmium, chromium, and nickel. Environ Health Perspect 1981;40:43-64.
2. Pesch B, Kendzia B, Hauptmann K, Van Gelder R, Stamm R, Hahn JU, et al. Airborne exposure to inhalable hexavalent chromium in welders and other occupations: Estimates from the German MEGA database. Int J Hyg Environ Health 2015;218:500-6.
3. André N, Paut O, Arditti J, Fabre P, Bremond V, Alhmana T, et al. Severe potassium dichromate poisoning after accidental nasal introduction. Arch Pediatr 1998;5:145-8.
4. Matey P, Allison KP, Sheehan TM, Gowar JP. Chromic acid burns: Early aggressive excision is the best method to prevent systemic toxicity. J Burn Care Rehabil 2000;21:241-5.
5. Jiao X, Teng Y, Zhan Y, Wu J, Lin X. Soil heavy metal pollution and risk assessment in Shenyang industrial district, Northeast China. PLoS One 2015;10:e0127736.
6. Bhownik AK, Alandar A, Katsoyiannis I, Shen H, Ali N, Ali SM, et al. Mapping human health risks from exposure to trace metal contamination of drinking water sources in Pakistan. Sci Total Environ 2015;538:306-16.
7. Nnorom IC, Osibanjo O. Heavy metal characterization of waste portable rechargeable batteries used in mobile phones. Int J Environ Sci Tech 2009;6:641-50.
8. Zendehdel R, Shetab-Boushehri SV, Azari MR, Hosseini V, Mohammadi H. Chemometrics models for assessment of oxidative stress risk in chrome-electroplating workers. Drug Chem Toxicol 2015;38:174-9.
9. Zhang KH, Zhang X, Wang XC, Jin LF, Yang ZP, Jiang CX, et al. Chronic occupational exposure to hexavalent chromium causes DNA damage in electroplating workers. BMC Public Health 2011;11:224.
10. Dayan AD, Paine AJ. Mechanisms of chromium toxicity, carcinogenicity and allergenicity: Review of the literature from 1985 to 2000. Hum Exp Toxicol 2001;20:439-51.
11. Kurosaki K, Nakamura T, Mukai T, Endo T. Unusual findings in a fatal case of poisoning with chromate compounds. Forensic Sci Int 1995;75:57-65.
12. Berndt WO. Renal chromium accumulation and its relationship to chromium-induced nephrotoxicity. J Toxicol Environ Health 1976;1:449-59.
13. Wedeen RP, Qian LF. Chromium-induced kidney disease. Environ Health Perspect 1991;92:71-4.
14. Franchini I, Mutti A, Cavatorta A, Corradi A, Cosi A, Olivetti G, et al. Nephrotoxicity of chromium. Remarks on an experimental and epidemiological investigation. Contrib Nephrol 1978;10:98-110.
15. Martin WR, Fuller RE. Suspected chromium picolinate-induced rhabdomyolysis. Pharmacotherapy 1998;18:860-2.
16. Bradberry SM, Vale JA. Therapeutic review: Is ascorbic acid of value in chromium poisoning and chromium dermatitis? Toxicol Clin Toxicol 1999;37:195-200.
17. Banner W Jr, Koch M, Capin DM, Hopf SB, Chang S, Tong TG. Experimental chelation therapy in chromium, lead, and boron intoxication with N-acetylcysteine and other compounds. Toxicol Appl Pharmacol 1986;83:142-7.
18. Illner N, Gerth J, Pfeiffer R, Bruns T, Wolf G. Nearly a stairway to heaven—severe dichromate intoxication in a young man. Clin Nephrol 2009;71:338-41.
19. Cerulli J, Grabe DW, Gauthier I, Malone M, McGoldrick MD. Chromium picolinate toxicity. Ann Pharmacother 1998;32(4):428-31.
20. Xiang J, Sun Z, Huan JN. Intensive chromic acid burns and acute chromium poisoning with acute renal failure. Chin Med J 2011;124:2071-3.
21. Schiff H, Weidmann P, Weiss M, Masry SG. Dialysis treatment of acute chromium intoxication and comparative efficacy of peritoneal versus hemodialysis in chromium removal. Miner Electrolyte Metab 1982;7:28-35.