Survival after acute ingestion of chromic acid complicated by acute kidney injury: a case report

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Poisoning with chromium is fatal and rarely reported. We report a case of a 25-year-old man who developed severe acidosis, gastrointestinal hemorrhage, and acute kidney and hepatic injury following homicidal ingestion of chromium. Patient improved after multiple cycles of alternate day hemodialysis.

Keywords:
acute kidney injury, chromium, hemodialysis

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Introduction

Chromic acid is a strong metal acid, and acute poisoning is very rare but very serious with severe skin injury and renal and liver failure. The majority of published cases were suicide attempts with lethal outcomes [1–3]. We describe the case of a 25-year-old man who presented to the Department of Medicine, JN Medical College, Aligarh with ingestion of 100 ml of chromic acid poison containing 1 g of chromium oxide used in industrial setup for chrome plating with homicidal intent. Patient was treated with hemodialysis for 20 days and was discharged after 22 days of hospitalization. The patient is now on regular follow-up in Nephrology outpatient department of JN Medical College with normal renal function tests (RFTs).

Case report

A 25-year-old man referred to our hospital 3 days after ingestion of chromium acid mixed with cold drink with homicidal intent. At presentation, his vitals were stable and he complained of abdominal discomfort. Renal and liver failure were absent at presentation but appeared later in the course of disease. There was absence of any evidence of caustic injury to the oral or upper gastrointestinal (GI) mucosa and his pupils were normally reacting. His complete blood counts, RFTs, and liver function tests were within normal limits on day 1. Day 2 postadmission, patient started having decreased sensorium and shortness of breath. Urine output decreased to 50 ml in 24 h. His RFTs were acutely deranged from baseline normal values to serum creatinine 8.2 mg/dl and blood urea 95 mg/dl. Arterial blood gas analysis revealed metabolic acidosis with pH 7.2, bicarbonate 12.5 mmol/l, serum potassium 5 mmol/l, serum sodium 135 mmol/l, and oxygen saturation 95.5. Ultrasonography of the abdomen showed normal-sized kidneys with maintained echotexture, and color Doppler study revealed normal renal arteries with resistive index 0.6.

Patient also developed upper GI bleeding for which he was immediately shifted to critical care ward (Medicine ICU) and taken for emergency hemodialysis. Upper GI fresh bleeding continued for 2 days, which was managed with 3 U of packed red blood cells, and 1 U of platelet-rich plasma was transfused. With this, platelet count rose to 88 000 × 10^3/μl from 5000 × 10^3/μl.

Liver function tests revealed elevated liver enzymes and bilirubin values (aspartate transaminase 95 IU/l, alanine transaminase 114 IU/l, alkaline phosphatase 16 KAU/100 ml, bilirubin total 3 mg/100 ml, direct 1.53 mg/100 ml, indirect 1.53 mg/100 ml, prothrombin time 14 s, and international normalized ratio 1.8).

He was treated with intermittent hemodialysis; no chelating agents or other methods for enhancing elimination were used. Patient underwent seven cycles of alternate day hemodialysis in which his RFT fluctuated. During the next 15 days, the renal function deteriorated, despite daily increase in urine output. RFT fluctuated from a peak of serum creatinine 15.6 mg/dl and blood urea 125 mg/dl to a nadir of serum creatinine 1.6 mg/dl and blood urea 52 mg/dl. Gradually, upper GI bleed subsided, and urine output returned to 1400 ml/day from a nadir of 50 ml/day (Table 1). Renal biopsy was discussed but was not considered due to refusal by patient party and deranged blood parameters. Patient was kept under close observation, and his blood counts, renal functions, and urine output gradually improved. Patient was discharged after 22 days of in-hospital treatment in stable condition.

Discussion

Solutions containing chromium ions have many industrial applications, which include chrome plating,

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Table 1 Clinical and biochemical profile of patient

Parameters	Day 1	Day 4	Day 10	Day 15	Day 22 (discharge)
Blood urea (mg/dl)	28	70	114	92	52
Serum creatinine (mg/dl)	0.7	8.2	11	5.8	1.6
Serum sodium (mmol/l)	132	143	132	126	134
Serum potassium (mmol/l)	3	4.5	3.6	4.4	3.8
AST (IU/l)	Normal	45			20
ALT (IU/l)	Normal	114			18
Bilirubin total, direct, indirect (mg/100 ml)	Normal	3.0			1.8
TLC ($10^3/\mu l$)	17	11	11.8	9.3	8.2
Hgb (g/dl)	12.8	10.7	11.5	11	10.3
Platelet ($10^3/\mu l$)	76	5	88	60	120
pH	7.2	7.3	7.37	7.4	7.34
HCO_3 (mmol/l)	9.3	16	24	20.5	22
Urine output (ml/24 h)	1500	50	260	800	1200

ALT, alanine transaminase; AST, aspartate transaminase; Hgb, hemoglobin; TLC, total lymphocyte count.

textile printing, and dyeing. Of the three valencies in use (Cr^{2+}, Cr^{3+}, Cr^{6+}), the hexavalent form (Cr^{6+}) is considered the most hazardous [4]. Hexavalent chromium readily crosses the cell membranes, causing cellular damage. The resulting cellular damage leads to GI bleeding, hemolysis, and hepatic and renal damage. In the present case report, aggressive hemodialysis between day 2 and 20 after injury caused a progressive decrease in chromium levels poisoning and renal failure with improvement in GI and liver functions.

To our knowledge, there is only one case reported of fatal chromic acid poisoning with acute renal failure in India by Varma *et al.* [5]. In another study, successful treatment of a patient suffering from severe acute potassium dichromate poisoning with liver transplantation was reported in Austria [6].

No proven antidote is available for chromium poisoning. Acute poisoning is often fatal regardless of therapy. Treatment in cases of acute high-level chromium exposure is usually supportive and symptomatic. Fluid and electrolyte balance is critical. Appropriate supportive measures may include ventilatory support, cardiovascular support, and renal and hepatic function monitoring [7].

Toxicological analysis of blood taken 5 h after chromium ingestion and before dialysis was initiated showed a chromium concentration of 34.0 mg/l, and after first dialysis session it was 18.0 mg/dl. The amount of 1 g chromium is believed to be the lethal dose for humans; there are reports suggesting that chromium blood concentrations of 10 mg/l or more are inevitably lethal [2].

Renal failure was the most serious complication. Progression to anuria is associated with poor prognosis. Initial oliguric/anuric phase could be explained by the combination of both direct chromic acid-induced intrinsic renal azotemia (both tubular and glomerular) and peripheral edema with volume depletion and hemoconcentration [4,7,8].

Affected patients should be monitored carefully for evidence of GI bleeding, hemolysis, coagulopathy, seizures, and pulmonary dysfunction [8].

Hemodialysis and charcoal hemoperfusion do not substantially enhance chromium removal from the body if renal function remains normal [7]. However, if renal failure ensues, hemodialysis may be necessary for management of the renal failure itself.

Peritoneal dialysis or hemodialysis is of great value to aid the removal of circulating chromium and is of use in treating established renal failure [9].

Conclusion

There are several interesting points to be noted in this case report: absence of caustic injury to the mucosa of the upper GI tract [10], course of organ failure with no specific elimination treatment attempted, and recovery after prolonged hospitalization with hemodialysis. The patient was discharged after 22 days of hospitalization in good condition, with normal liver and renal function but without the need for maintenance hemodialysis.

We emphasize the importance of timely intensive care for patients with accidental or unintentional poisoning with chromic acid or similar chemical substances. Early initiation of hemodialysis in chemical poisoning patients presenting with acute kidney injury may lead to early recovery of renal function, which may be many a times lifesaving.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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