Survival after Severe acute Chromic Acid Poisoning Complicated with Renal and Liver Failure

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Abstract

Chromic acid is a strong metal acid and acute poisoning is very rare, but very serious with severe skin injury, renal and liver failure. The majority of published cases were suicide attempts with lethal outcomes. We describe the case of a 55-year-old man who had accidentally taken a sip of 20% chromic acid (estimated chromium intake: 2.3 g). Renal and liver failure were not present at presentation, but appeared later in the course of disease. He was treated with hemodialysis, no chelating agents or other methods for enhancing elimination were used. Liver and renal function improved over the next 30 days and the patient was discharged after 45 days of hospitalization with no need for dialysis. In the follow-up period of eight months his renal function remained depressed, but stable.

Key words: chromic acid, liver and renal failure, survival


Introduction

Acute poisoning with hexavalent chromium (Cr) compounds is a very serious and often lethal condition with severe skin or gastrointestinal injury (depending on the route of poisoning), but also hepatic and renal failure which together lead to fatal outcome in most patients. It is not a very common acute poisoning due to limited accessibility of these compounds to general public.

Chromic acid is a strong mineral acid, which contains the most toxic form of chromium—the hexavalent (VI) form. It is used in industry for chromium plating, in the production of colored glass and ceramic glazes, in the band instrument repair industry and for “brightening” raw brass.

It causes ulceration to skin or mucosa, from which chromium (VI) ions are rapidly absorbed (1, 2). Cellular toxicity is associated with mitochondrial and lysosomal injury by biologically reactive intermediates: Cr (IV) and reactive oxygen species (3, 4).

Treatment options for chromic acid poisoning are limited (2). Oxidation of hexavalent to trivalent form with ascorbic acid (vitamin C) early (within the first two hours) after ingestion or skin exposure, may reduce absorption of chromium since the trivalent form does not cross cellular membranes so rapidly (5). Efficacy of activated charcoal has not been proven (5, 7). After the chromium (VI) has been absorbed, there is not much that can be done except to control the complications. Hemodialysis, exchange transfusions or application of chelating agents, such as EDTA, BAL or dimercaprol, have not been shown to be effective in the treatment of chromium poisoning (6, 7).

Fluid and electrolyte balance must be maintained especially in case of extensive skin or mucosal lesions which may lead to significant fluid loss and/or hemorrhage which usually is cause of early mortality. Respiratory, liver and renal function must be closely monitored as well as urine output. Hemodialysis must be applied if renal failure occurs, while anuria is usually associated with a poor prognosis (2). In cases of severe liver failure with hepatic necrosis, transplantation is the only option which can save the life of a patient (8).

There are only a few reports of chromium (VI) poisoning, mostly with dichromate salts, and even fewer of chromic acid poisoning (9-12) with only two published survival cases (13, 14).
A 55-year-old man with no significant medical history came to the emergency department about two hours after accidental swallowing of chromic acid. He declared that he had taken only one sip of liquid from an unmarked bottle, believing it to be wine. Immediately after recognising his mistake, he swallowed a large amount (approx. 2.5 L) of water and milk, and then induced vomiting, after which he again drank a lot of water and milk.

On examination, he was in good general condition, with no neurological deficits, normal blood pressure and pulse rate, without respiratory symptoms. There were no mucosal lesions visible by inspection or fibber-endoscopy in the mouth, throat, larynx or oesophagus. During the examination the patient started complaining of abdominal pain, nausea, vomited and started having watery diarrhoea. Initial laboratory values are shown in Table 1. The patient was clinically stable so he was admitted to the open ward. During the first few hours the patient developed chills, fever (38.5°C), tachycardia and hypotension. His urine output decreased and his stools became blood-watery and were almost continuous. Four hours after admission laboratory findings worsened so the patient was then transferred to the medical intensive care unit (ICU).

Parenteral antibiotic treatment (ciprofloxacin and metronidazol) was started due to the risk of mediastinitis and/or peritonitis after acid ingestion, although there were no clinical or radiological signs confirming this suspicion. Omeprazole was administered i.v. to lower the intrinsic acid production. It was not clear whether the fever had infectious origin or was just a symptom of chromium poisoning.

Urine output decreased rapidly to only 100 mL on the first day and turned to anuria in the next four days. Abdominal ultrasound showed enlarged kidneys with oedematous cortex and pronounced pyramids without other pathology. The patient was given fluid to correct hypovolemia, but continuous veno-venous hemodiafiltration (CVVHDF) was started as soon as the venous (subclavian) access was established and continued for the first two days in the ICU. On the third day we continued with continuous veno-venous hemodialysis (CVVHD).

For the first four days the patient continued to have large volumes (>2 L) of blood-watery stools, with a gradual decrease in red blood count, but with no need for blood transfusion. Inflammatory markers (ESR, CRP) normalized after 4 days of treatment, but antibiotic treatment was continued for 10 days. All microbiological samples (blood, urine and stool cultures) came back negative.

Liver lesion also worsened in the first days (Fig. 2): aminotransferases peaked on the fourth, bilirubin on the eight and GGT on the twelfth day (Table 1). Liver synthetic function (prothrombin time, proteins) was also impaired, which resulted in peripheral edema due to substantial capillary leak. Despite CVVHDF and later CVVHD, creatinine and urea concentrations rose during the first nine days (Fig. 1) to peak at creatinine: 748 μmol/L; urea: 31.1 mmol/L on the ninth day. On the tenth day in the ICU classical hemodialysis (HD) lasting for 4 hours was started and performed over the following three days which lowered creatinine (538 μmol/L) and urea (16 mmol/L). After four days of anuria urine output started to gradually increase and reached 800 mL on the tenth day, after which we suspended HD treatment in expectation of renal recovery. However, during the next 12 days urea and creatinine levels rose despite the further increase in daily urine output: on the 22nd day urine output was 3,400 mL (stimulated by large oral fluid intake), but urea and creatinine were at the highest (42.2 mmol/L and 1,540 μmol/L, respectively). The ultrasonographic appearance of the kidneys remained the same (enlarged, edematous cortex, pronounced pyramids). There
Table 1. Laboratory Findings in Emergency Department, 4h after Admission, 4 Days after Admission and at Discharge

<table>
<thead>
<tr>
<th></th>
<th>In Emergency room</th>
<th>4h after admission</th>
<th>4 days after admission</th>
<th>45th day (Discharge)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hgb (g/L)</strong></td>
<td>176</td>
<td>196</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Htc (%)</strong></td>
<td>50.6</td>
<td>56.4</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>L (× 10^9/L)</strong></td>
<td>Normal</td>
<td>14.2 (19% bands)</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Platelet (× 10^9/L)</strong></td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>ESR (mm/h)</strong></td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>CRP</strong></td>
<td>Normal</td>
<td>55</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>AST (U/L)</strong></td>
<td>55</td>
<td>96</td>
<td>1510</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>ALT (U/L)</strong></td>
<td>62</td>
<td>102</td>
<td>1470</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>GGT (U/L)</strong></td>
<td>59</td>
<td>94</td>
<td>105</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Bilirubin (μmol/L)</strong></td>
<td>Normal</td>
<td>35</td>
<td>90</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>PT</strong></td>
<td>Normal</td>
<td>0.25</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Creatinine (μmol/L)</strong></td>
<td>Normal</td>
<td>116</td>
<td>35</td>
<td>234</td>
</tr>
<tr>
<td><strong>Urea (mmol/L)</strong></td>
<td>6.6</td>
<td>9.0</td>
<td>10.1</td>
<td>11.0</td>
</tr>
<tr>
<td><strong>Total proteins (g/L)</strong></td>
<td>-</td>
<td>-</td>
<td>42</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Figure 2. Aspartate aminotransferase (AST, black triangles connected with full line), alanine aminotransferase (ALT, black diamonds connected with spotted line) and bilirubin (white diamonds connected with full line) in the serum of the patient over time.

was no electrolyte disturbance, but the patient complained of typical symptoms of uremia: nausea, skin rash, change of taste, constant tiredness. HD was therefore again started. Urine output remained normal (between 1,400 and 2,200 mL), urea and creatinine decreased and uremic symptoms disappeared. Arteriovenous fistula was created on the left forearm after 40 days of hospitalization to permit possible hemodialysis in the future, but the last HD was performed on the 41st hospital day, after which creatinine and urea remained stable (249 μmol/L and 8.6 mmol/L respectively). Renal biopsy was discussed but was not necessary as the patient’s condition improved. Ultrasonographic image of the kidneys was at that time closer to normal: only slight edema of the cortex, slightly enlarged kidneys. Creatinine clearance on the 43rd day was 29.3 mL/min.

The patient was discharged after 45 days of hospitalization in good condition, with normal liver function and reduced renal function but with no need for dialysis. Follow-up after eight months revealed further reduction of creatinine (186 μmol/L) and urea (8.2 mmol/L). Creatinine clearance remained 30 mL/min.

Analysis of the swallowed liquid proved it to be chromic acid with 20% of elementary chromium (20 grams per decilitre). We estimated the volume of swallowed poison: the patient was given a bottle of water, from which he had taken a sip, trying to reproduce the amount of liquid he drank from the bottle of poison. The procedure was repeated for 20 times, and a mean volume of 11.5±0.6 mL was established. The estimated intake of chromium from this volume was 2.3±0.1 g. Toxicological analysis of blood taken five hours after chromium ingestion and before dialysis was initiated showed a chromium concentration of 3.4 mg/L.

**Discussion**

There are several interesting points to be noted in this case report: absence of caustic injury to the mucosa of the upper gastrointestinal tract, course of organ failure with no specific elimination treatment attempted and recovery after prolonged hospitalization.
Because the patient sought medical assistance about two hours after ingestion of the poison, oxidation of the ingested chromium (VI) with ascorbic acid was not attempted. Activated charcoal was also not applied due to lack of supporting evidence. Gastric lavage was contraindicated due to the possibility of organ perforation.

Although chromic acid is a strong acid and the patient himself induced vomiting, there were no caustic lesions in the upper gastrointestinal tract. This may be attributed to dilution and neutralisation of the acid with large amounts of water and milk he had taken. We estimate that the ingested quantity of chromium was more than 2 g. The fraction of ingested chromium that entered the patient’s circulation is not known, but the blood concentration of chromium was 3.4 mg/L about five hours later. The amount of 1 g chromium ingested was more than 2 g. The fraction of water and milk he had taken. We estimate that the ingested chromium concentration of 10 mg/L or more are inevitably lethal (15).

Gastrointestinal symptoms were first to appear, within the first hours, but also first to subside. There was significant fluid loss to intestinal lumen, apparent from heavy hemoconcentration, followed by gastrointestinal bleeding, which did not require transfusion therapy. Sufficient fluid resuscitation prevented development of shock in the early hours after poisoning. There was probably a significant protein loss through the damaged mucosa which together with liver failure led to significant hypoproteinemia with peripheral edema. Renal failure with anuria certainly contributed to edema.

Liver lesion, though minimal, was already present at first examination in the emergency department and progressed in the first few days (hepatocellular necrosis and impaired liver synthetic function.) Liver enzymes normalized rather quickly, but it took more than thirty days for serum proteins to normalize.

Renal failure was the most serious complication. Initial oliguric/anuric phase could be explained by the combination of both direct chromic acid induced intrinsic renal azotemia (both tubular and glomerular) and prerenal azotemia due to frequent blood-watery stools and peripheral oedema with volume depletion and haemoconcentration. Vigorous and adequate i.v. fluid resuscitation corrected prerenal azotemia, so intrinsic azotemia became the principal reason of injury. The concentrating capability of excreted urine was inadequate (inadequate osmolarity), resulting in a large amount of produced and excreted urine not accompanied by fall of creatinine and urea; creatinine clearance remained 0 mL/min. It took 40 days for creatinine clearance to recover to 30 mL/min which resulted in increased but stable creatinine and urea levels and normal electrolyte concentrations. Limited evidence-based data and only a few reported cases prevent us from making any other conclusions.

Conclusion

We report a case of unintentional ingestion of a small amount of chromic acid. Although the amount of fluid was small it did a significant life threatening damage: liver and renal failure. Both conditions improved over time, after intensive supervision and treatment with hemodialysis. Renal function improved significantly but during the follow-up period of eight months, it did not normalize completely. We emphasize the importance of intensive care within the first couple of weeks for patients with accidental or unintentional poisoning with chromic acid or similar chemical substances.

References

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