

## CASE REPORT

# Transdermal exposure to Chrome-III oxide resulting in intoxications and morbidity in two tanning workers—a case report

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## Abstract

Here, we report two cases of chrome-III oxide intoxications following transdermal exposure in tanning workers. A 58-year-old man (case 1) and his 41-year-old co-worker (case 2) were brought to our ED with unconsciousness six hours after accidental transdermal exposure to chrome-III oxide crystals (Tankrom® AB, Chrome-III oxide 25.5% and Schorlemmer Basicity 33%). Upon arrival, both patients presented with agitation and chemical burns affecting 11% (case 1) and 3% (case 2) of their total body surface area, respectively. Chemical burns were mainly distributed on injured skin. Initial arterial blood gas analysis revealed moderate (case 1, pH: 7.268) to mild acidosis (case 2, pH: 7.352). One of our patients (case 1) had significant respiratory depression. Laboratory results of case 1 and case 2 showed increased white blood cell counts, (16,630 mm<sup>3</sup> and 19,950 mm<sup>3</sup>) and elevated blood glucose (178 mg/dL and 193 mg/dL), creatinine (1.33 mg/dL and 1.52 mg/dL), liver enzyme (aspartate aminotransferase/alanine aminotransferase of 416/322 U/L and 141/118 U/L) and creatine phosphokinase level (474 U/L and 566 U/L), respectively. Radiologic examinations revealed an orbital wall fracture and L2 compression fracture in case 1, while case 2 had a subdural hematoma, subarachnoid hemorrhage and scapular fracture and had to undergo an operation for external ventricular drainage. Both patients became alert on day three (case 1) and day six (case 2) and were discharged on day 27. We serially measured their serum and urinary chromium concentrations after hospital arrival. The calculated serum half-lives were 35.7 hours and 25.1 hours, and urinary half-lives were 2.3 hours and 2.5 hours in case 1 and case 2, respectively. We suggest that transdermal exposure to chrome-III oxide, especially to injured skin, may result in significant toxicity and morbidity. Therefore, it is essential to take necessary precautions and preventive measures to avoid transdermal exposure to chromium.

## Keywords

Chromium; Transdermal intoxication; Toxicokinetic

## 1. Introduction

Chromium is the sixth most abundant element on earth. It has been used commercially in chemical, metallurgical and refractory industries [1]. Hexavalent chromium is a strong oxidizing agent with irritant activity, which can cause acute or chronic toxicities in exposed persons [1, 2]. The clinical manifestations of acute chromium toxicity may include acute gastrointestinal symptoms, renal failure, hepatic failure and intravascular hemolysis, and in severe cases, coma and death may result [2–4]. Compared to hexavalent chromium, trivalent compounds except chromium sulfate have only weak irritant activity and are considered relatively nontoxic [1]. In this report, we present two cases of chrome-III oxide intoxication after accidental transdermal exposure in tanning workers.

## 2. Case presentation

### 2.1 Case 1

A 58-year-old man presented to our emergency department (ED) after a factory guard found him and his co-worker unconscious six hours after accidental exposure to chrome-III oxide crystals (Tankrom® AB, Chrome-III oxide 25.5% and Schorlemmer Basicity 33%). On arrival, he was agitated, had a Glasgow coma scale (GCS) of 11 (E2V4M5) and high blood pressure of 153/110 mmHg. Physical examinations revealed chemical burns on his face, trunk and both extremities, accounting for approximately 11% of his total body surface area (TBSA). Chemical burns were mainly distributed on injured skin with bleeding or abrasion. We immediately removed his contaminated clothes and decontaminated the patient.

He was given 5 L/minute of oxygen supply, and his arterial blood gas analysis (ABGA) revealed a pH of 7.268 (normal range: 7.35–7.45), partial pressure of carbon dioxide ( $\text{PCO}_2$ ) of 42.4 mmHg (normal range: 35–45), and partial pressure of oxygen ( $\text{PaO}_2$ ) of 205.9 mmHg (normal range: 75–100). Radiologic examination revealed an orbital wall fracture and an L2 vertebrae compression fracture.

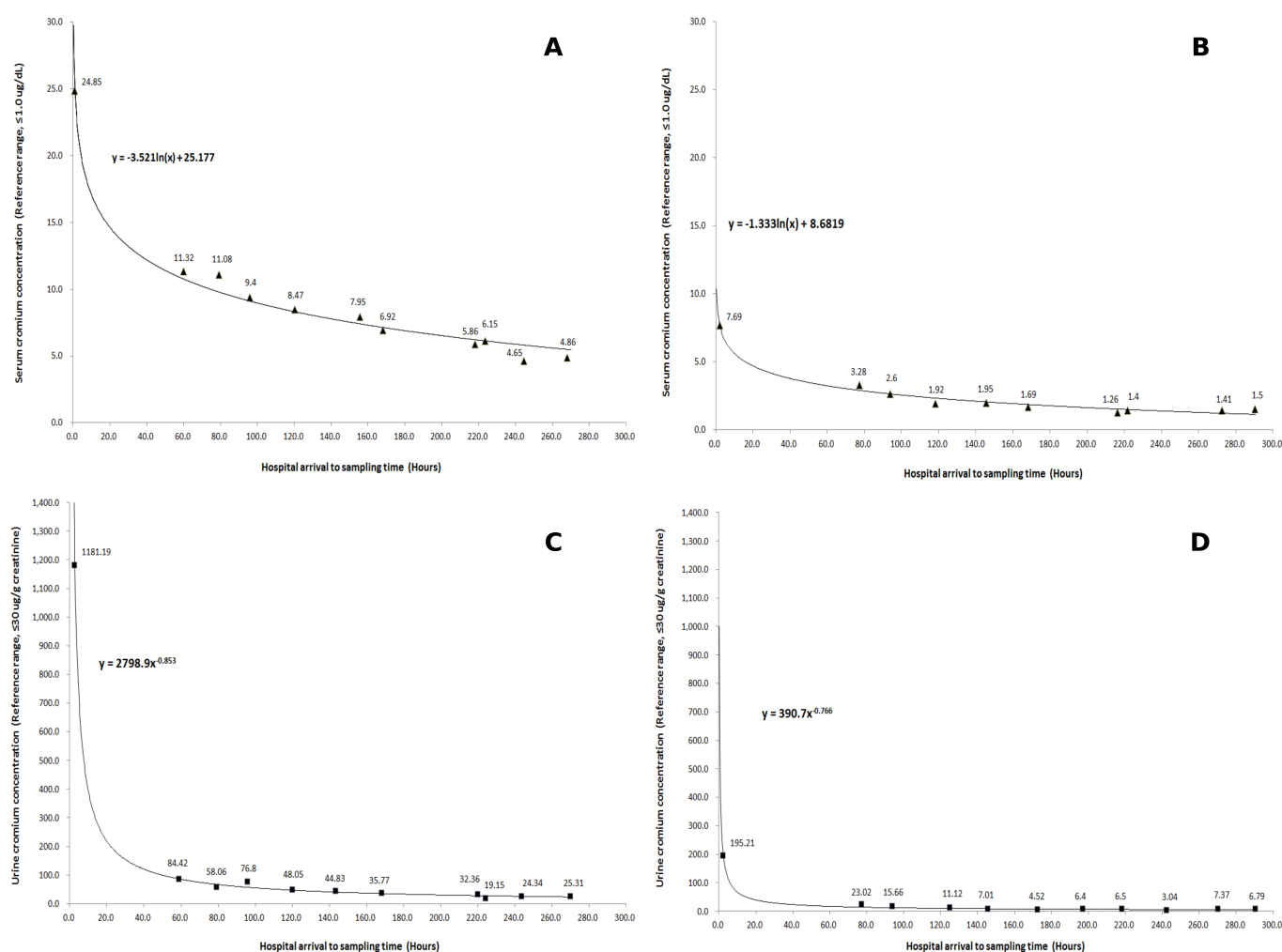
Laboratory results revealed increased white blood cell (WBC) counts of  $16,630 \text{ mm}^3$  (normal range: 4000–10,000) and elevated lactic acid (70.4 mg/dL; normal range: 4.5–18.0), glucose (178 mg/dL; normal range: 55–99), blood urea nitrogen (BUN) (25.1 mg/dL; normal range: 8.0–20.0), creatinine (1.33 mg/dL; normal range: 0.5–1.3), aspartate aminotransferase (AST) (416 U/L; normal range: 14–40), alanine aminotransferase (ALT) (322 U/L; normal range: 8–46) and creatine phosphokinase (CPK) (474 U/L; normal range: 43–244). Other test results were within normal limits. His serum ( $\leq 1.0 \text{ ug/dL}$ ) and urinary chromium concentrations ( $\leq 30.0 \text{ g/g creatinine}$ ) were measured from 1.0 hours (24.5 ug/dL, 1181.2 g/g creatinine) to 268.0 hours (4.68 ug/dL, 25.31 g/g creatinine) after the hospital arrival, as shown in Fig. 1A,C. Drug screening test results showed no significant

anomalities.

The patient was given supportive management, including painkillers, sedatives, prophylactic antibiotics, and fluid administration and was admitted to the intensive care unit (ICU) for close monitoring and critical care. On day three, he became alert, and the chemical burns had changed to brown-colored pigmented ulcers (chromic holes), not shown herein. We applied wet dressing using commercial products. Brain magnetic resonance angiography (MRA) performed on day five was normal, and he was discharged with good medical conditions on day 27.

## 2.2 Case 2

A 41-year-old man visited our ED with signs of agitation, a GCS of 13 (E4V3M6) and tachycardia (heart rate, 125 beats/minute) on arrival. Physical examinations revealed chemical burns on approximately 3% of his TBSA, following which the patient was decontaminated. ABGA with room air revealed a pH of 7.352,  $\text{PCO}_2$  of 38.6 mmHg, and  $\text{PaO}_2$  of 86.9 mmHg. Radiologic examination revealed that the patient had sustained traumatic subdural hematoma (SDH), subarachnoid hemorrhage (SAH), and a scapular fracture.



**FIGURE 1.** Serial measurements of chromium concentration in serum and urine. (A) Serially measured serum chromium concentration of case 1. (B) Serially measured serum chromium concentration of case 2. (C) Serially measured urinary chromium concentration of case 1. (D) Serially measured urinary chromium concentration of case 2.

Laboratory results revealed increased WBC counts (19,950 mm<sup>3</sup>), lactic acid (24.3 mg/dL), glucose (193 mg/dL), BUN (25.9 mg/dL), creatinine (1.52 mg/dL), AST (141 U/L), ALT (118 U/L) and CPK (566 U/L). His other test results were within normal limits.

We also measured his serum and urinary chromium concentrations from 4.0 hours (7.69 ug/dL, 195.21 g/g creatinine) to 292.0 hours (1.5 ug/dL, 6.79 g/g creatinine) after the hospital arrival, as shown in Fig. 1B,D. The patient underwent an operation for extraventricular drain (EVD) placement to control increased intracranial pressure and was subsequently admitted to the ICU for close monitoring and critical care. By day three, he was responsive to verbal commands, with a GCS of 11, and by day six, he became alert. He was discharged in good medical condition on day 27.

### 3. Discussion

After six hours of transdermal exposure to chrome-III oxide, the reported patients presented with agitation, hyperglycemia, and elevations of WBC counts, lactic acid, BUN, creatinine, liver enzyme and CPK but did not have severe toxicities [3–5]. However, one of our patients had significant respiratory depression, which did not progress to respiratory failure. Both patients developed chronic holes in their skins, a sign of transdermal chromium exposure (data not shown).

In Table 1, we present a comprehensive summary of the clinical characteristics and key findings of both patients, which were then compared with those of previously documented cases with severe intoxications following transdermal exposure. Our findings demonstrate that transdermal absorption of chromium is enhanced in instances of exposure to acid solutions or injured skin, as evidenced in the cases reported in this study [6, 7].

In Fig. 1, we present the serial measurements of the serum and urinary chromium concentrations of both patients after the hospital arrival. Case 1 exhibited a peak serum concentration that was approximately three times higher than that of case 2, which was consistent with the extent of their chromic holes. It is worth noting that nearly 60% of absorbed chromium is excreted by urine within 8 hours of ingestion [1]. Urine elimination of chromium in our patients revealed a rapid initial phase followed by a slow phase with long half-lives, consistent with a previous report on chrome platers [8]. Assuming a plasma volume of 3.75 liters, the calculated chromium absorptions *via* the skin for cases 1 and 2 were estimated as 0.95 mg and 0.33 mg, respectively (Table 1) [8].

The threshold of acute oral toxicity of trivalent chromium is 1990–3300 ug/kg, with only 0.5–2.0% of ingested chromium being absorbed from the gut [1, 9]. We estimate that the transdermal absorptions observed in our patients were close to the threshold of acute oral toxicity. The Urinary Chromium concentration ratio to the value of 30 g/g creatinine, the daily permitted urinary chromium levels at the end of a shift was 39.4 and 6.5 in cases 1 and 2, respectively [1]. Using the measured chromium concentrations, we estimated that cases 1 and 2 had a serum half-life of 35.7 hours and 25.1 hours and a urinary half-life of 2.3 hours and 2.5 hours, respectively, different from those of oral hexavalent chromium intoxication

[10]. Treatment of chromium intoxication is primarily based on the symptoms of the exposed patients. In this present study, both patients received supportive management after decontamination. We did not perform chelation, antioxidant administrations or hemodialysis. Their urine outputs were well maintained at a rate of 0.5–1.5 mL/kg/hour by the fourth day of hospitalization.

Following the transdermal chrome-III oxide intoxications, the reported patients revealed mild to moderate acute chromium toxicity. Notably, the calculated serum and urine half-lives in our patients differed from those of oral hexavalent chromium intoxication. Transdermal exposure to chrome-III oxide, especially to injured skin, may result in significant toxicity and morbidity. Therefore, it is essential to take necessary precautions and preventive measures to avoid transdermal exposure to chromium.

### AVAILABILITY OF DATA AND MATERIALS

The data are contained within this article.

### AUTHOR CONTRIBUTIONS

JTP—examined and diagnosed the patients; wrote the first version of the manuscript. KHC—approved the final version of the paper and performed the revisions. All authors contributed to the final version of the manuscript.

### ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol was approved by the Institutional Review Board of the Catholic University of Korea, Uijeonbu St. Mary's Hospital (IRB file no. UC18ZEI0101). Informed consent was obtained from the patient involved in the study.

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### CONFLICT OF INTEREST

The authors declare no conflict of interest.

**TABLE 1. A Comparison of key clinical features of chromium intoxication cases following transdermal exposure.**

	Case 1	Case 2	Lin <i>et al.</i> [7]	Kelly <i>et al.</i> [6]
Age/Sex	58/male	41/male	22/male	56/male
Occupation	Tanning worker	Tanning worker	Electroplating worker	Chromium processing
Type of chromium (concentration)	Chrome-III oxide, 25.5%	Chrome-III oxide, 25.5%	Chromic acid, 1.4 M	Chromium sulfate, 40%
pH & valence	Acid salt (pH 3), 3 <sup>+</sup>	Acid salt (pH 3), 3 <sup>+</sup>	Acid solution, 6 <sup>+</sup>	Acid solution, (pH 2.8), 3 <sup>+</sup>
Exposure-related event	Fell on the stalk of chromium	Fell on the stalk of chromium	Working in the tank with legs immersed	Immersion up to his neck in the solution
Exposure duration	>6 h	>6 h	Within 10 min	–
At initial presentation				
Chief complaint	Agitation	Agitation	Abdominal pain	–
Glasgow coma scale (GCS)	11 (E <sub>2</sub> V <sub>4</sub> M <sub>5</sub> )	13 (E <sub>4</sub> V <sub>3</sub> M <sub>6</sub> )	15 (E <sub>4</sub> M <sub>5</sub> V <sub>6</sub> )	15 (E <sub>4</sub> M <sub>5</sub> V <sub>6</sub> )
Vital signs	Stable	Stable	Stable	Shock
Skin findings	Erosion & bulla	Erosion & bulla	Pigmented ulcer	Variable degree of ulcer
Involved skin % of TBSA	11%	3%	15%	>70%
Chromium Conc., Serum (<1 ug/dL)	24.85 at 1.6 h	7.69 at 4.7 h	–	47 at 5.5 h
Suggested absorption <i>via</i> the skin (mg)*	0.934	0.289	–	176
Urine (<30 ug/g creatinine)	1181.2 at 3.4 h	195.21 at 4.7 h	88,208	–
Urinary Chromium cocentration ratio <sup>§</sup>	39.4	6.5	1960.1	–
Key laboratory findings				
Leukocytosis	Yes	Yes	Yes	Yes
Increased liver enzyme	Yes	Yes	Yes	Yes
Rhabdomyolysis	Yes	Yes	Yes	–
Renal failure with oliguria	No	No	Yes	Yes
Intravascular hemolysis	No	No	No	Yes
Treatments	Trauma care Conservative care	Trauma care EVD & ICP control Conservative care	Chelating agent Antioxidant CVVH & Plasmapheresis	Methylene blue Exchange transfusion
Clinical outcomes	Good recovery	Good recovery	Severe, but survived	Fatal

–: not described in the literature; TBSA: total body surface area; CVVH, continuous veno-venous hemofiltration; EVD: external ventricular drainage; ICP: increased intracranial pressure.

Suggested absorption *via* the skin (mg)\*: suggested amounts of chromium in serum absorbed through the skin assuming the plasma volume of 3.76 liters; Urinary Chromium concentration ratio<sup>§</sup>: ratio of urinary chromium concentration measured at initial presentation to the value of 30 g/g creatinine, the daily permitted urinary chromium levels at the end of shift.

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