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## A Case of Unintentional Isopropanol Poisoning via Transdermal Absorption Delayed by Weekly Hemodialysis

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# A Case of Unintentional Isopropanol Poisoning via Transdermal Absorption Delayed by Weekly Hemodialysis

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 Study Design A  
 Data Collection B  
 Statistical Analysis C  
 Data Interpretation D  
 Manuscript Preparation E  
 Literature Search F  
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**Patient:** Female, 67-year-old  
**Final Diagnosis:** Chronic renal failure • isopropanol poisoning  
**Symptoms:** Encephalopathy  
**Medication:** —  
**Clinical Procedure:** Dialysis  
**Specialty:** Nephrology • Toxicology

**Objective:** Unusual clinical course

**Background:** Isopropanol toxicity is the most common reported toxic alcohol ingestion in the United States and is well known to emergency physicians. Most toxicities result from unintentional ingestion of rubbing alcohol; however, an under-recognized mechanism of unintentional toxicity is transdermal absorption. Additionally, hemodialysis effectively removes isopropanol and its metabolites from circulation, so that in patients receiving regular hemodialysis, the manifestation of toxicity can be delayed.

**Case Report:** A 67-year-old woman with end-stage renal disease secondary to insulin-dependent type II diabetes on once-weekly hemodialysis presented to the Emergency Department via the Emergency Medical Service with acute encephalopathy, severe hypoglycemia, and hypothermia. Her daughter found her confused and lethargic, smelling of acetone, and with a bottle of rubbing alcohol in her hand. The patient had been topically applying large quantities of rubbing alcohol for several months as a home remedy for cramps and adamantly denied any oral ingestion. She had missed several hemodialysis appointments over the previous month. Upon arrival, the patient was confused, profoundly hypoglycemic, and hypothermic. Additional laboratory examination revealed an elevated plasma osmolality, osmolar gap, isopropanol level, and acetone level. She was treated supportively with glucose-containing fluids and external warming and was admitted to the Intensive Care Unit. Hemodialysis was resumed, and the patient was discharged 3 days after admission with stable blood glucose, regular body temperature, and baseline mental status.

**Conclusions:** Our report is unique as it presents both an under-recognized mechanism of isopropanol toxicity (transdermal absorption) and an uncommon presentation of chronic exposure with manifestations of toxicity delayed by regular hemodialysis.

**Keywords:** Acetone • Inhalation Exposure • Poisoning • Renal Dialysis • Skin Absorption

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/934529>

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## Background

Isopropanol (also known as isopropyl alcohol or 2-propanol) is a potent toxic alcohol which annually accounts for the most commonly reported toxic alcohol ingestion in the United States [1]. It is a clear, colorless liquid with an aromatic odor, resembling acetone [1,2]. It is found in many common household products, most notably rubbing alcohol, which commonly contains up to 70% isopropanol [2]. Other products containing isopropanol include hand sanitizers, paint thinners, detergents, jewelry cleaners, and skin/hair products [2]. Most events of toxicity are unintentional ingestions and occur in children as the liquid is clear, resembles water, and can be easily accessible [3]. However, some patients have ingested isopropanol abusively as an inexpensive ethanol substitute to achieve inebriation [1].

Unintentional and intentional ingestions of isopropanol are common presentations to the Emergency Department (ED) in the United States; however, there have been few cases reported on unintentional toxicity via transdermal absorption of the toxic alcohol [4,5]. A common yet ill-advised home remedy for various ailments such as cramps, neuropathy, bug bites, and rashes is the topical application of rubbing alcohol. Patients have been reported to apply it directly or soak absorbent materials such as cotton balls, pads, gauze, or towels in the alcohol and apply it to their skin for significant periods of time [4-9]. If applied in large amounts such as in these cases, transdermal absorption and inhalation of the substance have resulted in clinically significant toxicity. However, studies in rabbits suggest that inhalation plays a much smaller role in the development of toxicity relative to transdermal absorption [10].

We present a unique case of a patient who repeatedly applied large doses of isopropanol to her skin as a home remedy and developed unintentional isopropanol toxicity via transdermal absorption, exacerbated by missed hemodialysis. The symptoms of toxicity in this patient were delayed because she received hemodialysis once weekly for end-stage renal disease, which would remove the toxic metabolites of isopropanol from circulation, and did not become clinically apparent until dialysis was missed. This patient's toxicity likely contributed to the development of severe hypoglycemia and altered mental status, resulting in the admission to the Intensive Care Unit (ICU).

## Case Report

A 67-year-old woman presented to the ED via the Emergency Medical Service (EMS) with acute encephalopathy, severe hypoglycemia, and hypothermia, with a last known normal status 5 h prior to arrival. She had a past medical history of non-alcoholic steatohepatitis (NASH) cirrhosis, gastric antral vascular ectasia syndrome, hypothyroidism, insulin-dependent type

2 diabetes mellitus, hypertension, hyperlipidemia, essential tremor, and end-stage renal disease, with dialysis treatment on Saturdays. Her home medications included levothyroxine (200 mcg p.o. daily), furosemide (80 mg p.o. b.i.d.), nifedipine (30 mg p.o. b.i.d.), propranolol hydrochloride extended-release (120 mg p.o. daily), doxepin (25 mg p.o. daily), gabapentin (300 mg p.o. 3 times weekly), atorvastatin (40 mg p.o. daily), colestipol (3 g p.o. b.i.d.), pantoprazole (40 mg p.o. b.i.d.), and subcutaneous insulin (glargine 45 units daily in AM; lispro 10 units b.i.d., in AM and PM, plus sliding scale with meals). She was a former smoker and denied any history of alcohol or illicit drug use. The patient was found by her daughter lying on the couch, lethargic, confused, with breath smelling of acetone, and with a bottle of rubbing alcohol (isopropanol) in her hand. At the time, the patient was confused but adamant that she had not consumed the alcohol. The patient had been applying rubbing alcohol topically to her legs, chest, arms, and neck for several months to treat cramps. She would then use a heating pad after application. Her daughter stated that over the past month, the patient had been becoming more confused with breath that smelled of "nail polish remover". The patient had additionally missed several dialysis treatments throughout the past month due to an illness in her husband. Additionally, she had difficulty maintaining a stable blood glucose level, with large fluctuations between hyper- and hypoglycemia, as well as difficulty focusing her vision over the past several weeks. The patient had not eaten anything all day on the day of presentation and could not recall how much insulin she took.

Upon arrival to the ED, the patient was confused, hemodynamically stable, hypothermic with a rectal temperature of 34.4°C, and hypoglycemic with a blood glucose level of 66. The initial blood glucose was 33 per the EMS, and the patient was given intravenous (i.v.) 10% dextrose (D10) and glucose en route. The patient was administered an additional bolus of D10 and placed on a Bear-hugger to correct the hypoglycemia and hypothermia, respectively. On initial examination, the patient was alert and oriented with a Glasgow Coma Scale of 14, was in no acute distress, and had an intact airway, with no labored breathing, abdominal tenderness, or focal neurological deficits. Electrocardiogram (ECG) revealed sinus bradycardia. Laboratory workup revealed elevated plasma osmolality (330 mOsm/kg), plasma acetone (116 mg/dL), plasma isopropanol (29 mg/dL), and a calculated serum osmolar gap of 23.8 mOsm/kg. On repeat laboratory testing, the patient continued to have hypoglycemia and was placed on an i.v. D10 drip at 75 mL/h. Poison control was contacted and recommended supportive therapy, including i.v. fluids and management of hypoglycemia. Repeat laboratory testing 3 h later revealed a plasma isopropanol level of 10 mg/dL and acetone level of 148 mg/dL. The patient was then admitted to the ICU for management of hypoglycemia, hypothermia, and isopropanol toxicity. The Nephrology Department was consulted for hemodialysis.

On day 2 of admission, hemodialysis was resumed, the D10 drip was continued at 75 mL/h, and oral intake was encouraged while insulin was held. Insulin was resumed as the patient's blood glucose level was corrected. The Medical Toxicology Department was consulted for evaluation of the patient at bedside, and recommended continued supportive therapy. The patient's mental status and hypothermia improved within 24 h of admission with glycemic normalization, allowing for discontinuation of the Bear-hugger and i.v. D10 drip and transfer to the general medical floor. Post-hemodialysis laboratory examinations included electrolyte studies (which did not reveal any significant abnormalities) but did not include a repeat alcohol panel given the patient's improved mental status.

The patient was discharged from the hospital on day 3 of admission with baseline mental status, stable blood glucose, an adjusted home subcutaneous insulin regimen (glargine 25 units daily in AM [from 45 units], lispro 5 units b.i.d., in AM and PM [from 10 units], plus sliding scale), instructions to skip short-acting insulin if patient does not eat, a resumed once-weekly dialysis regimen, and counseling regarding safe use of rubbing alcohol. There were no complications during management of this patient.

## Discussion

This report highlights an interesting case of a patient who developed clinically significant isopropanol toxicity that was likely via transdermal absorption given the thin nature of her skin, large surface area, and frequency in which she was applying it. The toxicity ultimately resulted in an altered mental status, contributing to profound hypoglycemia and hypothermia, necessitating her admission to the ICU. This patient's presentation is especially unique as her symptoms of toxicity did not become apparent until after weekly dialysis was missed. This patient's hypoglycemia was likely the result of fasting with inappropriate insulin use, although isopropanol toxicity can result in hypoglycemia as the metabolism of the alcohol by hepatic alcohol dehydrogenase increases the NADH/NAD<sup>+</sup> ratio, inhibiting gluconeogenesis [2]. Nevertheless, her altered mental status likely contributed to this inappropriate use. Additionally, gastrointestinal irritation, secondary to isopropanol and its metabolite, acetone, may have resulted in poor appetite. Similarly, although severe toxicities can result in hypothermia, this patient's low body temperature was likely the result of her hypoglycemia; however, both hypoglycemia and hypothermia were indirectly related to isopropanol toxicity in this patient [3].

This case also reveals important key clinical and diagnostic features of isopropanol toxicity. Upon arrival, this patient showed signs of mental status depression with an accompanying acetonc odor, a clinical presentation that ought to cue

the physician to consider isopropanol toxicity as a diagnosis. Hallmark manifestations of toxicity include inebriation, central nervous system (CNS) depression, and gastric irritation which can result in hemorrhage [1,2]. Coma, respiratory depression, hypotension, renal failure, and rhabdomyolysis can be seen in severe cases [1,2]. The patient's laboratory findings of an elevated osmolar gap and ketonemia without acidosis were also hallmark diagnostic clues [11]. Unlike the other toxic alcohols (methanol and ethylene glycol), isopropanol does not cause acidosis. It is metabolized to acetone by hepatic alcohol dehydrogenase, which does not cause acidosis, although it can contribute to some level of CNS depression [2]. Acetone was once thought to be a primary contributor to CNS depression because it is metabolized from isopropanol; however, several case studies have reported improving mental status in the setting of rising acetone levels (as also seen in our patient), which would not be expected if it was a major CNS depressant [12-15]. These studies suggest that isopropanol itself is the primary CNS depressant in the setting of toxicity, rather than acetone which likely plays a less significant role.

Isopropanol follows first order kinetics and has a half-life between 2.5 and 8 h [16-18]. Given its metabolism by hepatic alcohol dehydrogenase, this can be prolonged by hepatic disease, as in our patient with NASH cirrhosis. It has been shown that hepatic alcohol dehydrogenase activity decreases with the severity of hepatic dysfunction [19]. However, the laboratory workup in our patient at 3 h revealed a decreasing plasma isopropanol level and increasing plasma acetone level, suggestive of hepatic metabolism. Its half-life can also be prolonged by renal disease (as in our patient), as it has been reported that up to 20% of the absorbed dose of isopropanol is renally excreted, unmetabolized [12]. Acetone, its metabolite, has a slower half-life, estimated to be between 7 and 24 h [17,18]. This can be prolonged by renal disease, as it is primarily renally excreted with some additional respiratory excretion, resulting in our patient's distinct acetonc breath and patient's presentation after missed dialysis [3]. However, acetone can be metabolized via alternative metabolic pathways in the setting of renal failure because it is a ketone body and can be metabolized as such.

The treatment of isopropanol toxicity is supportive with the removal of the offending agent, i.v. hydration, and correction of other abnormalities, such as the hypoglycemia and hypothermia in our patient [1,2]. Alcohol dehydrogenase inhibitors (eg, fomepizole) should not be used because they would delay the metabolism of isopropanol and prolong the symptoms of toxicity [2]. Hemodialysis is indicated in severe, life-threatening poisonings, toxicity that is refractory to supportive therapy, and isopropanol levels greater than 400 mg/dL [2,3,6].

The findings of this report are consistent with previous case reports regarding the development of toxicity from topically

applied rubbing alcohol [4-9]. In these previously published cases, the toxicity was unintentional, as the adult patients were pursuing home remedies for their various symptoms. Additionally, as transdermal absorption is a slow process, the development of toxicity requires prolonged exposure, as seen in our case [5,10,20]. These findings should raise the awareness of ED physicians to consider transdermal absorption as a possible mechanism of exposure in patients presenting with isopropanol toxicity. The results of the present case are also consistent with previous reports, as toxicity resolved quickly with supportive therapy and removal of offending agent [4,5]. These previous reports also note the possible potentiation of toxicity by inhalation of fumes from the rubbing alcohol. However, the present study is limited by its inability to determine to what degree the inhalation of the fumes played in developing toxicity.

This study reveals the need for future research to determine the doses of transdermal isopropanol exposure that result in toxicity, as well as the need for public education on the appropriate indications and safe uses of the topical application of rubbing alcohol.

## Conclusions

Our report is unique as it presents both an under-recognized mechanism of isopropanol toxicity (transdermal absorption) and an uncommon presentation of chronic exposure with manifestations of toxicity delayed by regular hemodialysis. Transdermal absorption may account for more presentations of isopropanol toxicity in the ED than currently recognized. This report highlights the need for increased awareness by ED physicians of this mechanism of toxicity and public education on the appropriate indications and safe uses of topical application of the alcohol.

## References:

1. Ashurst JV, Nappe TM. Isopropanol toxicity. [Updated 2021 Jun 26]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK493181/>
2. Cohen JP, Quan D. Alcohols. In: Tintinalli JE, Ma O, Yealy DM, et al eds. Tintinalli's Emergency Medicine: A comprehensive study guide, 9e. McGraw Hill; 2020. Available from: <https://accessemergencymedicine.mhmedical.com/content.aspx?bookid=2353&sectionid=220744565>
3. Slaughter RJ, Mason RW, Beasley DM, et al. Isopropanol poisoning. Clin Toxicol (Phila). 2014;52(5):470-78
4. Wolfshohl JA, Jenkins DA, Phillips TM. A case of toxic transdermal absorption of isopropyl alcohol with falsely elevated creatinine. Am J Emerg Med. 2021;377:e5-377
5. Leeper SC, Almatari AL, Ingram JD, Ferslew KE. Topical absorption of isopropyl alcohol induced cardiac and neurologic deficits in an adult female with intact skin. Vet Hum Toxicol. 2000;42(1):15-17
6. Garrison RF. Acute poisoning from use of isopropyl alcohol in tepid sponging. JAMA. 1953;152(4):317-18
7. Senz EH, Goldfarb DL. Coma in a child following use of isopropyl alcohol in sponging. J Pediatr. 1958;53(3):322-23
8. Wise, JR. Alcohol sponge baths. N Engl J Med. 1969;280(15):840
9. McFadden SW, Haddow JE. Coma produced by topical application of isopropanol. Pediatrics. 1969;43(4):622-23
10. Martinez TT, Jaeger RW, deCastro FJ, et al. A comparison of the absorption and metabolism of isopropyl alcohol by oral, dermal and inhalation routes. Vet Hum Toxicol. 1986;28(3):233-36
11. Sarai M, Tejani AM, Chan AH, et al. Magnesium for alcohol withdrawal. Cochrane Database Syst Rev. 2013;(6):CD008358
12. Gaudet MP, Fraser GL. Isopropanol ingestion: case report with pharmacokinetic analysis. Am J Emerg Med. 1989;7(3):297-99
13. Blanchet B, Charachon A, Lukat S, et al. A case of mixed intoxication with isopropyl alcohol and propanol-1 after ingestion of a topical antiseptic solution. Clin Toxicol (Phila). 2007;45(6):701-4
14. Mydler TT, Wasserman GS, Watson WA, Knapp JF. Two-week-old infant with isopropanol intoxication. Pediatr Emerg Care. 1993;9(3):146-48
15. Dua SL. Letter: Peritoneal dialysis for isopropyl alcohol poisoning. JAMA. 1974;230(1):35
16. Daniel DR, McAnalley BH, Garriott JC. Isopropyl alcohol metabolism after acute intoxication in humans. J Anal Toxicol. 1981;5(3):110-12
17. Natowicz M, Donahue J, Gorman L, et al. Pharmacokinetic analysis of a case of isopropanol intoxication. Clin Chem. 1985;31(2):326-28
18. Jones AW. Elimination half-life of acetone in humans: Case reports and review of the literature. J Anal Toxicol. 2000;24(1):8-10
19. Vidal F, Perez J, Morancho J, et al. Hepatic alcohol dehydrogenase activity in alcoholic subjects with and without liver disease. Gut. 1990;31(6):707-11
20. Dyer S, Mycyk MB, Ahrens WR, Zell-Kanter M. Hemorrhagic gastritis from topical isopropanol exposure. Ann Pharmacother. 2002;36(11):1733-35