

Isopropanol (Lexi-Tox)

CAS Registration

- 67-63-0

Use

Commercially available as isopropyl alcohol 70%, USP (rubbing alcohol); may also be found in concentrations up to 99%

May be found in nail polish remover, hairspray, hand sanitizers, cleaning fluids, lacquer, windshield washer fluids, inks, hand lotions, aftershave, body rubs, rubefacients, creosote, resins, otic water remover, component of folk medicines, topical medications, and cosmetics; may be utilized in the production of acetone, glycerol, and isopropyl acetate and also as an industrial solvent (eg, for gums, essential oils, paint strippers, shellac)

Clinical Presentation

Ingestion: Dermal contact is the most common exposure to isopropanol, but ingestion is the most common route that produces toxicity. Pediatric exposures are often the consequence of exposure to hand sanitizers (Santos 2017). Classic signs and symptoms of systemic toxicity resemble those found with ethanol intoxication: CNS depression, inebriation, nausea, and vomiting. Severe intoxication may cause myocardial depression and vasodilation leading to hypotension, hypothermia, and cardiovascular collapse; respiratory arrest and coma may also occur. An increased osmolal gap **without** an anion gap metabolic acidosis is consistent with isopropanol intoxication. Isopropanol is metabolized to acetone; expect acetone to be present in both the serum and urine once metabolism has occurred. Late in the course of intoxication, isopropanol may not be detectable; however, acetone concentrations may be significantly elevated. The development of ketonemia and ketonuria may be prolonged in patients who have coingested ethanol. Death typically occurs as a result of refractory hemodynamic collapse. Because the toxic effects of isopropanol are primarily due to the parent alcohol, CNS depression will progressively improve as isopropanol is metabolized and eliminated.

Inhalation: Prolonged inhalation of isopropanol may result in systemic toxicity characterized by CNS depression and inebriation.

Ocular: Ocular exposure may cause local irritation.

Dermal: Local irritation and skin drying may occur. Prolonged dermal exposure may result in systemic toxicity characterized by CNS depression; gastrointestinal signs and symptoms are typically absent. Intoxication has been reported following the sponging of children with isopropanol for fever control (Arditi 1987).

Comprehensive listing by system (listed alphabetically): **Note:** Unless otherwise noted, the symptoms listed are for exposures via ingestion.

Cardiovascular: Arrhythmias, cardiac arrest (severe intoxication), hypotension (severe intoxication), myocardial depression, shock (severe intoxication), tachycardia

Central nervous system: Ataxia, CNS depression, coma (severe intoxication), confusion, dizziness, drowsiness, headache, hypothermia (severe intoxication), impaired coordination, inebriation, seizures (rare), slurred speech, stupor

Endocrine & metabolic: Hyperglycemia, hypoglycemia, ketonemia, osmolal gap

Gastrointestinal: Abdominal pain, gastritis, hematemesis, melena, nausea, vomiting

Hematologic: Hemolytic anemia (rare)

Neuromuscular & skeletal: Deep tendon reflexes (absent or diminished) (severe intoxication), flaccid muscular paralysis (severe intoxication), gait instability

Ocular: Conjunctivitis, corneal abrasions (ocular exposure), irritation (ocular exposure), miosis, mydriasis, nystagmus

Renal: Acute tubular necrosis (rare), ketonuria, myoglobinuria (rare)

Respiratory: Cough, dyspnea, pulmonary edema, respiratory arrest (severe intoxication)

Miscellaneous: Fruity odor on breath

Mechanism of Toxicity

Isopropanol is oxidized by alcohol dehydrogenase (ADH) to acetone, a ketone that cannot be oxidized to an organic acid (eg, unlike methanol [formic acid]), resulting in ketonemia and ketonuria **without** significant acidemia.

Isopropanol is a potent CNS depressant with up to three times the CNS depressant effects of ethanol. Acetone may also contribute to CNS depression, but to a lesser extent. In addition, isopropanol is directly irritating to the gastrointestinal tract following ingestion.

Diagnosis

Diagnosis should be made based on patient history, physical examination, laboratory findings, and clinical suspicion. Pertinent laboratory data which may support a diagnosis of isopropanol intoxication include:

- Presence of serum isopropanol and/or acetone
- Unexplained osmolal gap
- Ketonemia or ketonuria without significant metabolic acidosis
- Absence of other known causes of ketosis including:
 - starvation
 - diabetic ketoacidosis
 - cyanide poisoning
 - infection

Patients will typically have a normal anion gap and pH with an elevated osmolal gap. Isopropanol and its metabolite, acetone, are osmotically active; therefore, an elevated osmolal gap may be present throughout the intoxication.

The presence of acetone in the urine and serum ketones is a result of the metabolism of isopropanol. Ketonuria and/or ketonemia in the absence of hyperglycemia are highly suggestive of isopropanol intoxication. It may take several hours for ketonuria and ketonemia to develop (may take longer if coingested with ethanol). Urine acetone may be detected at least 3 hours after ingestion by using Acetest tablets.

Serum isopropanol levels may help to confirm the diagnosis of isopropanol intoxication but do not reliably predict clinical effects and outcome; levels as low as 50 to 100 mg/dL (comparable to ethanol concentrations of 100 to 200 mg/dL) may result in significant toxicity. If the ingestion of an alcohol-containing compound is suspected in patients with anion gap metabolic acidosis, but isopropanol, acetone, and/or ethanol are not detected, consider the presence of methanol and/or ethylene glycol.

Note: Isopropanol-poisoned patients may have a falsely elevated serum creatinine level due to the presence of ketonemia; clinicians should be aware of this when evaluating a patient's renal function.

Note: Isopropanol-poisoned patients may have an anion gap acidosis related to other causes (eg, starvation/alcoholic ketoacidosis) and, therefore, careful examination of laboratory findings in the context of patient history and presentation is essential to avoid inappropriate antidote administration (Meng 2015).

Laboratory Testing/Diagnostic Procedures

- [Anion Gap, Serum or Plasma](#)
- [Arterial Blood Gases](#)
- [Ethanol Level](#)
- [Glucose, Serum or Plasma](#)
- [Isopropanol Level \(Includes Acetone\)](#)
- [Ketone Bodies, Serum or Plasma](#)
- [Ketones, Urine](#)
- [Osmolality, Calculated](#)
- [Osmolality, Serum or Plasma](#)

Exposure Control

Containment: All emergency workers (first responders) and hospital staff should follow appropriate precautions regarding exposure to hazardous materials including the use of protective clothing (wear neoprene or nitrile gloves) and goggles. Masks and respiratory equipment should be worn if the isopropanol is aerosolized.

Treatment: Stabilization

Initially, evaluate and correct immediate life-threatening complications (eg, airway, breathing, and circulation). The most common serious complications of isopropanol intoxication include CNS depression, cardiovascular collapse, hypothermia, and respiratory depression. Supportive care with an emphasis on airway protection and adequate tissue oxygenation is the cornerstone of treatment.

Treatment: Decontamination

All contaminated clothing and belongings should be bagged in liquid-occlusive containers and removed from patient care areas to protect health care providers from exposure.

Ingestion: If the patient presents within 1 hour of ingestion, consider the following decontamination procedure(s):

- Gastric lavage: In rare situations when gastric lavage is deemed appropriate, it is most effective if initiated within 1 hour of the ingestion; however, gastric lavage has not been proven to be beneficial and is not routinely recommended due to the risk of complications and the lack of demonstrated efficacy. Use is contraindicated in a patient with an unprotected airway, in a patient in whom its use increases the risk and severity of aspiration, and in a patient who is at risk of hemorrhage or gastrointestinal perforation due to pathology (Benson 2013; Vale 2004).

Based on experimental and clinical studies, the following decontamination procedures have **not** been shown to be beneficial:

- Activated charcoal: Isopropanol is a small molecular weight compound and is not adsorbed by activated charcoal; therefore, unless toxic coingestants are involved, activated charcoal has no role in the management of a patient exposed to isopropanol.
- Ipecac (AACT 2004; AAP 2003; Höjer 2013)
- Cathartics (AACT 2004)

Inhalation: Remove the patient from the source of exposure and into fresh air. Monitor for respiratory distress.

Ocular: Irrigate with copious amounts of tap water or normal saline for at least 15 minutes; remove contact lenses if easily removable without causing additional trauma to the eye.

Dermal: Gently wash exposed skin and/or hair with nontoxic, mild detergent and warm water; rinse thoroughly with water.

Treatment: Antidote(s)

No specific antidote exists. Do not administer an alcohol dehydrogenase inhibitor (eg, ethanol or fomepizole), as this may prolong isopropanol toxicity.

Treatment: Nonpharmacologic Supportive Therapy

In patients with adequate renal function, maintain good urine output to facilitate elimination of isopropanol and acetone.

Hemodialysis: Hemodialysis is an efficient means of elimination of both isopropanol and acetone and may shorten the duration of coma; however, most patients will not require hemodialysis.

Indications for use: Hemodialysis should be considered in patients with elevated isopropanol levels and **any** of the following: Hemodynamic instability despite intensive supportive care; prolonged coma; hepatic or renal failure.

Patient Disposition

Initial evaluation: The following individuals need to be evaluated in the emergency department (ED):

- Patients who expose themselves to isopropanol with the intent of self-harm or intentional abuse
- Victims of abuse or neglect who were exposed to isopropanol with malicious intent
- Most patients with suspected isopropanol intoxication require evaluation in the ED. Exceptions may exist; consider consultation with a poison control center to assist with referral decisions (eg, minimal exposures that may not require emergency department evaluation).

Criteria for emergency department discharge:

- Patients who present with an unintentional exposure and remain asymptomatic for 2 hours after exposure are unlikely to develop toxicity and may be considered for discharge.

Note: Patients should not be considered for discharge until isopropanol levels are negligible; patients with measurable isopropanol levels may still be intoxicated and incapable of driving, both physically and legally.

- Resolution of symptoms may be prolonged in patients who have coingested ethanol; these patients may require a longer observation period.
- Repeat serum isopropanol and acetone levels may be helpful in estimating individual clearance rates and the expected duration of intoxication.

Pharmacodynamics/Kinetics

Absorption: Rapid from GI tract or by inhalation; little absorption through intact skin

Ingestion: Rapid (~100%)

Inhalation: Well-absorbed

Ocular: Negligible

Dermal: Negligible through intact skin; prolonged exposure may lead to significant absorption

Distribution: V_d : 0.6 to 0.7 L/kg

Metabolism: Hepatic by alcohol dehydrogenase to acetone (80%) which is further metabolized to acetate, formate, and carbon dioxide. Acetone is present in the serum within 30 to 60 minutes and in the urine within 3 hours of ingestion.

Half-life, elimination:

Isopropanol: 4.2 hours (range: 2.6 to 16.2 hours; coingestion of ethanol prolongs the elimination of isopropanol) (Pappas 1991)

Acetone: 11.4 hours (range: 7.6 to 26.2 hours) (Pappas 1991)

Time to peak, serum: Within 0.5 to 2 hours

Excretion: Excreted renally, 25% to 50% unchanged

Isopropanol: Urine (~20% as unchanged drug); lungs (2% as unchanged drug)

Acetone: Urine (60% to 80%); lung (20% to 40%)

Complications of Exposure

Females: Pregnancy: Isopropanol was found to cross the placenta and cause symptoms of intoxication in a newborn following maternal ingestion prior to delivery (Wood 2007).

Additional Information

Optimal care decisions are made based upon specific patient details. Consider consultation with a poison control center. To reach poison control centers in the United States and its territories, call 1-800-222-1222.

Regulatory considerations: If exposure is a result of a work-related incident, patients and/or caregivers may be legally required to report the exposure; contact the state or local health department for more information.

When a release or spill occurs, the company responsible for the release, its response contractors, the local fire and police departments, and the local emergency response personnel provide the first line of defense.

If a chemical has been spilled following a transportation accident, Chemtrec may be involved depending on the size of the event. The phone number is: US: 1-800-424-9300; outside the US: 1-703-527-3887, collect calls are accepted.

Index Terms

1-Methylethanol; 2-Hydroxypropane; 2-Propanol; 2-Propyl Alcohol; Dimethyl Carbinol; IPA; Isopropyl Alcohol; Isopropyl Alcohol, Rubbing; Rubbing Alcohol

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