

Toxic Alcohol Poisoning

Toxic alcohol poisoning happens with methanol and ethylene glycol. They become real poison inside your body.

THE ONE-LINE TRUTH

Methanol and ethylene glycol are not toxic by themselves.

The chain — how it unfolds

1. Breathing speeds up to blow off CO₂ and balance the acid load. — why: Alcohol dehydrogenase is the only enzyme in the body that can break down methanol or ethylene glycol, so it converts them into formic acid or oxalic acid—toxins that the original alcohol was not.
2. The kidney tries to get rid of the acid but cannot keep up. — why: Fast breathing (hyperventilation) is the lungs trying to blow off CO₂ to balance out the acid flood, because the blood pH is dropping and the body is in metabolic acidosis (acid building up in the blood).
3. Once formate builds up your optic nerve gets hurt. — why: The kidneys try to filter out the acid through urine but cannot move fast enough to keep up with how much formate or oxalate is being made, so acid piles up in the blood.
4. Once oxalate builds up calcium gets used and kidney tubules block with crystals. — why: Once formate builds up in the blood, it crosses into the optic nerve and damages the cells that let you see, causing vision changes or blindness that is specific to methanol poisoning.
5. None of this is random. — why: Once oxalate builds up, it grabs calcium from the blood to form crystals, which both drops blood calcium and plugs up the kidney tubules with crystal debris, causing kidney failure.
6. The body is trying to keep blood flow, oxygen, or balance going — so each finding is the body trying to help, not the real sickness itself. — why: None of these findings—fast breathing, vision loss, low calcium, kidney failure—is random; each one is the body's damaged organ trying to compensate or the toxin attacking a specific target organ.

7. Trace it back: alcohol dehydrogenase normally clears ethanol, then the patient drank methanol like windshield fluid or moonshine, and this response is the body buying time. — why: Tracing backward: the patient drank methanol or ethylene glycol expecting it to be safe (or by accident), but alcohol dehydrogenase treated it like regular ethanol and made poison; the body's response is the race between damage and clearance.

Spot it — what you see, and why

- Confusion or acting drunk without drinking alcohol: The toxic alcohol breaks down into acids (like formic acid from methanol or glycolic acid from ethylene glycol) that flood the brain and block the cells from making energy, so thinking gets fuzzy and the person acts confused or drowsy.
- Fast, deep breathing (like trying to catch your breath after running): The body senses the blood has turned too acidic (metabolic too much acid in the blood (acidosis)) from the toxic breakdown products, so the lungs speed up to blow off carbon dioxide and try to bring the acid level back down.
- Strange or fruity smell on the breath (not the smell of regular alcohol): Toxic alcohols like isopropanol break down into acetone, which smells sweet or fruity, unlike ethanol (regular drinking alcohol) which has its own sharp smell. Methanol and ethylene glycol themselves have almost no smell.
- Blurry vision, seeing spots, or complete vision loss (especially with methanol): Methanol breaks down into formic acid, which directly poisons the optic nerve (the cable that sends pictures from the eye to the brain) and the retina (the light-sensing layer at the back of the eye), shutting down vision.
- Severe belly pain, nausea, and vomiting: The stomach and gut lining get irritated by the toxic alcohol itself, and the acids building up in the blood make the person feel sick and cause pain.
- Low calcium in the blood and tiny crystals in the urine (with ethylene glycol): Ethylene glycol breaks down into oxalic acid, which grabs onto calcium in the blood and forms sharp calcium oxalate crystals that show up in the urine and kidneys, dropping the calcium level.

Red flags — act now

- Vision loss or blurry vision (sign of methanol poisoning the optic nerve)
- No urine or very little urine (kidney failure from ethylene glycol)
- Seizures, unresponsive, or coma (severe brain poisoning)
- Blood pH below 7.1 or very high anion gap (life-threatening acid buildup)
- Fast breathing that will not slow down (body trying hard to fix dangerous acid level)

Treat it — in this order

1. Fomepizole (Antizol) IV loading dose 15 mg/kg, then 10 mg/kg every 12 hours → Blocks the liver enzyme alcohol dehydrogenase so methanol and ethylene glycol cannot turn into formic acid or oxalic acid. This stops the poisoning chain before the toxic acids form.
2. Hemodialysis (start if pH < 7.25, kidney injury, vision changes, or methanol/ethylene glycol level > 50 mg/dL) → Physically removes both the parent alcohol (methanol or ethylene glycol) and the toxic acid (formic acid or oxalic acid) from the blood much faster than the kidneys can clear them. Corrects severe too much acid in the blood (acidosis) within hours.
3. Sodium bicarbonate IV bolus 1-2 mEq/kg, then continuous infusion to keep pH > 7.30 → Raises blood pH to reduce too much acid in the blood (acidosis). Shifts formic acid into its charged form (formate), which the kidneys can clear faster in the urine. Buys time for dialysis and fomepizole to work.
4. Folinic acid (leucovorin) IV 1 mg/kg (up to 50 mg) every 4-6 hours for methanol poisoning → Provides a helper molecule (cofactor) that the liver needs to break down formic acid into carbon dioxide and water. Speeds up the body's own detox pathway.
5. Thiamine 100 mg IV and pyridoxine 100 mg IV every 6 hours for ethylene glycol poisoning → Provide helper molecules (cofactors) that help the liver convert glyoxylic acid (a toxic breakdown product of ethylene glycol) into safer chemicals like glycine instead of letting it become oxalic acid.

Your top priorities at the bedside

1. Check airway, breathing, and oxygen saturation every 15 minutes; prepare intubation equipment at bedside and call respiratory therapy — why: Severe metabolic acidosis (acid building up in the blood) (pH below 7.30) causes Kussmaul respirations where the patient breathes fast and deep to blow off carbon dioxide; if the patient tires out or pH drops further the brainstem stops sending breathing signals and respiratory arrest happens within minutes
2. Start two large-bore IV lines (18-gauge or larger) and draw blood for arterial blood gas (ABG), basic metabolic panel, osmolal gap, and toxic alcohol levels before giving any treatment — why: We need the osmolal gap (the difference between measured and calculated blood thickness) to prove toxic alcohol is present before lab results come back; fomepizole and dialysis change the numbers so we must get baseline labs first; large IVs allow rapid sodium bicarbonate infusion to fix too much acid in the blood (acidosis)

3. Give fomepizole 15 milligrams per kilogram IV loading dose over 30 minutes within the first hour, then prepare maintenance doses every 12 hours — why: Fomepizole blocks alcohol dehydrogenase (the liver enzyme that breaks down alcohols); this stops methanol from turning into formic acid and ethylene glycol from turning into oxalic acid; the poisons stay harmless and the kidneys pee them out unchanged