

Tricyclic Antidepressant (TCA) Overdose

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart beats because tiny gates called sodium channels open and close in a rhythm, letting electrical signals zip through the heart muscle so it squeezes in the right order. At the same time, acetylcholine (a chemical messenger) keeps your pupils small, your mouth wet, your bladder emptying, and your gut moving. Alpha receptors on the walls of your blood vessels keep them tight enough to hold your blood pressure steady. And GABA—the brain's main calming signal—acts like a brake pedal to keep nerve cells from firing too fast. When someone takes too much tricyclic antidepressant, the drug blocks all four of these systems at once. The sodium channels in the heart get jammed, so the electrical signal crawls instead of zipping—the heart's rhythm on the monitor gets wide and slow, and dangerous fast rhythms like ventricular a fast heart rate (tachycardia) or v-fib can start. The acetylcholine receptors shut off, so pupils blow wide open, the mouth goes bone-dry, the skin gets hot and flushed, the bladder can't empty, and thinking gets foggy or wild. The alpha receptors on blood vessels stop working, so the vessels relax and blood pressure drops. And GABA in the brain gets turned down, so the brake pedal lifts and seizures can explode. Every weird finding—wide QRS, big pupils, dry skin, low blood pressure, confusion, seizures—points back to this one upstream break: too much tricyclic blocking these four systems. The body tries to cope for a while, but once the heart rhythm tips into chaos or seizures drop the blood's pH, the window to recover closes fast. Sodium bicarbonate is the key treatment because it helps unblock the sodium channels and stabilize the heart.

What you might feel

- You find a patient who is drowsy
- QRS is wide
- Blood pressure is low
- Anticholinergic poisoning signs are there
- The electrocardiogram shows QRS over 100 or tall R in aVR
- This means arrhythmia risk

Call the doctor right away if...

- QRS complex wider than 100 milliseconds on the EKG — the single most important danger sign in TCA overdose. It predicts seizures and deadly heart rhythms, and it is the trigger to give IV sodium bicarbonate right away.
- A tall R wave in lead aVR (taller than 3 mm) — an EKG fingerprint of the sodium-channel poisoning TCAs cause in the heart's wiring.
- Rapid crash in the first 1 to 2 hours — a TCA patient can go from awake and talking to seizing, with low blood pressure (hypotensive), and unconscious (comatose) in minutes. Never trust an early 'looks fine.'
- Seizures — each seizure makes the blood more acidic, and too much acid in the blood (acidosis) makes the TCA grip the heart's sodium channels even harder (a vicious cycle: treat aggressively and re-check the EKG).
- Blood pressure that stays low after IV fluids — both the heart pump and the blood vessels are poisoned; norepinephrine is the vasopressor of choice.
- Falling level of consciousness — plan for early airway protection; slow, shallow breathing raises CO₂, worsens too much acid in the blood (acidosis), and feeds the cardiotoxicity loop.

What the care team will do

- Place patient on continuous cardiac monitor and obtain 12-lead ECG immediately, checking QRS width and looking for tall R wave in lead aVR
- Establish large-bore IV access and prepare sodium bicarbonate 1 to 2 milliequivalents per kilogram for immediate bolus if QRS is over 100 milliseconds or patient has low blood pressure or arrhythmia
- Check blood pressure every 5 to 15 minutes and start IV fluid bolus of normal saline 500 to 1000 milliliters; prepare norepinephrine infusion if systolic pressure stays below 90 despite fluids
- Assess and protect airway, position patient on side if drowsy, and have suction and bag-valve-mask at bedside; monitor respiratory rate and oxygen saturation continuously
- Keep benzodiazepines (lorazepam 2 to 4 milligrams IV or diazepam 5 to 10 milligrams IV) immediately available and give at first sign of muscle twitching or seizure activity
- Monitor core temperature every 30 minutes and look for signs of anticholinergic toxidrome: flushed dry skin, dilated pupils, urinary retention, decreased bowel sounds, confusion
- Recheck 12-lead ECG every 15 to 30 minutes after each sodium bicarbonate dose, measuring QRS width and monitoring for ventricular arrhythmias or heart blocks

- Prepare 20 percent lipid emulsion (Intralipid) 1.5 milliliters per kilogram bolus with infusion ready if patient develops ventricular a fast heart rate (tachycardia), ventricular fibrillation, or pulseless electrical activity unresponsive to sodium bicarbonate and defibrillation

Medicines you may be given — and why

Sodium bicarbonate

Tricyclic antidepressants (TCAs) block sodium channels in the heart, which slows the electrical signals that make the heart beat in rhythm. When the blood becomes more alkaline (less acidic), the TCA molecule loosens its grip on those sodium channels, freeing them up so the heart can conduct signals normally again. The extra sodium ions also flood the channels and help push the electrical current through despite some remaining blockade. This double effect—higher pH and more sodium—reverses the life-threatening heart rhythm problems and low blood pressure caused by TCA overdose.

Normal saline (0.9 % sodium chloride IV fluids)

TCA overdose causes blood vessels to relax and the heart to pump weakly, which drops blood pressure. Giving IV fluids fills up the blood vessels (like topping off a half-empty tank) so there is enough volume for the heart to pump forward. The extra sodium in normal saline also helps reverse the sodium-channel blockade in the heart, just like sodium bicarbonate does.

Norepinephrine

TCA overdose blocks alpha receptors on blood vessels (the switches that normally keep vessels snug) and weakens the heart, causing blood pressure to fall. Norepinephrine is a powerful signal that turns on both alpha receptors (to squeeze blood vessels tight) and beta receptors in the heart (to make it pump harder). This two-pronged push brings blood pressure back up and keeps blood flowing to the brain and kidneys.

Lorazepam or diazepam

TCA overdose lowers the seizure threshold by blocking brain receptors that normally keep electrical activity in check. Benzodiazepines turn up GABA (the brain's chief calming messenger) by making GABA receptors more sensitive, which puts the brakes on runaway electrical storms and stops the seizure.

Activated charcoal

Activated charcoal is a fine black powder with a huge surface area, like a sponge full of tiny pores. TCA molecules stick to the charcoal particles and are trapped, so they cannot be absorbed into the bloodstream. The charcoal and the bound drug then pass out in the stool.

This works only if there is still TCA sitting in the gut—usually within the first hour or two after ingestion.

Intravenous lipid emulsion (Intralipid 20 %)

TCAs are lipophilic (they love fat). When you infuse a concentrated fat emulsion into the bloodstream, it creates a 'lipid sink'—a fatty compartment that the TCA molecules are drawn into, pulling them away from the heart and brain where they are causing toxicity. This can restart the heart or stabilize dangerous rhythms when nothing else works. The exact mechanism is still debated, but clinical experience shows it can be life-saving in refractory cardiac arrest from TCA overdose.

Where this information comes from

- ACMT tricyclic antidepressant toxicity; ToxNet

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