

Acetaminophen poison

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally your liver breaks down acetaminophen into harmless pieces that leave in your urine. A tiny amount becomes NAPQI, a poisonous molecule, but your liver immediately grabs it with glutathione, a protective chemical that acts like a shield. When you swallow too much acetaminophen—either all at once or big doses over several days—the safe breakdown paths get overwhelmed. NAPQI piles up faster than glutathione can handle. Once glutathione runs out, NAPQI sticks directly to liver cells and kills them. Damage unfolds in four stages. Stage 1, the first 24 hours after swallowing, you may feel sick to your stomach or feel nothing at all. Stage 2, 24 to 72 hours later, brings pain in the right side of your belly under the ribs and liver blood tests start climbing because liver cells are quietly dying. Stage 3, 72 to 96 hours, is sudden massive liver failure—the liver stops making clotting proteins so you bruise and bleed easily, it stops clearing ammonia so confusion sets in, and kidneys may shut down. Stage 4 is either recovery as new liver cells grow back, or death without a transplant. Every problem you see traces back to one upstream break: too much acetaminophen exhausted glutathione and left NAPQI free to destroy the liver. The antidote, N-acetylcysteine (NAC), works by refilling glutathione stores and must start within 8 hours for the best chance of survival. Timing is everything—early treatment saves lives, delayed treatment may not.

What you might feel

- Nausea and vomiting (Stage 1, first 24 hours)
- Feeling better or no symptoms at all (Stage 2, 24 to 72 hours)
- Pain and tenderness in the right upper belly (Stage 2 to 3)
- Yellow skin and whites of the eyes (jaundice, Stage 3, 72 to 96 hours)
- Confusion, extreme sleepiness, or not responding (encephalopathy, Stage 3)
- Easy bruising, bleeding gums, or bleeding from the stomach or gut (Stage 3)
- Little or no urine output (oliguria, Stage 3)
- Fast heartbeat and low blood pressure (Stage 3 to 4)
- Belly swelling (ascites, Stage 3 to 4)

Call the doctor right away if...

- INR greater than 1.5 or liver enzymes (ALT or AST) climbing into the thousands—signals severe liver cell death and loss of clotting proteins
- Confusion, sleepiness, or flapping hand tremor when arms are held out (asterixis)—signs ammonia is poisoning the brain
- Blood lactate greater than 3.5 after giving fluids, or blood pH less than 7.30—means massive cell death and very high risk of dying
- Kidney function worsening (rising creatinine) or urine output dropping below 0.5 mL per kilogram per hour—acute kidney injury on top of liver failure
- Reported swallowing more than 10 grams total (or 150 mg per kg of body weight), or blood acetaminophen level plotting above the treatment line on the Rumack-Matthew nomogram—high risk for liver damage if the antidote NAC is not given quickly

What the care team will do

- Start the NAC antidote immediately if the acetaminophen level is above the treatment line on the nomogram, or right away if the patient is confused, jaundiced, or has liver enzymes in the thousands—do not wait for levels if poisoning happened more than 8 hours ago.
- Draw blood for acetaminophen level at least 4 hours after ingestion, and also send INR (clotting time), ALT and AST (liver enzymes), creatinine (kidney function), and basic metabolic panel with bicarbonate and anion gap.
- Monitor for anaphylactoid reactions (flushing, itching, hives, wheezing, low blood pressure) during the first IV NAC loading dose, and stop the infusion temporarily if these appear—then restart at a slower rate once symptoms resolve.
- Check mental status, hand tremor (hold arms out and watch for flapping), and measure urine output every 2 to 4 hours—report immediately if the patient becomes confused, very sleepy, or makes less than 0.5 milliliters per kilogram per hour of urine.
- Place the patient on continuous pulse oximetry and cardiac monitoring, and keep two large-bore IV lines patent with normal saline running.
- Hold all non-essential medicines that the liver breaks down, especially opioids, benzodiazepines, and sedatives, unless absolutely needed for pain or agitation.
- Keep the patient NPO (nothing by mouth) if they are vomiting, confused, or have an INR above 1.5, and notify the provider to consider a nasogastric tube for oral NAC or switch to IV NAC.
- Consult the regional poison control center (1-800-222-1222 in the United States) and notify the transplant service early if INR is greater than 2.0, creatinine is rising, pH is less than 7.30, or lactate is above 3.5 after fluid resuscitation.

Medicines you may be given — and why

Activated charcoal

Activated charcoal works like a powerful sponge in your stomach and intestines. Its surface is covered with millions of tiny holes that trap acetaminophen molecules before they can pass through the gut wall into your blood. Once trapped, the charcoal carries the poison out of your body in your stool. This only works if you give it early—within 1 hour after the person swallowed the pills—because after that, most of the acetaminophen has already been absorbed into the bloodstream.

N-acetylcysteine (NAC, Acetadote when given IV, Mucomyst when given by mouth)

N-acetylcysteine is the only antidote that saves the liver from acetaminophen poisoning. Here is how it works: normally, your liver breaks down acetaminophen using two safe pathways (sulfation and glucuronidation, like two clean garbage chutes), but a small amount goes through a third pathway run by an enzyme called CYP2E1 (mainly) and CYP3A4. This third pathway makes NAPQI, a highly toxic chemical. Your liver uses a molecule called glutathione (your body's main trash collector and shield) to immediately grab NAPQI, neutralize it, and flush it out. When you take too much acetaminophen, the safe pathways get overwhelmed, the liver makes much more NAPQI, and glutathione stores run empty—like a bucket with a hole draining faster than you can fill it. Without glutathione, NAPQI attacks and kills liver cells (hepatocytes) by destroying the proteins and membranes inside them, causing massive cell death (hepatic tissue death (necrosis)). NAC does three powerful things: First, it gives the liver cysteine, the key building block to make fresh glutathione and refill the empty tank. Second, NAC itself can bind directly to NAPQI and neutralize it, acting as a backup shield. Third, NAC improves blood flow (microcirculation) and oxygen delivery to the liver, helping damaged cells survive and repair. The earlier you give NAC, the more liver cells you save—it works best within 8 hours but should be given even days later because it still helps.

Where this information comes from

- American Association for the Study of Liver Diseases (AASLD) 2011 Position Paper on Acute Liver Failure
- Rumack-Matthew Nomogram (FDA-endorsed)
- American College of Medical Toxicology (ACMT) NAC dosing guidelines
- Cochrane Database of Systematic Reviews on acetaminophen poisoning management

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