

Acute Liver Failure

A plain-English guide for you and your family

WHAT IS HAPPENING

Your liver does three jobs that keep you alive every minute. First, it makes clotting factors—proteins that stop bleeding when you get cut. Second, it detoxifies ammonia, a poison made when your gut bacteria digest protein. Third, it stores sugar and releases it between meals to keep your brain and body running. Acute liver failure happens when something suddenly destroys most of your liver cells in weeks or less. The most common cause is acetaminophen (Tylenol) overdose, but viral hepatitis (A, B, or E), autoimmune attack, sudden loss of blood flow to the liver, mushroom poisoning, or Wilson disease (a genetic condition where copper builds up) can all do it. Once most liver cells die, all three jobs fail at once. Your blood stops clotting normally because clotting factors stop being made, so you bruise easily and bleed from your gums or nose. Ammonia piles up in your blood and poisons your brain, making you confused, sleepy, or—in severe cases—unconscious. Your blood sugar drops because the liver cannot release stored sugar. As toxins rise, your brain can swell dangerously, your blood pressure drops, your kidneys fail from low blood flow, and infections spread because your immune defenses weaken. Every strange symptom traces back to one catastrophe: your liver cells died faster than your body can adapt, and now multiple organs are failing in a cascade. This is a medical emergency. It requires intensive care and immediate evaluation for liver transplant, often within hours to days, because without a working liver the domino effect becomes unstoppable.

What you might feel

- Yellowing of the skin and eyes (Jaundice) (yellow skin, eyes, and under the tongue)
- Confusion, sleepiness, or coma (hepatic encephalopathy)
- Easy bruising, tiny red spots on the skin, bleeding gums, nosebleeds, or blood in vomit or stool
- Pain or tenderness below the right rib cage
- Nausea, vomiting, and complete loss of appetite
- Flapping hand tremor when arms are held out (asterixis)
- Fast heart rate (over 100 beats per minute) and low blood pressure (top number under 90 or mean arterial pressure under 65 mmHg)

- Very little urine (less than 400 mL in 24 hours or less than 0.5 mL per kilogram of body weight per hour for six hours or more)
- Rapid, deep breathing (Kussmaul respirations) or shortness of breath
- Swelling of the abdomen (ascites) or swelling of the ankles and legs (peripheral swelling (edema))

Call the doctor right away if...

- Stupor (arousable only to painful stimuli) or coma (no response to pain)—this is grade III or IV hepatic encephalopathy and signals high risk of brain swelling (cerebral swelling (edema)), pressure inside the skull rising above 20 mmHg, and the brain herniating (getting squeezed through the skull base). The person needs a breathing tube placed immediately, transfer to the ICU, and (in transplant centers) a device to monitor brain pressure.
- INR above 4.0 or active bleeding from the gut (vomiting blood, black tarry stool, or bright red blood from the rectum)—this means the blood has almost no clotting ability and bleeding can be deadly. Fresh frozen plasma, prothrombin complex concentrate, or recombinant factor VIIa must be given, and if bleeding is ongoing, a camera scope (endoscopy) must look inside the gut to find and stop the source.
- Blood sugar below 60 mg/dL despite continuous IV dextrose (sugar solution)—this means the liver has lost almost all ability to make new glucose (gluconeogenesis) or release stored glucose (glycogenolysis). Less than 10 percent of liver cells are still working. Without an emergency liver transplant, death is imminent.
- Creatinine above 2.0 mg/dL or climbing more than 0.3 mg/dL per day, plus very little urine —this strongly suggests hepatorenal syndrome type 1 or death of kidney tubule cells (acute tubular tissue death (necrosis)). Both mean multiple organs are failing. Without a liver transplant, 70 to 80 percent of patients will die.
- Blood pressure so low that it requires vasopressor drugs (norepinephrine or vasopressin) through an IV to keep mean arterial pressure at or above 65 mmHg—this is distributive shock and multiorgan dysfunction syndrome. The person must go to the ICU immediately for close monitoring and be evaluated urgently for emergency liver transplant. Without transplant, fewer than 20 out of 100 patients survive.

What the care team will do

- Check mental status every 1 to 2 hours using the Glasgow Coma Scale and document hepatic encephalopathy grade—alert (grade 0), confused (grade I-II), stuporous or only responding to pain (grade III), or no response to pain (grade IV)—and call the provider immediately if the grade worsens.

- Keep the head of the bed elevated 30 degrees at all times, avoid tight cervical collars or neck flexion, keep the room quiet and lights dim, and give sedation medicines only as ordered—never give benzodiazepines like lorazepam or diazepam because they worsen encephalopathy.
- Check blood glucose by fingerstick every 1 to 2 hours and hang continuous IV dextrose (D10 or D20 solution) to keep glucose between 100 and 150 mg/dL—never let it drop below 60 mg/dL.
- Inspect skin, gums, and all puncture sites every 2 to 4 hours for new bruises, pinpoint bleeding spots (petechiae) (tiny red dots), oozing, or active bleeding; use the smallest-gauge needles possible; apply pressure for 5 minutes after any blood draw or IV insertion; avoid rectal temperatures, suppositories, and intramuscular injections completely.
- Measure and record all urine output hourly with a Foley catheter, target at least 0.5 mL per kilogram per hour (about 30 to 40 mL per hour for an average adult), hang IV normal saline to match losses, and notify the provider if urine output drops below 0.3 mL/kg/hour for 2 hours in a row.
- Hang the N-acetylcysteine (NAC) IV infusion exactly as ordered—loading dose 150 mg/kg over 1 hour, then 50 mg/kg over 4 hours, then 100 mg/kg over 16 hours—and watch for anaphylactoid reactions (flushing, rash, wheezing, low blood pressure) in the first hour.
- Place the patient on contact-plus-droplet isolation (gown, gloves, mask) until hepatitis A, hepatitis B, and hepatitis E serology results return negative, and list the patient as NPO (nothing by mouth) with a bowel regimen (lactulose 30 mL every 6 hours by mouth or rectal enema) to produce 2 to 3 soft stools per day.
- Avoid giving any unnecessary medicines—especially sedatives, opioids, NSAIDs like ibuprofen, antibiotics like tetracycline or sulfa drugs, and herbal supplements—and double-check every ordered dose with pharmacy because the failing liver cannot metabolize (break down) most drugs, causing toxic buildup.

Medicines you may be given — and why

N-acetylcysteine (NAC) — for acetaminophen (Tylenol) poisoning and some other forms of sudden liver failure

Your liver uses a chemical called glutathione to neutralize poisons. Think of glutathione as a tiny sponge inside each liver cell that soaks up and disarms toxins before they can punch holes in the cell. When you take too much acetaminophen, your liver turns it into a toxic chemical called NAPQI (picture a sharp spike). Normally glutathione grabs every spike and makes it safe. But an overdose floods the liver with so many spikes that the glutathione sponges run out. Once the sponges are gone, free spikes stab liver cells and kill them. N-acetylcysteine gives your liver cysteine, the one ingredient your body cannot make fast enough on its own. With

fresh cysteine, liver cells quickly build new glutathione sponges in an hour or two. Those new sponges mop up the leftover spikes, so no more liver cells die, and the liver can start healing.

Where this information comes from

- American Association for the Study of Liver Diseases (AASLD) 2023 Guidance on Acute Liver Failure
- European Association for the Study of the Liver (EASL) Clinical Practice Guidelines: Acute Liver Failure 2017
- American College of Gastroenterology (ACG) Clinical Guideline: Diagnosis and Management of Acute Liver Failure 2020
- U.S. Acute Liver Failure Study Group (ALFSG)

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