

# Cirrhosis with Decompensation

Decompensated cirrhosis is when a scarred liver finally stops doing its jobs — blood backs up and bursts veins, fluid fills the belly, ammonia fogs the brain, kidneys shut down, and infections take over.

## THE ONE-LINE TRUTH

A scarred liver that can no longer hold things together.

## The chain — how it unfolds

1. Years of liver injury from alcohol, hepatitis, or fat buildup scar the liver into a hard, shrunken, lumpy organ. — why: Scar tissue replaces healthy liver cells and blocks the tiny blood channels inside the liver, so blood cannot flow through smoothly anymore. This is the ONE upstream break that causes everything else.
2. Portal vein pressure rises above 10 mmHg, creating portal high blood pressure (hypertension). — why: Blood flowing from the intestines and spleen tries to enter the liver but hits a wall of scar tissue. Pressure builds up like water behind a dam. The body cannot lower that pressure because the blockage is permanent.
3. Blood reroutes through thin, fragile collateral veins in the esophagus and stomach, forming varices. — why: The body opens backup routes to get blood around the liver and back to the heart. But these veins were never meant to handle high pressure or high flow, so they swell up like overinflated balloons. When pressure gets too high, or the person vomits or eats rough food, the varices burst and bleed — sometimes pouring a liter of blood into the stomach in minutes.
4. High portal pressure squeezes fluid out of blood vessels into the belly, causing fluid buildup in the belly (ascites). — why: The pressure pushes watery fluid (plasma) through the walls of capillaries in the intestines and liver surface. At the same time, the liver is making less albumin, so there is not enough protein to hold fluid inside the blood vessels. Gravity pulls the fluid down into the belly. The person's abdomen swells, they gain weight, and breathing gets harder because the fluid pushes up on the lungs.

5. The liver stops making enough clotting factors, so the person bleeds and bruises easily. — why: Clotting factors II, VII, IX, and X are made only in the liver. When the liver is too scarred to make them, even a tiny cut or needle stick can bleed for a long time. The INR (a measure of clotting time) goes up, often above 1.5 or even 2.0. This makes variceal bleeding even more dangerous because the blood will not clot to stop the bleeding.
6. Ammonia and other toxins build up in the blood and poison the brain, causing hepatic encephalopathy. — why: Normally, the liver breaks down ammonia (made by gut bacteria digesting protein) into urea, which the kidneys pee out. But a scarred liver cannot do that job, and some blood bypasses the liver entirely through collateral veins, so ammonia goes straight to the brain. Ammonia makes brain cells swell and stops them from sending signals properly. The person becomes confused, sleepy, forgetful, or even combative. In severe cases, they slip into a coma. Triggers include eating too much protein, getting constipated (so ammonia sits in the gut longer), bleeding into the gut (blood is protein), infection, or dehydration.
7. The kidneys shut down even though they are not diseased, causing hepatorenal syndrome. — why: The kidneys sense that blood flow is low because so much blood is backed up in the portal system or shunted through collateral veins. They think the body is dehydrated, so they release hormones that squeeze their own blood vessels shut to save water. This creates a vicious cycle: less blood flow means the kidneys clamp down harder, which makes blood flow even worse. Creatinine rises, urine output drops, and the person needs dialysis — but the kidneys themselves are fine. If the liver recovers or is transplanted, the kidneys wake up again.
8. Bacteria grow unchecked in the fluid in the belly or in the bloodstream, causing spontaneous bacterial peritonitis or sepsis. — why: The liver normally filters bacteria out of the blood and makes immune proteins. A failing liver cannot do either job well. Gut bacteria leak through the intestine wall into the fluid buildup in the belly (ascites) fluid, where they multiply freely because there are not enough white blood cells or antibodies to kill them. The person develops belly pain, fever, confusion, and low blood pressure. Without fast antibiotics, the infection spreads to the blood and becomes sepsis, which can be deadly.

## Spot it — what you see, and why

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- Fluid building up in the belly (ascites): The scarred liver cannot make enough albumin (a protein that holds fluid in the blood vessels), and high pressure in the portal vein (the main vein to the liver) pushes fluid out into the belly.
- Yellow skin and eyes (jaundice): The damaged liver cannot process bilirubin (a yellow waste from broken-down red blood cells), so it builds up in the blood and stains the skin and whites of the eyes.

- Confusion or sleepiness (hepatic encephalopathy): The failing liver cannot filter ammonia and other toxins from the blood, so these poisons travel to the brain and interfere with thinking and awareness.
- Vomiting blood or black, tarry stools: High pressure in the portal vein causes veins in the esophagus or stomach (varices) to swell like balloons and burst, leading to dangerous bleeding.
- Easy bruising or bleeding that won't stop: The liver makes clotting factors (proteins that help blood form clots), and when it fails, the body cannot plug holes in blood vessels quickly.
- Swollen legs and ankles (edema): Low albumin and high portal-vein pressure let fluid leak out of blood vessels into the tissues, especially in the legs where gravity pulls fluid down.

## Red flags — act now

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- Confusion, drowsiness, or not waking up easily (suggests toxins are affecting the brain)
- Vomiting blood or black, sticky stools like tar (life-threatening bleeding from swollen veins)
- Fever and belly pain with fluid buildup in the belly (ascites) (possible infection of the fluid in the belly)
- Making very little urine or none at all (kidneys may be failing)
- Trouble breathing or very fast breathing (fluid may be pressing on the lungs or infection is spreading)

## Treat it — in this order

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1. Octreotide IV drip + urgent upper endoscopy with band ligation or sclerotherapy for variceal bleeding → Octreotide squeezes blood vessels in the gut to slow bleeding. Banding physically ties off the torn veins so they stop leaking. Together they stop the person from bleeding to death and going into shock.
2. Ceftriaxone 2 g IV daily (or norfloxacin oral if stable) to prevent or treat spontaneous bacterial peritonitis (SBP) → Kills bacteria in the belly fluid and bloodstream before they cause sepsis (life-threatening infection). The damaged liver cannot fight infection well, so antibiotics buy time and prevent death.
3. Lactulose syrup (20–30 g every 1–2 hours until bowel movement, then adjusted) + rifaximin 550 mg twice daily for hepatic encephalopathy → Lactulose acidifies the gut and pulls ammonia out of the blood into stool. Rifaximin kills ammonia-making bacteria in the intestines. Together they lower brain ammonia and clear confusion.

4. IV albumin 1–1.5 g/kg on day 1, then 1 g/kg on day 3 + midodrine and octreotide (or terlipressin where available) for hepatorenal syndrome → Albumin fills blood vessels with protein so kidneys get enough blood flow. Midodrine and octreotide (or terlipressin) squeeze blood vessels to push blood pressure up and wake the kidneys before they fail permanently.
5. Spironolactone 100 mg daily + furosemide 40 mg daily (water pills) for fluid buildup in the belly (ascites), with low-salt diet (< 2 g sodium/day) → Water pills make the kidneys dump extra salt and water into urine, draining the belly and leg swelling. Low salt keeps fluid from building back up. This relieves breathing trouble and discomfort.

## Your top priorities at the bedside

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1. Check blood pressure, heart rate, and oxygen level every 1 to 2 hours if the patient has bleeding signs; place two large-bore IVs and keep type-and-cross blood ready. — why: Variceal bleeding (bleeding from swollen throat veins) can cause the patient to lose a large amount of blood very fast, dropping blood pressure and causing shock (not enough blood flow to organs); two IVs allow rapid fluid and blood transfusion to keep organs alive until endoscopy stops the bleeding.
2. Monitor mental status every 2 to 4 hours using a simple test; ask the patient to write their name or draw a star, and report any new confusion, sleepiness, or strange behavior immediately. — why: Hepatic encephalopathy (brain toxins from liver failure) can go from mild confusion to coma in hours; catching it early allows lactulose and rifaximin to clear ammonia (a toxin) from the bowels before brain swelling or coma happens.
3. Measure and record urine output every hour; notify the provider if urine is less than 30 milliliters per hour or if urine becomes very dark. — why: Low urine output means the kidneys are shutting down from hepatorenal syndrome (kidney failure caused by the sick liver) or dehydration; catching it early allows IV albumin and fluid adjustment to protect kidney function and prevent the need for dialysis.