

Cirrhosis

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THE STORY IN ONE SENTENCE

Long-term injury from alcohol, viral hepatitis, fatty liver disease, autoimmune attack, or inherited metal and metabolic problems slowly replaces healthy liver cells with hard scar tissue and bumpy regeneration nodules that cannot do the liver's work.

“Cirrhosis means the liver has been hurt for so long that healthy tissue turned into scar tissue. Blood cannot flow through properly. The liver cannot do its jobs. Problems stack up in waves.”

Normal anatomy — the body's job

The liver sits under your right ribs and does hundreds of jobs. It makes albumin to hold water in your blood, clotting factors to stop bleeding, and bile to digest fat. It cleans toxins, drugs, and ammonia from your blood. It stores sugar and vitamins. Blood flows in through the portal vein, moves slowly through tiny spongy channels past millions of liver cells that do the work, then flows out. Picture that slow, gentle flow through soft, spongy tissue—because in cirrhosis, scar tissue turns the liver hard and squeezes those channels shut.

BODY'S JOB

The liver is your body's factory and cleanup crew combined. It builds the proteins that keep fluid in your blood vessels and stop bleeding. It makes bile to help you digest fat. It breaks down every medicine and toxin you swallow, and it turns dangerous ammonia (made by gut bacteria) into safe urea that your kidneys can remove. If the liver stops working, toxins build up, you cannot clot blood, fluid leaks everywhere, and your brain gets foggy.

1. Blood carrying nutrients and bacteria flows from your intestines into the portal vein.
2. The portal vein carries blood into the liver at low pressure, like a gentle stream.

3. Inside the liver, blood moves slowly through millions of tiny spongy channels called sinusoids, past rows of liver cells.
4. Liver cells pull toxins, ammonia, and old hormones out of the blood and clean it.
5. Liver cells make albumin, clotting factors, and bile and release them into the blood or bile ducts.
6. Clean blood flows out of the liver through the hepatic vein into the heart.
7. Bile flows the opposite direction through tiny bile ducts, joins into bigger ducts, and empties into your intestine to help digest fat.

What breaks

Cirrhosis happens when something hurts the liver over and over for months or years—alcohol, hepatitis B or C virus, fatty liver from obesity or diabetes, autoimmune attack, or inherited problems like too much iron or copper. Each injury kills liver cells. The body tries to heal by laying down scar tissue, but scar tissue is stiff and useless—it cannot clean blood, make proteins, or let blood flow through. As scar piles up, it squeezes the blood channels inside the liver and chokes off flow. The liver shrinks, hardens, and gets bumpy with nodules trying to regrow. That stiff, scarred liver is the broken part—all the weird findings trace back to it.

CLINICAL TERM

Chronic hepatocellular injury from alcohol, viral hepatitis (HBV, HCV), metabolic dysfunction-associated steatohepatitis (MASH, formerly NAFLD/NASH), autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis, hemochromatosis, Wilson disease, alpha-1 antitrypsin deficiency, or chronic biliary obstruction triggers repeated cycles of tissue death (necrosis), inflammation, and fibrogenesis. Activated hepatic stellate cells deposit collagen, forming bridging fibrosis that distorts normal lobular architecture. Regenerative nodules surrounded by fibrous septa create a nodular, firm liver with loss of functional hepatocyte mass and disruption of sinusoidal blood flow. This structural distortion increases intrahepatic vascular resistance, leading to portal high blood pressure (hypertension). Simultaneously, the reduced functional hepatocyte mass causes synthetic failure (decreased albumin, clotting factors), impaired detoxification (hyperammonemia, drug accumulation), and cholestasis (hyperbilirubinemia). The upstream break is irreversible architectural distortion; the downstream manifestations are portal high blood pressure and hepatic insufficiency.

How the body compensates

The body tries hard to keep blood pressure, oxygen delivery, and balance steady even as the liver fails. It opens backup blood routes around the liver, holds onto salt and water to keep

blood volume up, and shifts blood flow. For weeks or months, vital signs can look okay and the patient feels tired but stable. That is the dangerous middle—because the body is working at max effort and one more hit (infection, bleeding, dehydration) can tip everything into a sudden crash. The job is to see that cliff coming before the patient goes over it.

1. The body opens collateral veins (backup routes) to let blood bypass the clogged liver and return to the heart, but these veins are weak and balloon into varices in the esophagus, stomach, and rectum.
2. The spleen swells because backed-up blood pools there, trapping and destroying platelets, which lowers the platelet count but partly protects fragile varices from clotting and rupturing early.
3. The kidneys sense low effective blood flow (because blood is pooling in the belly and dilated vessels) and hold onto salt and water, raising blood volume temporarily but pushing more fluid into the belly as fluid buildup in the belly (ascites).
4. The heart pumps faster and blood vessels dilate to try to keep oxygen delivery up, creating a hyperdynamic circulation with warm skin and bounding pulses even though effective blood flow to organs is falling.
5. The liver tries to regenerate by forming nodules, but these nodules are disorganized, do not restore normal architecture, and actually worsen the scarring and blood flow obstruction.
6. Gut bacteria make more ammonia, but the diseased liver cannot convert it to urea, so the body shifts some ammonia clearance to muscles (which is why muscle wasting accelerates) and kidneys, buying a little time.
7. These compensations keep the patient looking stable—normal or near-normal blood pressure, okay oxygen levels, awake and talking—while the liver and organ reserve silently drain away.
8. The crash point comes when varices rupture and bleed massively, infection (spontaneous bacterial peritonitis) tips the balance, the kidneys fail (hepatorenal syndrome), or the brain shuts down (hepatic encephalopathy grade III–IV). Once that happens, the window to save the patient without a transplant closes fast.
9. Watch trends, not snapshots: rising creatinine, worsening confusion, new infection, dropping platelets, or growing fluid buildup in the belly (ascites) are the cliff edge approaching. A stable-looking patient on a downward slope is not truly stable.

Domino effect

1. Scar tissue blocks blood flow through the liver, so pressure builds up in the portal vein. — Imagine water flowing through a sponge. Scar tissue turns the sponge into a hard rock, so blood cannot seep through the tiny channels anymore. Pressure upstream in the portal vein climbs from normal (5–8 mmHg) to high (>10 mmHg), like a clog in a drainpipe.

2. High portal pressure forces blood to find backup routes around the liver, ballooning weak veins into varices. — Blood backs up and reroutes through small veins in the esophagus (food tube), stomach, and rectum. These veins are not built for high pressure, so they swell into fragile, thin-walled varices that can burst and bleed suddenly, causing life-threatening bleeding (hemorrhage).
3. High pressure pushes watery fluid out of blood vessels into the belly, forming fluid buildup in the belly (ascites). — High pressure in the portal vein and low albumin (because the liver cannot make enough) let fluid leak out of blood vessels and pool in the abdominal cavity. The kidneys sense low blood flow and hold onto salt and water, making the fluid buildup in the belly (ascites) worse—a vicious cycle.
4. The spleen swells and traps platelets, so the platelet count drops. — Backed-up blood from the blocked portal vein pools in the spleen, stretching it. The swollen spleen (splenomegaly) traps and destroys platelets, causing a low platelet count (thrombocytopenia) (low platelet count), which adds to bleeding risk.
5. The liver cannot make enough albumin or clotting factors, so fluid leaks everywhere and bleeding starts easily. — Albumin is the sticky protein that holds water inside blood vessels; without enough, water leaks into tissues (peripheral swelling (edema) in legs, fluid buildup in the belly (ascites) in belly). Clotting factors (II, VII, IX, X) are made only in the liver; without them, blood cannot clot properly, so patients bruise easily, bleed from gums, or bleed dangerously after procedures.
6. The liver cannot clean ammonia and toxins, so they build up and poison the brain, causing hepatic encephalopathy. — Gut bacteria make ammonia. Normally the liver converts ammonia to urea (which kidneys remove safely). In cirrhosis, ammonia and other toxins bypass the liver through collateral veins or the liver simply cannot process them. Ammonia crosses into the brain and disrupts thinking, causing confusion, tremor (asterixis, a flapping hand tremor), slurred speech, sleepiness, and in severe cases, coma.
7. Bile backs up inside the liver, spilling bilirubin into the blood and turning skin and eyes yellow (jaundice). — Scar tissue squeezes bile ducts and liver cells cannot process bilirubin properly. Bilirubin (a yellow pigment from broken-down red blood cells) builds up in the blood, deposits in skin and eyes (jaundice), and spills into urine (dark, tea-colored urine). Bile salts in the skin cause intense itching.
8. The kidneys sense low blood flow, clamp down, and can spiral into hepatorenal syndrome, a form of kidney failure with no kidney disease. — Blood pools in dilated vessels and ascitic fluid, so less reaches the kidneys. The kidneys think the body is dehydrated and constrict their blood vessels to save water, which paradoxically drops kidney blood flow even more. Creatinine rises, urine output falls, but the kidneys themselves are not damaged—this is hepatorenal syndrome, driven purely by cirrhosis and circulatory dysfunction. It is reversible only with liver transplant or sometimes with vasoconstrictors plus albumin.

Symptoms · what the patient feels, and why

- **Yellow skin and eyes (jaundice):** The damaged liver cannot clear bilirubin (a yellow waste product from old red blood cells) from the blood, so it builds up and stains the skin and whites of the eyes.
- **Swollen belly (ascites):** Scar tissue blocks blood flow through the liver, so pressure builds up in the vein bringing blood from the intestines. This high pressure forces watery fluid out of blood vessels and into the belly.
- **Spider-shaped blood vessels on the chest and shoulders (spider angiomas):** The sick liver cannot break down estrogen (a hormone) properly, so estrogen levels rise and cause tiny blood vessels near the skin to swell and branch out like spider legs.
- **Swollen veins on the belly that look like twisted ropes (caput medusae):** High pressure in the blocked liver veins forces blood to find other paths around the liver, pushing belly-wall veins to swell and bulge under the skin.
- **Breast growth in men (gynecomastia):** The damaged liver cannot break down estrogen, so levels rise in men and cause breast tissue to grow.
- **Shaking hands (asterixis or flapping tremor):** The liver cannot clean ammonia (a toxic waste from digesting protein) out of the blood, so ammonia builds up and poisons the brain, causing the hands to jerk and flap when held out.
- **Red palms (palmar redness (erythema)):** High estrogen levels make blood vessels in the palms widen, turning the hands bright red, especially the base of the thumb and pinky.
- **Easy bruising and bleeding:** The liver makes clotting proteins that stop bleeding. When the liver is badly scarred, it cannot make enough of these proteins, so even small bumps cause bruises and cuts bleed longer.
- **Confusion or sleepiness (hepatic encephalopathy):** Ammonia and other toxins that the damaged liver cannot filter out build up in the bloodstream, travel to the brain, and slow down thinking, memory, and alertness.
- **Throwing up blood or black tarry stools:** High pressure in blocked liver veins causes veins in the esophagus (food tube) to swell like balloons (varices). These swollen veins can burst and bleed heavily into the stomach or intestines.

Focused exam

- **Yellow skin and whites of eyes (jaundice):** When red blood cells die, they release a yellow waste called bilirubin. A healthy liver cleans this out of the blood. In cirrhosis, scar tissue replaces working liver cells, so bilirubin piles up and stains the skin yellow. This tells you the liver has lost its ability to do its cleanup job.

- Spider-shaped blood vessels on the chest, face, and arms (spider angiomas): Scar tissue in the liver squeezes the portal vein (the big vein that carries blood from the gut to the liver). Pressure builds up, so blood finds new paths through tiny vessels in the skin. These spider shapes show portal pressure is high—a hallmark of cirrhosis.
- Belly swollen with fluid that sloshes when you tap it (ascites): High portal pressure pushes fluid out of blood vessels into the belly. At the same time, the sick liver cannot make enough albumin (a protein that holds water in the blood). Fluid buildup in the belly (Ascites) means cirrhosis is advanced and the liver is struggling to keep up.
- Large, firm spleen felt below the left rib cage (splenomegaly): When portal pressure rises, blood backs up into the spleen. The spleen swells and traps platelets, which can lead to easy bruising or bleeding. A big spleen is a reliable sign that portal pressure is high in cirrhosis.
- Bright red palms and white, flat nails (palmar redness (erythema) and Terry's nails): The failing liver cannot break down hormones that widen blood vessels in the hands. Red palms and pale nails are clues that liver function is poor and the body's balance of hormones and proteins is off.
- Hand tremor (asterixis) and confusion or sleepiness: When the liver fails, it cannot clean ammonia and other toxins from the blood. These toxins reach the brain and cause confusion, sleepiness, or a flapping tremor of the hands. This is hepatic encephalopathy, a sign of severe liver failure.
- Vomiting bright red blood or passing black, tarry stool (melena): High portal pressure makes veins in the esophagus and stomach swell into fragile balloons called varices. When a varix bursts, the patient bleeds heavily into the gut. This is a life-threatening emergency and requires immediate help to stop the bleeding.

Diagnostics · every test answers a question

- Complete blood count (CBC) → Are platelets low? Is the patient anemic from bleeding?
- Comprehensive metabolic panel (CMP) with liver enzymes (ALT, AST, bilirubin, albumin, alkaline phosphatase) → How badly is the liver damaged? Is it still making protein?
- Prothrombin time (PT) and INR → Can the liver still make clotting factors? Is the patient at risk for bleeding?
- Abdominal ultrasound with Doppler → Is the liver scarred and nodular? Is blood flow through the portal vein backward or slow?
- Paracentesis (belly tap) with ascitic fluid cell count, albumin, and culture → Is the belly fluid infected? Is it from cirrhosis or something else?
- Upper endoscopy (esophagogastroduodenoscopy, EGD) → Are there varices in the esophagus or stomach? Are they at risk of bleeding?

- Hepatitis panel (HBsAg, anti-HCV) and alcohol use history → What caused the cirrhosis?
- Alpha-fetoprotein (AFP) and liver ultrasound every 6 months → Is liver cancer developing?

Treatment logic

- Screen with upper endoscopy and prevent variceal bleeding with non-selective beta-blocker (propranolol or nadolol) or endoscopic variceal ligation (banding) → High portal pressure pushes blood backward into fragile veins in the esophagus, making them swell into varices. Beta-blockers lower the pressure inside these veins by slowing the heart and narrowing blood vessels. Banding physically ties off the varices so they cannot bleed. Both cut the risk of life-threatening bleeding by about half.
- Restrict salt to < 2 grams per day and give spironolactone (aldosterone antagonist) with furosemide (loop diuretic); if fluid buildup in the belly (ascites) is tense or > 5 liters, do large-volume paracentesis and give albumin 6–8 g per liter removed → The scarred liver cannot make albumin, so water leaks out of blood vessels into the belly. Salt holds onto water, making it worse. Spironolactone blocks a hormone (aldosterone) that tells the kidneys to keep salt; furosemide directly tells the kidneys to dump salt and water. Together, they pull fluid back into the blood. Paracentesis drains large amounts of fluid directly, and albumin keeps blood pressure stable during the drain.
- If ascitic fluid shows > 250 neutrophils/ μ L, start ceftriaxone 2 g IV daily (or cefotaxime); give albumin 1.5 g/kg on day 1 and 1 g/kg on day 3 → Bacteria sneak into ascitic fluid because the immune system is weak in cirrhosis. This infection (spontaneous bacterial peritonitis, SBP) can spread to the blood and cause sepsis. Antibiotics kill the bacteria. Albumin boosts blood volume and helps the kidneys work, which lowers the risk of kidney failure during infection.
- For hepatic encephalopathy, give lactulose 15–30 mL twice daily (titrate to 2–3 soft stools per day) and add rifaximin 550 mg twice daily → When the liver fails, ammonia from gut bacteria builds up in the blood and poisons the brain, causing confusion or coma. Lactulose is a sugar the body cannot absorb; it traps ammonia in the stool and pulls it out. Rifaximin is an antibiotic that stays in the gut and kills ammonia-making bacteria. Together, they lower brain toxin levels and clear confusion.
- For hepatorenal syndrome type 1, give albumin 1 g/kg on day 1 (max 100 g), then 20–40 g/day, plus vasoconstrictor (terlipressin, midodrine + octreotide, or norepinephrine); consider dialysis as bridge to transplant → In advanced cirrhosis, blood vessels in the gut widen and steal blood from the kidneys. The kidneys shut down even though they are structurally normal (hepatorenal syndrome). Albumin expands blood volume, and vasoconstrictors squeeze gut vessels to push blood back to the kidneys. This can restart urine flow and buy time for transplant.

- Calculate MELD-Na score (based on bilirubin, INR, creatinine, and sodium) and refer to liver transplant center when MELD ≥ 15 or if complications (variceal bleeding, SBP, encephalopathy, hepatorenal syndrome) occur → MELD-Na predicts 3-month mortality in cirrhosis. Patients with higher scores are sicker and die sooner without a transplant, so they get priority on the transplant list. Liver transplant is the only treatment that replaces dead, scarred liver with a healthy, working liver and can cure cirrhosis.
- Screen for hepatocellular carcinoma every 6 months with abdominal ultrasound $\pm$ alpha-fetoprotein (AFP); if tumor found, refer for resection, ablation, transplant, or transarterial chemoembolization depending on size and liver function → Cirrhosis raises the risk of liver cancer 20-fold. Screening catches tumors when they are small (< 3 cm) and confined to the liver, which means surgery, ablation, or transplant can cure it. Early detection improves survival.

Medications

Propranolol · Non-selective beta blocker

Lowers the pressure inside the veins that connect your gut to your liver (called portal veins). When the liver is scarred, blood backs up and these veins swell like overfilled balloons. Propranolol slows your heart and relaxes these veins so the pressure drops and the veins are less likely to burst and bleed.

1. Propranolol blocks beta receptors (tiny switches) on heart muscle and blood vessel walls.
2. Heart beats more slowly and with less force, so it pumps less blood into the portal system each minute.
3. Blood vessels in the gut and spleen relax and widen, so blood pools there instead of forcing its way through the scarred liver.
4. Pressure in the portal vein drops by about 10–20%, which shrinks the swollen veins (varices) in the esophagus and stomach and makes them less likely to tear open.

SAFETY WATCH

Can make asthma worse by tightening airways; do not use if you have active wheezing or severe lung disease. · Can slow heart rate too much (under 50 beats per minute) or drop blood pressure and make you dizzy. · Can mask low blood sugar warnings in people with diabetes. · Check heart rate and blood pressure before each dose increase; goal is to lower resting heart rate by about 25% without dropping blood pressure below 90/60.

Nadolol · Non-selective beta blocker

Works the same way as propranolol: slows the heart and relaxes gut blood vessels to lower pressure in the swollen veins around the esophagus and stomach. Nadolol lasts longer in the body, so you take it once a day instead of twice.

1. Nadolol blocks beta receptors on heart and blood vessel cells.
2. Heart rate drops and the amount of blood pumped per minute falls.
3. Gut and spleen blood vessels relax and hold more blood, relieving pressure in the portal vein.
4. Pressure in esophageal varices (swollen veins) drops, lowering the risk they will rupture and bleed.

SAFETY WATCH

Avoid in severe asthma or COPD with active wheezing. · Monitor heart rate and blood pressure; stop if heart rate falls below 50 or blood pressure drops too low. · Can hide signs of low blood sugar in diabetes. · Long half-life means it stays in the body longer; missed doses or sudden stops can cause rebound fast heart rate.

Carvedilol · Non-selective beta blocker with alpha-blocking activity

Lowers portal vein pressure even more than propranolol because it works two ways: slows the heart (beta block) and also relaxes the tiny muscles around liver blood vessels (alpha block). This double action drops pressure harder and may work better to prevent bleeding from varices.

1. Carvedilol blocks beta receptors on the heart, slowing heart rate and reducing the volume of blood pushed into the portal system.
2. It also blocks alpha-1 receptors on blood vessel walls, which relaxes the muscles and widens the vessels.
3. Blood flow meets less resistance inside and around the liver, so pressure in the portal vein drops by 15-25%.
4. Lower pressure shrinks varices and reduces bleeding risk more than propranolol or nadolol in many studies.

SAFETY WATCH

Can drop blood pressure more than other beta blockers; start at a low dose and check blood pressure sitting and standing. · Avoid in severe asthma or decompensated heart failure. · Use cautiously if kidney function is poor; dose may need adjustment. · Monitor heart rate; stop if it falls below 50 or if dizziness or fainting occurs.

Spironolactone · Aldosterone antagonist (potassium-sparing diuretic)

Blocks aldosterone, a hormone that tells the kidneys to hold onto salt and water. In cirrhosis, aldosterone levels are too high, so the body keeps piling up fluid in the belly (ascites). Spironolactone flips that switch off so the kidneys let go of salt and water through urine, which shrinks the belly fluid.

1. Spironolactone enters kidney cells and blocks aldosterone receptors.
2. Without aldosterone's signal, the kidney stops reabsorbing sodium from the urine back into the blood.
3. Sodium stays in the urine and pulls water with it (water follows salt).
4. More urine is made, less fluid stays in the body, and fluid buildup in the belly (ascites) shrinks over days to weeks.
5. Potassium is kept in the blood instead of lost in urine, which is why this drug is called potassium-sparing.

SAFETY WATCH

Can raise blood potassium to dangerous levels (hyperkalemia), especially if kidney function is poor or if you eat a lot of high-potassium foods (bananas, oranges, potatoes). Check potassium and creatinine every few days when starting, then weekly. · Can cause painful breast swelling (gynecomastia) in men because it blocks some testosterone receptors. · Stop if potassium goes above 5.5 mEq/L or creatinine rises sharply (sign of kidney injury). · Do not use with other potassium-sparing diuretics or potassium supplements unless closely monitored.

Furosemide · Loop diuretic

Blocks a sodium pump in the kidneys so salt and water pour out into the urine. Works faster and stronger than spironolactone. In cirrhosis, furosemide is paired with spironolactone (usual ratio 40 mg furosemide to 100 mg spironolactone) to drain fluid buildup in the belly (ascites) more quickly while keeping potassium balanced.

1. Furosemide enters the kidney tubule and blocks the Na-K-2Cl cotransporter (a pump that pulls sodium, potassium, and chloride back into the blood).
2. All three salts stay in the urine instead of being reabsorbed.
3. Water follows the salts, so a large volume of dilute urine is produced within an hour.
4. Belly fluid shrinks as the kidneys pull water from the blood, which then pulls water from the abdomen into blood vessels to be filtered and excreted.

SAFETY WATCH

Can drop potassium, sodium, and magnesium too low, especially when used alone; this is why it is always given with spironolactone in cirrhosis. · Can worsen kidney function if too much fluid is pulled off too fast (pre-renal injury); monitor creatinine and stop diuretics if creatinine rises more than 50% or weight drops more than 1 kg per day. · Can cause dizziness and fainting if blood pressure falls too low. · Check electrolytes (sodium, potassium, magnesium) and kidney function every few days when starting, then weekly.

Albumin (intravenous) · Plasma volume expander / colloid

A protein solution given through an IV to pull fluid back into blood vessels and keep blood pressure stable. In cirrhosis, albumin is used three ways: (1) after large-volume paracentesis (draining more than 5 liters of belly fluid) to prevent shock, (2) with antibiotics during spontaneous bacterial peritonitis to protect the kidneys, and (3) in hepatorenal syndrome to help restore kidney blood flow.

1. Albumin is a large protein that cannot cross blood vessel walls easily.
2. When infused into a vein, it increases the protein concentration in the blood.
3. Water moves from the belly or tissues into the blood to balance the concentration (osmosis).
4. Blood volume rises, blood pressure stabilizes, and kidneys get better blood flow so they are less likely to shut down.
5. Also binds toxins and inflammatory molecules, which may reduce kidney and brain injury in infection.

SAFETY WATCH

Can cause fluid overload and heart failure if given too fast or in too large a volume; watch for shortness of breath, leg swelling, or rapid weight gain. · Rarely, allergic reactions (rash, itching, low blood pressure) can occur. · Expensive and derived from human plasma, so reserve for guideline-backed indications: post-paracentesis (8 g per liter removed if >5 L), SBP (1.5 g/kg day 1, then 1 g/kg day 3), and hepatorenal syndrome (dose varies by protocol).

Lactulose · Osmotic laxative / ammonia-lowering agent

A synthetic sugar that gut bacteria break down into acids. These acids do two things: (1) trap ammonia (a toxin that causes confusion) in the colon so it leaves in stool instead of entering the blood, and (2) speed up bowel movements so less ammonia is made and absorbed. Lowers blood ammonia and clears brain fog (hepatic encephalopathy).

1. Lactulose is not absorbed in the small intestine; it travels to the colon intact.

2. Colon bacteria ferment lactulose into lactic acid and acetic acid.
3. These acids lower the pH (make the colon more acidic).
4. Ammonia (NH_3) in the colon is converted to ammonium (NH_4^+), which is charged and cannot cross back into the blood—it stays trapped in stool.
5. Acids also pull water into the colon (osmosis), which softens stool and triggers bowel movements, flushing out ammonia before bacteria can make more.

SAFETY WATCH

Can cause bloating, cramping, and diarrhea; dose is adjusted so the patient has 2–3 soft bowel movements per day—not watery diarrhea. · Too much diarrhea can dehydrate the patient and worsen kidney function or electrolyte imbalance. · Sweet taste can be hard to tolerate; can be mixed with juice or given by enema if the patient cannot swallow. · Does not work immediately—takes 24–48 hours to see improvement in confusion.

Rifaximin · Non-absorbable antibiotic

An antibiotic that stays in the gut and kills bacteria that produce ammonia. Less ammonia means less confusion. Rifaximin is added to lactulose when lactulose alone does not fully control encephalopathy or to prevent encephalopathy from coming back.

1. Rifaximin binds to bacterial RNA polymerase (the enzyme bacteria use to copy their genes).
2. Bacteria cannot make the proteins they need to grow and divide, so they die.
3. Fewer bacteria in the colon means less ammonia is produced from dietary protein.
4. Blood ammonia levels drop, and brain function improves.

SAFETY WATCH

Very safe because it is not absorbed; side effects are rare (mild nausea or headache). · Expensive; insurance may require prior authorization and proof that lactulose alone was tried first. · Should be used with lactulose, not instead of it, in most patients. · Stop if the patient develops severe diarrhea or *Clostridioides difficile* infection (though this is very rare with rifaximin).

Ceftriaxone · Third-generation cephalosporin antibiotic

Kills bacteria that infect the fluid in the belly (spontaneous bacterial peritonitis, or SBP). In cirrhosis, gut bacteria can leak into fluid buildup in the belly (ascites) and multiply. Ceftriaxone crosses into ascitic fluid and breaks down bacterial cell walls so the bacteria burst and die.

1. Ceftriaxone enters the bloodstream after IV or IM injection.
2. It diffuses into ascitic fluid and reaches bacteria floating there.

3. The drug binds to penicillin-binding proteins on the bacterial cell wall.
4. Bacteria cannot build or repair their cell walls, so they swell, rupture, and die.
5. Covers the most common SBP bacteria: *E. coli*, *Klebsiella*, and *Streptococcus* species.

SAFETY WATCH

Can cause allergic reactions, especially in patients allergic to penicillin (about 5–10% cross-reactivity); ask about penicillin allergy before giving. · Can cause diarrhea or *Clostridioides difficile* infection if used for a long time. · Can displace bilirubin from albumin in newborns (do not use in babies under 28 days); not relevant in adults with cirrhosis. · Dose does not need adjustment for liver disease, but reduce dose if kidney function is very poor (GFR under 15).

Norfloxacin · Fluoroquinolone antibiotic

An oral antibiotic used to prevent spontaneous bacterial peritonitis from happening again (secondary prevention (prophylaxis)) after a first episode, or to prevent a first episode in high-risk patients (those with very low ascitic protein or GI bleeding). Kills gut bacteria before they can leak into the belly fluid.

1. Norfloxacin is absorbed from the small intestine and enters the bloodstream.
2. It crosses into ascitic fluid and gut mucosa.
3. The drug blocks bacterial DNA gyrase and topoisomerase IV (enzymes bacteria need to unwind and copy their DNA).
4. Bacteria cannot divide, and they die.
5. Reduces the number of gut bacteria that could translocate (cross the gut wall) into fluid buildup in the belly (ascites).

SAFETY WATCH

Can cause tendon rupture (especially Achilles tendon) in older adults; stop if tendon pain or swelling occurs. · Can prolong the QT interval (heart rhythm issue); avoid in patients taking other QT-prolonging drugs or with known long QT. · Can cause nausea, headache, or dizziness. · Increasing bacterial resistance is a concern; some centers now use trimethoprim-sulfamethoxazole or ceftriaxone instead for prevention (prophylaxis).

Terlipressin · Vasopressin analog (splanchnic vasoconstrictor)

Tightens blood vessels in the gut and spleen so more blood flows to the kidneys instead. In hepatorenal syndrome, the kidneys shut down because blood is pooling in the belly vessels and

not reaching the kidneys. Terlipressin shifts blood flow back to the kidneys, which helps them start working again. Always given with albumin.

1. Terlipressin is a prodrug that is slowly converted to vasopressin (a hormone that tightens blood vessels).
2. It binds to V1 receptors on smooth muscle cells in gut blood vessels.
3. Those vessels constrict (narrow), so blood is squeezed out of the gut and spleen.
4. More blood returns to the heart and is pumped to the kidneys.
5. Kidney blood flow and filtration improve, creatinine drops, and urine output rises over 3–14 days.

SAFETY WATCH

Can cause starved blood flow (ischemia) (not enough blood) in the heart, gut, fingers, or toes because it tightens all arteries; watch for chest pain, belly pain, or cold, painful fingers. · Can cause dangerous heart rhythm problems or raise blood pressure too high. · Not approved in the United States as of 2025; used in Europe and other countries. In the US, norepinephrine or midodrine plus octreotide are alternatives. · Always give with IV albumin; terlipressin alone is less effective.

Norepinephrine · Vasopressor (alpha- and beta-adrenergic agonist)

Tightens blood vessels throughout the body, which raises blood pressure and pushes more blood to the kidneys. Used in hepatorenal syndrome when terlipressin is not available. Given through a central IV line in the ICU, along with albumin, to restore kidney function.

1. Norepinephrine binds to alpha-1 receptors on blood vessel smooth muscle.
2. Vessels constrict, blood pressure rises, and blood is redirected from low-priority areas (gut, skin) to vital organs (kidneys, brain, heart).
3. Kidney blood flow and filtration improve.
4. Also stimulates beta-1 receptors on the heart to increase heart rate and force, which helps pump blood to the kidneys.

SAFETY WATCH

Must be given through a central line in the ICU; can cause tissue death (necrosis) if it leaks out of a vein. · Can cause dangerous high blood pressure, fast heart rate, or irregular heart rhythms. · Can reduce blood flow to fingers, toes, or gut if dose is too high or used too long. · Requires continuous monitoring of blood pressure, heart rate, and urine output. · Always use with IV albumin.

Midodrine · Alpha-1 adrenergic agonist (oral vasopressor)

An oral pill that tightens blood vessels to raise blood pressure and improve kidney blood flow. Used off-label in hepatorenal syndrome, usually combined with octreotide and albumin. Less powerful than IV vasopressors but can be given outside the ICU.

1. Midodrine is absorbed from the gut and converted in the body to an active metabolite.
2. The metabolite binds to alpha-1 receptors on blood vessel smooth muscle.
3. Vessels constrict, blood pressure rises, and more blood reaches the kidneys.
4. Used three times a day, dose is increased every few days until blood pressure improves or maximum dose is reached.

SAFETY WATCH

Can cause dangerously high blood pressure when lying flat; take sitting or standing, and check blood pressure regularly. · Can cause scalp tingling, goosebumps, or chills (piloerection) from vessel constriction. · Not as effective as terlipressin or norepinephrine; often used as a bridge or when ICU care is not available. · Always combine with octreotide and albumin for hepatorenal syndrome.

Octreotide · Somatostatin analog

A synthetic version of somatostatin, a hormone that tightens blood vessels in the gut. Reduces blood flow to the gut and spleen so more blood flows to the kidneys. Also lowers portal pressure. Used in hepatorenal syndrome (with midodrine and albumin) and to control acute variceal bleeding (with endoscopy).

1. Octreotide binds to somatostatin receptors on blood vessels in the gut.
2. Blood vessels constrict, reducing blood flow to the gut and spleen.
3. Portal vein pressure drops, and more blood is available for the kidneys and other organs.
4. Also reduces secretion of gut hormones (like glucagon) that dilate blood vessels, which helps maintain blood pressure.

SAFETY WATCH

Can cause nausea, diarrhea, or belly cramps. · Can raise or lower blood sugar unpredictably (blocks insulin and glucagon); monitor glucose in diabetics. · Can rarely cause gallstones if used long-term. · Given as a continuous IV infusion or subcutaneous injection; always use with albumin (and midodrine in hepatorenal syndrome).

Red flags

- Throwing up bright red blood or coffee-ground material (bleeding from swollen veins in the food tube)
- Confusion, extreme sleepiness, or not making sense (ammonia poisoning the brain)
- Black, sticky, tar-like stools (bleeding inside the intestines)
- Sudden belly pain with fever (infection of belly fluid)
- Yellowing skin getting much worse quickly (liver failing fast)

Nursing priorities

1. Check mental status every 2 to 4 hours using a simple tool like asking the patient to draw a five-pointed star or copy overlapping circles; report any new confusion, sleepiness, or hand flapping (asterixis) immediately. — Ammonia crosses into the brain when the liver cannot clean it from blood; early hepatic encephalopathy (brain poisoning) can progress in hours to coma and brain swelling; lactulose must start right away to make the patient have 2 to 3 soft bowel movements per day and trap ammonia in stool.
2. Monitor for any vomiting and immediately check if the material is bright red, dark red, or looks like coffee grounds; keep the patient NPO (nothing by mouth), place two large-bore IVs (18-gauge or bigger), and call the provider stat for possible endoscopy. — Variceal bleeding (burst swollen veins in the food tube) kills 15 to 20 percent of patients in the first six weeks; octreotide (a vein-squeezing hormone) and antibiotics like ceftriaxone must start before endoscopy; blood transfusion target is hemoglobin 7 to 8 grams per deciliter because higher levels raise pressure in the portal vein and cause re-bleeding.
3. Measure and record belly size at the belly button (umbilicus) every day at the same time; weigh the patient daily before breakfast on the same scale; report weight gain over 2 pounds in one day or belly size increase over 2 centimeters. — Fluid buildup in the belly (Ascites) (belly fluid) builds up because the scarred liver cannot make albumin (a blood protein that holds water in vessels) and portal high blood pressure (hypertension) (high pressure in the liver vein) pushes fluid out; tracking helps adjust spironolactone (an aldosterone-blocking diuretic) and furosemide (a loop diuretic) doses; goal is weight loss of 0.5 to 1 pound per day.
4. Restrict sodium (salt) to less than 2 grams per day by teaching the patient to avoid processed foods, canned soups, deli meats, cheese, bread, and restaurant meals; read all food labels and count milligrams. — Aldosterone (a salt-saving hormone) is high in cirrhosis because the liver cannot break it down; extra salt makes the kidneys hold water and worsens fluid buildup in the belly (ascites); sodium restriction works better than diuretics alone and prevents the need for repeated paracentesis (belly fluid drainage).

5. Check for fever every 4 hours and report any temperature over 100.4 degrees Fahrenheit or new belly pain; prepare for diagnostic paracentesis (needle sample of belly fluid) to count white blood cells and culture bacteria. — Spontaneous bacterial peritonitis (belly fluid infection) happens because bacteria from the gut leak into fluid buildup in the belly (ascites) and the sick liver cannot make antibodies (infection-fighting proteins); even without symptoms, 10 to 30 percent of hospitalized cirrhosis patients have this infection; untreated infection causes septic shock and kidney failure (hepatorenal syndrome).
6. Inspect skin daily for new yellow color (jaundice), bruises, or small red spider-shaped blood vessels (spider angiomas); check sclera (white part of eyes) for yellow tint; report worsening yellowing of the skin and eyes (jaundice) or new bruising. — Bilirubin (a yellow waste product from broken-down red blood cells) builds up when the damaged liver cannot process it; rising bilirubin means liver function is getting worse; easy bruising happens because the liver cannot make clotting factors (proteins that stop bleeding) and the spleen destroys platelets (clotting cells).
7. Teach the patient to avoid all alcohol, acetaminophen over 2 grams per day, NSAIDs like ibuprofen and naproxen, and any herbal supplements without provider approval; review the medication list and stop any unnecessary drugs. — Alcohol causes direct liver cell death and inflammation even in small amounts; acetaminophen (Tylenol) is safe at low doses but toxic over 2 to 3 grams per day when the liver is damaged; NSAIDs (non-steroidal anti-inflammatory drugs) cause kidney failure and stomach bleeding in cirrhosis; many herbs and supplements contain toxins the sick liver cannot clear.
8. Ensure the patient eats small frequent meals with at least 1.2 to 1.5 grams of protein per kilogram of body weight per day and a late-evening snack with complex carbohydrates; consult nutrition if the patient is losing muscle mass. — Cirrhosis speeds up metabolism and breaks down muscle (sarcopenia) for energy because the liver cannot store sugar (glycogen); protein restriction is outdated and dangerous because it worsens muscle loss and does not prevent hepatic encephalopathy; branched-chain amino acids (special proteins in meat, eggs, and dairy) improve survival and brain function.

Sources

- AASLD Practice Guidance on Cirrhosis 2021
- Hepatology journal (official AASLD publication)
- Cochrane Hepato-Biliary Group
- AGA Clinical Practice Update on Cirrhosis 2022

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