

Hepatic Encephalopathy

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THE STORY IN ONE SENTENCE

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“Hepatic encephalopathy means ammonia and other toxins are getting into the brain and confusing it—sometimes to the point of coma.”

Normal anatomy — the body's job

The liver sits in the upper-right belly and acts as the body's main detox factory. Blood from the intestines (carrying nutrients and waste) flows through the portal vein into the liver, where liver cells scrub out toxins—especially ammonia—before the blood goes back to the heart and out to the rest of the body, including the brain.

BODY'S JOB

Gut bacteria digest protein and make ammonia. The liver's job is to grab that ammonia and turn it into urea through a set of chemical steps called the urea cycle. Urea is safe; the kidneys dump it into the pee. This keeps ammonia levels in the blood low (normally less than 35 micromoles per liter) so the brain stays clear and healthy.

1. Protein from food reaches the intestines.
2. Gut bacteria break down that protein and produce ammonia.
3. Ammonia enters the blood and travels through the portal vein to the liver.
4. Healthy liver cells run the urea cycle: ammonia + carbon dioxide → urea.
5. Urea (harmless) flows out in the blood, gets filtered by the kidneys, and leaves in the urine.
6. The rest of the blood—now clean—goes to the heart and out to the brain and body.

What breaks

The liver loses its ability to clean ammonia, or blood finds a shortcut around the liver, so ammonia floods into the bloodstream and crosses into the brain. Then a trigger—like bleeding, infection, or constipation—pushes ammonia even higher and tips the brain into confusion.

CLINICAL TERM

Chronic liver failure (cirrhosis) or portosystemic shunts (including surgical shunts like TIPS—transjugular intrahepatic portosystemic shunt) cause ammonia to bypass hepatic detoxification. Precipitating factors—gastrointestinal bleeding (protein load from blood), infection (increased catabolism), dehydration (reduced renal ammonia clearance), low potassium (hypokalemia) (renal ammoniogenesis), constipation (increased gut ammonia production), or CNS-depressant drugs (benzodiazepines, opioids)—further elevate serum ammonia and unmask or worsen encephalopathy. The downstream result is elevated blood ammonia crossing the blood-brain barrier, impairing astrocyte function, and causing cerebral dysfunction ranging from subtle cognitive changes (West Haven grade I) to coma (grade IV). In acute liver failure, cytotoxic cerebral swelling (edema) and intracranial high blood pressure (hypertension) can develop rapidly.

How the body compensates

The body has no good way to compensate for a failing liver. Vital signs (heart rate, blood pressure) often stay normal because the brain problem is metabolic, not a failure of the heart or lungs. That steadiness is dangerous—it hides how sick the brain really is. The key is to spot the trajectory: worsening confusion, rising ammonia, deepening coma, or signs of brain swelling in acute failure. Once the brain swells or herniates, the window to save the patient closes fast.

1. The heart and lungs usually keep working normally, so blood pressure and oxygen levels look fine on the monitor.
2. Ammonia levels rise in the blood, but there is no alarm bell—labs have to be checked deliberately.
3. The brain tries to adapt at first: astrocytes (star-shaped brain support cells) soak up ammonia and convert it to glutamine, which causes the cells to swell with water.
4. Early confusion and sleep changes are the first visible signs, but they are easy to miss or blame on other things (pain medicine, infection elsewhere).
5. As ammonia keeps climbing, astrocyte swelling worsens, and neurons (the brain's signal cells) start to misfire—causing asterixis (flapping tremor), slurred speech, and agitation or lethargy.

6. In chronic liver disease, the brain may partly adapt over time, so the same ammonia level causes less confusion than it would in acute failure.
7. In acute liver failure, there is no time to adapt—ammonia and brain swelling rise together within hours to days, pushing intracranial pressure (pressure inside the skull) dangerously high.
8. The worst crash point is brain herniation (the swollen brain shifts and crushes the brainstem, which controls breathing and heart rate) or aspiration (the unconscious patient chokes on vomit or saliva).
9. The team must track the trend—worsening grade of encephalopathy, rising ammonia, pupil changes, or posturing—because once herniation starts, survival drops fast even with intensive care.
10. A patient who looks stable but is one grade worse than yesterday is on a downhill slope and needs urgent treatment and possible transplant evaluation.

Domino effect

1. Ammonia and other toxins (mercaptans, manganese, inflammatory cytokines) build up in the blood because the liver cannot detoxify them. — In cirrhosis, scar tissue replaces working liver cells, so the urea cycle slows or stops. In portosystemic shunts, blood skips the liver entirely. Either way, ammonia that should be turned into urea stays in the bloodstream and rises.
2. Ammonia crosses the blood-brain barrier and enters the brain. — Ammonia is a small molecule that slips easily from blood into brain tissue. Once inside, it is taken up by astrocytes (the brain's support cells), which try to detoxify it by converting ammonia to glutamine.
3. Astrocytes swell with water (cytotoxic swelling (edema)) as glutamine accumulates inside them. — Glutamine is osmotically active—it pulls water into the cell. Swollen astrocytes press on neurons (the brain's signal cells) and disrupt normal brain chemistry, especially in the cortex (the outer thinking layer) and basal ganglia (deep motor-control centers).
4. Early encephalopathy appears: subtle confusion, poor attention, personality changes, sleep-wake reversal (awake at night, sleepy during the day). — The prefrontal cortex (front of the brain, responsible for focus and judgment) and the reticular activating system (brainstem network that controls wakefulness) are sensitive to ammonia. Even modest levels disrupt their signaling, causing mild cognitive slips and flipped sleep cycles (West Haven grade I–II).
5. Asterixis (flapping hand tremor) and slurred speech develop; confusion deepens. — Ammonia interferes with the basal ganglia and motor cortex, causing brief lapses in muscle tone—seen as the flapping tremor when the patient holds their arms out. As more neurons misfire, speech becomes slurred and confusion worsens (grade II–III).

6. The patient becomes lethargic, then stuporous, then unresponsive (coma). — High ammonia levels and swollen astrocytes disrupt the thalamus (the brain's relay station) and brainstem, shutting down consciousness. The brain cannot maintain normal electrical activity, and the patient slips into coma (grade IV).
7. In acute liver failure, cerebral swelling (edema) (brain swelling) and intracranial high blood pressure (hypertension) (high pressure inside the skull) develop rapidly. — Without time to adapt, ammonia-driven astrocyte swelling happens fast and globally. The skull is a rigid box, so swelling raises pressure inside. High pressure crushes blood vessels (reducing oxygen delivery) and can push the brainstem downward (herniation), causing sudden death.
8. Every sign—confusion, asterixis, coma, and brain swelling—is the brain's direct reaction to ammonia toxicity, not a separate disease. — This is one chain of injury. Lowering ammonia (with lactulose to trap it in the gut, rifaximin to reduce ammonia-producing bacteria, and treating triggers like infection or bleeding) can reverse early stages and prevent progression. Liver transplant is the only cure when the liver is too damaged to recover.

Symptoms · what the patient feels, and why

- Confusion and trouble thinking clearly: When the liver is damaged, it cannot clean ammonia (a toxic waste from breaking down protein) out of the blood. That ammonia travels to the brain and poisons the neurons (brain cells), making it hard to think, remember, or focus.
- Sleeping during the day but awake at night (flipped sleep schedule): The ammonia and other toxins confuse the brain's internal clock (the part that tells you when to sleep and wake). This flips the normal day-night pattern so the person feels sleepy during the day and restless at night.
- Hand tremor or flapping (asterixis): The toxins disrupt the brain's motor cortex (the region that controls smooth muscle movements) and the basal ganglia (deep brain structures that coordinate motion). The muscles in the wrists and hands lose steady control, causing a jerky flapping motion when the person holds their arms out.
- Slurred speech or mumbling: Ammonia damages the neurons in the brain areas that plan and coordinate speech. The tongue and lips do not move precisely, so words come out unclear or jumbled.
- Personality changes (irritability, apathy, or acting out of character): The prefrontal cortex (the front of the brain that manages emotions and social behavior) is especially sensitive to ammonia. When it is poisoned, the person may lose their usual self-control, becoming short-tempered, withdrawn, or behaving strangely.
- Slow responses or seeming 'out of it': Ammonia slows the electrical signals between neurons all over the brain. Messages take longer to travel, so the person reacts late to questions or events and seems distant or 'foggy.'

- **Unsteady walk or trouble balancing:** The cerebellum (the back part of the brain that keeps you balanced) and basal ganglia are disrupted by the toxins. This makes it hard to walk in a straight line or stand without swaying.
- **Deep sleepiness or coma (in severe cases):** Very high ammonia levels flood the brain and shut down the reticular activating system (the network of neurons in the brainstem that keeps you awake and alert). When this system fails, the person cannot stay awake and may slip into a coma.

Focused exam

- **Asterixis (flapping tremor when the patient holds both arms out straight with wrists bent back):** This is the classic sign that ammonia and other toxins are poisoning the brain. The liver is not cleaning them out of the blood, so they cross into the brain and scramble the signals that keep muscles steady. Think of it like static interrupting a radio — the brain tries to hold the position, but the signal keeps cutting out.
- **Confusion, slowed thinking, or trouble staying on topic:** Early hepatic encephalopathy starts with subtle thinking problems — forgetting names, losing the thread of a conversation, or taking longer to answer simple questions. This is your first warning that ammonia is rising and reaching the brain.
- **Reversed sleep-wake cycle (awake and restless at night, sleeping during the day):** Ammonia and other toxins mess up the brain's internal clock, scrambling the normal sleep rhythm. This often appears before obvious confusion and is a key early clue.
- **Fetor hepaticus (breath that smells sweet and musty, like rotten fruit or freshly mown grass):** When the liver fails or blood bypasses it through shunts, chemicals called mercaptans (normally filtered out) escape through the lungs. This distinctive smell is a direct sign of severe liver dysfunction.
- **Slurred or slow speech, trouble finding words:** Ammonia slows the brain's ability to coordinate the muscles needed for clear speech. This is part of the toxin's effect on motor control and thinking centers in the brain.
- **Yellowing of the skin and eyes (Jaundice) (yellow skin and eyes), easy bruising, spider veins on the skin, or swollen belly from fluid:** These signs show chronic liver disease — the upstream problem. The liver is not making clotting factors, not processing bilirubin (the yellow pigment), and blood is backing up, raising pressure in the belly veins. These confirm the liver is severely damaged.

Diagnostics · every test answers a question

- **Serum ammonia level** → Is ammonia elevated, confirming that the liver is not clearing toxins?

- Comprehensive metabolic panel (CMP) including sodium, potassium, creatinine, glucose, and liver enzymes (ALT, AST, alkaline phosphatase, bilirubin) → Are there triggers like low potassium, low sodium, high creatinine (kidney failure), or high bilirubin (worsening liver failure)?
- Complete blood count (CBC) → Is there a sign of infection (high white count) or bleeding (low hemoglobin)?
- Blood culture and urinalysis with culture → Is there a hidden infection triggering the encephalopathy?
- Abdominal ultrasound with Doppler or CT abdomen/pelvis → Is there a structural problem — portal vein clot, new liver mass, or worsening cirrhosis?
- Upper endoscopy (EGD) if there is evidence of GI bleeding → Is there active bleeding from varices (swollen veins in the esophagus or stomach)?
- Electroencephalogram (EEG) in unclear cases → Is there diffuse slowing of brain waves, supporting the diagnosis of metabolic encephalopathy?

Treatment logic

- Identify and treat the trigger — bleeding, infection, dehydration, low potassium, constipation, sedating drugs, or high protein load → The liver or shunt was already broken, but the trigger pushed ammonia over the edge. Removing the trigger stops the ammonia spike. For example, stopping a GI bleed removes the protein flood in the gut; treating infection lowers inflammation and metabolic stress; fixing dehydration and low potassium restores kidney function so ammonia can be cleared.
- Lactulose by mouth (15–30 mL every 1–2 hours initially, then adjust) or rectal enema to achieve 2–3 soft bowel movements per day → Lactulose is a synthetic sugar that gut bacteria ferment into acid. The acid traps ammonia in the colon (turning it into ammonium, which cannot be absorbed) and speeds it out in stool. It also feeds beneficial bacteria that produce less ammonia.
- Rifaximin 550 mg by mouth twice daily (added to lactulose for moderate to severe or recurrent encephalopathy) → Rifaximin is a non-absorbed antibiotic that kills ammonia-producing bacteria in the gut, lowering the ammonia source. It works without wiping out bacteria throughout the body because it stays in the intestines.
- Correct electrolytes — replace potassium (goal > 4.0 mEq/L) and restore normal hydration with IV fluids if needed → Low potassium and dehydration worsen kidney function, which traps ammonia in the blood. Correcting them helps the kidneys clear ammonia and removes common metabolic triggers.

- Stop or reverse any bleeding — transfuse blood if hemoglobin < 7 g/dL, perform urgent upper endoscopy for varices, and give octreotide and ceftriaxone if variceal bleeding is suspected → Blood in the gut is a massive protein load — bacteria break it down into ammonia. Stopping the bleed and clearing blood from the intestines with lactulose removes the ammonia source. Octreotide lowers portal pressure, and antibiotics prevent infection.
- Avoid sedating medications (benzodiazepines, opioids, anticholinergics); switch dietary protein to plant-based sources and maintain at least 1.2 g/kg/day unless absolutely contraindicated → Sedatives worsen confusion and coma by directly suppressing the brain. Protein restriction was once standard but is now avoided because it causes muscle wasting; plant protein (legumes, soy) produces less ammonia than animal protein and is preferred.
- Treat infections aggressively — start empiric antibiotics for suspected spontaneous bacterial peritonitis (SBP) or other infections while awaiting cultures → Infection is a leading trigger of hepatic encephalopathy. Treating it lowers systemic inflammation, reduces metabolic stress, and removes a key ammonia driver. For SBP, third-generation cephalosporins (e.g., ceftriaxone) plus albumin are standard.

Medications

Lactulose · Non-absorbable disaccharide (a sugar the gut cannot pull into the blood)

Lactulose traps ammonia (the toxic waste that builds up when the liver fails) inside the colon and pulls it out of the body in stool. It does this two ways: first, gut bacteria break lactulose down into acids that make the colon more acidic, and ammonia cannot cross back into the blood in that acid environment—it stays trapped. Second, lactulose draws water into the colon (acts like a gentle laxative), so stool moves faster and carries the ammonia out before the body can reabsorb it. The goal is three soft bowel movements every day to keep ammonia low.

1. You swallow lactulose (or it is given through a feeding tube or as an enema into the rectum).
2. Lactulose reaches the colon, where bacteria break it into lactic acid and acetic acid.
3. These acids lower the pH (make the colon more acidic).
4. Ammonia (NH_3) in the colon picks up a hydrogen ion and becomes ammonium (NH_4^+), which cannot cross the colon wall back into blood.
5. Lactulose also pulls water into the colon by osmosis (water follows the sugar), softening stool and speeding up transit.
6. Ammonia leaves the body in stool instead of traveling to the brain.

SAFETY WATCH

Diarrhea, bloating, cramping, and gas are common because lactulose pulls water into the gut. · Too much lactulose can cause severe diarrhea and dehydration; the dose must be adjusted so the patient has three soft stools per day, not constant watery diarrhea. · Electrolyte imbalances (low potassium, low sodium) can happen with prolonged diarrhea—labs should be checked. · Very sweet taste; some patients find it hard to tolerate. Mixing it with juice or soda can help. · Do NOT stop lactulose suddenly in a patient with hepatic encephalopathy—ammonia will climb again and confusion can return.

Rifaximin · Non-absorbable antibiotic (stays in the gut; very little enters the bloodstream)

Rifaximin kills or slows down the gut bacteria that make ammonia from protein in food. Normally, when you eat protein, bacteria in the colon break it down and release ammonia as waste. In liver failure, the liver cannot clean that ammonia out of the blood, so it builds up and poisons the brain. Rifaximin reduces the number of ammonia-producing bacteria, so less ammonia is made in the first place. Because rifaximin is not absorbed into the blood, it works only in the gut and causes very few side effects in the rest of the body.

1. You swallow rifaximin tablets.
2. Rifaximin stays in the gut lumen (the inside tube of the intestine) and is NOT absorbed into the bloodstream.
3. It binds to bacterial RNA polymerase (the enzyme bacteria use to copy their genes).
4. This stops bacteria from making proteins they need to survive and reproduce.
5. Ammonia-producing bacteria die off or stop multiplying.
6. Less ammonia is made from dietary protein, so blood ammonia levels drop.
7. Lower ammonia means less reaches the brain, and confusion improves.

SAFETY WATCH

Very well tolerated because almost none is absorbed into the body. · Nausea, bloating, or mild belly discomfort can happen but is uncommon. · Rare risk of *Clostridioides difficile* (C. diff) infection, like with any antibiotic, though rifaximin is less likely to cause this than antibiotics that are absorbed. · Expensive; insurance may require prior authorization or may cover it only after lactulose alone has failed. · Do NOT use rifaximin as the only treatment—lactulose is always first-line.

Zinc sulfate (supplemental zinc) · Trace mineral supplement

Zinc is a mineral the liver needs to run enzymes that help clear ammonia. One key enzyme, ornithine transcarbamylase, works in the urea cycle (the liver's ammonia-disposal system).

Many patients with cirrhosis are low in zinc because the sick liver does not store or use it well. Giving extra zinc can help the remaining liver cells work a bit better at turning ammonia into urea (a safer waste product that leaves in urine). Zinc will NOT cure hepatic encephalopathy or replace lactulose, but it may help as an add-on in patients who are zinc-deficient.

1. You swallow zinc sulfate tablets.
2. Zinc is absorbed in the small intestine.
3. It travels through the blood to the liver.
4. Zinc binds to urea-cycle enzymes and helps them fold into the right shape to work.
5. These enzymes convert ammonia (NH_3) into urea (a less toxic compound).
6. Urea is released into the blood and filtered out by the kidneys into urine.
7. Blood ammonia levels may drop slightly, especially if the patient was zinc-deficient to start.

SAFETY WATCH

Nausea and stomach upset, especially if taken on an empty stomach; take with food. · Too much zinc (chronic overdose) can cause copper deficiency, leading to anemia and nerve problems—do not exceed the prescribed dose. · Some formulations (zinc acetate, zinc sulfate) are better absorbed than others; follow the prescription exactly. · Not all guidelines recommend routine zinc for every patient—it is used more often if a zinc level is low or if encephalopathy does not improve with standard treatment.

L-ornithine L-aspartate (LOLA) · Amino acid combination supplement

LOLA provides two amino acids (ornithine and aspartate) that help the body detoxify ammonia through two backup pathways outside the damaged liver. Ornithine feeds into the urea cycle in any remaining healthy liver cells, helping turn ammonia into urea. Aspartate helps muscle cells convert ammonia into glutamine (a safe storage form of nitrogen) so ammonia is pulled out of the blood. Muscles act like a sponge for ammonia when the liver cannot do its job. LOLA is used in some countries (especially in Europe) but is NOT FDA-approved in the United States; guidelines vary on its use.

1. LOLA is given by mouth or by intravenous infusion.
2. Ornithine enters the bloodstream and reaches the liver, where it boosts the urea cycle by increasing the substrate available for ammonia detoxification.
3. Aspartate enters muscle cells and combines with ammonia to form glutamine (via the enzyme glutamine synthetase).
4. Glutamine is a safe, neutral compound that stores nitrogen until the kidneys can excrete it.
5. Ammonia is pulled out of the blood into muscle, lowering blood ammonia and reducing brain toxicity.

SAFETY WATCH

Generally well tolerated. · Nausea and diarrhea can occur, especially with oral formulations. · Intravenous LOLA must be given slowly; rapid infusion can cause nausea or vomiting. · Not widely available in the United States; more commonly used in Europe and Asia. · Should NOT be used as monotherapy—always used WITH standard ammonia-lowering treatment.

Treat the trigger (antibiotics, blood products, electrolyte repletion, stop sedatives) · Not a single drug—this is a bundle of treatments targeting precipitating causes

Hepatic encephalopathy almost always has a trigger that tips a barely-compensated liver over the edge. The most common triggers are infection (which releases inflammatory signals that worsen brain swelling and increase ammonia production), gastrointestinal bleeding (blood in the gut is digested into protein, which gut bacteria turn into ammonia), dehydration or electrolyte imbalances (low potassium or sodium worsen confusion and can stress the kidneys), constipation (ammonia sits in the colon longer and gets reabsorbed), and sedating medications like benzodiazepines or opioids (these directly suppress brain function and unmask encephalopathy). Fixing the trigger is AS important as lowering ammonia—if you only give lactulose but leave an infection untreated, the encephalopathy will not improve. Antibiotics kill the infection, stopping the inflammatory cascade. Blood transfusion or endoscopy stops GI bleeding, cutting off the protein load to the gut. IV fluids and electrolyte replacement restore kidney function and help the body clear toxins. Stopping sedatives lets the brain wake up.

1. Identify the trigger through history, exam, and labs (infection: fever, high white count, positive cultures; bleeding: low blood count, black or bloody stool; dehydration: high creatinine, low urine output; sedatives: recent prescriptions or drug screen).
2. For infection: start broad-spectrum antibiotics (e.g., third-generation cephalosporin for spontaneous bacterial peritonitis, fluoroquinolone for urinary tract infection) to kill bacteria and stop the release of endotoxins and cytokines that worsen brain inflammation.
3. For GI bleeding: give packed red blood cells to replace lost blood; perform upper endoscopy to band or sclerose varices; give IV proton-pump inhibitor (e.g., pantoprazole) to heal ulcers and reduce re-bleeding.
4. For dehydration/electrolyte loss: give IV normal saline or lactated Ringer's to restore volume; replace potassium (oral or IV) and correct sodium slowly to avoid central pontine myelinolysis.
5. For constipation: increase lactulose dose or give enema to flush out stool and trapped ammonia.

6. For sedative use: stop benzodiazepines, opioids, or sleeping pills; consider reversal agents (flumazenil for benzos, naloxone for opioids) ONLY in controlled settings because they can cause withdrawal or seizures in chronic users.
7. The brain clears as the trigger resolves and ammonia production or accumulation drops.

SAFETY WATCH

Antibiotics: watch for allergic reactions, *Clostridioides difficile* diarrhea, and kidney injury (especially aminoglycosides or vancomycin). · Blood transfusion: risk of volume overload in patients with fluid buildup in the belly (ascites) or heart failure; transfuse slowly and consider diuretics if needed; risk of transfusion reactions (fever, rash, rarely severe). · Electrolyte replacement: NEVER correct sodium too fast (no more than 6–8 mEq/L in 24 hours) to avoid osmotic demyelination syndrome (permanent brain damage); monitor potassium closely—too much IV potassium can stop the heart. · Stopping sedatives: abrupt withdrawal from benzodiazepines can cause seizures; taper if the patient has been on them long-term; stopping opioids can cause severe withdrawal (agitation, pain, diarrhea) but is rarely dangerous. · Endoscopy for bleeding: small risk of perforation or aspiration; done urgently but carefully, often with airway protection (intubation) if the patient is encephalopathic and cannot protect their airway.

Red flags

- Sudden deep confusion or inability to wake the person (coma) — the brain is being poisoned fast and may swell or stop working.
- New seizures or uncontrolled shaking — extremely high ammonia or swelling in the brain can trigger abnormal electrical storms.
- Vomiting blood or black tarry stools in a person with liver disease — bleeding inside the gut dumps more protein (which becomes ammonia) into the system and can trigger or worsen brain poisoning.
- Fever and worsening confusion together — may signal an infection (like spontaneous bacterial peritonitis) that is pushing the sick liver over the edge.
- Very slow breathing or gasping — the brainstem centers that control breathing may be shutting down from severe toxin buildup.

Nursing priorities

1. Check airway and breathing every 15 to 30 minutes in confused or sleepy patients; place patient on their side; have suction ready; check oxygen saturation — High ammonia slows brain signals to breathing muscles and patients can vomit and breathe it into their lungs causing aspiration pneumonia; patients in grade 3 or 4 coma may need a breathing tube

2. Give lactulose every hour until the first bowel movement, then adjust to get two to three soft stools per day; give by mouth, feeding tube, or enema if patient cannot swallow — Lactulose turns into acid in the colon which traps ammonia as ammonium and pulls it out in stool; also works as a laxative to speed removal of ammonia-making bacteria and protein
3. Look for and report bleeding signs including checking all stools for black color or blood, measuring blood pressure and heart rate every 2 to 4 hours, and watching for pale skin or low hemoglobin — Gastrointestinal bleeding releases protein that becomes ammonia and is the number one trigger; needs urgent blood transfusion and endoscopy to stop bleeding and remove blood from intestines
4. Monitor potassium and sodium levels daily and replace as ordered; give IV fluids carefully to avoid overload in patients with fluid buildup in the belly (ascites); measure strict intake and output — Low potassium makes kidneys produce more ammonia; dehydration concentrates ammonia in blood; but too much IV fluid worsens belly swelling and dilutes sodium which also triggers encephalopathy
5. Watch for infection signs including temperature every 4 hours, check belly for new pain or tenderness, send urine and blood cultures if fever occurs, and report white blood cell count changes — Infection causes protein breakdown and immune chemicals that raise ammonia production; SBP happens in 10 to 30 percent of cirrhosis patients with fluid buildup in the belly (ascites) and often has no clear symptoms except confusion
6. Hold all sedating medicines including opioids, benzodiazepines, and sleeping pills; check medication list with pharmacy; use non-drug methods for pain and anxiety like repositioning and family presence — The damaged liver cannot metabolize these drugs so they accumulate and directly suppress brain function on top of ammonia toxicity; even small doses can push patients into coma
7. Assess mental status using West Haven grades every 4 to 8 hours; test for asterixis by having patient hold arms out with wrists bent back; document sleep-wake pattern; use confusion assessment tools — Tracking encephalopathy grade shows if treatment is working or patient is getting worse and needs ICU care; asterixis is a flapping tremor caused by ammonia disrupting brain signals to muscles
8. Give rifaximin 550 mg by mouth twice daily as ordered for grade 2 or higher encephalopathy or frequent repeat episodes; continue even after mental status improves — Rifaximin kills ammonia-producing bacteria in the gut but stays in the intestines and does not absorb into the blood; reduces encephalopathy episodes by 50 percent when used with lactulose

Sources

- AASLD/EASL Practice Guideline on Hepatic Encephalopathy 2014
- American Journal of Gastroenterology

- Hepatology journal (official journal of AASLD)
- UpToDate clinical decision support

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