

Ascites and Spontaneous Bacterial Peritonitis

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THE STORY IN ONE SENTENCE

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“In any cirrhosis patient with fluid buildup in the belly (ascites), new fever, belly pain, or confusion means SBP until you prove otherwise—start antibiotics right away and give IV albumin to protect the kidneys and save their life.”

Normal anatomy — the body's job

The peritoneum is a smooth, two-layer membrane that lines your belly and wraps your organs. It makes less than 50 milliliters (about three tablespoons) of clear, sterile fluid every day to let organs glide without rubbing. Your gut wall is a tight fence of cells glued together by proteins called tight junctions; these keep the trillions of bacteria living inside your intestines from leaking into your blood or belly. Your liver makes albumin (a protein that holds water inside blood vessels like a sponge), clotting factors, and immune proteins (complement, antibodies) that tag and kill any bacteria that slip through.

BODY'S JOB

Tight gut fence plus strong immune proteins equals a clean, dry belly with no extra fluid and zero bacteria outside the bowel.

1. Peritoneum secretes a few drops of lubricating fluid → organs slide smoothly.
2. Gut tight junctions lock bacteria inside the bowel → bloodstream and belly stay sterile.
3. Liver makes albumin → water stays in blood vessels, not the belly.

4. Liver makes complement and antibodies → any stray bacteria are tagged and eaten by white blood cells before they multiply.

What breaks

Cirrhosis scars and stiffens the liver, so blood backs up in the portal vein (the big vein from your gut to your liver). That high pressure pushes water and protein out of blood vessels into your belly, forming fluid buildup in the belly (ascites). At the same time, cirrhosis means your liver cannot make enough albumin or infection-fighting proteins, so the fluid buildup in the belly fluid is weak and cannot kill bacteria. Cirrhosis also makes your gut wall leaky—swelling and low bile flow loosen the tight junctions between gut cells, letting bacteria sneak through into the belly fluid. Once bacteria land in fluid buildup in the belly, they multiply fast because there are not enough white blood cells or antibodies in the fluid to stop them. That runaway infection is spontaneous bacterial peritonitis.

CLINICAL TERM

Portal high blood pressure (hypertension) (portal vein pressure ≥ 10 mmHg above inferior vena cava pressure) drives fluid buildup in the belly (ascites) by raising hydrostatic pressure (pushing water out) and lowering oncotic pressure (protein pull that holds water in). Cirrhosis cuts synthesis of albumin, complement C3, and opsonins, so ascitic fluid has total protein < 1 g/dL and poor opsonic activity. Bacterial translocation occurs when gut-wall permeability rises (from bowel swelling (edema), reduced bile salts, and small-intestinal bacterial overgrowth) and gut bacteria—most often *Escherichia coli* ($\approx 40\%$ of cases), *Klebsiella pneumoniae* ($\approx 10\%$), *Streptococcus pneumoniae*, and other streptococci—cross into mesenteric lymph nodes, then seed fluid buildup in the belly via bacteremia or direct migration. Spontaneous bacterial peritonitis is diagnosed when ascitic polymorphonuclear white blood cell (leukocyte) (PMN) count is ≥ 250 cells/ μ L, even if culture is negative (culture-negative neutrocytic fluid buildup in the belly). Mono-microbial infection (one bacterial species) distinguishes SBP from secondary peritonitis (perforation or abscess), which is usually polymicrobial.

How the body compensates

Early SBP often causes only mild fever or vague belly discomfort because the large volume of fluid buildup in the belly (ascites) dilutes bacterial toxins and the cirrhotic liver cannot mount a strong immune response. Vital signs may stay near normal for hours while the body squeezes blood vessels and speeds the heart to hold blood pressure up. Once that compensation breaks,

sepsis triggers widespread vessel dilation and fluid leak, and blood pressure crashes suddenly—kidneys shut down, confusion deepens, and the patient can die within hours.

1. {'what': 'Fever appears, often low-grade at first.', 'why': 'Bacterial endotoxin (lipopolysaccharide from Gram-negative cell walls) and cytokines (IL-1, IL-6) reset the hypothalamus thermostat upward. Because cirrhotic patients are often malnourished and immune-blunted, the fever may be only 38–38.5 °C instead of spiking high.'}
2. {'what': 'Abdominal pain and tenderness may be mild or absent.', 'why': 'The large pool of fluid buildup in the belly (ascites) cushions the peritoneum so inflammation does not always cause sharp pain. Cirrhosis can blunt pain signals, and some patients have hepatic encephalopathy that dulls their awareness.'}
3. {'what': 'Mental status worsens—confusion, sleepiness, or agitation.', 'why': 'Infection boosts ammonia production in the gut and triggers cytokine release; both poison the brain. The cirrhotic liver cannot clear ammonia, so blood levels climb and encephalopathy deepens from grade 0–I to grade II–III.'}
4. {'what': 'Heart rate climbs and vessels constrict to hold blood pressure steady (compensated shock).', 'why': 'The body releases epinephrine and norepinephrine to squeeze vessels and pump harder, trying to keep mean arterial pressure above 65 mmHg so organs get enough blood.'}
5. {'what': 'Compensation fails → systemic inflammatory response syndrome (SIRS) → low blood pressure (hypotension), a fast heart rate (tachycardia), organ failure.', 'why': 'Cytokines (TNF- α , IL-1, nitric oxide) dilate blood vessels everywhere and open gaps between endothelial cells so plasma leaks out. Blood pressure drops, kidneys lose blood flow (perfusion), and acute kidney injury (AKI) begins. Lactate rises as tissues switch to anaerobic metabolism.'}
6. {'what': 'Monitor vital signs, lactate, creatinine, urine output, and mental status every 2–4 hours.', 'why': 'A patient who looks stable now can decompensate in hours. Rising lactate (>2 mmol/L), falling urine output (<0.5 mL/kg/h), climbing creatinine, or dropping MAP signal early shock—catch it before multi-organ failure locks in.'}

Domino effect

1. Cirrhosis → portal high blood pressure (hypertension) → fluid buildup in the belly (ascites) forms in the belly. — Scar tissue in the liver blocks blood flow like a dam. Pressure in the portal vein climbs above 10–12 mmHg. That high pressure forces water, sodium, and a little protein out of the capillaries in your gut and liver into the space around your organs. At the same time, the sick liver makes less albumin, so blood loses its sponge-like pull and fluid leaks even faster.

2. Cirrhosis weakens the gut wall → bacteria cross from bowel into fluid buildup in the belly (ascites). — Portal high blood pressure (hypertension) makes the gut swell with fluid, and low bile flow means fewer bile salts to keep bacteria in check. The protein glue (tight junctions) between gut cells loosens. Live bacteria—mostly *E. coli* and *Klebsiella*—slip through the gut wall into lymph channels and bloodstream, then land in the fluid buildup in the belly (ascites) pool.
3. Bacteria multiply unchecked in fluid buildup in the belly (ascites) → infection (peritonitis) begins. — Normal ascitic fluid in a healthy person would have antibodies and complement proteins to tag bacteria and call in white blood cells. In cirrhosis, the fluid buildup in the belly (ascites) is low in protein (<1 g/dL) and almost empty of immune fighters, so bacteria double every 20–30 minutes without resistance.
4. Bacterial toxins trigger body-wide inflammation → fever, confusion, and blood-vessel dilation. — Endotoxin (lipopolysaccharide from bacterial cell walls) and live bacteria flip on your immune system's alarm—white blood cells dump cytokines (IL-1, IL-6, TNF- α) into the blood. These reset your brain's thermostat (fever), poison brain cells (worsening encephalopathy), and tell blood vessels to open wide (vasodilation), which drops blood pressure.
5. Low blood pressure and inflammation → kidneys fail (acute kidney injury and hepatorenal syndrome). — When blood pressure falls, kidneys get less flow. At the same time, cytokines make the vessels inside the kidneys squeeze shut while vessels everywhere else stay wide open. That mismatch starves the kidney's filters (nephrons) of oxygen. Creatinine climbs, urine output drops, and without quick treatment the kidneys shut down completely (hepatorenal syndrome type 1).
6. Untreated SBP → septic shock → multi-organ failure and death. — Bacteria keep multiplying and spilling endotoxin. Blood pressure crashes despite maximum squeezing by stress hormones. Organs (brain, liver, kidneys, gut) lose blood flow, cells die, lactate builds up, and the patient spirals into irreversible shock. Mortality without treatment approaches 90 % within days.
7. Early antibiotic (ceftriaxone) + IV albumin → infection cleared, kidneys protected, survival jumps. — A third-generation cephalosporin (ceftriaxone 2 g IV daily, or cefotaxime) kills the gut bacteria within 24–48 hours. IV albumin (1.5 g/kg at diagnosis, then 1 g/kg on day 3) pulls fluid back into blood vessels, raises blood pressure, binds toxins, and shields kidney tubules from starved blood flow (ischemia). Landmark trials show albumin cuts kidney injury and death by about 30 % in high-risk patients (creatinine >1 mg/dL, blood urea nitrogen >30 mg/dL, or total bilirubin >4 mg/dL). After one episode, daily norfloxacin or twice-weekly ciprofloxacin prevents recurrence and keeps the patient alive long enough to reach liver transplant.

8. Long-term: secondary prevention (prophylaxis) prevents recurrence; liver transplant is the only cure. — SBP recurs in 70 % of survivors within one year if you do nothing. Daily norfloxacin (400 mg) or ciprofloxacin (750 mg once weekly, though some centers use twice weekly) cuts recurrence to ≈ 20 %. Because cirrhosis itself is the root cause—leaky gut, weak immunity, fluid buildup in the belly (ascites)—only a new liver (transplant) can truly fix the problem and stop the cycle.

Symptoms · what the patient feels, and why

- Fever (temperature ≥ 38.0 °C / 100.4 °F): Bacteria release a toxin called endotoxin (a piece of their outer wall) into the belly fluid. This toxin travels through the blood to the brain's thermostat (the hypothalamus) and tells it to turn up the body's temperature, like cranking up a furnace. The brain does this by making chemicals called cytokines (IL-1 and IL-6) that reset the temperature higher to fight the infection.
- Belly pain or tenderness: The peritoneum (the thin lining that wraps the inside of your belly and all your organs) gets swollen and irritated by the infection. When it swells, it presses on tiny pain nerves in that lining, sending 'ouch' signals to your brain. The pain is often dull and spread out because the pool of fluid in the belly (ascites) acts like a cushion, so it doesn't hurt as sharply as if the lining were dry.
- Worsening confusion or altered mental status (hepatic encephalopathy): When you have an infection, your gut bacteria make more ammonia (a poisonous waste gas), and your already-damaged liver can't clean it out of the blood fast enough. Ammonia and other toxins float up to the brain and damage the nerve cells, making you confused, sleepy, or act strangely. The infection also releases inflammatory chemicals (cytokines) that directly harm brain cells, making the confusion worse.
- Low blood pressure (systolic < 90 mmHg or a drop of ≥ 40 mmHg from your usual): Bacterial toxins tell blood vessels all over your body to relax and widen (vasodilation), like opening floodgates. At the same time, tiny holes open in the vessel walls (capillary leak), so fluid leaks out into tissues. Both mean less blood flows through the pipes to vital organs, and your blood pressure drops—this is the start of septic shock.
- Sudden kidney injury (creatinine rises ≥ 0.3 mg/dL or urine drops to < 0.5 mL per kilogram per hour): When blood pressure falls, the kidneys get less blood flow—like a garden hose with weak pressure. At the same time, inflammatory chemicals from the infection squeeze the arteries feeding the kidneys even tighter. The kidney cells (nephrons) run out of oxygen and nutrients, so they stop working and can't filter waste or make urine. In cirrhosis, this can also trigger hepatorenal syndrome, where the kidneys shut down even more because the liver is so sick.

- Very low body temperature (hypothermia $<36.0^{\circ}\text{C}$ / 96.8°F): If the infection overwhelms your body, your cells run out of energy (like a phone battery dying) and can no longer burn fuel to make heat. Your core temperature drops because the body's furnace shuts down. This is a red flag that you are in severe septic shock and your organs are failing.
- Few or no symptoms at all (happens in up to 30 % of people): The large pool of fluid in your belly dilutes the bacteria and their toxins, so there's less to irritate the lining. Also, cirrhosis weakens your immune system so it can't mount a strong fever or inflammation response. You might feel only a little off, or nothing at all—the only way to catch the infection is to test the fluid with a needle (paracentesis).
- A stalled gut (Ileus) (belly bloating, no bowel sounds, nausea, vomiting): Inflammation of the peritoneum sends signals through nerves and releases chemicals (cytokines) that tell the intestines to stop their normal wave-like squeezing (peristalsis). The gut goes quiet and stops moving food, so you feel bloated, nauseated, and can't pass gas or stool. It's like the conveyor belt in your belly shuts off.
- Diarrhea: Infection and inflammation in the belly can irritate the gut lining and speed up movement of fluid through the intestines, so stool comes out loose and watery. Toxins from bacteria can also trigger the gut to dump water into the bowel instead of absorbing it.

Focused exam

- Perform paracentesis (inserting a needle into the abdomen to withdraw fluid) and send the sample to the lab for cell count with differential, Gram stain, culture (inoculate blood culture bottles at bedside for best yield), total protein, and albumin: In spontaneous bacterial peritonitis (SBP, infection of fluid buildup in the belly (ascites) fluid without an obvious hole or leak in the belly organs), the fluid buildup in the belly fluid contains more than 250 polymorphonuclear white blood cells (leukocytes) (PMNs, a type of white blood cell that fights bacteria) per cubic millimeter. A bacterial culture may be positive. Paracentesis is the only definitive way to confirm the fluid is infected rather than sterile (germ-free). Even if the culture is negative, a PMN count above 250 diagnoses SBP and means you must start antibiotics immediately.
- Palpate the abdomen for focal tenderness, rigidity (stiff, board-like abdominal wall muscles), masses, or rebound tenderness (sharp pain when you release pressure quickly after pushing in): Localized tenderness, a palpable lump, or peritoneal signs (inflammation of the abdominal lining causing rigidity and rebound) suggest secondary bacterial peritonitis (infection from a perforated viscus, meaning a hole in the intestine or another organ) or an abscess (a walled-off pocket of pus) rather than spontaneous bacterial peritonitis. These conditions require surgery to fix the hole or drain the abscess, not antibiotics alone.

- Assess mental status for confusion, lethargy (decreased alertness or sleepiness), or asterix (flapping tremor of the hands when the wrists are extended): Hepatic encephalopathy (brain dysfunction caused by liver failure) worsens in SBP because infection increases ammonia and other toxins that the failing liver cannot clear. The infection triggers systemic inflammation, raising the risk of acute kidney injury (sudden loss of kidney function) and hepatorenal syndrome (kidney failure triggered by severe liver disease). Altered mental status is a red flag that the infection is causing organ dysfunction.
- Measure blood pressure lying flat and standing to detect orthostatic low blood pressure (hypotension) (a drop of 20 mmHg or more in systolic pressure, or 10 mmHg or more in diastolic pressure, when the patient stands up): Low blood pressure (Hypotension) (low blood pressure) in SBP indicates sepsis (life-threatening infection causing organ dysfunction) or impending septic shock (infection causing dangerously low blood pressure that does not respond to fluids). Orthostatic low blood pressure signals intravascular volume depletion (not enough blood volume in the vessels), which predicts acute kidney injury and hepatorenal syndrome. Low blood pressure means the kidneys are not getting enough blood flow.
- Inspect the skin and sclera (whites of the eyes) for yellowing of the skin and eyes (jaundice) (yellow discoloration from bilirubin buildup) and spider angiomas (small dilated blood vessels radiating from a central red point, usually on the chest or face): Yellowing of the skin and eyes (Jaundice) and spider angiomas are visible signs of chronic liver disease and cirrhosis (scarring that replaces normal liver tissue). Cirrhosis causes portal high blood pressure (hypertension) (high pressure in the portal vein entering the liver), which leads to fluid buildup in the belly (ascites). It also impairs immune function—the liver produces fewer complement proteins and opsonins (molecules that tag bacteria so white blood cells can destroy them)—making SBP more likely.
- Palpate and percuss the abdomen to assess for tense fluid buildup in the belly (ascites) (fluid under pressure causing a firm, swollen abdomen) and shifting dullness (a percussion sign confirming free fluid in the belly cavity): Tense fluid buildup in the belly (ascites) in a patient with cirrhosis and fever indicates large-volume infected fluid. When infection triggers systemic inflammation, blood vessel walls become leaky, worsening fluid buildup in the belly and raising pressure inside the abdomen. This combination accelerates translocation (movement) of bacteria from the gut into the peritoneal space and predicts worse outcomes.

Diagnostics · every test answers a question

- Diagnostic paracentesis with ascitic fluid analysis: cell count with differential, Gram stain, bacterial culture (inoculate blood culture bottles at bedside), total protein, and albumin → Does the ascitic fluid contain more than 250 polymorphonuclear white blood cells (leukocytes) per cubic millimeter, confirming spontaneous bacterial peritonitis? Is the serum-fluid buildup in the belly (ascites) albumin gradient (SAAG, calculated by subtracting ascitic fluid albumin from serum albumin) 1.1 grams per deciliter or higher, indicating portal high blood pressure (hypertension) as the cause of fluid buildup in the belly?
- Serum creatinine, blood urea nitrogen (BUN), and electrolytes (sodium, potassium) to assess kidney function and volume status → Is acute kidney injury developing? Is the patient at risk for hepatorenal syndrome (type 1: creatinine doubles to above 2.5 mg/dL in less than 2 weeks; type 2: slower rise)? Does low sodium (hyponatremia) (serum sodium below 135 mEq/L) indicate dilutional volume overload (too much water diluting the sodium) or impaired free water clearance (the kidneys cannot get rid of excess water)?
- Complete blood count (CBC) with differential, prothrombin time/INR (international normalized ratio), and liver function tests (total and direct bilirubin, AST, ALT, alkaline phosphatase, albumin) → Does a high white blood cell count (leukocytosis) (high white blood cell count, typically above 11,000 per microliter) or bandemia (increased immature neutrophils, called bands) confirm systemic infection? Does a low platelet count (thrombocytopenia) (platelet count below 150,000 per microliter) reflect portal high blood pressure (hypertension) and splenic sequestration (trapping of platelets in an enlarged spleen)? Does prolonged INR (above 1.5) indicate impaired hepatic synthetic function (the liver cannot make clotting factors)?
- Blood cultures (two sets, aerobic and anaerobic bottles) drawn before starting antibiotics → Is there concurrent bacteremia (bacteria in the bloodstream)? Can culture and susceptibility testing guide antibiotic narrowing after starting empiric (best-guess) coverage?
- Ascitic fluid lactate dehydrogenase (LDH), glucose, total protein, albumin, carcinoembryonic antigen (CEA), and alkaline phosphatase if secondary bacterial peritonitis is suspected → Does the ascitic fluid chemistry suggest a perforated viscus (hole in the bowel) rather than spontaneous bacterial peritonitis? Do Runyon criteria—ascitic fluid with total protein above 1 g/dL, glucose below 50 mg/dL, and LDH above the upper limit of normal for serum—suggest secondary peritonitis requiring surgery?

- Abdominal ultrasound or CT (computed tomography) scan only if secondary bacterial peritonitis is suspected based on focal tenderness, peritoneal signs, or atypical fluid chemistry → Is there evidence of bowel perforation (free intraperitoneal air), abscess (fluid collection with an enhancing rim on contrast CT), or another intra-abdominal surgical emergency? Does imaging confirm fluid buildup in the belly (ascites) if the clinical exam is equivocal (unclear)?
- Repeat paracentesis 48 hours after starting antibiotics if clinical improvement is absent or if initial PMN count was very high (above 1,000 per cubic millimeter) → Is the PMN count falling appropriately (expected drop of at least 25 percent at 48 hours)? Does failure to respond suggest resistant organisms, secondary peritonitis, or inadequate antibiotic dosing?

Treatment logic

- Perform diagnostic paracentesis immediately, send fluid for cell count with differential, Gram stain, culture (inoculate blood culture bottles at bedside for highest yield), total protein, and albumin. Start empiric intravenous ceftriaxone 2 grams every 24 hours (or cefotaxime 2 grams every 8 hours) without waiting for culture results. → Ceftriaxone is a third-generation cephalosporin (a beta-lactam antibiotic) with excellent coverage of *Escherichia coli*, *Klebsiella* species, and *Streptococcus pneumoniae*—the organisms causing more than 80 percent of community-acquired SBP. Starting antibiotics within 6 hours of presentation reduces mortality because bacterial toxins and inflammatory cytokines (signaling proteins like tumor tissue death (necrosis) factor-alpha and interleukin-6) damage the kidneys, heart, and brain. Paracentesis confirms the diagnosis and rules out secondary peritonitis (which requires surgery). If the patient was on fluoroquinolone prevention (prophylaxis) or has healthcare-associated infection, empiric coverage should include piperacillin-tazobactam or a carbapenem to cover resistant gram-negative organisms.

- Administer intravenous albumin 1.5 grams per kilogram of body weight within 6 hours of diagnosis (day 1), then 1.0 gram per kilogram on day 3. Use 25 percent albumin solution. → Albumin is an oncotic agent (a large protein that holds water in the blood vessels by exerting osmotic pressure, like a sponge pulling water back into the bloodstream). Infection triggers systemic inflammatory response syndrome (SIRS), which increases vascular permeability (blood vessel walls become leaky) and causes effective arterial blood volume to drop. This activates the renin-angiotensin-aldosterone system and sympathetic nervous system, which narrow the renal arteries and reduce blood flow to the kidneys, causing acute kidney injury or hepatorenal syndrome. Albumin expands intravascular volume, improves renal blood flow (perfusion) (blood flow to the kidneys), and reduces the risk of kidney failure and death by approximately 30 percent compared to antibiotics alone. This benefit is most pronounced in patients with baseline creatinine above 1 mg/dL, BUN above 30 mg/dL, or total bilirubin above 4 mg/dL.
- Discontinue nephrotoxic medications: non-steroidal anti-inflammatory drugs (NSAIDs like ibuprofen, which block renal prostaglandins and reduce glomerular filtration), ACE inhibitors and angiotensin receptor blockers (ARBs, which dilate the efferent renal arteriole and drop filtration pressure), and aminoglycosides (antibiotics toxic to renal tubules). Temporarily hold diuretics (furosemide, spironolactone) until infection clears and blood pressure stabilizes. → In cirrhosis with infection, renal blood flow depends on compensatory prostaglandin-mediated vasodilation (widening of renal arteries by prostaglandin E₂, like opening a valve to let more blood through). NSAIDs block this, causing acute tubular tissue death (necrosis) (kidney tubule cell death from starved blood flow (ischemia), meaning inadequate blood flow). ACE inhibitors drop the glomerular filtration rate by dilating the efferent arteriole (the vessel leaving the filtering unit), which lowers the pressure gradient needed for filtration. Diuretics worsen intravascular volume depletion by pulling more fluid out of the blood vessels. Stopping these drugs protects kidney function during the high-risk period of infection.
- After infection resolves (repeat paracentesis showing PMN count below 250 per cubic millimeter and negative culture, or clinical resolution of fever and abdominal pain), start secondary prevention (prophylaxis) with oral norfloxacin 400 milligrams once daily (or ciprofloxacin 750 milligrams once weekly, or trimethoprim-sulfamethoxazole one double-strength tablet daily). Continue indefinitely or until liver transplantation. → Patients who survive SBP have a 70 percent one-year recurrence rate without prevention (prophylaxis) because the underlying cirrhosis, portal high blood pressure (hypertension), and gut bacterial overgrowth persist. Norfloxacin selectively suppresses gram-negative bacteria (*E. coli*, *Klebsiella*) in the intestinal lumen, reducing bacterial translocation (movement of bacteria from the gut into the bloodstream and fluid buildup in the belly (ascites)). Prevention reduces recurrence by approximately 60 percent and improves one-year survival. Patients with ascitic fluid total protein below 1.5 g/dL are at especially high risk and benefit most from prevention, even if they have never had SBP.

- If performing large-volume paracentesis (removing 5 liters or more of ascitic fluid to relieve tense fluid buildup in the belly (ascites) and respiratory distress), administer intravenous albumin 6 to 8 grams per liter of fluid removed immediately after the procedure. → Removing large volumes of ascitic fluid causes sudden shifts in intra-abdominal pressure and fluid distribution, triggering post-paracentesis circulatory dysfunction (PPCD, a syndrome of effective hypovolemia—not enough blood volume in the vessels—renal vasoconstriction, and sodium retention). Albumin replacement prevents PPCD by maintaining intravascular oncotic pressure (the pull that keeps water in the blood vessels), reducing the risk of acute kidney injury, low sodium (hyponatremia) (low sodium from water retention), and early fluid buildup in the belly (ascites) recurrence. PPCD occurs in up to 70 percent of large-volume paracentesis without albumin replacement.
- If fluid buildup in the belly (ascites) becomes refractory (does not respond to maximum-dose diuretics: spironolactone 400 milligrams and furosemide 160 milligrams daily, with sodium restriction below 2 grams per day) and the patient has recurrent SBP or requires frequent large-volume paracentesis (3 or more times per month), refer for transjugular intrahepatic portosystemic shunt (TIPS, a radiologic procedure placing a stent between the portal vein and hepatic vein to bypass the cirrhotic liver and lower portal pressure). → TIPS reduces portal pressure from above 12 mmHg to 8 to 10 mmHg, decreasing the hydrostatic force (pressure pushing fluid out of blood vessels) that drives fluid buildup in the belly (ascites) formation. Lower portal pressure reduces bacterial translocation because splanchnic venous congestion (backup of blood in intestinal veins) and bowel wall swelling (edema) (swelling that disrupts the gut barrier) improve. TIPS decreases fluid buildup in the belly recurrence and the need for repeated paracentesis. Some studies suggest it may reduce SBP risk by improving gut barrier function, though data are mixed.
- Evaluate for liver transplantation in all patients with SBP, especially those with MELD score above 15, refractory fluid buildup in the belly (ascites), hepatorenal syndrome, or recurrent SBP despite prevention (prophylaxis). → Liver transplantation is the only definitive cure for cirrhosis and its complications. It replaces the scarred liver with a healthy one, normalizing portal pressure, restoring immune function (complement and opsonin production), repairing gut barrier function, and eliminating the risk of SBP recurrence. One-year survival after transplant exceeds 90 percent at experienced centers. SBP is a sentinel event signaling decompensated cirrhosis and should prompt transplant evaluation unless contraindications (active substance use, advanced malignancy, severe cardiopulmonary disease) exist.

Medications

Ceftriaxone IV (third-generation cephalosporin antibiotic) · Third-generation cephalosporin antibiotic

Ceftriaxone locks onto tiny protein machines (called penicillin-binding proteins) that bacteria use to build their outer shell. When these machines stop working, the bacteria's wall gets weak and bursts open, like a balloon with a hole in it—and the bacteria dies. In spontaneous bacterial peritonitis (when bacteria from the gut sneak across the intestine wall into belly fluid and start multiplying), the most common invaders are gut bacteria such as *E. coli* and *Klebsiella*. Ceftriaxone kills these bugs in the belly fluid and stops the infection.

1. After you get the IV, ceftriaxone travels through your blood and soaks into the belly fluid where bacteria are multiplying.
2. The drug finds bacterial cells and sticks tightly to their penicillin-binding proteins—these are the enzymes bacteria use to weave together the tough net (called peptidoglycan) that forms their outer wall.
3. With those enzymes blocked, bacteria can't finish building or fixing their wall. Water rushes into the weak cell, and the bacteria burst like an overfilled water balloon.
4. Dead bacteria release fewer toxins (called endotoxins), so inflammation in your body starts to calm down and you begin to feel better.

SAFETY WATCH

Can cause thick, sludgy bile or even fake gallstones (called pseudolithiasis) because some of the drug leaves your body through bile. Usually goes away on its own. · May let a dangerous germ called *C. diff* grow in your colon (because the antibiotic also kills helpful bacteria), causing severe watery diarrhea. Wash hands and watch for new diarrhea. · Rare allergic reactions—rash, itching, or trouble breathing. Tell your doctor if you've ever had an allergy to penicillin or other cephalosporin antibiotics (about 5 to 10 people out of 100 with a penicillin allergy will also react to ceftriaxone). · In newborn babies, never mix ceftriaxone with IV fluids that contain calcium—tiny crystals can form in the blood and lungs and be deadly. (This warning does not apply to adults.)

IV albumin (25% human albumin solution) · Plasma volume expander and kidney-protective protein

Albumin is a big protein that normally floats in your blood and acts like a sponge, holding water inside your blood vessels so your blood pressure and kidney blood flow stay steady. When you have spontaneous bacterial peritonitis, the infection makes blood vessels throughout your body relax too much (like a garden hose going limp), and at the same time the tiny arteries in your kidneys clamp down tight. Blood pressure drops and your kidneys get starved

of blood. Giving IV albumin pulls water back into your blood vessels from the tissues, raises your blood pressure, and keeps blood flowing to your kidneys so they don't shut down (a dangerous complication called hepatorenal syndrome type 1).

1. Albumin is given through an IV—usually 1.5 grams per kilogram of your body weight on day 1 of infection treatment, then 1 gram per kilogram on day 3.
2. These large albumin molecules stay in your blood vessels and attract water (because water likes to cluster around big proteins). Water moves out of your tissues and into your bloodstream.
3. Your blood volume goes up, which raises your blood pressure and the amount of blood your heart pumps every minute.
4. More blood reaches your kidneys. That tells your kidneys to stop releasing a cascade of stress hormones (renin → angiotensin II → aldosterone) that were trying to squeeze every drop of blood to vital organs.
5. With better blood flow and less squeezing of kidney arteries, your kidney cells get the oxygen they need. Your creatinine (a waste product that piles up when kidneys fail) stays stable or comes down.

SAFETY WATCH

Can overload your body with fluid if your heart is weak—watch for new shortness of breath, crackling sounds in the lungs, or swollen legs. Your doctor will listen to your lungs and check your oxygen. · Very rare severe allergic reaction (anaphylaxis) if you have a condition called IgA deficiency. Symptoms include throat swelling, hives, or a sudden drop in blood pressure. · Because albumin is made from donated human blood (although it's heat-treated and screened), there is a tiny risk of transfusion reactions—fever, chills, or itching. · Albumin is expensive. Guidelines from the American Association for the Study of Liver Diseases (AASLD) say to reserve it for patients whose creatinine is above 1 mg/dL, blood urea nitrogen (BUN) is above 30 mg/dL, or total bilirubin (a marker of liver function) is above 4 mg/dL—these are signs your kidneys are at high risk of failure.

Avoid nephrotoxic drugs, ACE inhibitors, ARBs, and NSAIDs · Drug avoidance strategy (harm reduction)

In cirrhosis, your kidneys are already struggling because blood vessels in your gut are too relaxed (from scar tissue blocking flow through the liver), so less blood reaches the kidneys. Your body tries to keep blood pressure steady by releasing hormones that squeeze kidney blood vessels, but this leaves your kidneys on the edge of shutting down. NSAIDs like ibuprofen block chemicals (prostaglandins) that normally keep the inflow pipe to the kidney filter open, so the filter clogs. ACE inhibitors and ARBs block a hormone (angiotensin II) that keeps the outflow pipe squeezed just enough to maintain filtering pressure—without that

squeeze, pressure drops and the filter stalls. Other kidney-toxic drugs (certain antibiotics, IV dye for CT scans, some transplant medicines) directly poison the delicate kidney cells. Any of these can tip you into sudden kidney failure (acute kidney injury) or hepatorenal syndrome.

1. In cirrhosis, your baseline kidney blood flow is already low because gut blood vessels are dilated (relaxed) and steal blood away from kidneys.
2. NSAIDs block an enzyme called cyclooxygenase (COX), so your kidneys stop making prostaglandins PGE₂ and PGI₂—chemicals that keep the inflow arteriole open.
3. The inflow arteriole tightens, dropping the pressure inside the kidney filter (glomerulus), so less blood gets cleaned and waste products pile up.
4. ACE inhibitors or ARBs stop angiotensin II from squeezing the outflow arteriole. Without that squeeze, the pressure across the filter falls even more.
5. Kidney-toxic drugs (like the antibiotic gentamicin or IV contrast dye) directly damage the cells lining the kidney tubules, causing inflammation and cell death, which clogs the tubules and shuts down urine production.

SAFETY WATCH

NSAIDs also make your body hold onto salt and water, making belly fluid and leg swelling worse. · ACE inhibitors and ARBs can cause potassium to climb dangerously high (hyperkalemia) in cirrhosis, especially if your kidneys are starting to fail—your doctor will check potassium levels often. · Antibiotics like gentamicin need careful dosing based on kidney function and blood-level monitoring; avoid them unless no other antibiotic will work. · IV contrast dye for CT scans is especially risky—your doctor should use the smallest dose possible, give you extra IV fluids beforehand, and may give a protective medicine called N-acetylcysteine, following KDIGO acute kidney injury guidelines.

Norfloxacin or ciprofloxacin daily (fluoroquinolone antibiotic prevention (prophylaxis)) · Fluoroquinolone antibiotic

Norfloxacin and ciprofloxacin jam the tiny molecular machines (called DNA gyrase and topoisomerase IV) that bacteria in your gut use to unzip and copy their DNA. When these machines break, bacteria can't multiply and they die off. The goal is to thin out the crowd of gut bacteria—especially the troublemakers like *E. coli* and *Klebsiella*—so fewer can sneak across your leaky gut wall into your belly fluid and cause another infection. This daily prevention antibiotic is given to people at high risk of getting spontaneous bacterial peritonitis again, such as those who've already had one infection, have very low protein in their belly fluid (less than 1.5 grams per deciliter), or had bleeding from varices.

1. You take one pill by mouth every day—norfloxacin 400 milligrams or ciprofloxacin 500 milligrams (sometimes ciprofloxacin 750 once a week is used instead).

2. The antibiotic enters bacteria in your gut and locks onto DNA gyrase, the enzyme that untwists the coiled-up DNA so it can be read and copied.
3. With DNA gyrase blocked, the DNA breaks into pieces and the bacteria can't repair it or divide, so they die.
4. Gram-negative bacteria (the main family that includes *E. coli* and *Klebsiella*) drop in number inside your gut.
5. Fewer bacteria means fewer invaders are available to slip through the gut wall (which is extra leaky in portal high blood pressure (hypertension) because of high pressure and inflammation) and into your belly fluid.
6. With fewer bacteria translocating, the risk of infection in your belly fluid drops sharply.

SAFETY WATCH

Over time (usually after 6 to 12 months), bacteria in your gut can become resistant to the antibiotic—up to 30 to 40 people out of 100 develop fluoroquinolone-resistant *E. coli* or *Klebsiella*, according to AASLD data. If you get another infection, it may be harder to treat.

- Can cause tendon swelling and even tendon rupture (most often the Achilles tendon in your heel)—risk is higher if you're over 60, take steroids, or have kidney problems. Stop the drug and call your doctor if you feel sudden heel or leg pain.
- Can lengthen the electrical signal in your heart (QT prolongation on an ECG), which may trigger a dangerous rhythm called torsades de pointes. Risk goes up if you take other medicines that do the same thing, or if your potassium or magnesium is low.
- Like any antibiotic, can allow *C. diff* to overgrow in your colon, causing severe watery diarrhea. Wash hands and report new diarrhea right away.
- Rarely causes nerve damage (peripheral neuropathy)—tingling, numbness, or pain in hands or feet that can be permanent. Stop the drug immediately if this starts.
- AASLD guidelines say to use daily prevention antibiotics ONLY if you've had SBP before, OR your belly-fluid protein is less than 1.5 g/dL and your Child-Pugh score is 9 or higher or creatinine is 1.2 mg/dL or higher, OR you have cirrhosis and just had bleeding from varices or stomach ulcers.

Large-volume paracentesis with IV albumin replacement (8 g albumin per liter removed if >5 L) · Procedural intervention with colloid replacement

Paracentesis means putting a needle through your belly wall into the fluid-filled space (peritoneal cavity) and draining the fluid out. Removing large amounts of belly fluid relieves the pressure that makes it hard to breathe and hard to eat (because your stomach gets squashed). But when you take out more than 5 liters (about 1.3 gallons), your blood vessels lose so much water so fast that your blood pressure can drop and your kidneys can fail. This happens because fluid in your blood vessels rushes into the belly to try to replace what was drained, leaving less blood for your heart to pump. Giving albumin through an IV—8 grams for every liter of fluid removed—keeps water in your blood vessels, props up your blood pressure,

and protects your kidneys from shutting down (a problem called post-paracentesis circulatory dysfunction).

1. Your doctor uses ultrasound (a sound-wave picture) to find a safe spot—usually in the lower side of your belly, away from organs and big blood vessels—and slides a needle in.
2. Fluid drains out by gravity or gentle suction until the tightness is gone, or sometimes all the fluid is removed if it keeps coming back despite medicine.
3. When more than 5 liters come out, water from your bloodstream shifts into your belly to fill the empty space, like water flowing downhill.
4. Your blood volume drops, triggering your kidneys to release stress hormones (renin, then angiotensin II, then aldosterone) to try to hang onto every bit of fluid and keep your blood pressure from crashing.
5. Albumin is infused into your vein—a 25% solution, calculated as 8 grams per liter of fluid removed, usually given at the end of the procedure or within 6 hours.
6. Albumin's big protein molecules pull water into your blood vessels and hold it there, stopping blood volume from dropping further. This calms the hormone cascade, keeps blood flowing to your kidneys, and holds your blood pressure steady.

SAFETY WATCH

Bleeding into your belly or belly wall is rare (less than 1 person in 100) and INR and platelet count do not reliably predict who will bleed. · Poking a hole in your bowel or bladder can happen if the anatomy is unusual or the fluid is trapped in pockets (loculated)—always use ultrasound to guide the needle. · Infection risk is very low if sterile technique is used; you do NOT need to take antibiotics just because you're having the procedure. · Post-paracentesis circulatory dysfunction happens in 15 to 20 people out of 100 if albumin is not given. You may see rising creatinine (kidney waste product), low sodium in your blood, low blood pressure, and fast re-accumulation of belly fluid. · Albumin is expensive. Some guidelines allow cheaper synthetic fluids (like gelatin or starch solutions) if less than 5 liters are removed, but AASLD prefers albumin for removals over 5 liters because studies show it lowers the risk of kidney failure and death.

Spironolactone and furosemide (aldosterone antagonist and loop diuretic combination) · Aldosterone antagonist (spironolactone) and loop diuretic (furosemide)

In cirrhosis, your body mistakenly thinks you don't have enough blood volume (because blood pools in your gut vessels), so it releases a hormone called aldosterone. Aldosterone tells the last part of the kidney's filter (the collecting duct) to pull salt and water back into your blood and throw potassium into the urine. This makes belly fluid and leg swelling worse.

Spironolactone blocks aldosterone's signal, so your kidneys dump salt and water into urine

instead of saving it—and they keep potassium. Furosemide works earlier in the kidney filter (in a loop-shaped tube called the loop of Henle) and blocks a transporter that reabsorbs salt, chloride, and water—so even more salt and water leave in your urine, but furosemide also dumps potassium. Using both drugs together in a 100 mg spironolactone to 40 mg furosemide ratio mirrors how cirrhotic kidneys behave and keeps potassium balanced—spironolactone's potassium-saving cancels furosemide's potassium-wasting. Together they shrink belly fluid and leg swelling without making electrolytes swing wildly.

1. In cirrhosis, high aldosterone opens tiny gates (called ENaC channels) in the collecting-duct cells, letting sodium flood back into your blood; water follows the sodium, and potassium and hydrogen ions are pushed into the urine.
2. Spironolactone grabs onto the aldosterone receptor (the lock where aldosterone would normally fit) and blocks it, so ENaC channels don't open as much.
3. More sodium stays in the urine tube and is peed out; water follows by osmosis (water always chases salt), shrinking your blood volume and belly fluid.
4. Potassium is saved instead of dumped—spironolactone is called potassium-sparing.
5. Furosemide blocks a molecular pump (the Na-K-2Cl cotransporter) that would normally haul sodium, potassium, and chloride out of the urine and back into the blood in the loop of Henle.
6. With the pump shut down, sodium, chloride, and water pour into the urine; potassium is lost too (furosemide is potassium-wasting).
7. The 100:40 ratio (100 mg spironolactone : 40 mg furosemide) keeps potassium from climbing too high or dropping too low, while achieving steady urine output and gradual shrinking of belly fluid and leg swelling.

SAFETY WATCH

Potassium can climb too high (hyperkalemia, above 5.5 milliequivalents per liter) from spironolactone—especially risky if your kidneys are starting to fail, you take an ACE inhibitor or ARB, or you eat a lot of high-potassium foods (bananas, oranges, potatoes). Your doctor will check potassium and creatinine within 3 to 5 days of starting the pills or increasing the dose. · Potassium, sodium, and magnesium can drop too low (hypokalemia below 3.5, low sodium (hyponatremia) below 130, low magnesium (hypomagnesemia)) from furosemide; your doctor will replace them with pills or IV if needed. · Kidneys can fail suddenly (acute kidney injury) if you lose weight too fast—safe rate is no more than 0.5 kilograms (about 1 pound) a day if you don't have leg swelling, or 1 kilogram (about 2 pounds) a day if you do have swelling. Weigh yourself every morning and call if weight drops faster. Your doctor will check creatinine regularly. · Brain fog and confusion (hepatic encephalopathy, caused by ammonia and toxins crossing into the brain) can worsen if diuretics cause dehydration and kidney waste (urea) to pile up. If you become confused, your doctor will give lactulose (a syrup that traps ammonia in stool) and rifaximin (an antibiotic that kills ammonia-making gut bacteria). · Breast swelling in men (gynecomastia) and irregular periods in women can happen because spironolactone also blocks male hormones (androgens). Usually mild but can be bothersome. · AASLD guidelines say to start spironolactone 100 mg plus furosemide 40 mg once daily, then increase both together (keeping the ratio) every 3 to 5 days if you're not losing enough weight and electrolytes are stable. Maximum doses are spironolactone 400 mg and furosemide 160 mg daily.

Transjugular intrahepatic portosystemic shunt (TIPS) · Interventional radiology procedure (mechanical portal pressure reduction)

TIPS is a small metal tube (stent) that a doctor threads through a vein in your neck and places inside your liver to connect the portal vein (the high-pressure vein draining blood from your gut, spleen, and pancreas) directly to a hepatic vein (the low-pressure vein draining your liver into the main vein returning to your heart). Normally, blood flows through the liver's tiny, delicate vessels (sinusoids), but in cirrhosis those vessels are choked by scar tissue, causing pressure to build up—like a dam in a river. The stent creates a shortcut around the dam, dropping the pressure in the portal vein from dangerously high levels (often above 12 millimeters of mercury) down toward normal (5 to 10 mmHg). Lower pressure means less fluid is squeezed out of blood vessels into your belly, and your body's stress hormones (renin-angiotensin-aldosterone) calm down. TIPS is used for belly fluid that won't go away despite the strongest diuretics (refractory fluid buildup in the belly (ascites)) or keeps coming back rapidly after needle taps, and for bleeding from varices that medicine can't control.

1. Under live X-ray guidance (fluoroscopy), a doctor slides a catheter (thin tube) through a vein in your neck down into a hepatic vein inside your liver.

2. A special needle pokes through the liver tissue from the hepatic vein into the nearby portal vein.
3. A guidewire is threaded through, and a balloon is inflated to widen the tunnel; then a self-expanding metal stent (like a tiny spring) is placed to hold the tunnel open permanently.
4. Blood from your gut and spleen now flows through the low-resistance stent into the hepatic vein, skipping most of the scarred liver sinusoids.
5. Pressure in the portal vein drops. The goal is to reduce the pressure gradient between the portal vein and the vein leaving the liver (hepatic venous pressure gradient, or HVPG) to below 12 mmHg—this stops varices from bleeding and stops fluid from leaking into the belly.
6. With lower portal pressure, the force squeezing fluid across capillary walls into the peritoneum drops, and your kidneys get the message to stop releasing so many stress hormones. Belly fluid stops forming as fast, and diuretics can finally work.

SAFETY WATCH

Brain fog and confusion (hepatic encephalopathy) develop or worsen in 20 to 40 people out of 100 after TIPS—risk is higher if you're older, have had encephalopathy before, or have low albumin. Your doctor will treat this with lactulose (to trap ammonia in your stool) and rifaximin (an antibiotic that kills ammonia-producing bacteria), and may adjust your protein intake. · TIPS can push a very sick liver over the edge into sudden liver failure if your liver function is already very poor. Risk is high if your MELD score (a formula predicting liver-failure risk) is above 18, or if you have Child-Pugh class C cirrhosis with a MELD above 15, according to AASLD. · The stent can narrow (stenosis) or clot off (thrombosis) over time—happens in 10 to 20 people out of 100 with older bare-metal stents. Newer covered stents (lined with a Teflon-like material called ePTFE) clog less often and are now the preferred type. · Suddenly rerouting a large volume of blood back to the heart can overload the right side of the heart, causing high-output heart failure—especially risky if you already have heart disease. Your cardiologist will check your heart function before TIPS. · AASLD and EASL (European liver society) guidelines say TIPS for fluid buildup in the belly (ascites) is appropriate if: your belly fluid does not respond to the maximum doses of diuretics (spironolactone 400 mg plus furosemide 160 mg daily) AND you've needed frequent large-volume paracentesis (three or more taps in the past 3 months), AND you do not have severe heart or lung disease or uncontrolled encephalopathy.

Red flags

- Low blood pressure (systolic <90 mmHg) or signs of septic shock: cold arms and legs, blotchy skin, blood lactate >4 mmol/L (waste acid building up because organs aren't getting enough oxygen)
- Sudden kidney injury: creatinine rises ≥ 0.3 mg/dL in 48 hours, or urine output drops below 0.5 mL per kilogram per hour for 6 hours straight
- Severe confusion or unresponsiveness (hepatic encephalopathy grade III–IV): very sleepy, can't be woken easily, or in a coma
- High kidney waste or liver breakdown markers at diagnosis—creatinine ≥ 1 mg/dL, blood urea nitrogen (BUN) ≥ 30 mg/dL, or total bilirubin ≥ 4 mg/dL—all predict high risk of death and mean you need IV albumin (a protein infusion) to support blood pressure and kidneys
- Seeing lots of bacteria on a Gram stain of the belly fluid, or free air on a CT scan—this suggests the infection is NOT spontaneous bacterial peritonitis but secondary peritonitis from a hole in the bowel or an abscess, and you need emergency surgery

Nursing priorities

1. Notify the physician immediately and prepare for diagnostic paracentesis within the next hour—gather sterile kit, position patient supine or slightly tilted, and have blood-culture bottles ready at bedside. — Paracentesis (needle insertion to remove belly fluid) is the only way to diagnose SBP. We count white blood cells in the fluid: ≥ 250 polymorphonuclear cells (neutrophils, a type of infection-fighting white blood cell) per cubic millimeter confirms infection. Inoculating blood-culture bottles at the bedside increases bacterial growth by 80 percent compared to sending fluid in a plain tube.
2. Start intravenous antibiotics (ceftriaxone 2 grams every 24 hours or cefotaxime 2 grams every 8 hours) within 1 hour of suspected SBP, before culture results return. — Gut bacteria (especially *Escherichia coli*, *Klebsiella*, and *Streptococcus*) cause SBP. Delaying antibiotics allows bacteria to multiply in the bloodstream, leading to septic shock and multi-organ failure. Third-generation cephalosporins (a class of antibiotics) kill these bacteria and reduce death rates from 90 percent to under 20 percent when given early.
3. Administer intravenous albumin (a blood protein) 1.5 grams per kilogram of body weight within 6 hours of diagnosis, then repeat 1.0 gram per kilogram on day three. — Albumin prevents kidney failure (hepatorenal syndrome, a deadly complication where kidneys shut down due to poor blood flow) by expanding blood volume and improving circulation. Studies show albumin cuts kidney injury and death by 40 percent in SBP patients, especially those with creatinine ≥ 1.0 mg/dL, blood urea nitrogen ≥ 30 mg/dL, or bilirubin (a waste product from broken-down red blood cells) ≥ 4.0 mg/dL.

4. Monitor vital signs every 1 to 2 hours: blood pressure, heart rate, temperature, respiratory rate, and oxygen saturation—escalate immediately if systolic blood pressure drops below 90 mmHg or mean arterial pressure falls below 65 mmHg. — Septic shock (severe infection causing dangerously low blood pressure) develops in 30 percent of SBP patients. Low blood pressure means organs—brain, kidneys, liver, heart—are not getting enough oxygen-rich blood. Mean arterial pressure below 65 mmHg requires vasopressors (medications that squeeze blood vessels to raise pressure) and intensive-care monitoring.
5. Measure and record urine output every hour using a Foley catheter—notify the physician if output drops below 0.5 milliliters per kilogram per hour for six consecutive hours, or if creatinine rises ≥ 0.3 mg/dL from baseline. — Acute kidney injury occurs in 30 percent of SBP cases due to sepsis and poor kidney blood flow. Decreased urine output (oliguria) is the earliest warning sign. Kidney failure dramatically increases mortality and may require dialysis (a machine that filters waste from blood when kidneys cannot). Early detection allows us to give more intravenous fluids and albumin.
6. Assess mental status every 2 to 4 hours using a standardized tool (such as the Glasgow Coma Scale or the Hepatic Encephalopathy Grading scale)—watch for increased confusion, drowsiness, or inability to follow commands. — Hepatic encephalopathy (brain dysfunction from liver failure) worsens with SBP because infection increases ammonia and other toxins in the blood. Ammonia crosses into the brain and causes swelling (cerebral swelling (edema)). Worsening encephalopathy may signal sepsis spreading to the brain or herald liver failure, both requiring urgent treatment with lactulose (a sugar that traps ammonia in the gut) and rifaximin (an antibiotic that kills ammonia-producing bacteria).
7. Hold all diuretics (water pills such as furosemide and spironolactone) and nephrotoxic medications (drugs that harm kidneys, including NSAIDs like ibuprofen and aminoglycosides like gentamicin) until the infection clears and kidney function stabilizes. — Diuretics reduce blood volume and kidney blood flow, worsening kidney injury during infection. NSAIDs and aminoglycosides directly damage kidney tubules (the tiny tubes in kidneys that filter blood). Stopping these drugs protects fragile kidneys while albumin and antibiotics work to reverse the infection.
8. Educate the patient and family that after SBP resolves, lifelong daily antibiotics (norfloxacin 400 milligrams once daily or ciprofloxacin 500 milligrams once daily) are required to prevent recurrence, and discuss liver-transplant evaluation if not already listed. — Without preventive (prophylactic) antibiotics, 70 percent of patients suffer another SBP within one year. Daily antibiotics reduce recurrence by 60 percent. However, SBP signals advanced cirrhosis with high one-year mortality (30 to 50 percent), making liver transplantation the only cure. Starting this conversation early prepares patients for the next step in care.

Sources

- AASLD Practice Guidance on Ascites, Spontaneous Bacterial Peritonitis, and Hepatorenal Syndrome (2021)
- European Association for the Study of the Liver (EASL) Clinical Practice Guidelines on Ascites (2018)
- Infectious Diseases Society of America (IDSA)
- Surviving Sepsis Campaign 2021

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