

Ascites and Spontaneous Bacterial Peritonitis

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.

THE ONE-LINE TRUTH

Spontaneous bacterial peritonitis (SBP) is a bacterial infection of belly fluid (ascites) that happens when gut bacteria slip into the fluid—no hole in the bowel or other source you can fix with surgery.

The chain — how it unfolds

1. Cirrhosis → portal high blood pressure (hypertension) → fluid buildup in the belly (ascites) forms in the belly. — why: 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). — why: 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. — why: 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. — why: 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). — why: 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. — why: 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. — why: 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. — why: 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 $\approx$ 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.

Spot it — what you see, 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 °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.

Red flags — act now

- 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

Treat it — in this order

1. 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.

2. 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.
3. 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.
4. 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.

5. 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.

Your top priorities at the bedside

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. — why: 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. — why: 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. — why: 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.