

Acute Cholangitis

Acute cholangitis means the common bile duct is blocked and infected; bacteria multiply in stagnant bile, then rising pressure pumps infected bile into the bloodstream, causing sepsis (life-threatening infection response throughout the body).

THE ONE-LINE TRUTH

A gallstone, scar, or tumor blocks the bile duct so bile cannot drain; bacteria from the intestine swim up into the trapped bile and multiply, then high pressure pushes infected bile into the bloodstream — turning a plumbing problem into a body-wide infection.

The chain — how it unfolds

1. A gallstone (most common, about 75 percent of cases), benign stricture (scar from prior surgery, chronic pancreatitis, or primary sclerosing cholangitis), or malignant tumor (cholangiocarcinoma, pancreatic cancer, or ampullary cancer) blocks the common bile duct. — why: Physical obstruction stops bile from flowing into the duodenum, so bile pools upstream in the intrahepatic and extrahepatic ducts; pressure inside the duct rises from the normal 5 to 10 cm H₂O up to 20 to 40 cm H₂O or higher because the liver keeps making bile (500 to 1000 milliliters per day) with nowhere for it to go.
2. Bacteria from the small intestine — normally kept out by forward bile flow and the sphincter of Oddi — swim upstream through the ampulla of Vater and colonize the stagnant bile. — why: Without flow to flush them out, enteric bacteria (*Escherichia coli* 25 to 50 percent, *Klebsiella* 15 to 20 percent, *Enterococcus* 10 to 15 percent, *Enterobacter*, and anaerobes such as *Bacteroides* in some cases) ascend from the duodenum where trillions live harmlessly; they multiply rapidly in the nutrient-rich bile (which contains amino acids, bilirubin, and cholesterol) and form biofilms on the duct wall.
3. High pressure in the obstructed duct exceeds the pressure in the tiny veins (hepatic sinusoids) that drain the liver, forcing infected bile backward into those veins — a phenomenon called cholangiovenous reflux. — why: Normal sinusoidal pressure is about 5 cm H₂O; when bile-duct pressure hits 20 to 40 cm H₂O, the pressure gradient reverses and bile (now full of bacteria and endotoxin) is pushed into the sinusoids, then into hepatic veins, then into the inferior vena cava and right heart — seeding bacteria directly into the systemic bloodstream and bypassing the liver's usual detoxification.

4. Bacteremia (bacteria in the blood) triggers the immune system to release cytokines (interleukin-1, interleukin-6, tumor tissue death (necrosis) factor-alpha), and body temperature spikes to 38.5 to 40 degrees Celsius (101 to 104 Fahrenheit) or higher. — why: Bacterial endotoxin (lipopolysaccharide from the outer membrane of gram-negative bacteria) binds Toll-like receptor 4 on macrophages and monocytes; these cells release pyrogens (fever-causing cytokines) that act on the hypothalamus to reset the body's thermostat higher; fever slows bacterial replication and speeds white-cell activity, but it also signals systemic infection.
5. Right upper quadrant (RUQ) pain develops because the liver capsule and bile ducts stretch from rising pressure and inflammation. — why: The liver capsule (Glisson capsule) and bile-duct walls have nociceptors (pain receptors) that fire when stretched; intraductal pressure rises and the liver swells (hepatomegaly); inflammation also irritates the peritoneum (abdominal lining) nearby, causing sharp visceral pain that the brain localizes to the right upper abdomen and may radiate to the right shoulder (referred pain via the phrenic nerve, which shares sensory roots C3 to C5 with the diaphragm).
6. Yellowing of the skin and eyes (Jaundice) (yellow skin and sclera) appears because conjugated bilirubin cannot drain through the blocked duct, so it backs up into the bloodstream and deposits in tissues. — why: Hepatocytes conjugate (attach glucuronic acid to) bilirubin to make it water-soluble so it can be excreted in bile; when the duct is obstructed, conjugated (direct) bilirubin spills back into sinusoidal blood; blood levels rise above 2 to 3 milligrams per deciliter, and bilirubin deposits in skin (yellow) and sclera (the whites of the eyes turn yellow) because these tissues have high elastin, which binds bilirubin; urine turns dark brown (tea- or cola-colored) because the kidneys filter and excrete the excess conjugated bilirubin, and stool turns pale (clay-colored) because no bilirubin reaches the intestine to be converted to stercobilin, the pigment that makes stool brown.
7. Blood tests show elevated direct (conjugated) bilirubin (often 2 to 20 milligrams per deciliter or higher), alkaline phosphatase (ALP, often 300 to 1000 units per liter, normal less than 120), and gamma-glutamyl transferase (GGT, often 200 to 1000 units per liter, normal less than 50) — the cholestatic (bile-flow obstruction) pattern. — why: Cells lining the bile ducts (cholangiocytes) and hepatocytes make ALP and GGT; when ducts are blocked and cells are damaged by pressure and inflammation, these enzymes leak into the bloodstream; conjugated bilirubin rises because it cannot exit into the intestine; aminotransferases (AST and ALT) may rise modestly (100 to 300 units per liter) from hepatocyte injury, but the ALP and GGT elevation is out of proportion — this pattern distinguishes duct obstruction from hepatitis (where AST and ALT would be in the thousands).

8. White blood cell count climbs (leukocytosis, often 15 000 to 25 000 cells per microliter or higher, normal 4000 to 11 000) and the differential may show a left shift (more than 10 percent band forms, which are immature neutrophils). — why: Bone marrow releases neutrophils in response to cytokines (granulocyte colony-stimulating factor, interleukin-3) produced during infection; when demand is high, the marrow releases immature forms (bands, metamyelocytes) into the blood before they fully mature — this left shift signals acute bacterial infection and stress on the marrow.

Spot it — what you see, and why

- Fever (often $>38.5^{\circ}\text{C}$ / 101.3°F) with shaking chills (rigors): Bacteria from infected bile spill into the bloodstream. Your immune cells release chemicals called cytokines (like alarm signals), which tell the brain's thermostat (the hypothalamus) to turn the body's temperature up. Rigors — violent shaking — are your muscles firing fast to make heat and reach that new higher setting.
- Right upper quadrant (RUQ) abdominal pain: The bile duct is blocked, so pressure builds up inside. The stretched duct walls and the liver's capsule (its outer wrapper) have pain nerves that fire when pulled tight. Inflammation also irritates the peritoneum, the thin lining around your organs.
- Yellowing of the skin and eyes (Jaundice) (yellowing of skin and whites of the eyes): The liver makes a yellow pigment called bilirubin and normally dumps it into bile, which flows to your intestine. When the duct is blocked, bilirubin backs up into the blood like water behind a dam. The yellow color spills into your skin and the whites of your eyes.
- Dark urine (tea- or cola-colored): Extra bilirubin in your blood gets filtered by your kidneys into your pee. Because bilirubin is yellow-brown, your urine turns dark like strong tea. This happens because bile can't leave through stool anymore.
- Pale or clay-colored stool: Bilirubin normally gives poop its brown color. When the bile duct is blocked, bilirubin never reaches your intestine. Without that pigment, stool comes out pale, gray, or clay-like.
- Nausea and vomiting: Pain and inflammation tickle the vagus nerve, a long nerve that connects your gut to your brainstem. That triggers the vomiting center in your brain. Bacterial toxins (poisons from germs) in your blood also make you feel sick.

Red flags — act now

- Low blood pressure (Hypotension) (systolic blood pressure <90 mm Hg) or signs of septic shock — bacteria are overwhelming your circulation; needs immediate ICU care, IV fluids, antibiotics, and urgent drainage of the blocked duct.

- Altered mental status or confusion — the brain isn't getting enough oxygen, or toxins are building up; part of Reynolds pentad (five-sign pattern) and warns of high death risk without fast treatment.
- Acute kidney injury (creatinine rising, urine output dropping) — sepsis and low blood pressure starve the kidneys of blood; signals that organs are starting to fail.
- Persistent fever or rising white blood cell count despite antibiotics — means the bile duct is still blocked and acting like a sealed abscess full of germs; drainage (ERCP or a needle through the skin) is essential.
- Impaired blood clotting (Coagulopathy) (INR elevated, platelet count low) — the sick liver can't make clotting proteins well, and sepsis uses up platelets; raises bleeding risk during procedures and means severe disease.

Treat it — in this order

1. Intravenous fluids immediately (normal saline or lactated Ringer solution, 1-2 liters in the first hour, then adjusted based on blood pressure, heart rate, and urine output) → Restores blood volume and raises blood pressure so oxygen reaches the brain, kidneys, and heart. Helps dilute bacteria and toxins in the bloodstream. Supports the kidneys so they can make urine and filter waste. Improves tissue blood flow (perfusion) (blood flow to cells), which lowers lactate.
2. Broad-spectrum intravenous antibiotics started within one hour (common choices: piperacillin-tazobactam 3.375-4.5 g every 6 hours, OR ceftriaxone 1-2 g every 24 hours PLUS metronidazole 500 mg every 8 hours, OR a carbapenem such as meropenem 1 g every 8 hours if the patient has severe sepsis or risk of resistant bacteria) → Kills gut bacteria (especially *Escherichia coli*, *Klebsiella*, *Enterococcus*, and anaerobes like *Bacteroides*) that traveled up into the bile ducts and entered the bloodstream. Stops infection from spreading and causing septic shock and organ failure. Lowers fever and white blood cell count as bacteria die.
3. Biliary drainage within 24 hours by endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy (cutting the muscle at the duct opening) and stone removal or stent placement; drain sooner (within 6-12 hours) if the patient is in septic shock or not improving with fluids and antibiotics → Opens the blocked bile duct so bile flows into the small intestine again. Lowers pressure in the duct, which stops bacteria and toxins from being forced into the bloodstream. Fixes the root cause of the infection. Most patients improve quickly once bile is drained. ERCP is the preferred method because it is less invasive than surgery.

4. Percutaneous transhepatic biliary drainage (PTBD, a tube placed through the skin and liver into the bile duct under imaging guidance) if ERCP fails, cannot be done safely, or is not available → Drains backed-up bile from outside the body through an external tube, lowering pressure in the duct and allowing infected bile to exit. Buys time for the patient to stabilize before definitive surgery or repeat ERCP. Works even when the duct cannot be reached from inside the intestine.
5. Cholecystectomy (surgical removal of the gallbladder) 2–6 weeks after the acute infection is controlled, if gallstones caused the episode and the patient is healthy enough for surgery → Removes the gallbladder so new stones cannot form and travel into the bile duct, preventing future episodes of acute cholangitis and acute cholecystitis (gallbladder inflammation). Reduces lifetime risk of biliary complications. Usually done laparoscopically (through small incisions) as an outpatient or short-stay procedure.

Your top priorities at the bedside

1. Establish large-bore intravenous access (18-gauge or larger) and infuse 1 to 2 liters of normal saline or lactated Ringer solution over the first hour, then titrate (adjust) rate to keep systolic blood pressure ≥ 90 mm Hg and urine output ≥ 0.5 mL per kilogram per hour. — why: Sepsis (body-wide infection) causes blood vessels to leak and dilate (widen), dropping blood pressure; fast IV fluid fills the circulation so the heart can pump oxygen to the brain, kidneys, and liver before organs fail.
2. Administer broad-spectrum intravenous antibiotics within one hour of recognition (common regimens: piperacillin-tazobactam 3.375 to 4.5 grams every 6 hours, OR ceftriaxone 1 to 2 grams every 24 hours PLUS metronidazole 500 milligrams every 8 hours). — why: Bacteria multiply in the trapped bile and spill into the bloodstream; every hour of delay before antibiotics increases the risk of septic shock, organ failure, and death by approximately 7 to 10 percent.
3. Monitor blood pressure, heart rate, respiratory rate, temperature, and oxygen saturation every 15 to 30 minutes initially, then every 1 to 2 hours once stable; immediately notify the physician if systolic blood pressure falls below 90 mm Hg or mental status changes. — why: Acute cholangitis can progress to septic shock (severe infection causing dangerously low blood pressure) within hours; early detection of deterioration allows the team to escalate to ICU care and emergency bile-duct drainage before irreversible organ damage.