

Acute Cholangitis

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THE STORY IN ONE SENTENCE

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“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).”

Normal anatomy — the body's job

The liver makes bile continuously — about 500 to 1000 milliliters every day — and sends it down the common bile duct into the duodenum (the first part of the small intestine) through a small opening called the ampulla of Vater. Bile carries bilirubin (a yellow breakdown product of old red blood cells) and bile acids (detergents that break fats into tiny droplets so enzymes can digest them). Steady forward flow keeps the ducts flushed clean and prevents bacteria from the intestine climbing backward.

BODY'S JOB

When bile flows freely, it digests fats so your body can absorb vitamins A, D, E, and K; removes waste bilirubin so it leaves in your stool (which is why stool is brown); and washes any bacteria that wander in back down into the gut — so the bile ducts stay sterile (germ-free) and pressure stays normal (about 5 to 10 cm of water).

1. Hepatocytes (liver cells) make bile and pump it into tiny canals called canaliculi — like water flowing into the smallest branches of a stream.
2. Canaliculi merge into bigger and bigger bile ducts inside the liver, joining to form the right and left hepatic ducts.

3. The right and left hepatic ducts meet just outside the liver to form the common hepatic duct.
4. The cystic duct from the gallbladder joins the common hepatic duct to make the common bile duct (CBD) — the final highway carrying bile toward the intestine.
5. The common bile duct runs down behind the pancreas and joins the pancreatic duct (which carries digestive enzymes from the pancreas) at the ampulla of Vater.
6. The ampulla of Vater opens into the duodenum through a muscular valve called the sphincter of Oddi, which relaxes after you eat to let bile and pancreatic juice flow in and help digest your meal.
7. When you are not eating, the sphincter of Oddi stays partly closed, so some bile backs up into the gallbladder to be stored and concentrated until the next meal.

What breaks

A gallstone (about 75 percent of cases), scar (stricture from old surgery or chronic inflammation), or tumor (cholangiocarcinoma, pancreatic cancer, or ampullary cancer) plugs the common bile duct. Bile cannot drain, so it backs up under pressure like water behind a closed valve. Bacteria from the small intestine — normally harmless there — swim upstream into the stagnant bile and multiply because there is no flow to wash them away. High pressure then forces infected bile and bacteria backward into tiny veins in the liver, dumping them straight into the bloodstream. That flood of bacteria and their toxins triggers sepsis (body-wide inflammation), and if pressure stays high the infection cannot clear even with antibiotics — the duct must be drained.

CLINICAL TERM

Biliary obstruction (blocked bile duct) halts antegrade (forward) bile flow, creating bile stasis (pooling). Enteric bacteria — most commonly *Escherichia coli*, *Klebsiella* species, *Enterobacter*, and *Enterococcus* — ascend from the duodenum and proliferate in the static bile. Elevated intraductal pressure (often exceeding 20 to 30 cm H₂O) causes cholangiovenous reflux: infected bile enters hepatic sinusoids and hepatic veins, seeding the bloodstream. This produces bacteremia (bacteria circulating in blood) and triggers systemic inflammatory response syndrome (SIRS). When bacterial endotoxins (lipopolysaccharides from gram-negative bacteria) and cytokines (interleukin-1, interleukin-6, tumor tissue death (necrosis) factor-alpha) overwhelm compensatory mechanisms, distributive shock (septic shock) develops — widespread vasodilation, capillary leak, and tissue poor blood flow (hypoperfusion) lead to multiorgan dysfunction syndrome (MODS) and, without intervention, death.

How the body compensates

Early on, the sympathetic nervous system and immune system work to keep blood pressure and oxygen delivery stable even while bacteria spread. Heart rate climbs, blood vessels squeeze, and the spleen releases extra white cells. Vital signs may look acceptable — this is the dangerous hidden window when the patient appears stable but infection is building. Once bacterial toxins overwhelm the vessels and blood pressure crashes, organs lose oxygen and compensation fails fast.

1. The sympathetic nervous system detects falling blood pressure or rising cytokines and releases epinephrine (adrenaline) and norepinephrine to raise heart rate (tachycardia, often 100 to 120 beats per minute or higher) and constrict blood vessels (peripheral vasoconstriction), temporarily maintaining blood pressure.
2. The spleen contracts and releases stored red cells and white cells into circulation; bone marrow ramps up production of neutrophils (leukocytosis, white count often 15 000 to 25 000 cells per microliter or higher) to fight infection.
3. The liver increases synthesis of acute-phase proteins (C-reactive protein, procalcitonin) to tag bacteria and activate complement; fever is generated by cytokine-driven hypothalamic set-point elevation to slow bacterial replication and speed immune-cell activity.
4. The kidneys retain sodium and water (via renin-angiotensin-aldosterone and antidiuretic hormone) to expand blood volume and support blood pressure; urine output may drop (oliguria, less than 0.5 milliliters per kilogram per hour).
5. This compensation hides how sick the patient is — blood pressure may be 100 to 110 systolic, so the team does not yet recognize septic shock, but lactate is climbing (above 2 millimoles per liter signals cells are using anaerobic metabolism because oxygen delivery is marginal) and mental status may be quietly worsening.
6. The tipping point is septic shock: when endotoxin and cytokines (especially nitric oxide released by damaged endothelium) overwhelm alpha-adrenergic receptors, blood vessels dilate despite maximal sympathetic drive; capillaries leak fluid into tissues; effective circulating volume drops; blood pressure falls below 65 millimeters of mercury mean arterial pressure even with fluid resuscitation; and organs (brain, kidneys, liver, gut) do not get enough oxygen — lactate rises above 4, urine stops, confusion deepens, and multiorgan failure begins.
7. Think of obstructed infected bile like a deep abscess: antibiotics reach blood concentrations of 10 to 50 micrograms per milliliter, but inside the walled-off duct under high pressure, drug penetration is poor and bacteria keep multiplying — you must drain the duct to drop pressure, let antibiotics in, and remove the source.

8. Watch trends, not single numbers: serial lactates (rising from 2 to 4 to 6 millimoles per liter), climbing white count with left shift (more bands than mature neutrophils), worsening confusion (Glasgow Coma Scale dropping), rising creatinine (kidneys failing), or falling urine output all warn that compensation is failing and the window to intervene is closing.
9. A patient who looks stable now but is on a downward slope — heart rate climbing, blood pressure drifting down, lactate up, altered mental status — is not truly stable; this is compensated shock about to decompensate, and the mortality without urgent drainage (ERCP or percutaneous transhepatic cholangiography) within 24 hours can exceed 30 to 50 percent once multiorgan failure sets in.

Domino effect

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. — 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. — 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. — 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. — 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. — 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. — 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. — 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). — 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.
9. If the duct is not drained within 24 to 48 hours, bacterial toxins and cytokines trigger widespread vasodilation (blood vessels widen and leak), blood pressure drops below 65 millimeters of mercury mean arterial pressure despite intravenous fluids, and organs lose blood flow (perfusion) — this is septic shock. — Endotoxin and cytokines (especially nitric oxide and prostaglandins released by damaged endothelium) cause arterioles to dilate and capillaries to become leaky; fluid shifts out of blood vessels into the interstitial space (third-spacing); effective circulating volume drops; the heart pumps faster but cannot maintain pressure (distributive shock); organs (brain, kidneys, liver, gut) do not get enough oxygen; cells switch to anaerobic metabolism, producing lactate (rising above 2 then 4 millimoles per liter); and mitochondria fail, leading to cell death.
10. Confusion or altered mental status develops when the brain is underperfused or when toxins cross the blood-brain barrier. — Mean arterial pressure below 65 millimeters of mercury reduces cerebral blood flow (perfusion) pressure (normally autoregulated at 50 to 150 mmHg systolic); neurons are exquisitely sensitive to low oxygen (hypoxia) (low oxygen) and begin to malfunction within minutes; high bilirubin (above 10 to 20 milligrams per deciliter) and ammonia (which rises when the liver fails to clear gut-derived toxins) can directly impair neurotransmission (a component of hepatic encephalopathy); bacterial endotoxin may also cross the blood-brain barrier and activate brain microglia, causing delirium.
11. Kidneys fail (acute kidney injury): creatinine rises (from baseline 0.8 to 1.2 milligrams per deciliter up to 2, 3, 4 or higher), urine output drops below 0.5 milliliters per kilogram per hour (oliguria), and potassium and acid accumulate in the blood. — Septic shock drops renal blood flow (perfusion) pressure; glomerular filtration rate falls; tubular cells become blood-starved (ischemic) (not enough oxygen) and die (acute tubular tissue death (necrosis)); cytokines and endotoxin directly injure the kidney microcirculation; without filtration, waste products (urea, creatinine) and electrolytes (potassium) accumulate, and acid-base balance tips toward metabolic acidosis (acid building up in the blood).

- 12.** Lungs may fail (acute respiratory distress syndrome, ARDS): oxygen levels drop, the patient breathes fast and hard, and chest X-ray shows white-out (bilateral infiltrates) as fluid leaks into the alveoli. — Cytokines damage the pulmonary capillary endothelium and alveolar epithelium, causing the blood-air barrier to leak; protein-rich fluid floods the alveoli (non-cardiogenic pulmonary edema (fluid flooding the lungs)); surfactant is inactivated, so alveoli collapse (atelectasis); oxygen cannot diffuse into blood; the patient becomes hypoxemic (oxygen saturation dropping below 90 percent on room air) and may require intubation and mechanical ventilation with high positive end-expiratory pressure (PEEP) to keep alveoli open.
- 13.** Without urgent biliary drainage — endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy and stone extraction or stent placement (preferred), or percutaneous transhepatic cholangiography (PTC) if ERCP fails — the obstructed infected duct continues to pump bacteria into the bloodstream, and multiorgan failure progresses to death in 20 to 50 percent of severe cases (Tokyo Guidelines grade III). — Antibiotics kill bacteria circulating in blood and tissues but cannot reach therapeutic concentrations inside the high-pressure obstructed duct; the duct acts as a protected reservoir (like a walled abscess) where bacteria keep multiplying; drainage drops intraductal pressure (from 30 to 40 cm H₂O back toward normal 5 to 10), allows bile to flow, stops cholangiovenous reflux, removes the bacterial source, and lets antibiotics penetrate — that is why current Tokyo Guidelines (2018) and American Society for Gastrointestinal Endoscopy (ASGE) guidelines recommend urgent (within 24 hours) or emergent (within 12 hours for grade III shock) ERCP plus broad-spectrum antibiotics (piperacillin-tazobactam, or a carbapenem, or ceftriaxone plus metronidazole, tailored to local resistance patterns and culture results) to maximize survival.

Symptoms · what the patient feels, and why

- Fever (often >38.5 °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.
- Low blood pressure (Hypotension) (low blood pressure, systolic <90 mm Hg): Bacterial toxins and immune chemicals make blood vessels relax and widen everywhere. Fluid also leaks out of vessels into tissues. Together, this drops the pressure in your pipes — that's septic shock, and it means infection is overwhelming your circulation.
- Altered mental status or confusion: Low blood pressure means less oxygen-rich blood reaches your brain. Bacterial toxins and high bilirubin can also cross into brain tissue and interfere with how nerve cells work, causing confusion or sleepiness.
- A fast heart rate (Tachycardia) (fast heart rate, often >100 beats per minute): Your body releases adrenaline to pump harder and faster, trying to keep blood pressure up and deliver oxygen. Fever and infection also speed up your metabolism, so your heart has to beat faster to keep up.
- Itching (Pruritus) (itching skin): Bile contains acids (bile salts) that normally flow into your gut. When bile backs up, these salts spill into your blood and settle under your skin. They tickle nerve endings there, causing itching all over.

Focused exam

- Fever, often higher than 39°C (102.2°F): Bacteria from the small intestine have traveled backward up into bile ducts that are blocked. Fever means the immune system is fighting that infection spreading into the bloodstream. Fever is one part of Charcot triad (fever + yellowing of the skin and eyes (jaundice) + right upper belly pain), which points directly to infected bile ducts (acute cholangitis).
- Right upper quadrant pain (pain just under the right ribs): A gallstone, scar tissue (stricture), or tumor is blocking the bile duct. Bile backs up behind the blockage, stretching the duct and the liver's covering (capsule), which is packed with pain nerves. This stretch causes sharp or aching pain. This pain is the second part of Charcot triad.

- Yellowing of the skin and eyes (Jaundice) (yellow color in the skin and whites of the eyes): Bile cannot drain from the liver into the small intestine because the duct is blocked. Bilirubin (a yellow pigment made when old red blood cells break down) backs up into the blood instead of leaving the body in stool. High bilirubin in the blood turns skin and eyes yellow. Yellowing of the skin and eyes (Jaundice) is the third part of Charcot triad.
- Low blood pressure (hypotension, systolic below 90 mmHg) and fast heart rate (tachycardia, over 100 beats per minute): Bacteria and their toxins have leaked from the infected bile duct into the bloodstream, causing septic shock (life-threatening infection in the blood). Toxins make blood vessels relax and leak fluid, dropping blood pressure. The heart beats faster to try to pump enough oxygen to the brain, kidneys, and other organs. These signs mean the infection is severe and organs are at risk.
- Confusion or altered mental status (change in thinking, awareness, or behavior): When septic shock worsens, the brain does not receive enough oxygen and glucose. Confusion or sleepiness signals that organs are starting to fail. This finding, along with fever, yellowing of the skin and eyes (jaundice), belly pain, and shock, makes up Reynolds pentad (five signs of severe acute cholangitis that need emergency drainage).

Diagnostics · every test answers a question

- Complete blood count (CBC) → Is the body fighting an infection by making extra white blood cells?
- Liver function tests (total and direct bilirubin, alkaline phosphatase, gamma-glutamyl transferase, alanine aminotransferase, aspartate aminotransferase) → Is the bile duct blocked, forcing bile and bilirubin to back up into the liver and blood?
- Blood cultures (two sets from different sites, drawn before starting antibiotics) → Are bacteria circulating in the bloodstream (bacteremia)?
- Serum lactate → Are the tissues getting enough oxygen, or is the patient in shock?
- Right upper quadrant ultrasound → Is the bile duct dilated (swollen wider than normal)? Is there a stone or mass blocking it?
- Magnetic resonance cholangiopancreatography (MRCP) or endoscopic ultrasound (EUS) → Where exactly is the blockage, and what is causing it (stone, stricture, or tumor)?
- Basic metabolic panel (BMP, including sodium, potassium, creatinine, and blood urea nitrogen) → Are the kidneys still working, and are electrolytes balanced?

Treatment logic

- 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.
- 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.
- 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.
- 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.
- 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.

- Vasopressors (norepinephrine, started at 0.05–0.1 mcg/kg/min and titrated to maintain mean arterial pressure above 65 mmHg) if blood pressure remains low despite intravenous fluids → Tightens blood vessels to raise blood pressure and restore blood flow to vital organs (brain, heart, kidneys). Prevents organs from failing due to low oxygen delivery. Used in septic shock when fluids alone do not raise blood pressure enough.
- Supportive care (oxygen therapy, pain control with intravenous opioids such as morphine or fentanyl, monitoring in an intensive care unit if the patient has septic shock or organ failure) → Keeps oxygen levels normal so tissues can heal. Relieves pain so the patient can breathe deeply and rest. Close monitoring detects worsening infection, falling blood pressure, or kidney failure early. Allows the medical team to adjust fluids, antibiotics, and vasopressors quickly.

Medications

Broad-spectrum intravenous antibiotics · Anti-infective agents (combination therapy targeting bile duct pathogens)

Bacteria trapped inside a blocked bile duct multiply fast and release toxins into the bloodstream, causing sepsis (infection spreads through the whole body and blood pressure drops dangerously). Antibiotics are powerful germ-killers given through an IV line so they reach the blood and liver within minutes. We use combinations that kill the three main types of bacteria found in infected bile: gram-negative rods like *E. coli* and *Klebsiella* (shaped like tiny sticks, very common in gut and bile), gram-positive cocci like *Enterococcus* (round bacteria), and anaerobes like *Bacteroides* (bacteria that live without oxygen). Standard first choices are piperacillin-tazobactam (a penicillin plus a helper that stops bacteria from breaking down the penicillin) or a cephalosporin like ceftriaxone or cefepime combined with metronidazole (which specifically kills anaerobes). These drugs punch holes in bacterial cell walls or stop bacteria from building new walls, so the germs burst and die. Antibiotics buy time and stop sepsis from getting worse, but they cannot cure cholangitis alone—the bile duct **MUST** be drained because antibiotics cannot reach high enough levels inside a blocked, pus-filled duct.

1. IV antibiotic enters a vein and spreads through the bloodstream in seconds.
2. Drug molecules travel to the liver and inflamed bile duct walls.
3. Antibiotic attaches to bacterial cell walls or enters bacteria.
4. Piperacillin-tazobactam, ceftriaxone, and cefepime block enzymes bacteria need to build strong cell walls.
5. Metronidazole (if used) enters anaerobic bacteria and damages their DNA so they cannot reproduce.
6. Bacteria lose their protective wall, burst, or stop dividing, and the immune system clears dead germs.

7. Bacterial toxin levels drop, sepsis begins to stabilize, but infection will NOT clear until the bile duct blockage is removed.

SAFETY WATCH

Allergic reactions: About 1 in 10 people report penicillin allergy; true severe allergy (anaphylaxis with throat swelling, shock) is rare but life-threatening. Always ask about prior reactions. If real penicillin allergy exists, switch to a fluoroquinolone like ciprofloxacin plus metronidazole, or a carbapenem like meropenem. · Kidney injury: Piperacillin-tazobactam and cephalosporins are cleared by the kidneys. In sepsis, kidneys often struggle (acute kidney injury from low blood pressure). Doses must be adjusted down if creatinine (kidney waste marker in blood) rises. Monitor creatinine daily. · Clostridioides difficile colitis (C. diff): Broad-spectrum antibiotics kill good gut bacteria, allowing C. difficile (a toxin-making bacteria) to overgrow and cause severe watery diarrhea, cramping, and colon inflammation. Risk is highest in older adults and hospitalized patients. Watch for new diarrhea during or after antibiotics. · Liver toxicity: Rarely, piperacillin-tazobactam can cause drug-induced hepatitis (liver inflammation). Monitor liver enzymes (ALT, AST) if they start rising beyond the cholangitis baseline. · Drug resistance: Using broad antibiotics when not needed or for too long breeds resistant bacteria (superbugs). Once bile is drained and cultures return, narrow antibiotics to target the specific bacteria grown. Typical treatment duration is 4 to 7 days after successful drainage.

Intravenous crystalloid fluids (normal saline or lactated Ringer's) · Volume expanders / resuscitation fluids

Sepsis makes blood vessels leaky—fluid seeps out of the bloodstream into tissues, so blood volume drops and blood pressure falls (septic shock). The heart pumps faster but there is not enough fluid left in the vessels to carry oxygen to organs. Kidneys, brain, and liver start to fail. IV crystalloid fluids are sterile saltwater solutions given rapidly through a large IV catheter to refill the bloodstream. Normal saline is water with the same salt concentration as blood; lactated Ringer's also contains potassium, calcium, and lactate (a buffer that helps correct blood acid buildup). We give 30 milliliters per kilogram of body weight in the first 3 hours (for a 70 kg adult, that is about 2 liters)—sometimes more if blood pressure stays low. This fluid pushes blood pressure back up, restarts urine flow (a sign kidneys are working again), clears confusion (brain gets oxygen), and keeps organs alive until antibiotics and drainage fix the infection.

1. Nurse or doctor inserts a large-bore IV catheter (thick needle) into a vein, often in the arm or central vein in the chest or neck.
2. Crystalloid fluid bags hang on an IV pole; fluids flow by gravity or are pushed in under pressure to speed delivery.

3. Fluid enters the bloodstream and spreads throughout the vascular system in minutes.
4. Blood volume rises, stretching vessel walls and triggering sensors that tell the heart to beat more effectively.
5. Blood pressure climbs back toward normal (goal mean arterial pressure ≥ 65 mm Hg).
6. Restored pressure drives blood into organs: kidneys restart urine production, brain gets oxygen (confusion improves), liver blood flow (perfusion) improves.
7. Monitoring continues: if blood pressure does not rise after 30 mL/kg, clinicians add vasopressor medications (drugs that squeeze blood vessels) like norepinephrine, and hunt for ongoing bleeding or fluid loss.

SAFETY WATCH

Fluid overload: Too much IV fluid makes the heart pump against excessive volume, and fluid backs up into the lungs (pulmonary swelling (edema)). Watch for new shortness of breath, crackles heard with a stethoscope over the lungs, and low oxygen saturation. Elderly patients and those with heart failure are highest risk. Stop or slow fluids if overload signs appear; give diuretics (water pills like furosemide) if needed. · Electrolyte imbalance: Large volumes of normal saline can cause hyperchloremic metabolic acidosis (acid building up in the blood) (too much chloride makes blood acidic). Lactated Ringer's is often preferred because it contains lactate that buffers acid. Monitor blood sodium, potassium, chloride, and bicarbonate daily. · Dilutional anemia: Rapid fluids dilute red blood cells in the bloodstream, so hemoglobin (oxygen-carrying protein) drops on lab tests. This is usually not dangerous unless the patient was already anemic or is bleeding. · Hypothermia: Cold IV fluids lower body temperature, especially when given fast. Use a fluid warmer in shock or if patient is already cold. · Tissue swelling (edema): Fluid leaks into tissues, causing swollen arms, legs, or face. This usually improves as sepsis resolves and vessel walls heal.

Biliary drainage via ERCP or percutaneous transhepatic cholangiography (PTC) · Procedural intervention (definitive treatment, not a drug)

A blocked bile duct full of infected bile and pus acts like a closed abscess—antibiotics cannot clear the infection until the blockage is removed and bile drains out. ERCP (endoscopic retrograde cholangiopancreatography) is the first-choice procedure: a flexible camera scope goes through the mouth, down the esophagus and stomach, into the duodenum (first part of the small intestine) where the bile duct opens. The doctor injects dye into the duct to see the blockage on X-ray, then uses tiny tools passed through the scope to remove gallstones, cut open a narrowed duct opening (sphincterotomy), or place a plastic or metal stent (small tube) to hold the duct open so bile flows into the intestine. ERCP should happen within 24 hours of diagnosis; if the patient has septic shock or altered mental status (confusion, sleepiness), drainage must happen within 6 to 12 hours—this is a true emergency. If ERCP fails or cannot

be done (twisted anatomy, tumor blocking access, or unstable patient who cannot tolerate sedation for the scope), a radiologist performs percutaneous transhepatic cholangiography (PTC): using ultrasound or CT guidance, they push a needle through the skin into the liver and into a bile duct, then thread a drain catheter that carries infected bile out of the body into a collection bag. Once bile drains, infection pressure drops fast, bacteria counts fall, fever breaks, and antibiotics can finish the job. Surgery (open or laparoscopic bile duct exploration) is a backup if both ERCP and PTC fail, but it carries higher risk in a septic patient.

1. Patient is sedated (usually moderate sedation or sometimes general anesthesia for ERCP; local numbing plus light sedation for PTC).
2. ERCP: Endoscope passes into duodenum. Catheter is fed into the bile duct opening. Contrast dye is injected and X-ray images show the blockage (stone, stricture, tumor). If a stone is seen, a wire basket or balloon grabs and pulls it out. If the opening is tight, a sphincterotome (electric wire) cuts the muscle to widen it. A stent may be placed to keep the duct open.
3. PTC: Ultrasound finds a bile duct inside the liver. Needle punctures skin, liver, and duct. Guidewire is threaded through the needle. Drain catheter slides over the wire and is left in place, with the outer end taped to skin and connected to a drainage bag.
4. Infected bile and pus flow out immediately—pressure inside the duct drops.
5. Bacteria no longer multiply in a closed space; immune system and antibiotics clear remaining infection.
6. Patient's fever, white blood cell count, and bilirubin start to fall within 24 to 48 hours.
7. Drain or stent stays in place until infection clears; stones or strictures may need repeat procedures or surgery later.

SAFETY WATCH

Post-ERCP pancreatitis: ERCP can irritate the pancreas (which shares the same duct opening), causing acute pancreatitis (severe belly pain, nausea, vomiting, rising amylase and lipase enzymes) in 3 to 5 percent of cases. Risk is higher in young patients, patients with sphincter of Oddi dysfunction, and those needing repeated cannulation attempts. Rectal indomethacin (a non-steroidal anti-inflammatory drug) given right after ERCP cuts pancreatitis risk by about half. · ERCP perforation: The scope or tools can tear the duodenum or bile duct (rare, under 1 percent). Signs are severe new belly pain, fever, and air seen outside the intestine on CT scan. Small perforations may heal with antibiotics and bowel rest; large tears need emergency surgery. · Bleeding: Sphincterotomy cuts tissue and can cause bleeding into the intestine. Most bleeding stops on its own; severe bleeding may need endoscopy to cauterize (burn shut) the vessel or inject epinephrine to constrict it. · Cholangitis worsening: Injecting dye into a blocked duct without draining it can briefly raise pressure and spread bacteria into the bloodstream, worsening sepsis. That is why drainage (stone removal or stent placement) must happen in the same session as the diagnostic part of ERCP. · PTC risks: Bleeding from the liver puncture site (can cause bleeding into the abdomen or bile ducts), bile leak into the abdomen (bile peritonitis, needs emergency surgery), infection spreading along the catheter track, or catheter dislodgement. The external drain must be flushed daily and kept clean to prevent clogging or skin infection. · Contrast allergy: IV iodinated contrast used in fluoroscopy during ERCP or PTC can cause allergic reactions (hives, throat swelling, anaphylaxis) or worsen kidney function. Pre-medicate with steroids and antihistamines if prior allergy, use lowest dye volume, and hydrate well. · Sedation risks: Moderate sedation or anesthesia can suppress breathing or drop blood pressure, especially in septic or elderly patients. Monitoring (pulse oximeter, blood pressure, heart monitor) is mandatory; anesthesiologist standby or intubation may be needed for high-risk patients.

Vasopressors (norepinephrine, first-line) · Sympathomimetic / alpha-adrenergic agonist

When septic shock is so severe that IV fluids alone do not raise blood pressure enough (mean arterial pressure still below 65 mm Hg after 30 mL/kg fluids), vasopressors are added. These are powerful drugs that squeeze blood vessels to push blood pressure up. Norepinephrine is the first-choice vasopressor for septic shock. It works by attaching to alpha-adrenergic receptors on the muscle layer of artery walls. This binding makes the muscle cells contract (tighten), narrowing the vessel like squeezing a garden hose. Narrower vessels mean higher resistance, so the same amount of blood pumped by the heart flows under higher pressure. Norepinephrine also mildly stimulates beta-1 receptors in the heart, slightly increasing heart squeeze strength, but the main effect is vessel constriction. The drug is given as a continuous IV infusion through a central line (a catheter placed into a large vein in the neck, chest, or

groin) because it is too strong and irritating for small peripheral arm veins—if it leaks into skin, it can cause tissue death. Vasopressors are a temporary bridge to keep organs perfused (blood flowing to brain, heart, kidneys) while fluids, antibiotics, and biliary drainage fix the underlying sepsis. Once infection is controlled and vessels heal, the drug is slowly turned down and stopped.

1. Norepinephrine infusion starts at a low dose (typically 0.01 to 0.05 micrograms per kilogram per minute) through a central venous catheter.
2. Drug molecules travel in bloodstream to arteriole smooth muscle cells.
3. Norepinephrine binds to alpha-1 adrenergic receptors on the cell surface.
4. Receptor activation triggers a cascade inside the cell: calcium ions flood in, causing muscle fibers to contract.
5. Arterioles constrict, increasing systemic vascular resistance (the resistance the heart pumps against).
6. Blood pressure rises within seconds to minutes.
7. Blood flow is redirected to vital organs (brain, heart, kidneys); skin and limbs may get less blood (cool, pale extremities).
8. Dose is titrated up or down every few minutes to keep mean arterial pressure ≥ 65 mm Hg.
9. As sepsis improves, natural vessel tone returns and norepinephrine is weaned off over hours to days.

SAFETY WATCH

Tissue starved blood flow (ischemia) and tissue death (necrosis): Excessive vasoconstriction starves tissues of oxygen. Fingers, toes, nose, and ears are most vulnerable (can turn black and die). Intestinal starved blood flow causes severe belly pain and bloody diarrhea. Limb starved blood flow can require amputation. Use the LOWEST dose that achieves blood pressure goal and wean as soon as able. · Arrhythmias (irregular heartbeats): Norepinephrine stimulates the heart and can trigger dangerous rhythms like ventricular a fast heart rate (tachycardia) or atrial fibrillation (an irregular, quivering heartbeat). Continuous heart monitoring (telemetry) is required. Correct low potassium and low magnesium, which make arrhythmias more likely. · Extravasation (drug leaking into tissue): If the IV catheter is not perfectly in the vein, norepinephrine leaking into surrounding skin causes severe tissue damage and tissue death (necrosis). This is why a central line is mandatory. If extravasation occurs, inject phentolamine (an alpha-blocker antidote) into the affected tissue immediately to reverse constriction and save the tissue. · Renal starved blood flow (ischemia): Too much constriction reduces blood flow to kidneys, worsening acute kidney injury. Monitor urine output and creatinine closely. Balance vasopressor dose with adequate fluid resuscitation. · Myocardial starved blood flow (ischemia) (heart attack): High doses increase the heart's oxygen demand and can reduce blood flow in coronary arteries, triggering chest pain or heart attack in patients with underlying heart disease. Watch for new chest pain, EKG changes, or rising troponin. · Rebound low blood pressure (hypotension): If norepinephrine is stopped suddenly, blood pressure can crash. Always wean slowly, reducing the dose stepwise while monitoring closely.

Red flags

- 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.

Nursing priorities

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. — 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). — 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. — 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.
4. Keep the patient NPO (nothing by mouth) and place a nasogastric tube if the patient is vomiting, to decompress (empty) the stomach and prevent aspiration (breathing in) of stomach contents into the lungs. — Most patients will need ERCP (endoscopic retrograde cholangiopancreatography), a procedure threading a camera through the mouth into the bile duct to remove the stone or place a drain; an empty stomach reduces the risk of vomiting and aspiration pneumonia during sedation.
5. Draw blood for complete blood count (white cell count), comprehensive metabolic panel (liver enzymes and bilirubin), lactate (a waste product that rises when tissues are starved of oxygen), and blood cultures (two sets from separate sites) before giving the first antibiotic dose. — Blood cultures identify the specific bacteria and guide antibiotic changes after 48 to 72 hours; lactate above 2 millimoles per liter signals severe sepsis and predicts which patients need ICU admission; liver enzymes and bilirubin levels help confirm duct blockage and track improvement after drainage.

6. Measure and record urine output every hour using a Foley catheter (bladder drainage tube); target output ≥ 0.5 milliliters per kilogram per hour (for a 70-kilogram patient, that is ≥ 35 milliliters per hour). — The kidneys filter waste only when blood pressure and blood flow are adequate; falling urine output is an early warning that IV fluids are insufficient or septic shock is worsening, and it guides how much additional fluid to give.
7. Position the patient with the head of the bed elevated 30 to 45 degrees unless blood pressure is critically low, and turn every 2 hours if the patient cannot reposition independently. — Head elevation reduces the risk of aspiration (stomach contents flowing backward into the airway) and helps the lungs expand fully; turning prevents pressure ulcers (bedsores) on the sacrum and heels in patients who are too weak or sedated to move.
8. Prepare for urgent biliary drainage within 24 hours (or immediately if the patient has septic shock or altered mental status) by confirming that the gastroenterology or surgery team is notified, consent is obtained, and pre-procedure checklist (coagulation studies, antibiotic timing, IV access) is complete. — Antibiotics and fluids alone cannot cure acute cholangitis; the blocked duct must be drained (usually by ERCP with stone removal or stent placement) to relieve pressure, stop bacteria from multiplying, and allow infected bile to escape; delay beyond 24 hours doubles the risk of death.

Sources

- Tokyo Guidelines 2018 (TG18) – international consensus on acute cholangitis diagnosis and management
- American College of Gastroenterology (ACG) Clinical Guideline: Management of Acute Cholangitis
- European Society of Gastrointestinal Endoscopy (ESGE) Guideline on Biliary Drainage
- Surviving Sepsis Campaign 2021 – for septic shock resuscitation and antibiotics

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