

Acute Cholecystitis

Acute cholecystitis means your gallbladder—a small pouch under your liver that stores bile—is blocked, swollen, and infected because a stone got stuck in the exit tube and won't let bile out, so pressure builds and germs grow inside.

THE ONE-LINE TRUTH

A gallstone plugs the cystic duct (the narrow tube that drains the gallbladder), trapping bile inside so pressure climbs, the wall swells, and bacteria multiply—turning a simple blockage into an infected, inflamed gallbladder that may die, tear, or spill into the belly if not drained or removed.

The chain — how it unfolds

1. A gallstone—or thick bile sludge—wedges into the cystic duct or gallbladder neck and plugs it completely. — why: The stone acts like a cork jammed in the neck of a bottle. Bile from the liver keeps flowing into the gallbladder at a steady rate (about 20 to 50 milliliters per hour), but nothing can drain out. Pressure inside the gallbladder starts to climb within hours, stretching the wall like air pumped into a balloon. This is the single broken thing upstream—every domino that falls next traces back to this one stuck stone.

2. Steady, unrelenting pain in the right upper quadrant (RUQ) that does not come and go. — why: The stretched gallbladder wall presses against the parietal peritoneum (the thin, pain-sensitive lining covering the inside of your abdominal wall). Stretch receptors and nociceptors (NO-sih-SEP-torz, pain nerves) fire continuously, sending signals up the intercostal nerves (the nerves between your ribs) and the phrenic nerve (the nerve supplying the diaphragm) to your spinal cord, then to the thalamus (the brain's relay station for sensory signals, deep in the center of your brain), and finally to the somatosensory cortex (the strip of brain that maps body sensation, running ear-to-ear across the top of your head). Unlike biliary colic—where a stone moves in and out, causing waves of crampy pain that come and go over minutes—the pain of cholecystitis is constant because the blockage is fixed. The pain sits in the RUQ because the gallbladder lives under your right ribs, just below the liver. It often radiates (spreads) to the right shoulder tip or right scapula (shoulder blade) because the phrenic nerve—which senses the diaphragm and gallbladder area—shares nerve roots with nerves that supply the shoulder (C3, C4, C5 nerve roots in your neck). Your brain interprets signals from both areas as coming from the shoulder—this is called referred pain.
3. The gallbladder wall swells with swelling (edema) (fluid buildup) and fills with inflammatory cells. — why: Trapped bile is chemically toxic. Bile salts—like chenodeoxycholate and deoxycholate—and bile acids are detergents. They dissolve the protective mucus layer and punch holes in the lipid membranes of epithelial cells lining the gallbladder, killing them. Dead and dying cells release damage-associated molecular patterns (DAMPs, fragments of broken cells that act like alarm signals) and cytokines (interleukin-1, interleukin-6, tumor tissue death (necrosis) factor-alpha—alarm proteins that call for help). These signals make nearby capillaries (tiny blood vessels) leaky by loosening the tight junctions between endothelial cells (the cells lining the vessel walls). Plasma (the liquid part of blood) leaks out into the tissue, causing swelling (edema)—the wall puffs up and thickens, going from under 3 millimeters normally to 5, 8, 10 millimeters or more. At the same time, neutrophils (the first-responder white blood cells) squeeze out of capillaries—a process called diapedesis (DIE-uh-puh-DEE-sis, meaning cells slipping through gaps)—and flood into the wall. Neutrophils release enzymes (elastase, myeloperoxidase) and reactive oxygen species (superoxide, hydrogen peroxide—bleach-like chemicals) to kill germs and clean up dead cells, but these same chemicals also damage healthy tissue, amplifying inflammation. Macrophages follow hours later, gobbling up debris and dead cells. All this—swelling, neutrophils, macrophages—makes the wall swell, stiffen, and hurt.

4. Bacteria migrate into the stagnant bile and multiply rapidly, turning sterile inflammation into infection. — why: Normal flowing bile acts like a river current, sweeping bacteria backward out of the gallbladder and down into the intestine. When bile sits still, bacteria—mostly *Escherichia coli* (*E. coli*), *Klebsiella pneumoniae*, *Enterobacter cloacae*, *Enterococcus faecalis*, and sometimes *Bacteroides fragilis* (an oxygen-hating germ)—drift up from the duodenum through the sphincter of Oddi and common bile duct, or seed the gallbladder from the bloodstream. Stagnant bile is a warm (37°C body temperature), nutrient-rich broth with no flow to flush germs away—it is a perfect culture medium. Bacteria double every 20 to 60 minutes under these conditions. Within 24 to 48 hours, bacterial counts climb into the millions or billions per milliliter. The gallbladder fills with pus—called empyema (em-pye-EE-muh, meaning pus in a body cavity). Bacteria release endotoxins (lipopolysaccharide fragments from Gram-negative cell walls) and exotoxins (secreted proteins) that trigger fever, a high white blood cell count (leukocytosis), and systemic inflammation. Without antibiotics and source control (removing the blockage), infection spreads into the wall—called suppurative cholecystitis (pus-forming inflammation)—and can spill into the bloodstream (bacteremia, sepsis).
5. Fever and a high white blood cell count (leukocytosis) (high white blood cell count) develop. — why: Bacterial endotoxins and cytokines—especially interleukin-1 and interleukin-6 released by macrophages and neutrophils—travel through the bloodstream to the hypothalamus (high-po-THAL-uh-muss, your brain's thermostat, located at the base of your brain just above the pituitary gland). They bind to receptors in the preoptic area of the hypothalamus and trigger the release of prostaglandin E₂ (PGE₂, a fatty acid signal), which resets the thermostat upward. Your brain now thinks 38 to 39°C (100 to 102°F) is normal instead of 37°C, so it makes you shiver to generate heat and constricts blood vessels in your skin to hold heat in—you feel cold and feverish at the same time. Fever speeds up your immune system (white cells move and kill faster at higher temperatures) and slows bacterial growth (most bacteria grow best at 37°C). At the same time, cytokines signal your bone marrow (the spongy tissue inside bones where blood cells are made) to release stored neutrophils from the reserve pool and to crank up production of new white cells. Your white blood cell count climbs above the normal range of 4,000 to 11,000 per microliter, often reaching 12,000 to 20,000 or higher in severe infection. A very high count (above 20,000) or a left shift (seeing immature neutrophils called bands on the blood smear) suggests severe or spreading infection.

6. Positive Murphy sign on physical exam. — why: You place your fingers under the patient's right ribcage, just below the edge of the liver, and press firmly inward and upward. Then you ask the patient to take a deep breath in. As the diaphragm (the dome-shaped breathing muscle separating chest from belly) pushes down, it pushes the liver and the inflamed gallbladder downward onto your fingers. The swollen, tender gallbladder hits your hand, and sudden sharp pain shoots through the patient—they gasp, stop breathing mid-inhalation, and may grimace or pull away. That involuntary stop is a positive Murphy sign. It happens because the inflamed gallbladder wall is packed with activated pain nerves; pressing on it triggers a flood of pain signals. A positive Murphy sign is about 65% sensitive (it is positive in 65 out of 100 people who truly have cholecystitis) and 87% specific (if it is positive, there is an 87% chance cholecystitis is present), making it a useful bedside clue. If Murphy sign is negative and the patient has no RUQ tenderness at all, cholecystitis is much less likely (high negative predictive value).
7. Ultrasound or CT shows a thickened gallbladder wall—greater than 3 to 4 millimeters, often 5 to 10 millimeters or more. — why: Swelling (Edema) fluid and inflammatory cells (neutrophils, macrophages, plasma proteins) pack into the wall, swelling it up. On ultrasound, the normal gallbladder wall is a thin bright line less than 3 millimeters thick. In cholecystitis, it looks thick, hypoechoic (dark because fluid does not reflect ultrasound waves well), and sometimes striated or layered—three distinct layers (mucosa, swollen (edematous) muscularis, serosa) separated by fluid, giving a bull's-eye or target sign. On CT with IV contrast, the wall enhances (lights up white) because inflamed vessels are leaky and contrast leaks into the tissue. Wall thickness greater than 4 millimeters is about 50 to 70% sensitive and 80 to 90% specific for acute cholecystitis when combined with other findings. Very thick walls—8 to 10 millimeters or more—or irregular, uneven thickening suggest severe inflammation or early gangrene.
8. Pericholecystic fluid—fluid around the gallbladder—appears on imaging. — why: Inflammation makes the capillaries and small vessels in and around the gallbladder wall leaky. Plasma and inflammatory fluid seep out into the pericholecystic space (the narrow gap between the gallbladder and the liver or omentum). On ultrasound, fluid appears as a dark (anechoic or hypoechoic) rim or crescent around the gallbladder. On CT, it looks like a bright halo (because fluid is denser than fat on CT). A small amount of pericholecystic fluid is common in acute cholecystitis and just means inflammation is severe. A large amount, especially if loculated (walled off in pockets) or with gas bubbles, suggests localized perforation, abscess formation, or gangrenous cholecystitis.

Spot it — what you see, and why

- Steady pain in the right upper belly lasting more than 6 hours: A gallstone plugs the cystic duct (the tube that drains bile out of the gallbladder). Bile backs up inside and stretches the gallbladder wall like an overfilled water balloon. The stretched lining sends steady pain signals to your brain.
- Pain shooting to the right shoulder or shoulder blade: The phrenic nerve senses both your diaphragm and your gallbladder, and it also carries feeling from your shoulder. Your brain mixes up the signals and you feel pain in your shoulder even though the real problem is in your belly. Doctors call this mix-up referred pain.
- Fever (temperature above 100.4°F or 38°C): Bacteria trapped in the gallbladder release toxins. These toxins tell the hypothalamus (your brain's thermostat) to turn up your body heat. Higher temperature helps your white blood cells kill the bacteria.
- Nausea and throwing up: Pain signals and inflammation chemicals trigger the vomiting center in your brainstem (the part at the base of your brain that controls basic body functions). The vagus nerve (a large nerve that connects your gallbladder to your stomach) carries the alarm. Your stomach slows down and you feel sick.
- Murphy sign (you gasp and stop breathing when the doctor presses your right upper belly while you breathe in deeply): When you breathe in, your diaphragm pushes down and shoves your swollen, tender gallbladder right onto the examiner's hand. The sudden sharp pain makes you gasp and freeze mid-breath.
- High white blood cell count (above 10,000 cells per microliter): Your bone marrow pumps out extra white blood cells—especially neutrophils (the bacteria-fighting kind)—into your bloodstream to battle the infection inside your gallbladder.

Red flags — act now

- Rigid belly, rebound tenderness, or guarding—signs the gallbladder may have torn open and bile is leaking into the belly (peritonitis)
- Low blood pressure, racing heart, or confusion—signs of septic shock from infection in the bloodstream
- Gas bubbles in the gallbladder wall on ultrasound or CT (emphysematous cholecystitis)—much higher risk the tissue is dying (gangrene) and will tear open, especially in people with diabetes
- Yellow skin and eyes with high bilirubin level—suggests a stone is blocking the common bile duct or pressing on it (Mirizzi syndrome)
- Severe pain that seems worse than what the physical exam shows—raises concern for dead gallbladder tissue (gangrenous cholecystitis) or blood flow cut off (ischemia)

Treat it — in this order

1. Intravenous fluids (normal saline or lactated Ringer solution) and nothing by mouth (NPO) → IV fluids replace water and salt lost from vomiting, fever, and not drinking. Most patients need 1 to 2 liters in the first few hours, then 100 to 150 mL per hour to keep up with ongoing losses. Keeping the patient NPO stops the intestine from releasing cholecystokinin (a hormone made by I-cells in the duodenum, the first part of the small intestine, when fat or protein arrives). Cholecystokinin tells the gallbladder to squeeze. When you stop that signal, the inflamed gallbladder can rest instead of contracting against a blocked exit, which would spike the pressure inside and worsen pain and swelling.
2. Broad-spectrum intravenous antibiotics: ceftriaxone 1 to 2 grams once daily plus metronidazole 500 mg every 8 hours, or piperacillin-tazobactam 3.375 grams every 6 hours (or 4.5 grams every 8 hours for severe infection), or a carbapenem (meropenem 1 gram every 8 hours or ertapenem 1 gram daily) if very sick, septic, or resistant bacteria are suspected → Antibiotics soak into the inflamed gallbladder tissue and bile, killing common bacteria such as *E. coli*, *Klebsiella*, *Enterococcus*, and anaerobes like *Bacteroides fragilis* (bacteria that live without oxygen in the gut). Ceftriaxone is a third-generation cephalosporin that kills gram-negative rods (rod-shaped bacteria) by punching holes in their cell walls; metronidazole kills anaerobes by damaging their DNA. Piperacillin-tazobactam covers both groups in one medicine. This stops infection from spreading into the bloodstream (sepsis), the liver (hepatic abscess), or through the gallbladder wall (gangrenous cholecystitis or perforation). The 2018 Tokyo Guidelines and 2010 Surgical Infection Society guidelines recommend coverage of gram-negative rods and anaerobes for community-acquired infection, and broader coverage (carbapenems or piperacillin-tazobactam) for hospital-acquired or high-risk cases (diabetes, immunosuppression, age over 70).
3. Pain medicine (IV ketorolac 15 to 30 mg every 6 hours for up to 5 days, or opioids like morphine 2 to 10 mg IV every 2 to 4 hours or hydromorphone 0.5 to 2 mg IV every 2 to 4 hours) and anti-nausea drugs (ondansetron 4 to 8 mg IV every 8 hours) → Pain medicine blocks pain signals. Ketorolac is an NSAID (nonsteroidal anti-inflammatory drug) that stops cyclooxygenase (an enzyme that makes prostaglandins, the chemicals that send pain and swelling messages), cutting pain and inflammation at the injury site. Use it carefully — no more than 5 days — because it can hurt the kidneys or cause stomach bleeding, especially in older adults. Opioids like morphine and hydromorphone bind to mu-opioid receptors in the spinal cord and periaqueductal gray (a pain-control center in the midbrain), shutting down pain signals before they reach consciousness. Anti-nausea drugs like ondansetron block serotonin (a brain chemical messenger) at 5-HT₃ receptors in the chemoreceptor trigger zone and vomiting center in the medulla oblongata (the brainstem), cutting nausea and preventing more fluid loss from throwing up.

4. Laparoscopic cholecystectomy (keyhole surgery to remove the gallbladder) within 72 hours of symptom start → Surgery takes out the entire gallbladder, stones and all, ending the obstruction and infection permanently. The surgeon makes 3 to 4 small cuts in the belly, inflates it with carbon dioxide gas to make room, and uses a camera and long instruments to clip off the cystic duct and cystic artery (the blood vessel feeding the gallbladder), then pulls the gallbladder out through one of the holes. The 2018 Tokyo Guidelines and randomized trials (ACDC 2013, INTACT 2020) show operating within 72 hours (early surgery) shortens hospital stay by 3 to 5 days, lowers complication rates (from 25% to 10%), and cuts readmissions (from 20% to under 5%) compared to waiting 6 to 12 weeks (delayed surgery). After the gallbladder is gone, bile flows straight from the liver through the common hepatic duct (the liver's main drain pipe) into the common bile duct and then the duodenum — you do not need a gallbladder to digest fat; the liver makes bile continuously (about 500 to 1,000 mL per day) and it drips steadily into the gut instead of being stored and squirted in bursts.
5. ERCP (endoscopic retrograde cholangiopancreatography) with sphincterotomy and stone removal if imaging or labs suggest a stone in the common bile duct → A flexible camera scope (an endoscope) goes down the throat, through the stomach, and into the duodenum (the first part of the small intestine) to reach the ampulla of Vater (the opening where the common bile duct and pancreatic duct empty into the intestine). The doctor threads a thin wire into the bile duct, injects contrast dye, and takes X-rays to see stones. Then a wire with electric current cuts the sphincter of Oddi (the muscular valve that controls bile and pancreatic juice flow), widening the opening. A tiny wire basket or balloon catheter is used to pull out any stones, restoring bile flow from liver to gut. This prevents ascending cholangitis (infection climbing up the bile ducts into the liver), which causes fever, yellowing of the skin and eyes (jaundice), and confusion (called Charcot triad) and can lead to septic shock. The 2019 American Society for Gastrointestinal Endoscopy (ASGE) guidelines recommend ERCP when total bilirubin is above 4 mg/dL, the common bile duct is dilated above 6 mm on ultrasound (or above 8 mm in older patients or those who had previous surgery), or there is yellowing of the skin and eyes plus very high alkaline phosphatase (above 3 times normal, around 360 U/L).

Your top priorities at the bedside

1. Start intravenous normal saline or lactated Ringer solution at 125–150 mL per hour and keep the patient NPO (nothing by mouth). — why: The inflamed gallbladder needs rest; eating or drinking makes it squeeze and worsens pain and pressure—IV fluids prevent dehydration from vomiting and maintain blood pressure while the patient waits for surgery.

2. Give the first dose of broad-spectrum IV antibiotics within 1 hour: ceftriaxone 1–2 grams plus metronidazole 500 mg, or piperacillin-tazobactam 3.375 grams. — why: Bacteria (usually *E. coli*, *Klebsiella*, or *Enterococcus*) grow rapidly in trapped bile—early antibiotics stop the infection from spreading into the bloodstream (sepsis) and reduce the chance the gallbladder wall will die (gangrene) before surgery.
3. Assess vital signs every 1–2 hours and watch for fever above 101°F, heart rate above 110, blood pressure below 90 systolic, or oxygen saturation below 94 percent. — why: Rising fever and heart rate with falling blood pressure signal worsening sepsis or septic shock (infection overwhelming the body)—catching these early lets the team give more fluid, start vasopressors (medicines that raise blood pressure), and move surgery up.