

Acute Cholecystitis

A plain-English guide for you and your family

WHAT IS HAPPENING

Your gallbladder is a pear-shaped pouch about the size of a small kiwi tucked under the right side of your liver. Its job is to store bile—a yellow-green digestive juice your liver makes all day—and squeeze it out when you eat fatty food so your intestine can break the fat down and absorb it. Bile flows out through a narrow tube called the cystic duct, and that flowing bile also sweeps bacteria backward out of the gallbladder like a river current washing leaves away. Now picture what happens when a gallstone—a hard pebble made of cholesterol or bile pigments—rolls into that tube and gets stuck like a cork in a bottle. Bile keeps flowing in from the liver, but nothing can get out. Pressure builds inside the gallbladder, stretching the wall like air pumped into a balloon. The trapped bile is chemically harsh—bile salts and acids act like dish soap, stripping away the protective lining and killing cells. Damaged cells leak alarm chemicals that make nearby blood vessels leaky, so fluid seeps into the wall and it swells up thick and puffy. At the same time, bacteria from your intestine—mostly *E. coli* and its cousins—drift up the ducts or hitch a ride through your bloodstream and land in the stagnant bile. Because the bile isn't flowing anymore, those bacteria multiply fast, turning sterile swelling into full-blown infection. That one stuck stone upstream explains everything you feel downstream: the steady, unrelenting pain in your right upper belly that won't quit (because the stretched wall is pressing on pain nerves), the fever (because bacteria release toxins that reset your brain's thermostat), the nausea (because pain and inflammation signals travel to your brainstem's vomit center), and the jump in your white blood cell count (because your bone marrow is flooding your bloodstream with infection-fighting cells). When the doctor presses under your right ribs and asks you to take a deep breath, your swollen gallbladder gets pushed down onto their fingers and you gasp in pain—that's called Murphy sign, and it's a bedside clue that your gallbladder is angry. Ultrasound or CT imaging shows the gallbladder wall thick and swollen—normally paper-thin at under 3 millimeters, now ballooned to 5 or 10 millimeters or more—often with fluid pooling around it like a moat. None of this is random. Every single finding—the pain, the fever, the thick wall, the fluid—flows directly from that one plugged tube. Your immune system is buying time, walling off the infection and flooding the area with white cells, but it can't fix the root problem: the stone is still stuck. If the blockage isn't removed, pressure keeps climbing, squeezing the blood vessels in the wall shut so oxygen can't reach the tissue. Without oxygen, cells die and the wall turns black—that's called gangrene. Dead tissue can tear open (perforation), spilling infected bile into your belly, or gas-forming bacteria can invade the dying wall (emphysematous cholecystitis), making the infection race ahead even faster. Both are surgical emergencies. The treatment is to remove the stone and drain or take out the gallbladder, usually by laparoscopic surgery (small incisions and a camera), plus antibiotics to kill the bacteria. Trace every symptom back to that one stuck stone, and the whole picture snaps into focus and makes sense.

What you might feel

- Steady pain in the right upper belly lasting more than 6 hours
- Pain shooting to the right shoulder or shoulder blade
- Fever (temperature above 100.4°F or 38°C)
- Nausea and throwing up
- Murphy sign (you gasp and stop breathing when the doctor presses your right upper belly while you breathe in deeply)
- High white blood cell count (above 10,000 cells per microliter)
- Loss of appetite
- Yellow skin and eyes (jaundice) in about 10 to 15 out of 100 people
- Severe pain with a rigid, board-like belly and rebound tenderness (pain when the doctor lets go after pressing)
- Confusion, dizziness, or very low blood pressure with a racing heart

Call the doctor right away if...

- 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)

What the care team will do

- Start intravenous normal saline or lactated Ringer solution at 125–150 mL per hour and keep the patient NPO (nothing by mouth).
- 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.
- 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.

- Perform a focused abdominal exam every 4 hours: palpate gently for Murphy sign, check for rebound tenderness, guarding, or a rigid belly.
- Administer IV pain medicine (ketorolac 15–30 mg every 6 hours or morphine 2–4 mg every 2–4 hours) and an anti-nausea drug (ondansetron 4 mg IV).
- Monitor intake and output hourly; report urine output below 30 mL per hour or dark, concentrated urine.
- Check the first set of labs: complete blood count (CBC) for white count above 10,000 cells per microliter, comprehensive metabolic panel (CMP) for elevated bilirubin or liver enzymes, and lipase to rule out pancreatitis.
- Prepare the patient for urgent ultrasound of the right upper abdomen, watching for a thickened gallbladder wall, pericholecystic fluid (fluid around the gallbladder), or stones.

Medicines you may be given — and why

Intravenous fluids (normal saline or lactated Ringer's solution)

When you have acute cholecystitis, you cannot eat or drink because your gut needs to rest, and you may throw up a lot. Your body loses water and salt. IV fluids are sterile salt-water mixtures that run through a thin tube placed in a vein. They flow straight into your bloodstream to replace what you lost. They keep your blood pressure steady, help your kidneys flush out waste, and make sure every organ gets enough oxygen-rich blood. Think of it like watering a wilting plant—the water travels through the roots to every leaf, bringing the plant back to life.

Ondansetron (Zofran)

When the gallbladder is blocked and swollen, pain and irritation in your belly trigger nerve signals that tell your brain to make you vomit. Cells in your gut release a chemical messenger called serotonin (5-HT₃), and that serotonin docks onto special landing spots (5-HT₃ receptors) on the vagus nerve (the main nerve highway running from your gut to your brainstem) and in the chemoreceptor trigger zone (the CTZ, a vomit sensor in the brainstem that reads signals in the blood). When serotonin binds to these receptors, the vomiting control center in your brainstem (the nucleus tractus solitarius) gets the message and tells your stomach muscles to heave. Ondansetron blocks those 5-HT₃ receptors—it sits on them so serotonin cannot land. Without serotonin locking on, the vomit signal never gets loud enough to make you throw up. Think of it like unplugging a fire alarm—the smoke (the nausea signal) is still there, but the bell cannot ring.

Ketorolac (Toradol)

When the gallbladder is blocked and swollen, damaged cells release an enzyme called cyclooxygenase (COX—comes in two forms, COX-1 and COX-2). COX enzymes take a fatty acid from broken cell membranes (arachidonic acid) and turn it into chemicals called

prostaglandins. Prostaglandins make pain nerve endings more sensitive (lower the pain threshold) and increase swelling and heat in the tissue. Ketorolac blocks both COX-1 and COX-2, so fewer prostaglandins get made. With less prostaglandin around, pain nerves send weaker signals, inflammation drops, and you feel less pain. Think of prostaglandins as the volume knob on a loudspeaker broadcasting pain—ketorolac turns the knob down so the signal is quieter.

Morphine

Severe pain from the swollen gallbladder travels up through nerves to the spinal cord and then to the brain, where you consciously feel it. Morphine is a drug that mimics your body's natural pain-relief chemicals (endorphins and enkephalins). It locks onto mu-opioid receptors (special docking proteins) on pain nerve endings in the belly, on relay neurons in the spinal cord, and on neurons in the brain (especially the periaqueductal gray, the thalamus, and the limbic system—the parts that process pain intensity and the emotional suffering it causes). When morphine activates these receptors, it opens potassium channels (potassium ions flow out of the nerve cell) and closes calcium channels (calcium cannot get in). This makes the nerve less excitable (hyperpolarized), so it is harder for the nerve to fire a pain signal. Morphine also reduces the release of pain-transmitting chemicals (substance P and glutamate) from nerve endings. The result: fewer pain signals leave the belly, fewer reach the brain, and the brain cares less about the signals that do arrive. Think of it like turning down the brightness on a screen—the image (pain) is still there, but it is dimmer and easier to tolerate.

Piperacillin-tazobactam (Zosyn)

When bile is trapped in the gallbladder, bacteria from the intestines (especially *Escherichia coli*, *Klebsiella pneumoniae*, *Enterococcus* species, and anaerobes like *Bacteroides fragilis*) can swim backward up the bile ducts and multiply in the stagnant bile, causing infection.

Piperacillin kills these bacteria by breaking their cell walls. Bacteria build their walls out of a tough mesh called peptidoglycan, which is assembled by enzymes called penicillin-binding proteins (PBPs). Piperacillin is shaped like the natural building block of peptidoglycan, so it tricks the PBPs into grabbing it. Once the PBPs bind to piperacillin, they get stuck and cannot finish building the wall. The wall develops holes and weak spots. Water rushes into the bacteria (osmosis), the bacteria swell up like a balloon, and they burst (lysis). Some bacteria fight back by making enzymes called beta-lactamases that cut piperacillin apart before it can work.

Tazobactam is a 'bodyguard' drug—it binds permanently to beta-lactamase enzymes and inactivates them (suicide inhibition). With tazobactam protecting piperacillin, the antibiotic can kill even bacteria that try to resist it. Together, piperacillin-tazobactam covers a broad range of gram-positive, gram-negative, and anaerobic bacteria.

Ceftriaxone (Rocephin) plus metronidazole (Flagyl)

This is a two-drug combination that together covers the main bacteria causing acute cholecystitis. Ceftriaxone kills aerobic bacteria (bacteria that need oxygen to survive), especially gram-negative rods like *Escherichia coli* and *Klebsiella pneumoniae*, and some gram-positive bacteria like *Streptococcus* species. It works the same way as piperacillin: it binds to penicillin-binding proteins (PBPs) in the bacterial cell wall, blocks them from finishing the peptidoglycan mesh, weakens the wall, and causes the bacteria to burst when water rushes in. Metronidazole kills anaerobic bacteria (bacteria that live in low-oxygen or no-oxygen environments), especially *Bacteroides fragilis* and *Clostridium* species, which are common in bile and gut contents. Metronidazole is a prodrug—it enters the bacteria and is activated by bacterial enzymes called nitroreductases (which only exist in anaerobic bacteria). Once activated, metronidazole forms toxic free radicals (highly reactive molecules with unpaired electrons) that attack and shred the bacteria's DNA, proteins, and cell membranes. The bacteria's DNA breaks into fragments, the bacteria cannot copy themselves, and they die. Together, ceftriaxone and metronidazole cover almost all the bacteria that cause cholecystitis.

Where this information comes from

- Tokyo Guidelines 2018 (TG18)
- American College of Gastroenterology
- World Society of Emergency Surgery 2020
- American College of Surgeons

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