

Acute Pancreatitis

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THE STORY IN ONE SENTENCE

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“Pancreatic enzymes turn on while still inside the pancreas, causing autodigestion (the organ eats its own tissue) and triggering a body-wide inflammatory storm that can shut down the lungs, kidneys, and heart.”

Normal anatomy — the body's job

Acinar cells in the pancreas make digestive enzymes in an inactive, locked form called zymogens. The pancreatic duct—a tube running through the middle of the gland—carries these inactive enzymes safely to the duodenum (the first part of your small intestine), where an enzyme in the intestinal wall called enterokinase unlocks trypsinogen and turns it into active trypsin. Trypsin then activates all the other enzymes, and together they break down the proteins, fats, and carbohydrates in your food. This controlled handoff keeps the pancreas safe. Keep this picture in mind, because every abnormal finding in pancreatitis is a departure from this safe, step-by-step process.

BODY'S JOB

The pancreas is both a factory and a delivery service: it makes the enzymes, stores them in a safe (inactive) form, and ships them through a protected duct to the place where they are supposed to work—the small intestine. If the enzymes activate too early, the factory starts digesting itself.

1. Acinar cells (enzyme-making cells) produce trypsinogen, chymotrypsinogen, proelastase, phospholipase, and other inactive enzymes and package them into granules.
2. These granules move through tiny branch ducts that merge into the main pancreatic duct, which runs the length of the pancreas like a river collecting tributaries.

3. The main pancreatic duct joins the common bile duct (carrying bile from the liver and gallbladder) at the ampulla of Vater, a shared exit door in the wall of the duodenum.
4. A ring of muscle called the sphincter of Oddi guards the ampulla and opens when food enters the duodenum, releasing both bile and inactive pancreatic enzymes into the intestinal space.
5. Once in the duodenum, enterokinase (an enzyme anchored in the intestinal lining) clips trypsinogen and converts it to active trypsin.
6. Trypsin then activates all the other enzyme precursors, so proteins, fats, and starches in your food are broken down into pieces small enough to absorb.
7. The enzymes never touch living pancreatic tissue under normal conditions because they stay inactive until they leave the gland and reach the intestine.

What breaks

Usually one upstream problem explains all the downstream findings together. In acute pancreatitis, that upstream break is premature activation of digestive enzymes inside the pancreas instead of in the intestine, which leads to the gland digesting itself and spilling inflammatory chemicals into the bloodstream.

CLINICAL TERM

Gallstones blocking the ampulla of Vater (the shared exit for bile and pancreatic ducts) cause about 40 percent of cases. Chronic alcohol use, which damages acinar cells and disrupts enzyme packaging, accounts for another 30 percent. Hypertriglyceridemia (triglycerides above 1,000 milligrams per deciliter) causes toxic free fatty acids that injure acinar cells. High calcium (Hypercalcemia) (high blood calcium) activates trypsinogen prematurely. Drugs—including azathioprine, valproic acid, thiazide diuretics, and others—trigger immune or toxic injury. Post-ERCP manipulation (a scope procedure to clear bile ducts) causes trauma and swelling (edema) at the ampulla. Autoimmune pancreatitis occurs when the body's immune system attacks the pancreas. With no known cause (Idiopathic) means no cause is found even after full work-up. The key break in every case is premature activation of trypsinogen to trypsin inside the pancreas, which then activates all the other digestive enzymes in a domino effect. This local autodigestion releases pro-inflammatory cytokines—tumor tissue death (necrosis) factor alpha, interleukin one, interleukin six—into the bloodstream. Read every clinical finding backward from this upstream break: they all point to enzyme activation and cytokine release.

How the body compensates

The body works hard to keep vital signs stable even while organs are being damaged. Heart rate, blood pressure, and oxygen saturation can look near-normal for hours to days because your body squeezes blood vessels, releases adrenaline and cortisol, and shifts fluid around. This stable phase is dangerous because it hides how sick the patient really is—compensation can mask impending organ failure. Your job is to see the tipping point coming before the patient crashes.

1. {'what': 'The body keeps heart rate, blood pressure, and oxygen saturation near normal for hours to days by constricting blood vessels in the skin and gut, releasing stress hormones (adrenaline, cortisol), and moving fluid from cells and tissues into the bloodstream.', 'why': 'The brain and heart have top priority, so the body sacrifices less critical areas to keep blood flowing to vital organs. This buys time but uses up reserves.'}
2. {'what': 'Watch for persistent a fast heart rate (tachycardia) (heart rate above 90 to 100 even at rest), rising hematocrit (above 44 percent), and falling urine output (less than 0.5 milliliters per kilogram per hour)—these are early warnings that compensation is wearing out.', 'why': 'A fast heart rate means the heart is working overtime to move blood that is too thick because plasma has leaked out. High hematocrit means fluid loss is not being replaced fast enough. Low urine output means the kidneys are not getting enough blood flow.'}
3. {'what': 'Lactate rises (above 2 millimoles per liter) when tissues switch to anaerobic metabolism because oxygen delivery is falling short.', 'why': 'When cells do not get enough oxygen, they burn sugar without oxygen and produce lactic acid as a waste product. Rising lactate is a red flag that organs are starving.'}
4. {'what': 'Intervene early with aggressive intravenous fluid resuscitation: 250 to 500 milliliters per hour of lactated Ringer solution in the first 12 to 24 hours, guided by heart rate, blood pressure, urine output, and hematocrit.', 'why': 'Replacing lost plasma volume before compensation fails can prevent shock, acute kidney injury, and multi-organ failure. Lactated Ringer is preferred over normal saline because it more closely matches the body's natural fluid and does not cause harmful too much acid in the blood (acidosis).'} }
5. {'what': 'The crash point to watch for is persistent organ failure lasting more than 48 hours: respiratory failure needing supplemental oxygen or a ventilator, shock needing vasopressor medicines to keep blood pressure up, or acute kidney injury with creatinine rising above 1.9 milligrams per deciliter.', 'why': 'Organ failure that does not improve within two days means the inflammatory storm is overwhelming the body's ability to recover, and mortality jumps from under 3 percent in mild cases to 20 to 30 percent.'}

6. {'what': 'Infected pancreatic tissue death (necrosis) (usually appearing 7 to 10 days after symptom onset) carries 15 to 30 percent mortality even with intensive care and surgical removal of dead tissue.', 'why': 'Dead pancreatic tissue has no blood flow, so white blood cells and antibiotics cannot reach it. Bacteria from the gut migrate across the damaged intestinal barrier, colonize the dead tissue, and cause sepsis.'}
7. {'what': 'Abdominal compartment syndrome—intra-abdominal pressure above 20 millimeters of mercury measured through a bladder catheter—compresses blood vessels and organs, causing starved blood flow (ischemia) and organ failure. This is a surgical emergency requiring decompression.', 'why': 'Massive fluid leaking into the belly and swollen intestines increase pressure inside the abdominal cavity like overfilling a balloon. High pressure squeezes the kidneys, liver, and blood vessels, stopping blood flow.'}
8. {'what': 'Watch trends over hours—rising heart rate, falling urine output, climbing lactate, worsening low oxygen (hypoxia), hematocrit staying high despite fluids—not just single values, because the direction tells you whether compensation is winning or losing.', 'why': 'One abnormal number can be noise, but a pattern of worsening numbers means the body is losing the fight and you need to escalate care—transfer to intensive care, add pressors, intubate—before full collapse.'}

Domino effect

1. Premature activation of trypsinogen to trypsin inside the pancreas triggers a cascade that activates all the other digestive enzymes—chymotrypsin, elastase, phospholipase A2, lipase—still inside the gland. — Trypsinogen normally converts to active trypsin only in the duodenum, where enterokinase flips the switch. But when trypsinogen activates early—because of a blocked duct, toxic injury, or reflux—trypsin cuts and activates all the other enzyme precursors (zymogens) that are still stored in the pancreas. It is like dominoes: one enzyme tips and all the others fall.
2. Activated enzymes digest pancreatic tissue (autodigestion): lipase breaks down fat, elastase breaks down blood vessel walls and connective tissue, and phospholipase attacks cell membranes. — These enzymes are designed to break down food, but now they are breaking down living pancreatic cells. Fat tissue death (necrosis) releases free fatty acids, which bind calcium and can cause low blood calcium. Blood vessel damage causes bleeding into and around the pancreas.
3. Dying pancreatic cells release damage-associated molecular patterns (DAMPs) and activate immune cells, which pour pro-inflammatory cytokines—tumor tissue death (necrosis) factor alpha, interleukin 1, interleukin 6—into the bloodstream. — When cells die by injury (not normal programmed death), they spill their contents, and the immune system reads those spilled molecules as danger signals. Macrophages and neutrophils rush in and release cytokines—chemical alarm bells that call more immune cells and amplify inflammation.

4. Systemic cytokines cause widespread capillary leak (increased vascular permeability), so plasma shifts out of blood vessels into the interstitial space (third-spacing) and the peritoneal cavity. — Inflammatory cytokines make endothelial cells (the lining of blood vessels) pull apart slightly, creating gaps. Plasma—the liquid part of blood—leaks through those gaps into tissues and the belly, but red blood cells are too big to escape. Think of it like a screen door with holes that let water through but not marbles.
5. Hematocrit rises (hemoconcentration) because red blood cells stay in the vessels while plasma volume drops. A hematocrit above 44 percent on admission or failure to drop below 44 percent at 24 hours predicts severe disease. — When fluid leaves the bloodstream, the percentage of red cells in each drop of blood goes up—like removing water from a sponge makes it denser. High hematocrit tells you the patient is losing plasma faster than they are getting it back, and the blood is becoming thick and sluggish.
6. Effective circulating volume falls, so cardiac output and organ blood flow (perfusion) drop, triggering a fast heart rate (tachycardia) (fast heart rate) and eventually low blood pressure (hypotension) (low blood pressure). — With less fluid in the pipes, the heart has less blood to pump. It tries to compensate by beating faster to move what little volume is left, but eventually the tank runs too low and blood pressure falls. Organs do not get enough oxygen and nutrients.
7. Systemic Inflammatory Response Syndrome (SIRS) develops: body temperature above 38 degrees Celsius or below 36, heart rate above 90, respiratory rate above 20, white blood cell count above 12,000 or below 4,000 per microliter. — Cytokines reach every organ and reset the body's thermostat (causing fever or, in severe cases, hypothermia), speed up the heart and lungs, and mobilize or exhaust white blood cells. This is the body's whole-body alarm—not just a local pancreas problem—and it means the inflammatory storm has gone systemic.
8. In severe cases, acute respiratory distress syndrome (ARDS), acute kidney injury (AKI), and shock occur because organs far from the pancreas fail from low blood flow (perfusion) and cytokine injury. — Cytokines damage the lining of lung capillaries, causing fluid to flood the air sacs (alveoli) and block oxygen exchange—that is ARDS. Low blood flow and cytokine toxins kill kidney tubule cells, so the kidneys stop filtering waste and creatinine rises—that is AKI. The heart muscle itself becomes depressed by cytokines, and blood vessels dilate when they should constrict, so blood pressure crashes—that is distributive shock.

9. Pancreatic and peripancreatic tissue death (necrosis) (tissue death) becomes infected after 7 to 14 days, usually with gut bacteria (*Escherichia coli*, *Klebsiella*, *Enterococcus*) that translocate across the damaged intestinal barrier. — Dead tissue has no blood flow, so white blood cells and antibiotics cannot reach it to fight infection. At the same time, inflammation and low blood flow damage the gut lining, making it leaky. Bacteria that normally live in the colon migrate through the leaky gut wall, travel through lymph or blood, colonize the dead pancreas, and cause sepsis with very high mortality—15 to 30 percent even with intensive care and surgery to remove the infected tissue.

Symptoms · what the patient feels, and why

- Sudden, severe pain in the upper-middle belly that shoots straight through to the back: The pancreas sits deep in your belly, right against the back wall. When it swells up and starts digesting itself (the enzymes meant for food attack the pancreas instead), that irritates the nerves running to your back. The pain is steady and boring—like a drill—not crampy waves.
- Nausea and throwing up: The inflamed pancreas releases alarm chemicals (cytokines) that travel to your brain and flip the vomit switch. Also, the swollen pancreas presses on your stomach and gut, making you feel sick. The vomiting can be relentless and makes you lose even more fluid.
- Belly is tender when pressed, especially just below the ribs in the center; muscles tighten up automatically (guarding): Leaked pancreatic enzymes and swelling irritate the lining of your abdomen (the peritoneum—a thin sheet wrapping your organs). When the doctor presses, it hurts, and your belly muscles lock up on their own to guard the sore spot.
- Belly looks swollen and feels tight; bowel sounds are very quiet or silent (ileus): The alarm chemicals slow down your gut's squeezing movements, so it goes quiet. At the same time, liters of fluid leak out of your blood vessels into your belly (third-spacing—fluid hiding in the wrong place), making your abdomen puff up like a balloon.
- Fast heart rate—over 90 to 100 beats per minute (tachycardia): Your heart speeds up to make up for low fluid in your blood vessels. It's trying to pump harder and faster to keep your blood pressure up and deliver oxygen everywhere. If the heart stays racing, it means you're still losing fluid or not getting enough back in.
- Low blood pressure—top number below 90 (hypotension)—and signs of shock like cold hands and feet, slow color return when you press a fingernail (delayed capillary refill): So much fluid has leaked out of your blood vessels that there's not enough left to fill the system. Your heart can't keep the pressure up, so blood flow to your skin, kidneys, and brain drops dangerously low. This is shock.

- Very little urine or none at all—less than half a milliliter per kilogram of body weight per hour (oliguria or no urine output (anuria)): When blood pressure falls, your kidneys don't get enough blood flow to make urine. First they try to hold on to every drop (prerenal—before the kidney problem), but if shock continues, the kidney's filters get damaged (acute tubular tissue death (necrosis)) and stop working.
- Low oxygen in the blood (hypoxemia) and trouble breathing (dyspnea): Alarm chemicals from the pancreas travel to the lungs and damage the walls of the tiny air sacs (alveoli). Fluid and protein leak into those sacs, blocking oxygen from getting into your blood. This is called ARDS (acute respiratory distress syndrome) and can lead to respiratory failure.
- Confusion, being very sleepy, or not knowing where you are (altered mental status): Your brain needs a steady supply of oxygen and fuel. When blood pressure drops, oxygen is low, and waste products pile up (like acid), brain cells can't work right. Confusion is a red flag that many organs are failing.
- Fever—temperature above 38°C (100.4°F)—or, in very sick patients, a dangerously low temperature below 36°C (96.8°F): Alarm chemicals reset your body's thermostat higher, causing fever as your immune system fights. But if you're in septic shock (infection in the blood) or overwhelming inflammation, your body loses control and temperature drops—a very bad sign of collapse.

Focused exam

- Epigastric tenderness with pain radiating straight through to the back: The pancreas sits behind your stomach in the retroperitoneum (the space behind the belly organs, against the back wall of your abdomen). When the pancreas gets inflamed, it swells and presses on nerves that run to the back. The pain starts in the upper middle part of the belly and shoots straight through to the back—like a belt of pain wrapping around. This pattern tells you the pancreas is the problem, not the stomach or intestines.
- Fast heart rate above 100 beats per minute (tachycardia) and low blood pressure with the top number below 90 mmHg (hypotension): When the pancreas is injured, it releases chemicals called cytokines (signaling proteins) that make blood vessel walls leaky—like tiny holes punched in a garden hose. Fluid leaks out of the bloodstream into the tissues around it (called third-spacing). Now there is less fluid inside the blood vessels to pump around. The heart speeds up to try to keep enough blood flowing to your organs, but blood pressure drops because there is simply less fluid to push through the system. This combination—fast heart with low blood pressure—tells you the pancreatitis is moderately severe or severe and the patient is losing fluid fast.

- Very little urine output—less than 0.5 milliliters per kilogram of body weight per hour (oliguria): Your kidneys need steady blood flow to filter waste and make urine. When fluid leaks out of the blood vessels (third-spacing), blood flow to the kidneys drops. The kidneys respond by holding onto every drop of water they can—so urine output falls. Low urine output (Oliguria) (less than 0.5 mL/kg/h for at least 6 hours) is a red flag that the patient is volume-depleted (not enough fluid in the bloodstream) and the kidneys are starting to fail. You must give intravenous fluids immediately to restore blood flow and prevent acute kidney injury.
- Bruising on the flanks (the sides of the lower back, called Grey Turner sign) or around the belly button (called Cullen sign): In severe pancreatitis, activated digestive enzymes—especially one called elastase—eat through blood vessel walls in the tissues around the pancreas. Blood leaks out and tracks through tissue planes until it reaches the skin, where you see it as bruises. These bruises appear 24 to 48 hours after symptoms start. Their presence means necrotizing pancreatitis (the pancreas is dying and bleeding)—a life-threatening complication.
- Swollen, tight belly (abdominal distension) with the muscles involuntarily tightening when you press (guarding and rigidity): Severe pancreatitis causes swelling behind the abdominal organs and fluid buildup inside the belly cavity. When pressure inside the abdomen climbs above 20 mmHg, abdominal compartment syndrome can happen—the high pressure squeezes blood vessels and organs, cutting off their blood supply. Guarding (reflex muscle tightening) and rigidity mean the lining of the abdomen (peritoneum) is irritated and inflamed. This combination warns of compartment syndrome or peritonitis (infection of the abdominal lining), both surgical emergencies.
- Confusion, drowsiness, or altered mental status: Severe pancreatitis triggers a bodywide inflammatory storm called systemic inflammatory response syndrome (SIRS). Cytokines damage tiny blood vessels throughout the body, including in the brain. Blood flow to the brain drops (cerebral poor blood flow (hypoperfusion)), starving brain cells of oxygen. This causes encephalopathy (brain dysfunction)—the patient becomes confused, slow to respond, or hard to wake up. It signals that multiple organs are failing and the risk of death is high.

Diagnostics · every test answers a question

- Serum lipase or serum amylase → Is the pancreas injured right now, and how much digestive enzyme is leaking into the blood?
- Blood urea nitrogen (BUN) at admission and at 24 hours → Is the patient volume-depleted, and are the kidneys conserving fluid because blood flow is too low?
- Hematocrit at admission and again at 24 hours → Is third-spacing getting worse, and is the fluid resuscitation working?

- C-reactive protein (CRP) at 48 to 72 hours after symptom onset → How strong is the bodywide inflammatory response, and is this pancreatitis likely to turn severe?
- Contrast-enhanced CT scan of the abdomen and pelvis → Is there dead pancreatic tissue (necrosis), fluid collections, blood clots in vessels, or signs of infection that change what we do next?
- Serum triglycerides (fasting or random) → Are very high blood fat levels (hypertriglyceridemia) the cause of this pancreatitis, and does it need emergency treatment?
- Transabdominal ultrasound → Are there gallstones in the gallbladder, and is the common bile duct wider than normal (suggesting a stone is blocking it)?
- Serum calcium (corrected for albumin) → Is the calcium level dropping because fat tissue death (necrosis) is consuming it, signaling severe pancreatitis?

Treatment logic

- Aggressive intravenous fluid resuscitation with lactated Ringer solution at 5 to 10 mL/kg/h (or 250 to 500 mL/h in most adults), started immediately and titrated to urine output above 0.5 mL/kg/h, heart rate below 120 beats per minute, and mean arterial pressure 65 to 85 mmHg → Replaces the plasma (liquid part of blood) that leaked out of blood vessels into tissues (third-spacing). This restores blood volume so the heart can pump enough blood to the kidneys, brain, and other organs. Adequate early resuscitation (within the first 12 to 24 hours) cuts the risk of persistent organ failure, pancreatic tissue death (necrosis), and death. Lactated Ringer solution is better than normal saline because it does not cause too much acid in the blood (acidosis) (too much acid in the blood) or worsen the inflammatory response (SIRS).
- ERCP (endoscopic retrograde cholangiopancreatography) within 24 hours if there is cholangitis (infected bile duct with fever and yellowing of the skin and eyes (jaundice)) or persistent biliary obstruction (bilirubin rising above 4 mg/dL with a dilated bile duct on imaging) → Removes a gallstone stuck at the ampulla of Vater (the opening where the bile duct and pancreatic duct drain into the duodenum). Once the stone is out, pancreatic enzymes can flow normally into the intestine instead of backing up and activating inside the pancreas. Early ERCP (within 24 hours) in gallstone pancreatitis complicated by cholangitis or persistent obstruction reduces mortality and complications, according to multiple randomized trials and current ACG/AGA guidelines.

- Opioid analgesia—intravenous morphine, hydromorphone, or fentanyl—titrated to keep pain controlled (goal pain score 3 or less on a 0-to-10 scale) → Blocks opioid receptors in the brain and spinal cord, stopping pain signals from reaching conscious awareness. Good pain control reduces the body's stress response (sympathetic nervous system activation)—this allows normal breathing, early movement out of bed, and tolerance of food, all of which improve outcomes. Older teaching that morphine worsens pancreatitis by tightening the sphincter of Oddi (the muscle at the bile duct opening) has been disproven—no opioid is contraindicated in pancreatitis.
- Early oral or enteral feeding—starting within 24 to 48 hours as soon as nausea improves and the patient can tolerate intake—with a regular low-fat solid diet or nasogastric/nasojejunal tube feeding if the patient cannot eat by mouth → Keeps the intestinal lining (gut mucosa) healthy and prevents gut bacteria from crossing a weakened intestinal wall into the bloodstream (bacterial translocation). Early feeding reduces infectious complications (especially infected pancreatic tissue death (necrosis)), shortens hospital stay, and lowers mortality compared to keeping the patient fasting (NPO) or using intravenous nutrition (parenteral nutrition). There is NO benefit to pancreatic rest (keeping the pancreas unstimulated by fasting)—this old concept has been abandoned. Multiple randomized trials and current guidelines (ACG, AGA, ESPEN) strongly recommend early enteral feeding.
- Cholecystectomy (surgical removal of the gallbladder) during the same hospital admission for mild gallstone pancreatitis without tissue death (necrosis), or 6 weeks after discharge for moderately severe or severe pancreatitis once inflammation and fluid collections have resolved → Removes the gallbladder that contains stones, eliminating the source of future gallstone pancreatitis. Without cholecystectomy, 25 to 30% of patients have another attack within weeks. Timing depends on severity: in mild cases (no organ failure, no tissue death (necrosis)), same-admission cholecystectomy is safe and preferred per ACG/AGA guidelines; in severe cases, surgery is delayed until the pancreas heals (usually 6 weeks) to avoid surgical complications from inflamed, friable tissue.
- Antibiotics (carbapenems such as meropenem or imipenem, or a fluoroquinolone such as ciprofloxacin plus metronidazole) ONLY if imaging or clinical evidence confirms infected pancreatic tissue death (necrosis) or another documented infection (cholangitis, bacteremia, aspiration pneumonia) → Kills bacteria colonizing dead pancreatic tissue or causing bloodstream infection (bacteremia), preventing progression to septic shock (life-threatening infection causing multiorgan failure). Infected tissue death (necrosis) typically appears 7 to 10 days after symptom onset with new or worsening fever, rising white blood cell count, and organ failure, and is confirmed by gas in dead (necrotic) tissue on CT or positive bacterial cultures from fine-needle aspiration or drainage. Once infection is documented, antibiotics reduce mortality.

- Minimally invasive step-up approach (percutaneous or endoscopic drainage followed by minimally invasive necrosectomy if needed) for infected or symptomatic pancreatic tissue death (necrosis), delaying intervention until at least 4 weeks after symptom onset when a wall forms around the collection (walled-off tissue death) → Drains infected fluid and removes dead tissue (necrosectomy) using small incisions, endoscopes, or catheters instead of open surgery. The step-up approach—starting with drainage alone and adding necrosectomy only if drainage fails—reduces complications (new-onset organ failure, fistulas, bleeding) and death compared to immediate open necrosectomy, per the PANTER and PENGUIN randomized trials. Delaying intervention until a fibrous wall forms around the tissue death (necrosis) (usually 4 weeks) makes drainage safer and more effective.

Medications

Lactated Ringer's solution (IV fluid) · Isotonic crystalloid

When the pancreas is inflamed, the blood vessels around it and throughout the body become leaky—fluid escapes from inside the blood vessels into the surrounding tissue spaces, like water leaking out of a cracked pipe. This loss of fluid from the blood causes the blood volume to drop, so less blood reaches vital organs like the kidneys and brain. Lactated Ringer's is a salt-and-water solution given through an IV that refills the blood vessels, restores blood volume, and helps blood flow (perfusion) reach all organs again. It contains electrolytes (salts like sodium, potassium, and chloride) in amounts close to normal blood, plus lactate, which the liver turns into bicarbonate to help balance acid in the blood.

1. IV fluid enters the bloodstream through a vein.
2. The fluid spreads throughout the blood vessels, raising the total volume of blood.
3. Higher blood volume increases blood pressure and pushes blood forward to organs.
4. Kidneys receive more blood flow and begin producing urine again (target: at least 0.5 mL per kilogram of body weight per hour).
5. Organs like the heart, brain, and pancreas get the oxygen and nutrients they need.

SAFETY WATCH

Monitor urine output every hour—aim for 0.5 mL/kg/h. Too little means the kidneys are not getting enough blood; too much may mean fluid overload. · Watch for fluid overload: swelling in the legs (edema), trouble breathing, or crackles when listening to the lungs (sign of fluid in the lungs). · Check electrolytes (blood salts) daily—too much or too little sodium, potassium, or calcium can cause muscle weakness, heart rhythm problems, or confusion. · In the first 12–24 hours, give 5–10 mL/kg/h, then adjust down to 3 mL/kg/h or less once the patient is stable. · Use caution in patients with heart failure or kidney failure—they cannot handle large fluid volumes and may need slower, smaller amounts.

Morphine, hydromorphone, or fentanyl (opioid pain relievers) · Opioid analgesic

Acute pancreatitis causes severe, constant abdominal pain because the inflamed pancreas swells and stretches its outer covering (the capsule), and inflammatory chemicals irritate nearby nerves. Opioids are strong pain medicines that attach to special docking sites called mu-opioid receptors on nerve cells in the brain and spinal cord. When an opioid locks onto a receptor, it blocks the nerve from sending pain signals up to the brain, so the person feels much less pain—like unplugging a fire alarm so the loud noise stops.

1. Opioid enters the bloodstream (IV, under the skin, or by mouth).
2. It travels to the brain and spinal cord.
3. The opioid binds to mu-opioid receptors on nerve cells.
4. Binding the receptor shuts down the nerve's ability to send pain signals forward.
5. The brain receives fewer pain messages, and the patient feels relief.

SAFETY WATCH

Respiratory depression (slowed breathing)—opioids can slow the brain's breathing center, especially at high doses. Watch breathing rate; if it drops below 8–10 breaths per minute, stop the opioid and give naloxone (a reversal drug). · Constipation—opioids slow gut movement. Give a stool softener and laxative from day one. · Nausea and vomiting—common in the first few days. Anti-nausea medicine (ondansetron) often helps. · Sedation (drowsiness)—normal at first but should improve. If the patient cannot wake up, stop the opioid. · Addiction risk—rare in short-term hospital use for acute pain, but taper the dose as pain improves and switch to non-opioid pain relievers as soon as possible. · Avoid in patients with very low blood pressure or severe breathing problems (like advanced COPD) unless closely monitored.

Early enteral nutrition (feeding by mouth or tube into the stomach or small intestine) · Nutritional therapy (not a drug, but a critical treatment)

The gut (intestines) has a lining that acts like a fence, keeping bacteria and toxins inside the intestine and out of the bloodstream. In acute pancreatitis, if the gut is not fed, the lining weakens and bacteria can cross into the blood (bacterial translocation), leading to infection and organ failure. Feeding the gut—even small amounts—keeps the lining strong and the good bacteria healthy. It also lowers inflammation throughout the body. Old teaching said 'rest the gut' (NPO—nothing by mouth), but studies show that actually INCREASES infection and death. Start oral or tube feeding as soon as the patient can tolerate it, usually within 24–48 hours.

1. Food (or liquid nutrition) enters the stomach or small intestine by mouth or feeding tube.
2. Nutrients are absorbed into the gut lining cells, giving them energy to stay healthy.

3. The gut barrier remains strong, trapping bacteria inside the intestine.
4. Fewer bacteria cross into the blood, so infection risk drops.
5. The body gets calories and protein to fuel healing and prevent muscle breakdown.

SAFETY WATCH

Start small—try clear liquids or a low-fat oral diet first. If the patient vomits or has severe pain, place a feeding tube past the stomach (nasojejunal tube) and start at a slow rate (10–20 mL/hour). · Watch for feeding intolerance: vomiting, bloating, or high residual volumes (fluid left in the stomach). If this happens, slow the rate or move the tube farther down into the small intestine. · Avoid high-fat formulas early—fat can stimulate the pancreas and worsen pain. Use a low-fat or elemental (pre-digested) formula if needed. · If the patient cannot tolerate any enteral feeding after 5–7 days, consider parenteral nutrition (TPN—IV nutrition), but this has higher infection risk. · Track weight, albumin, and prealbumin to make sure the patient is getting enough nutrition.

ERCP with sphincterotomy and stone extraction (endoscopic procedure, not a drug) · Therapeutic procedure

ERCP stands for endoscopic retrograde cholangiopancreatography—a scope procedure where a flexible camera tube goes down the throat, through the stomach, and into the first part of the small intestine (duodenum). The doctor threads a thin tool through the scope into the bile duct and pancreatic duct. If a gallstone is stuck in the bile duct and blocking the flow of bile and pancreatic juice, the doctor cuts the muscle valve at the duct opening (sphincterotomy) and pulls the stone out with a basket or balloon. This relieves the blockage, lets bile and pancreatic juice drain, and stops the backup that caused the pancreatitis. It also prevents bacteria in the bile duct (cholangitis—a life-threatening infection) from spreading.

1. The patient is sedated (asleep or very drowsy).
2. The endoscope (flexible camera tube) is passed through the mouth, down the esophagus, through the stomach, and into the duodenum.
3. The doctor finds the ampulla of Vater (the drainage opening) and inserts a small catheter (thin tube) into the bile duct.
4. Contrast dye is injected and X-rays are taken to see the stone.
5. The doctor cuts the muscle at the ampulla (sphincterotomy) to widen the opening.
6. A wire basket or balloon is used to grab and remove the stone.
7. Bile and pancreatic juice can now drain freely, and the blockage is gone.

SAFETY WATCH

ERCP is **ONLY** indicated (needed) if there is cholangitis (fever, yellowing of the skin and eyes (jaundice), right upper belly pain—Charcot triad) OR persistent bile duct obstruction (bilirubin and liver enzymes staying high, dilated bile duct on imaging). Do **NOT** do ERCP in mild gallstone pancreatitis without these signs—it does not help and raises risk. ·

Timing: Perform ERCP within 24 hours if cholangitis is present. If no infection but obstruction persists, ERCP within 48–72 hours. · Complications: post-ERCP pancreatitis (5–10% risk—inflammation worsens after the procedure), bleeding from the sphincterotomy cut (1–2%), perforation (tear in the intestine, <1%), infection. · Place a temporary plastic stent (small tube) in the bile duct if the stone cannot be removed or if swelling blocks drainage. · After ERCP, schedule cholecystectomy (gallbladder removal surgery) during the same hospital stay or within 2–4 weeks to prevent another attack.

Cholecystectomy (surgical removal of the gallbladder) · Surgical procedure (not a drug, but definitive treatment)

In gallstone pancreatitis, a stone from the gallbladder travels down the bile duct and blocks the pancreatic duct opening, causing pancreatitis. Even after the stone passes or is removed, the gallbladder still contains more stones that can cause another attack. Cholecystectomy (removing the entire gallbladder) eliminates the source of stones and prevents recurrence. For mild pancreatitis, surgery is done during the same hospital admission once the inflammation settles (usually 48–72 hours after admission, when pain improves and labs trend down). For severe pancreatitis with tissue death (necrosis) or organ failure, delay surgery until the patient is stable and inflammation resolves (often 6 weeks or more).

1. The patient is given general anesthesia (put to sleep).
2. The surgeon makes small incisions (cuts) in the abdomen and inserts a camera and instruments (laparoscopic surgery).
3. The gallbladder is carefully separated from the liver and bile duct.
4. The cystic duct (the tube connecting the gallbladder to the bile duct) and cystic artery are clipped and cut.
5. The gallbladder is removed through one of the small incisions.
6. Bile now flows directly from the liver into the bile duct and intestine—no storage in the gallbladder, but digestion continues normally.

SAFETY WATCH

Timing: For MILD gallstone pancreatitis, perform cholecystectomy during the same hospital stay, once pain and labs improve (usually 48–72 hours after admission). Do NOT delay—if the patient goes home without surgery, the risk of recurrent pancreatitis is 25–30% within weeks. · For SEVERE pancreatitis (necrosis, organ failure, fluid collections), delay surgery until inflammation fully resolves (often 6+ weeks). Operating too early in severe disease increases complications and death. · Surgical risks: bleeding, infection, bile leak (bile leaking from the cut duct), injury to the bile duct (rare but serious—may need another surgery to repair), injury to nearby organs (intestine, liver). · Post-op complications: some patients develop post-cholecystectomy syndrome—ongoing abdominal pain, bloating, or diarrhea. Usually mild and improves with time. · If the patient is too sick for surgery (very old, multiple organ failure, severe heart or lung disease), consider long-term ERCP with stent placement instead, though recurrence risk is higher.

Antibiotics (e.g., carbapenems like meropenem or imipenem, or fluoroquinolone + metronidazole) · Antibacterial agents

Antibiotics kill or stop bacteria from growing. In acute pancreatitis, antibiotics are ONLY used when bacteria invade dead pancreas tissue (infected tissue death (necrosis)) or when there is infection outside the pancreas (like pneumonia or a urinary tract infection). Infected tissue death happens when bacteria from the gut cross into the bloodstream and seed the dead tissue. Carbapenems and fluoroquinolones penetrate (reach) the pancreas well and cover the most common bacteria (*E. coli*, *Klebsiella*, *Enterococcus*, *Bacteroides*). Metronidazole is added to cover anaerobic bacteria (bacteria that live without oxygen) from the gut. Antibiotics do NOT help sterile (no bacteria) pancreatitis and do NOT prevent infection—early studies thought preventive (prophylactic) (preventive) antibiotics might help, but large trials showed no benefit and found antibiotics can select for resistant bacteria and fungal infections.

1. Antibiotic is given by IV and enters the bloodstream.
2. The drug travels to the site of infection (necrotic pancreas, lung, etc.).
3. Carbapenems bind to bacterial cell wall-building enzymes (penicillin-binding proteins) and break the wall, causing the bacteria to burst and die.
4. Fluoroquinolones block bacterial DNA gyrase (an enzyme bacteria need to copy their DNA), so bacteria cannot multiply.
5. Metronidazole enters anaerobic bacteria and disrupts their DNA, killing them.
6. Bacterial load drops, and the immune system can clear the remaining infection.

SAFETY WATCH

Confirm infection before starting antibiotics: order blood cultures, check white blood cell count and inflammatory markers (CRP, procalcitonin), and look for gas in dead (necrotic) tissue on CT scan (a sign of bacteria making gas). · If infected tissue death (necrosis) is confirmed, start IV carbapenem (meropenem 1 g every 8 hours or imipenem 500 mg every 6 hours) OR a fluoroquinolone (ciprofloxacin or levofloxacin) PLUS metronidazole. · Duration: treat for 7–14 days, depending on clinical response (fever resolving, white count dropping). If the patient does not improve, they may need drainage or surgery to remove the infected dead tissue. · Watch for side effects: carbapenems can cause diarrhea, rash, seizures (rare, usually only at very high doses or in kidney failure); fluoroquinolones can cause tendon rupture (especially the Achilles tendon), QT prolongation (heart rhythm problem), and confusion; metronidazole can cause a metallic taste, nausea, and peripheral neuropathy (nerve damage causing numbness and tingling) with long-term use. · Adjust doses in kidney or liver failure. · If the patient grows resistant bacteria or fungus, switch to targeted therapy based on culture results.

Insulin (for hypertriglyceridemia-induced pancreatitis) · Hormone (glucose-lowering and triglyceride-lowering agent)

When blood triglycerides (a type of fat) rise above 1000 mg/dL, they can trigger acute pancreatitis by clogging tiny blood vessels in the pancreas and releasing toxic free fatty acids that damage pancreatic cells. Insulin drives triglycerides down fast by activating an enzyme called lipoprotein lipase (LPL) on the walls of blood vessels. LPL breaks down triglycerides in the blood into smaller pieces (free fatty acids and glycerol) that cells can use for energy. Insulin also pushes glucose and fat into cells for storage, which lowers the amount of fat circulating in the blood. IV insulin infusion (continuous drip) plus IV dextrose (sugar water, to prevent low blood sugar) can drop triglycerides by hundreds of points within hours, stopping ongoing pancreas damage.

1. IV insulin is started as a continuous infusion (0.1–0.3 units/kg/hour).
2. Insulin binds to receptors on blood vessel endothelial cells (the inner lining of vessels).
3. This activates lipoprotein lipase, which chops triglycerides into free fatty acids and glycerol.
4. Triglyceride level in the blood drops rapidly (goal: under 500 mg/dL).
5. Insulin also signals the liver to stop making new triglycerides and signals fat cells to store fat instead of releasing it.
6. IV dextrose (D5W or D10W) is given at the same time to keep blood glucose in a safe range (prevents low blood sugar (hypoglycemia)).

SAFETY WATCH

Check blood glucose every 1–2 hours during insulin infusion—risk of low blood sugar (hypoglycemia) (low blood sugar, <70 mg/dL) is high. Give IV dextrose continuously to prevent this. If glucose drops, increase dextrose rate or decrease insulin rate. · Check triglyceride level every 4–6 hours. Once it falls below 500 mg/dL, taper insulin and transition to subcutaneous (under the skin) insulin if the patient still has diabetes. · Watch for low potassium (hypokalemia) (low potassium)—insulin pushes potassium into cells, lowering blood potassium. This can cause dangerous heart rhythms. Check potassium every 4–6 hours and replace if low. · After the acute episode, find and treat the underlying cause: uncontrolled diabetes (start or adjust diabetes medications), high-fat diet (strict diet counseling), alcohol (stop completely), hypothyroidism (start thyroid hormone), or genetic lipid disorder (start fenofibrate or omega-3 fatty acids). · Do NOT use insulin infusion if triglycerides are normal—it will only cause low blood sugar and does not help.

Plasmapheresis (therapeutic plasma exchange) · Extracorporeal blood purification procedure (not a drug)

Plasmapheresis is a procedure where blood is removed from the body, passed through a machine that separates out the liquid part (plasma, which contains the triglycerides), and then the cleaned blood (without the fatty plasma) is returned to the body along with replacement fluid (donor plasma or albumin). This physically removes triglycerides from the blood in 2–4 hours—much faster than insulin alone. It is reserved for SEVERE hypertriglyceridemia (triglycerides >2000–3000 mg/dL) when the patient is critically ill, not responding to insulin, or has organ failure. Plasmapheresis buys time for other treatments (insulin, diet, medications) to take over.

1. A large IV catheter (central line) is placed in a vein in the neck or groin.
2. Blood flows out of the body into the plasmapheresis machine.
3. The machine spins the blood in a centrifuge (like a fast spinning washing machine) to separate red cells from plasma.
4. The fatty, triglyceride-loaded plasma is discarded.
5. The red cells are mixed with replacement fluid (donor plasma or albumin solution) and returned to the patient.
6. Triglyceride level drops by 50–70% in one session.

SAFETY WATCH

Reserved for triglycerides >2000 mg/dL with severe pancreatitis or organ failure, when insulin infusion is not enough. · Requires ICU-level care and a trained apheresis team. · Complications: bleeding (the catheter can injure blood vessels), infection (the central line can introduce bacteria), low calcium (the replacement fluid contains citrate, which binds calcium and can cause muscle cramps, tingling, or heart rhythm problems—give IV calcium during the procedure), allergic reaction to donor plasma, blood clots. · After plasmapheresis, continue IV insulin infusion and start long-term triglyceride-lowering therapy immediately. · Check triglycerides 4–6 hours after the procedure and daily afterward. If they rise again, repeat plasmapheresis or intensify medical therapy.

Red flags

- Organ failure (lungs, heart, or kidneys not working) that lasts longer than 48 hours—this defines severe acute pancreatitis and raises the risk of dying to 20–30 percent
- Blood pressure stays low even after giving 2 to 3 liters of IV fluid—means the blood vessels are wide open (distributive shock) and the patient likely needs ICU care and drugs to squeeze vessels (vasopressors)
- Little or no urine, or creatinine (kidney waste number) going up despite lots of IV fluid—signals acute kidney injury and the patient may need dialysis to clean the blood
- Oxygen saturation below 90 percent on room air, or needing extra oxygen—early warning of ARDS; the lungs can fail fast and require a breathing tube within hours
- Confusion, a belly so swollen it's rock-hard, or a rigid abdomen—can mean abdominal compartment syndrome (pressure inside the belly is crushing organs), dead bowel, or infected dead pancreas tissue; needs emergency CT scan and possibly surgery

Nursing priorities

1. Start aggressive intravenous fluid resuscitation with lactated Ringer solution at 250 to 500 mL per hour (or 5 to 10 mL/kg/h) immediately, using a large-bore IV catheter—18-gauge or larger. — The inflamed pancreas leaks fluid into the belly and surrounding tissue, pulling fluid out of the blood vessels and causing shock—early aggressive fluids in the first 12 to 24 hours cut the risk of organ failure and death by restoring blood flow to the pancreas and kidneys.

2. Measure urine output every hour using a Foley catheter (bladder tube), and titrate IV fluids to keep output above 0.5 mL per kilogram per hour (about 30 to 40 mL/h in most adults). — Urine output is the best bedside sign that the kidneys are getting enough blood flow—if output stays low despite fluids, it signals worsening shock or kidney failure and the patient needs immediate escalation to ICU care.
3. Monitor vital signs every 1 to 2 hours, targeting heart rate below 120 beats per minute and mean arterial pressure (MAP) between 65 and 85 mmHg—notify the provider immediately if blood pressure stays low after 2 to 3 liters of IV fluid. — Persistent low blood pressure after aggressive fluids means distributive shock (blood vessels are too wide open) from systemic inflammation—this requires vasopressors (medicines that squeeze vessels) and ICU-level monitoring to prevent organ death.
4. Keep the patient NPO (nothing by mouth)—no food, no liquids—and place a nasogastric tube (NG tube, a tube through the nose into the stomach) if the patient is vomiting repeatedly or the belly is very swollen. — Eating or drinking stimulates the pancreas to release more digestive enzymes, which worsens inflammation and pain—resting the pancreas by keeping the gut empty allows healing, and an NG tube removes stomach contents to prevent vomiting and aspiration (breathing stomach contents into the lungs).
5. Give intravenous pain medicine—usually an opioid like fentanyl (a strong synthetic pain medicine) or hydromorphone—on a scheduled basis, not just as needed, and reassess pain every 1 to 2 hours. — Acute pancreatitis causes severe, unrelenting pain that triggers a stress response—high heart rate, high blood pressure, and shallow breathing—which worsens oxygen delivery and increases the work the heart and lungs must do; controlling pain reduces this stress and helps the patient breathe deeply to prevent lung collapse (atelectasis).
6. Monitor oxygen saturation continuously with pulse oximetry, keeping it above 92 percent, and listen to lung sounds every 4 hours—notify the provider immediately if the patient becomes short of breath, oxygen drops, or you hear crackles (wet lung sounds). — Acute pancreatitis triggers systemic inflammation that can damage the lungs, causing fluid to leak into the air sacs (acute respiratory distress syndrome or ARDS)—early detection and oxygen support prevent respiratory failure, which is the most common organ failure in severe pancreatitis.
7. Check blood glucose (blood sugar) every 4 to 6 hours, and give insulin as ordered if glucose rises above 180 mg/dL. — The inflamed pancreas stops making insulin properly, and the stress response raises blood sugar even higher—high glucose worsens inflammation, impairs immune function, and increases infection risk, so keeping glucose controlled supports healing.

8. Watch for signs of infection—fever above 100.4°F (38°C), rising white blood cell count, confusion, or worsening abdominal pain after initial improvement—and notify the provider immediately if any appear. — Infected pancreatic tissue (infected tissue death (necrosis)) or infected bile (cholangitis) dramatically raise the risk of death and require immediate antibiotics, possible drainage procedures, or emergency ERCP—early recognition saves lives.

Sources

- American College of Gastroenterology (ACG) Clinical Guideline: Management of Acute Pancreatitis 2013
- American Gastroenterological Association (AGA) Institute Guideline on Initial Management of Acute Pancreatitis 2018
- International Association of Pancreatology / American Pancreatic Association (IAP/APA) Guidelines 2013
- Cochrane systematic reviews on fluid resuscitation and feeding in acute pancreatitis

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