

Acute Pancreatitis

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.

THE ONE-LINE TRUTH

Digestive enzymes wake up too early—inside the pancreas instead of waiting until they reach the small intestine—so the gland starts digesting itself.

The chain — how it unfolds

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. — why: 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. — why: 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. — why: 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. — why: 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. — why: 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). — why: 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. — why: 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. — why: 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.

Spot it — what you see, 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.

Red flags — act now

- 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

Treat it — in this order

1. 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).
2. 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.
3. 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.

4. 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/nasojunal 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.
5. 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.

Your top priorities at the bedside

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. — why: 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). — why: 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. — why: 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.