

Acute Abdomen

Acute abdomen means a belly problem where the smooth lining inside your belly is inflamed. Time is critical.

THE ONE-LINE TRUTH

Acute abdomen is sudden, severe belly pain with signs that the belly lining is angry and inflamed — a surgical emergency until proven otherwise.

The chain — how it unfolds

1. Belly lining inflammation triggers body-wide alarm system. — why: The parietal peritoneum is inflamed by a hole, blocked blood flow, bleeding, twisted blockage cutting off blood, or infection. Inflammatory chemicals (cytokines like TNF-alpha and interleukins) flood into the bloodstream and make blood vessel walls leaky, so fluid shifts out of blood vessels into the belly and tissue spaces where it cannot help deliver oxygen.
2. Heart rate rises early. — why: Blood volume drops from fluid leaking into the belly, vomiting (which loses stomach acid and water), and third-spacing (fluid trapped in tissue and the belly cavity where it cannot circulate). The heart must beat faster to push the remaining blood around the body and keep oxygen delivery to vital organs alive. A fast heart rate (Tachycardia) (heart rate above 100 beats per minute) is often the first vital sign change.
3. Blood pressure falls if fluid loss continues. — why: Fluid loss becomes so severe the heart cannot compensate even at top speed. The body shunts blood away from the gut, kidneys, and skin to save the brain and heart (centralization), but eventually pressure falls and shock begins. Low blood pressure (Hypotension) (systolic blood pressure below 90 millimeters of mercury) is a late and ominous sign.
4. Belly muscles freeze and become hard as wood on exam. — why: Pain nerves in the parietal peritoneum sense danger and trigger a reflex arc through the spinal cord that locks the abdominal wall muscles (involuntary guarding). The belly becomes rigid — hard as a wooden board — because the brain is trying to splint the area and stop any movement that would stretch the inflamed lining and spread the damage. This is different from voluntary guarding, where the patient tenses when you press but relaxes between exams.

5. Bowel stops moving. — why: Inflammatory chemical signals and pain signals shut down the myenteric plexus (the nerve network in the bowel wall that controls squeeze waves). The gut is paralyzed by inflammation, not by a mechanical blockage. Bowel sounds become absent or very quiet. This paralysis is called a stalled gut (ileus). Gas and fluid build up, distending (swelling) the intestines and making the belly look swollen.
6. Patient lies perfectly still. — why: Any stretching or jarring of the inflamed peritoneum is agony. Even breathing deeply or coughing hurts, so the patient takes shallow breaths and lies motionless. This is a protective reflex. Contrast this with the thrashing, restless patient with a kidney stone — renal colic causes waves of severe pain but no peritoneal involvement, so movement does not make it worse.
7. Rebound tenderness appears on exam. — why: When you press on the belly, you compress the inflamed peritoneum against the abdominal wall. When you let go suddenly, the peritoneum snaps back and stretches, triggering a sharp stab of pain. This is rebound tenderness, and it is a hallmark of peritoneal inflammation. The patient may wince, cry out, or guard even more.
8. Trace it back to find the one upstream break. — why: Each sign — fast heart rate, low blood pressure, paralyzed bowel, rigid belly, stillness, and rebound tenderness — is the body's attempt to survive the crisis by preserving blood flow, limiting oxygen debt, and stopping the spread of inflammation. Reading the signs backward tells you the single upstream cause: a hole, blocked blood flow, bleeding, twisted blockage, or infection of the belly lining. That cause determines whether the patient needs emergency surgery (perforation, strangulation, AAA), urgent imaging and possible intervention (ischemia), or intensive medical management and possible drainage (some infections). The window to reverse the damage is measured in hours, not days.

Spot it — what you see, and why

- Severe, sharp, localized abdominal pain: Inflammation of the parietal peritoneum (belly wall lining) is sensed by somatic nerves (pain nerves) that give precise, intense pain at the exact site of injury, unlike the dull, vague pain from visceral organs (organs themselves) alone.
- Rebound tenderness (pain worse when examiner releases pressure): Pressing slowly compresses the inflamed peritoneum, but sudden release causes it to snap back and stretch, triggering sharp pain—a classic sign of peritoneal irritation (belly lining is inflamed).
- Involuntary guarding and rigidity (board-like abdomen): Somatic nerves (pain nerves in the belly wall) sense peritoneal inflammation and trigger a reflex that locks the abdominal wall muscles tight to prevent movement and limit spread of irritation and pain.

- Absent or diminished bowel sounds: Inflammatory mediators (chemical signals like cytokines) and pain signals shut down the normal the gut's squeezing waves (peristalsis) (squeeze wave) of the intestines, causing a stalled gut (ileus) (paralyzed bowel) so the gut stops making noise.
- A fast heart rate (Tachycardia) (heart rate greater than 100 beats per minute): Fluid loss from vomiting, third-spacing (fluid leaking into belly where it cannot help), and bleeding drops blood volume, so the heart beats faster to maintain oxygen delivery to vital organs.
- Low blood pressure (Hypotension) (systolic blood pressure less than 90 mmHg) in severe cases: Ongoing fluid loss and inflammatory shock (body-wide response to inflammation) overwhelm the heart's ability to compensate, blood pressure falls, and the body shunts blood away from the gut to preserve the brain and heart.

Red flags — act now

- Low blood pressure (Hypotension) (systolic BP less than 90 mmHg) or signs of shock (cold, clammy skin, confusion, weak pulse)
- Rigid, board-like abdomen with severe rebound tenderness
- Pain out of proportion to physical exam findings (suggests mesenteric starved blood flow (ischemia)—blocked blood vessels feeding the bowel)
- Rising lactate (greater than 2 to 4 mmol/L) or worsening metabolic acidosis (acid building up in the blood) (acid buildup in blood from dying tissue)
- Free air on imaging (gas outside the gut seen on X-ray or CT, suggests perforation requiring emergent surgery)

Treat it — in this order

1. IV fluids (normal saline or lactated Ringer's—1 to 2 liters rapid bolus, then continuous infusion at 125 to 200 mL per hour) → Replaces lost fluid from vomiting, third-spacing (fluid leaking into the belly and bowel wall like water soaking into a towel), and inflammation. Restores blood pressure and tissue blood flow (perfusion) (blood flow feeding organs with oxygen) so the heart doesn't have to race and organs stay alive.
2. Broad-spectrum IV antibiotics (e.g., piperacillin-tazobactam 3.375 g every 6 hours, or ceftriaxone 1 to 2 g every 24 hours plus metronidazole 500 mg every 8 hours) → Kills aerobic (oxygen-loving) and anaerobic (oxygen-hating) bacteria spilling from a perforated bowel or crossing through blood-starved (ischemic) bowel wall before they cause septic shock (life-threatening infection in the bloodstream, like poison spreading everywhere). Antibiotics cut death rates in abdominal infection per IDSA and Surgical Infection Society guidelines.

3. Emergency surgery or interventional radiology drainage (based on imaging and how stable the patient is) → Removes or repairs the broken part: drains a pus pocket, cuts out dead bowel, patches or removes a perforated segment, relieves a blockage, or stops active bleeding. Source control is the only way to eliminate the cause of peritoneal inflammation and stop the downward spiral.
4. Pain medicine (IV opioids, e.g., morphine 2 to 4 mg IV or fentanyl 25 to 50 mcg IV, adjusted to pain level) → Relieves pain so the patient can breathe deeply, cooperate with physical exam, and tolerate movement. Multiple studies and ACEP guidelines confirm that pain control does NOT hide peritoneal signs if you examine carefully—it actually makes the exam easier and more accurate.
5. NPO (nothing by mouth) and nasogastric tube if vomiting or a stalled gut (ileus) is present → Stops the stomach from pushing more fluid and air into a blocked or perforated bowel, reduces vomiting and risk of aspiration (breathing in stomach contents, like inhaling water while swimming), and decompresses swollen bowel loops by suctioning out fluid and gas.

Your top priorities at the bedside

1. Establish large-bore IV access (18-gauge or larger in both arms if possible) and start rapid fluid resuscitation with normal saline or lactated Ringer's—give 1 to 2 liters as fast as it will run, then reassess blood pressure and heart rate every 15 minutes. — why: Acute abdomen causes third-spacing (fluid leaks from blood vessels into the belly and tissues) and possible bleeding; hypovolemia (low blood volume) drops oxygen delivery to organs and worsens shock; large IVs let you give blood products quickly if needed.
2. Keep the patient NPO (nothing by mouth—no food, no water, no ice chips) and place a nasogastric tube (NG tube—a tube through the nose into the stomach) to suction if bowel sounds are absent or the patient is vomiting repeatedly. — why: An obstructed or perforated bowel cannot move contents forward; continuing to eat or drink increases vomiting, aspiration risk (breathing stomach contents into the lungs), and pressure that can worsen perforation; NG suction decompresses (relieves pressure in) the stomach and reduces these dangers.
3. Administer broad-spectrum IV antibiotics within 1 hour of recognition—common regimens include piperacillin-tazobactam 3.375 g every 6 hours or ceftriaxone 1 to 2 g every 24 hours plus metronidazole 500 mg every 8 hours. — why: Perforated or blood-starved (ischemic) (oxygen-starved) bowel spills bacteria into the sterile peritoneal cavity (belly space), causing peritonitis and sepsis (life-threatening infection response); early antibiotics lower mortality by controlling bacterial growth before and after surgery.