

PUD and GERD

PUD and GERD — acid ends up where it doesn't belong, and unprotected tissue pays the price.

THE ONE-LINE TRUTH

Acid belongs in the stomach, where a thick mucus shield protects the lining from being burned.

The chain — how it unfolds

1. The lower esophageal sphincter (LES) weakens or relaxes inappropriately, or increased belly pressure forces stomach contents upward. — why: Causes include hiatal hernia (stomach slides through the diaphragm, losing the normal angle), obesity or pregnancy (both raise intra-abdominal pressure), smooth-muscle dysfunction, or drugs/foods that relax the LES (calcium-channel blockers, nitrates, chocolate, caffeine). The result: acid and pepsin wash back into the esophagus, which has no mucus protection.
2. Acid touching the esophageal lining causes heartburn (burning pain behind the breastbone) and regurgitation (sour or bitter fluid backing up into the throat or mouth). — why: The esophagus is lined with squamous epithelium (thin, flat cells) designed only for swallowing, not for resisting acid. Acid activates pain nerves in the esophageal wall, creating the classic burning sensation. Regurgitation happens when a large volume of stomach contents overcomes the weak LES, especially when lying flat or bending over.
3. Repeated acid exposure inflames the esophageal lining, causing esophagitis (redness, swelling, erosions visible on endoscopy). — why: Chronic inflammation damages the squamous cells, causing them to die and slough off. Patients may feel pain with swallowing (odynophagia) or trouble swallowing (dysphagia) if swelling narrows the tube. Healing with scar tissue can lead to a stricture (a tight ring that blocks food passage).
4. Over months to years, chronic acid injury triggers the esophageal lining to change from normal squamous cells into columnar cells that look like stomach lining—this is Barrett's esophagus (intestinal metaplasia). — why: The body tries to protect the esophagus by replacing the fragile squamous lining with tougher, mucus-producing columnar cells (a survival adaptation). Unfortunately, these metaplastic cells have a small but real risk (about 0.5 % per year) of turning into esophageal adenocarcinoma, so Barrett's esophagus requires regular surveillance endoscopy.

5. In PUD, *H. pylori* bacteria or NSAIDs break down the stomach's mucus-bicarbonate shield, leaving the lining exposed to acid and pepsin. — why: *H. pylori* releases urease (which makes ammonia), proteases, and toxins (VacA, CagA) that dissolve mucus, kill epithelial cells, and trigger chronic inflammation. NSAIDs block cyclooxygenase (COX-1), stopping prostaglandin production; without prostaglandins, mucus and bicarbonate secretion drop, and blood flow to the lining falls, leaving it defenseless.
6. Acid and pepsin digest a crater—an ulcer—into the stomach (gastric ulcer) or duodenal wall (duodenal ulcer). Pain appears in the upper belly (epigastrium). — why: Gastric ulcers often hurt worse with eating (food stimulates acid secretion, which bathes the open sore). Duodenal ulcers classically feel better with food (food buffers acid briefly) but hurt 2–3 hours later when the stomach empties and acid floods the duodenum again. The pain comes from acid touching exposed nerve endings in the ulcer base.
7. If the ulcer erodes into a blood vessel, bleeding occurs—showing as black tarry stool (melena) (black, tarry, foul-smelling stool from digested blood) or vomiting blood (hematemesis) (vomiting blood or coffee-ground material). — why: Stomach acid turns blood dark. Small-vessel bleeding oozes slowly, giving time for digestion, so stool turns black. Large-vessel bleeding (e.g. into the gastroduodenal artery) floods the stomach, causing vomiting of bright-red or dark coffee-ground blood and can lead to shock (low blood pressure, fast heart rate, pale skin, confusion) from volume loss.
8. If the ulcer eats all the way through the stomach or duodenal wall, perforation occurs—stomach and duodenal contents spill into the peritoneal cavity, causing peritonitis. — why: Chemical peritonitis (from acid and bile) triggers sudden, severe, knife-like abdominal pain; the abdomen becomes board-rigid as the abdominal muscles guard. Air escaping into the peritoneum can be seen as free air under the diaphragm on an upright chest X-ray. Bacterial peritonitis and sepsis follow within hours if the perforation is not surgically repaired.

Spot it — what you see, and why

- Burning behind the breastbone (heartburn) that gets worse when you lie down or bend over: Stomach acid flows backward into the esophagus (the tube from your mouth to your stomach) because the muscle ring at the bottom of the esophagus is weak or relaxes too often. Lying flat lets gravity pull acid up more easily.
- Gnawing or burning pain in the upper belly, often between meals or at night: An open sore (ulcer) in the stomach lining or the first part of the small intestine is exposed to stomach acid. When your stomach is empty, the acid bathes the raw spot and causes pain.
- Pain that gets better when you eat food or take antacids: Food and antacids act like a sponge or a blanket, soaking up or coating over the acid so it stops irritating the ulcer for a little while.

- Nausea or feeling full quickly after eating a small amount: Inflammation or swelling around the ulcer can slow the stomach from emptying normally, so food sits longer and makes you feel stuffed or sick.
- Bloating or burping more than usual: Extra acid and gas build up in the stomach when acid production is high or when the stomach does not empty smoothly, pushing air back up.
- Trouble swallowing or food getting stuck: Repeated acid exposure can scar and narrow the esophagus, or a large ulcer can swell and block the passage. This is a warning sign that needs checking right away.

Red flags — act now

- Black, tarry stools or vomit that looks like coffee grounds (signs of bleeding)
- Trouble swallowing or food getting stuck
- Unintended weight loss (losing weight without trying)
- Severe, sudden belly pain that does not go away (may mean a hole has formed in the stomach or intestine)
- New symptoms starting after age 60

Treat it — in this order

1. Proton pump inhibitor (PPI) such as omeprazole 20–40 mg daily for 8 weeks → Shuts off the proton pumps (tiny acid-making machines) in the stomach lining cells. Acid production drops by 90%, giving the damaged tissue time to heal instead of being constantly bathed in acid.
2. H. pylori eradication therapy: triple therapy (PPI + clarithromycin + amoxicillin) or quadruple therapy (PPI + bismuth + tetracycline + metronidazole) for 10–14 days → Kills the H. pylori bacteria that are eating through the stomach lining. Removes the root cause in most PUD cases. Once the germ is gone, ulcers heal and usually do not come back.
3. Stop NSAIDs completely, or switch to acetaminophen; if NSAIDs are unavoidable (e.g., for heart protection), take the lowest dose with a PPI → Removes the poison that is stripping the stomach lining's protective mucus layer. Prevents new ulcers from forming and allows existing ones to heal.
4. Lifestyle changes for GERD: lose weight if overweight, elevate the head of the bed 6–8 inches, avoid meals within 3 hours of bedtime, avoid trigger foods (chocolate, mint, caffeine, alcohol, fatty foods) → Reduces pressure inside the belly that pushes acid upward. Gravity and timing keep acid in the stomach where it belongs. Weight loss reduces abdominal pressure on the lower esophageal sphincter.

5. Urgent upper endoscopy if alarm signs are present: age > 60 with new symptoms, unintended weight loss, trouble swallowing, vomiting blood, black stool, or anemia → Finds cancer, deep bleeding ulcers, or Barrett esophagus (pre-cancerous tissue in GERD) early. Allows immediate treatment like cautery to stop bleeding or biopsy to diagnose cancer.

Your top priorities at the bedside

1. Check hemoglobin and hematocrit levels and look at stool color every shift if the patient reports black stools or vomiting blood — why: A dropping hemoglobin below 7 grams per deciliter means active bleeding that steals oxygen delivery to organs; black stool contains digested blood and signals upper GI bleeding that can become life-threatening within hours
2. Hold the next dose of aspirin, ibuprofen, naproxen, or any NSAID and notify the provider — why: NSAIDs block prostaglandins (protective chemicals) that make mucus in the stomach lining; continuing them turns a small erosion into a bleeding ulcer that perforates through the stomach wall
3. Give the prescribed PPI 30 to 60 minutes before breakfast every day, not at bedtime — why: PPIs like omeprazole only work on active proton pumps (acid-making cells); these pumps turn on when you eat, so morning dosing before food blocks acid production for 24 hours and heals ulcers in 8 weeks