

PUD and GERD

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THE STORY IN ONE SENTENCE

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

Normal anatomy — the body's job

The stomach is a muscular bag that churns food and bathes it in acid made by parietal cells in the stomach wall. That acid is strong enough to dissolve metal, so the stomach protects itself with a thick mucus coat and bicarbonate (a base that neutralizes acid). At the top, the lower esophageal sphincter (LES)—a ring of muscle—acts like a trapdoor, keeping stomach contents from splashing back up into the esophagus. At the bottom, the pyloric sphincter controls the release of partly digested food into the duodenum (the first part of the small intestine).

Prostaglandins (local chemical messengers) tell the stomach lining to make mucus and to pump blood to the lining so it stays healthy. When all these parts work together, acid stays in the stomach, digestion runs smoothly, and nothing gets burned.

BODY'S JOB

The stomach's job is to grind food into a soupy mix and use acid and enzymes to start breaking down proteins. The mucus-bicarbonate barrier keeps the stomach lining safe from its own acid. The LES keeps that acidic soup from washing back into the esophagus, which has no protective coating. The duodenum receives the acidic food and quickly neutralizes it with bicarbonate from the pancreas. Every weird finding in PUD or GERD makes sense only when you picture this normal setup first—then you can see exactly what broke and why the body is struggling.

1. You swallow food, and it travels down the esophagus (a simple muscular tube with a thin, delicate lining).

2. The lower esophageal sphincter (LES) relaxes to let food drop into the stomach, then squeezes shut like a drawstring to keep stomach acid from splashing back up.
3. Parietal cells in the stomach wall pump out hydrochloric acid (HCl) into the stomach chamber; other cells release pepsin (a protein-cutting enzyme) and mucus.
4. The mucus layer coats the stomach lining like a thick blanket; underneath, bicarbonate is secreted to neutralize any acid that sneaks through.
5. Prostaglandins (local chemical signals) keep blood flowing to the lining and tell the mucus factories to stay on.
6. Food mixes with acid and enzymes, turning into chyme (a thick, acidic paste).
7. The pyloric sphincter at the bottom of the stomach releases small squirts of chyme into the duodenum, where pancreatic bicarbonate neutralizes the acid so enzymes can work safely.
8. Everything flows one direction: mouth → esophagus → stomach → duodenum. Acid never touches the esophagus in the normal state.

What breaks

Two big breaks cause most trouble. In GERD, the lower esophageal sphincter (the muscle door at the top of the stomach) gets weak or lazy, or increased belly pressure (obesity, pregnancy, hiatal hernia) forces acid up into the esophagus, which has no mucus shield and gets burned. In PUD, either *H. pylori* bacteria bore through the stomach's mucus layer and damage the lining directly, or NSAIDs turn off prostaglandin production, which shuts down mucus and bicarbonate output and starves the lining of blood—leaving it naked against acid. Once the mucus shield is breached, acid eats a hole (an ulcer) into the stomach or duodenal wall. Every symptom downstream—heartburn, regurgitation, upper belly pain, black stool, bloody vomit—flows from one of these two upstream breaks.

CLINICAL TERM

GERD: lower esophageal sphincter (LES) incompetence or transient LES relaxations (TLESRs), often worsened by increased intra-abdominal pressure (obesity, pregnancy) or hiatal hernia. Acid refluxes into the esophagus, causing esophagitis, and chronic exposure can lead to Barrett's esophagus (intestinal metaplasia) and, rarely, adenocarcinoma. PUD: mucosal defense breakdown due to *H. pylori* infection (which releases urease, ammonia, and proteases that disrupt the mucus-bicarbonate layer and trigger inflammation) or NSAID use (which inhibits cyclooxygenase, blocking prostaglandin synthesis and thereby reducing mucus, bicarbonate, and mucosal blood flow). Loss of defense allows acid and pepsin to ulcerate the gastric or duodenal mucosa. Complications include bleeding (hemorrhage) (from erosion into submucosal vessels), perforation (through-and-through hole causing peritonitis), and gastric outlet obstruction (from pyloric-channel scarring).

How the body compensates

The body tries to protect itself by making extra mucus, slowing stomach emptying, and tightening blood vessels to limit bleeding. These moves can keep symptoms mild and vital signs stable for weeks or months, which is dangerous because it hides how deep the damage is going. The tipping point comes when an ulcer erodes into a large artery (causing sudden massive bleeding), punches through the stomach wall (spilling contents into the belly and causing peritonitis), or when chronic reflux scars the esophagus into a tight stricture that blocks food. Watch for red flags—persistent vomiting, black tarry stool, throwing up blood, new severe belly pain, weight loss, trouble swallowing—because once the compensations fail, the window to reverse the damage closes fast.

1. The stomach tries to make extra mucus and bicarbonate to patch weak spots in the lining, buying time against acid damage.
2. Blood vessels in the stomach wall constrict (narrow) to slow bleeding if an ulcer nicks a small vessel; clotting factors rush in to plug the hole.
3. The stomach slows its emptying (delayed gastric emptying) to reduce acid production triggers and give the lining a chance to heal.
4. The body may reduce appetite or cause nausea to limit food intake, decreasing acid secretion overall.
5. In GERD, the esophagus thickens its lining (acanthosis) and tries to increase saliva production (saliva contains bicarbonate) to wash and neutralize refluxed acid.
6. These compensations can keep pain tolerable and vital signs normal, masking the fact that the ulcer is getting deeper or reflux damage is progressing to Barrett's changes.
7. The crash point to watch for: an ulcer erodes into the gastroduodenal artery or another large vessel → sudden massive bleeding (hemorrhage), shock, black stool, vomiting blood (hematemesis) (throwing up blood). Or the ulcer perforates the stomach/duodenal wall → peritonitis with board-like rigid abdomen, fever, sepsis. Or chronic GERD causes a stricture → trouble swallowing (dysphagia) (trouble swallowing) and weight loss, or Barrett's esophagus → risk of esophageal adenocarcinoma.
8. Monitor hemoglobin and hematocrit (to catch slow bleeding early), vital signs (tachycardia and low blood pressure are late signs of volume loss), and symptom patterns (new severe pain, vomiting, trouble swallowing (dysphagia), weight loss all signal decompensation).
9. Treat the root cause—kill *H. pylori* with antibiotics (clarithromycin + amoxicillin + proton-pump inhibitor for 14 days per ACG 2017), stop NSAIDs, reduce acid with a PPI (omeprazole, pantoprazole), lose weight and elevate the head of the bed for GERD—before compensations fail and complications become irreversible.

Domino effect

1. The lower esophageal sphincter (LES) weakens or relaxes inappropriately, or increased belly pressure forces stomach contents upward. — 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). — 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). — 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). — 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. — *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). — 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). — 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. — 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.

Symptoms · what the patient feels, 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.
- Unintended weight loss: Pain or fear of pain may make you eat less, or a hidden bleed from an ulcer can drain your body's stores. Weight loss can also signal a more serious problem like cancer.

- Black, tarry stools or vomit that looks like coffee grounds: Blood from a bleeding ulcer mixes with stomach acid and turns dark. Black stool means blood has traveled through the intestines; coffee-ground vomit means partly digested blood. Both are emergencies.
- New heartburn or belly pain starting after age 60: Older age raises the chance that symptoms come from something more serious, like cancer, rather than simple reflux or an ulcer, so doctors check more carefully.

Focused exam

- Tenderness when pressing on the upper belly (epigastric area): In PUD and GERD, the stomach or lower food-tube lining is damaged and inflamed. When you press, it hurts because the raw lining is sensitive and the body is protecting the injured area.
- Black, sticky, tar-like stool (melena): An ulcer in PUD can bleed. The blood travels through the gut and gets digested, turning it black and sticky. This means the ulcer has eaten deep enough to hit a blood vessel — a dangerous sign.
- Visible blood in vomit (hematemesis) — looks like coffee grounds or bright red: Active bleeding from a PUD ulcer is spilling into the stomach right now. The stomach cannot hold it, so the body throws it up. This is a medical emergency.
- Heartburn that gets worse lying flat or after big meals: In GERD, the lower esophageal sphincter (the ring muscle at the bottom of the food tube that keeps acid down) is weak. When you lie flat, gravity stops helping, and acid splashes back up. The esophagus burns because it is not built to handle acid like the stomach is.
- Upper belly pain that changes with eating — worse when hungry or worse right after eating: In PUD, duodenal ulcers (in the first part of the small intestine) hurt more when the stomach is empty because acid hits the raw spot with no food to buffer it. Gastric ulcers (in the stomach itself) often hurt more right after eating because food stretches the damaged lining. The pattern tells you where the break is.
- Rigid, board-like belly with rebound tenderness (pain when you let go after pressing): If a PUD ulcer eats all the way through the stomach or intestine wall (perforation), stomach contents and bacteria spill into the belly cavity. The body responds with severe inflammation (peritonitis) and the belly muscles tighten like a board. This is a surgical emergency.

Diagnostics · every test answers a question

- H. pylori testing (urea breath test, stool antigen test, or biopsy during endoscopy) → Is the bacterium *Helicobacter pylori* present in the stomach?
- Complete blood count (CBC) with hemoglobin and hematocrit → Is there bleeding from the ulcer that has caused anemia?

- Upper endoscopy (esophagogastroduodenoscopy, EGD) → Where exactly is the ulcer or damage, how deep is it, and is there cancer or pre-cancer?
- Fecal occult blood test (FOBT) or fecal immunochemical test (FIT) → Is there hidden blood in the stool that you cannot see?
- Serum gastrin level → Is there too much gastrin hormone, which drives the stomach to make massive amounts of acid?
- NSAID medication history review → Is the patient taking drugs that strip away the stomach lining's protective mucus?

Treatment logic

- 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.
- *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.
- 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.
- 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.
- 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.
- Nissen fundoplication surgery if GERD symptoms persist despite 8 weeks of PPI and full lifestyle changes → Wraps the top of the stomach around the lower esophagus to tighten the weak lower esophageal sphincter. Physically fixes the anatomic break that lets acid splash back up.

- Intravenous PPI (e.g., pantoprazole 80 mg bolus, then 8 mg/hour infusion) and urgent endoscopy for active upper GI bleeding → Rapidly shuts down acid production and stabilizes clot formation over a bleeding vessel. Endoscopy finds and stops the bleeding (cautery, clips, or injection).

Medications

Omeprazole (Prilosec), lansoprazole (Prevacid), esomeprazole (Nexium), pantoprazole (Protonix) · Proton pump inhibitor (PPI)

A PPI shuts down the tiny pumps inside the stomach lining cells that spray acid into the stomach. When those pumps stop, acid levels drop sharply and the raw spots in the stomach or food tube (esophagus) can heal.

1. You swallow the pill; it travels through the stomach into the small intestine and gets absorbed into the blood.
2. The drug circulates to the parietal cells in the stomach lining.
3. Inside those cells, the PPI finds the proton pump and locks onto it permanently.
4. That pump can no longer push acid out, so acid production drops by 90–95 percent.
5. With much less acid splashing around, the injured stomach or esophagus lining stops getting burned and begins to repair itself.
6. New pumps are built slowly over 1–2 days, which is why one dose per day keeps acid low all day.

SAFETY WATCH

Take it 30–60 minutes before breakfast (or the first meal of the day) so the drug is in your blood when the stomach pumps wake up after eating. · Long-term use (many months to years) may slightly raise the risk of bone fractures, low magnesium, low vitamin B₁₂, and gut infections (including *C. difficile*). · Check magnesium and B₁₂ levels if someone uses a PPI for more than a year. · Once the ulcer or esophagitis heals (usually 4–8 weeks), try to step down to the lowest dose that controls symptoms or stop if possible; not everyone needs lifelong therapy. · PPIs are very safe for short courses and during pregnancy when needed (category B).

Ranitidine (Zantac—withdrawn 2020), famotidine (Pepcid), nizatidine (Axiid), cimetidine (Tagamet) · H₂-receptor antagonist (H₂ blocker)

An H₂ blocker sits on histamine docking sites (H₂ receptors) on the stomach's acid-making cells and blocks histamine from landing. Histamine is a messenger that tells those cells to pump acid. When histamine is blocked, acid output drops by about 70 percent.

1. You swallow the pill; it is absorbed in the small intestine and enters the bloodstream.
2. The drug reaches the parietal cells in the stomach wall.
3. It plugs the H₂ receptor, so histamine released by nearby cells cannot attach.
4. Without histamine's signal, the cell makes much less acid.
5. Acid levels fall within 30–60 minutes and stay low for 6–12 hours (depending on the drug).
6. Lower acid gives the stomach or esophagus lining time to heal.

SAFETY WATCH

Ranitidine was pulled from the market worldwide in 2020 because of a contaminant (NDMA); do not use it. · Famotidine is now the most common H₂ blocker and is very safe for short-term and long-term use. · Cimetidine can interfere with many other drugs (it slows liver enzymes) and may cause confusion in older adults; famotidine is safer. · H₂ blockers are often used on-demand for mild heartburn or at bedtime for nighttime acid breakthrough. · They are safe in pregnancy (category B).

Sucralfate (Carafate) · Mucosal protectant

Sucralfate is a sticky medicine that turns into a paste when it meets stomach acid. The paste glues itself over ulcers and raw spots, forming a protective bandage that shields the injury from acid, pepsin (a digestive enzyme), and bile while healing happens underneath.

1. You swallow the tablet or liquid on an empty stomach (so food does not block it).
2. Sucralfate reaches the stomach and meets acid.
3. Acid activates the drug, turning it into a thick, sticky gel.
4. The gel preferentially binds to proteins in the ulcer crater, coating the raw tissue.
5. This physical barrier blocks acid and pepsin from touching the wound.
6. The coating stays in place for about 6 hours, giving the tissue a break to repair.
7. Sucralfate also may release small amounts of aluminum that stimulate protective mucus and bicarbonate secretion.

SAFETY WATCH

Take it on an empty stomach—1 hour before meals and at bedtime—so it can coat the ulcer without interference. · Do not take other medicines within 2 hours, because sucralfate can bind them and block absorption (especially thyroid hormone, some antibiotics, and antacids). · Constipation is the most common side effect (the aluminum slows the bowel). · Avoid in people with kidney failure, because aluminum can build up. · It is very safe in pregnancy (category B) and is sometimes used when a PPI is not wanted.

Aluminum/magnesium hydroxide (Maalox, Mylanta), calcium carbonate (Tums, Rolaids) · Antacid

An antacid is a base (the opposite of an acid). When you swallow it, it drops into the stomach and chemically neutralizes the acid already sitting there, raising the pH quickly. Symptoms improve within minutes, but the effect is short because the stomach keeps making new acid.

1. You chew or swallow the tablet; it dissolves in the stomach.
2. The base (aluminum hydroxide, magnesium hydroxide, or calcium carbonate) meets hydrochloric acid.
3. A neutralization reaction occurs: acid + base → salt + water.
4. The pH rises from around 1–2 toward 3–4, which is much less harsh.
5. Pain and burning ease within 5–15 minutes.
6. The effect lasts 30–60 minutes, because the stomach soon makes more acid and the antacid is emptied into the small intestine.

SAFETY WATCH

Aluminum-based antacids cause constipation; magnesium-based antacids cause diarrhea. Combination products (Maalox, Mylanta) try to balance the two. · Calcium carbonate can cause rebound acid (the stomach senses higher pH and pumps out extra acid later) and constipation. · Antacids can interfere with absorption of many drugs (tetracycline, fluoroquinolones, iron, some heart medicines); space them 2 hours apart. · Safe for occasional use; not ideal for daily long-term use. · Safe in pregnancy for short-term symptom relief.

Amoxicillin + clarithromycin + PPI (or metronidazole instead of amoxicillin if penicillin allergy); or bismuth + metronidazole + tetracycline + PPI · H. pylori eradication therapy (antibiotic regimen)

H. pylori is a spiral bacterium that burrows into the stomach lining and makes the tissue vulnerable to acid damage, leading to ulcers. Killing the bacteria lets the ulcer heal and sharply lowers the chance it will come back. Because H. pylori hides under mucus and can

resist single antibiotics, we use a combination of two or three antibiotics plus a PPI (to raise pH and help the antibiotics work).

1. You take the full combination—usually 3 or 4 drugs—twice a day for 10–14 days.
2. The PPI lowers stomach acid, which makes the environment less hostile to the antibiotics and may slow bacterial growth.
3. Amoxicillin (or metronidazole) breaks down the bacterial cell wall.
4. Clarithromycin (or tetracycline) stops the bacteria from making proteins they need to survive.
5. Bismuth (if used) coats the stomach, has some direct antibacterial effect, and may protect the lining.
6. Together, the drugs kill the bacteria in more than 80–90 percent of people when taken exactly as prescribed.
7. Once *H. pylori* is gone, the ulcer heals and the risk of a new ulcer drops from roughly 60–80 percent per year to less than 5 percent.

SAFETY WATCH

Take every dose on time for the full 10–14 days; stopping early lets resistant bacteria survive. · Common side effects: metallic taste (clarithromycin, metronidazole), diarrhea, nausea, black tongue or stool (bismuth). · Avoid alcohol during metronidazole therapy (it can cause severe nausea and vomiting). · Tetracycline causes sun sensitivity—use sunscreen—and should not be used in pregnancy or children under 8 (stains teeth). · Confirm eradication 4 weeks after finishing therapy with a urea breath test or stool antigen test (not antibody blood test, which stays positive for years). · If the first regimen fails, a second-line regimen with different antibiotics (often levofloxacin-based or high-dose dual PPI-amoxicillin) is used.

Misoprostol (Cytotec) · Prostaglandin analog

Misoprostol is a synthetic copy of prostaglandin E₁, a natural chemical that tells the stomach lining to make thick protective mucus and bicarbonate (a base that neutralizes acid). NSAIDs block prostaglandin production, which is why they cause ulcers; misoprostol replaces the missing prostaglandin and restores the stomach's defenses.

1. You take misoprostol (usually 4 times a day with meals and at bedtime).
2. The drug binds to prostaglandin receptors on the surface of stomach lining cells.
3. Those cells respond by pumping out more mucus and bicarbonate.
4. The thicker mucus layer shields the tissue from acid and pepsin.
5. Bicarbonate neutralizes acid right at the lining surface.

6. Misoprostol also slightly lowers acid secretion by the parietal cells.
7. Together, these effects prevent NSAID-induced ulcers in people who must keep taking NSAIDs.

SAFETY WATCH

Diarrhea and abdominal cramping are very common (up to 30–40 percent) because prostaglandins speed gut movement; taking it with food helps. · Absolutely contraindicated in pregnancy—misoprostol causes uterine contractions and miscarriage (it is actually used intentionally for medical abortion). · Women of childbearing age must use reliable contraception. · Because of the side effects and dosing burden (4 times daily), PPIs are now preferred for NSAID ulcer prevention in most patients. · Misoprostol is still used when a PPI cannot be used or in very high-risk patients on NSAIDs.

Metoclopramide (Reglan) · Prokinetic agent (dopamine antagonist)

Metoclopramide blocks dopamine receptors in the gut and stimulates receptors that make the stomach and esophagus muscles squeeze harder and faster. This tightens the lower esophageal sphincter (reducing reflux), speeds stomach emptying (so there is less to reflux), and pushes food forward through the intestine.

1. You take metoclopramide by mouth or injection.
2. The drug enters the bloodstream and reaches the gut muscle and nerves.
3. It blocks dopamine receptors, which normally slow gut movement, so blocking them speeds things up.
4. It also stimulates 5-HT₄ receptors, which trigger muscle contractions.
5. The lower esophageal sphincter squeezes tighter, reducing the chance that stomach contents slosh back up.
6. The stomach contracts more forcefully and empties faster into the small intestine.
7. With less sitting in the stomach, there is less pressure and less material available to reflux.

SAFETY WATCH

Use only for short courses (less than 12 weeks) because long-term use can cause tardive dyskinesia—a serious movement disorder with involuntary tongue, face, or limb movements that may be permanent. · Can cause drowsiness, restlessness, anxiety, and depression (it blocks dopamine in the brain). · Contraindicated if there is a gut blockage, perforation, or bleeding. · Avoid in people with Parkinson disease (worsens symptoms) or a history of seizures. · Pregnancy category B, but use only if clearly needed. · Because of the risk profile, metoclopramide is now reserved for cases of GERD with delayed stomach emptying (gastroparesis) that do not respond to acid suppression alone; it is rarely first-line.

Red flags

- 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

Nursing priorities

1. Check hemoglobin and hematocrit levels and look at stool color every shift if the patient reports black stools or vomiting blood — 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 — 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 — 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
4. Teach the patient to raise the head of the bed 6 to 8 inches using blocks under the bed legs, not just pillows — Gravity keeps stomach acid below the lower esophageal sphincter (the valve between stomach and esophagus) when the whole torso is tilted; pillows alone bend the waist and increase abdominal pressure that pushes acid upward

5. Document the last meal time and make sure no food is given within 3 hours of bedtime — A full stomach increases pressure that forces the lower esophageal sphincter open; lying down with food inside lets acid wash into the esophagus because gravity cannot help anymore
6. Verify that *H. pylori* testing was done with stool antigen, breath test, or biopsy during endoscopy, and confirm antibiotic therapy is ordered if positive — *H. pylori* bacteria burrow into stomach mucus and release urease (an enzyme) that creates ammonia and weakens the lining; 60% of ulcers come from this infection, and triple therapy with two antibiotics plus PPI kills it in 14 days to prevent ulcer return
7. Prepare the patient for urgent upper endoscopy if they are over 60 years old with new symptoms or have any alarm features like weight loss or anemia — Gastric cancer looks identical to benign ulcers on symptoms alone; endoscopy lets the provider take biopsies to check for malignant cells, and age over 60 raises cancer risk five times
8. Teach portion control and refer to weight-loss resources if BMI is over 30 — Obesity increases intra-abdominal pressure (pressure inside the belly) that squeezes the stomach and forces acid past the lower esophageal sphincter; losing 10% of body weight cuts reflux episodes in half

Sources

- ACG Clinical Guideline: Treatment of *Helicobacter pylori* Infection (2017)
- ACG Clinical Guideline: Diagnosis and Management of Gastroesophageal Reflux Disease (2022)
- American Gastroenterological Association
- UpToDate Clinical Guidelines

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