

GERD and Peptic Ulcer Disease

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THE STORY IN ONE SENTENCE

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“GERD and PUD mean acid in the wrong place. Stop the acid with a PPI (proton pump inhibitor, a medicine that turns off acid pumps in the stomach) and kill H. pylori bacteria if it is there.”

Normal anatomy — the body's job

The lower esophageal sphincter (LES) — a ring of smooth muscle at the bottom of the esophagus — stays closed between swallows to keep stomach acid from splashing up. Meanwhile, the stomach and duodenum coat themselves in a thick layer of mucus and bicarbonate (a base that neutralizes acid) so their own acid does not burn through. Keep that picture in your head, because every weird finding is just a change from this normal setup.

BODY'S JOB

The LES acts like a one-way valve: it opens to let food down, then squeezes shut to trap acid below. The stomach lining makes hydrochloric acid to break down food and kill bacteria, but it also secretes mucus and bicarbonate as a shield so the acid does not eat the stomach itself. The duodenum continues that mucus shield and gets extra bicarbonate from the pancreas to neutralize any acid that passes through. This system keeps acid working where it helps and blocked where it would hurt.

1. Food enters the esophagus and travels downward.
2. The LES relaxes to let food drop into the stomach, then contracts shut.
3. Stomach parietal cells (acid-making cells in the stomach wall) pump out hydrochloric acid and pepsin (a protein-digesting enzyme).

4. At the same time, mucus-secreting cells coat the stomach lining with a thick gel layer, and bicarbonate (a base) sits in that gel to neutralize any acid that touches the wall.
5. Partly digested food and acid move into the duodenum, where the pancreas squirts in bicarbonate to neutralize the acid and mucus cells continue the protective coat.
6. The LES stays closed between meals so acid cannot flow backward into the unprotected esophagus.

What breaks

GERD happens when the lower esophageal sphincter (LES) becomes weak or a hiatal hernia pulls it open, so acid splashes up into the esophagus. Peptic ulcer disease happens when *H. pylori* bacteria burrow into the stomach lining and weaken the mucus shield, or when NSAIDs (nonsteroidal anti-inflammatory drugs like ibuprofen, naproxen, or aspirin) block the chemicals that build mucus, leaving the lining naked against acid. Either way, acid ends up touching tissue that cannot handle it.

CLINICAL TERM

GERD results from transient lower esophageal sphincter relaxations (TLESRs) — spontaneous LES openings unrelated to swallowing — or from anatomic disruption by a hiatal hernia that reduces LES pressure. Peptic ulcer disease results from *H. pylori* infection (the bacteria produce urease, which raises local pH and allows colonization, then releases toxins and proteases that disrupt the mucus-bicarbonate barrier) or NSAID use (NSAIDs inhibit cyclooxygenase-1, blocking prostaglandin E₂ synthesis; prostaglandin E₂ normally stimulates mucus and bicarbonate secretion and maintains mucosal blood flow, so its absence leaves the mucosa vulnerable to acid injury).

How the body compensates

The body tries to heal by increasing mucus production, boosting blood flow to the injured area to bring repair cells, and triggering cough or throat-clearing to protect the airway from refluxed acid. These responses can mask the severity of the underlying break — heartburn may feel mild even as the esophagus is being scarred, and ulcer pain may come and go as the lining tries to patch itself between acid attacks. The danger is that compensatory mechanisms buy time but do not fix the root cause, so the injury silently deepens until a complication suddenly appears.

1. The stomach ramps up mucus and bicarbonate secretion to try to rebuild the protective layer over an ulcer or inflamed area.
2. Blood flow increases to the damaged mucosa, bringing white blood cells and growth factors to patch the hole; this is why ulcers can partially heal between flares.

3. In GERD, the esophagus tries to thicken its lining and increase saliva production (saliva is slightly alkaline and helps neutralize small amounts of refluxed acid).
4. The body triggers cough, throat-clearing, or even laryngospasm (vocal cord closure) when acid reaches the throat or airways, protecting the lungs from aspiration.
5. None of these compensations fix the upstream problem — the weak LES, the hiatal hernia, the bacteria, or the NSAID — so the injury continues in cycles of partial healing and re-injury.
6. The crash point to watch for is: a bleeding ulcer (erosion into a blood vessel, causing vomiting blood (hematemesis) or black tarry stool (melena)), perforation (a hole through the stomach or duodenal wall into the abdominal cavity, causing peritonitis), gastric outlet obstruction (scar tissue from repeated ulcers blocks the stomach exit), esophageal stricture (scar tissue narrows the esophagus, causing trouble swallowing (dysphagia)), or Barrett esophagus with dysplasia (chronic acid exposure turns esophageal squamous cells into intestinal-type columnar cells, some of which become precancerous).
7. Watch for the crash point before it happens: a patient with new-onset anemia, black stools, or trouble swallowing is no longer stable, even if pain is mild.

Domino effect

1. The lower esophageal sphincter (LES) relaxes too often or a hiatal hernia pulls it open. — Normally the LES stays closed to trap acid in the stomach; when it opens without a swallow (transient LES relaxation) or is mechanically stretched by a hernia, acid flows up into the esophagus, which has no mucus shield and burns immediately.
2. Acid touches the esophageal lining, causing heartburn and regurgitation. — The esophagus is lined with squamous epithelium (flat cells) designed for swallowing, not for acid; hydrochloric acid burns these cells, triggering pain signals (heartburn) and sometimes washing back into the mouth (regurgitation) or throat (sour taste).
3. Acid vapor or droplets reach the throat and airways, causing chronic cough, hoarseness, or asthma-like wheezing. — Even tiny amounts of acid or acidic gas irritate the larynx (voice box) and can trigger bronchospasm (airway tightening) through vagal reflexes (nerve signals that make airways squeeze shut); this is called laryngopharyngeal reflux or atypical GERD.
4. Meanwhile, *H. pylori* bacteria or NSAIDs break the mucus-bicarbonate shield in the stomach or duodenum. — *H. pylori* secretes urease, ammonia, and toxins (VacA, CagA) that punch holes in the mucus layer and damage epithelial cells; NSAIDs block prostaglandin E₂, so the stomach stops making mucus and bicarbonate and mucosal blood flow drops, leaving the lining naked.

5. Stomach acid digs into the unprotected lining, forming an ulcer (a crater in the mucosa). — Without the mucus shield, hydrochloric acid and pepsin (a protein-digesting enzyme) erode the epithelium down to the submucosa or even the muscle layer; you feel this as gnawing epigastric pain, often worse when the stomach is empty because acid has nothing else to digest.
6. If the ulcer erodes into a blood vessel, bleeding begins — you see vomiting blood (hematemesis) (vomiting blood), black tarry stool (melena) (black, tarry stool from digested blood), or anemia. — Blood vessels run through the submucosa; when acid eats deep enough to hit a vessel, it opens and bleeds; small bleeds cause chronic anemia, large bleeds cause sudden vomiting of blood or passage of black stool.
7. If the ulcer burns all the way through the stomach or duodenal wall, perforation occurs — a hole opens into the abdominal cavity. — The wall is only a few millimeters thick; if acid and pepsin digest through all layers (mucosa, submucosa, muscle, serosa), stomach contents spill into the peritoneum, causing sudden, severe pain (peritonitis) and free air under the diaphragm on X-ray.
8. Chronic inflammation and healing cycles cause scarring; in the stomach, scar tissue can block the pylorus (gastric outlet obstruction); in the esophagus, it can narrow the tube (stricture). — Every time the lining heals, it lays down collagen (scar tissue); repeated cycles build up thick scar that does not stretch, so food cannot pass easily — you feel early satiety and vomiting (gastric outlet obstruction) or trouble swallowing (dysphagia) and food sticking (esophageal stricture).
9. In GERD, years of acid exposure can transform esophageal squamous cells into intestinal-type columnar cells (Barrett esophagus), and some of those cells may develop dysplasia (precancerous changes). — Chronic injury triggers metaplasia (the body swaps in a tougher cell type to survive the acid), but intestinal-type cells in the esophagus can accumulate DNA mutations and progress to adenocarcinoma (esophageal cancer); Barrett with high-grade dysplasia is the last stop before cancer.

Symptoms · what the patient feels, and why

- Burning feeling behind the breastbone (heartburn) that often comes after eating or when lying down: The ring of muscle at the bottom of the esophagus (the lower esophageal sphincter, which normally acts like a one-way gate) relaxes too much or at the wrong times, so stomach acid splashes backward up into the esophagus (the swallowing tube). The esophagus lining isn't built to handle acid, so it feels like burning.
- Sour or bitter taste in the mouth (acid regurgitation): Stomach acid and partly digested food flow back up into the throat and mouth when the lower esophageal sphincter (the one-way gate at the bottom of the esophagus) opens when it shouldn't. You literally taste the acid.

- Pain in the upper middle belly (epigastric pain), often a gnawing or burning feeling that comes and goes with meals: An ulcer (a sore spot or crater) has formed in the lining of the stomach or duodenum (the first part of the small intestine right after the stomach). Stomach acid touches the raw spot, causing pain. The pain may ease right after eating (if the ulcer is in the duodenum, because food buffers the acid) or get worse after eating (if the ulcer is in the stomach).
- Feeling uncomfortably full or bloated after eating even a small amount of food: Acid irritation and inflammation in the stomach or duodenum can slow the stomach's normal squeezing and emptying (gastric motility). Food sits longer than it should, making you feel stuffed and heavy.
- Nausea or throwing up: Acid irritation and inflammation upset the stomach lining, triggering signals in the vagus nerve (a large nerve that connects the gut to the brainstem's vomiting center). The brain interprets these signals as 'something is wrong' and turns on nausea or vomiting to try to empty the stomach.
- Chronic cough or hoarse voice that won't go away: Acid reflux reaches high enough to touch the vocal cords in the larynx (the voice box) or drip into the trachea (the windpipe), irritating the lining. The vocal cords swell and the airways react by coughing to clear the irritation.
- Trouble swallowing or food feeling stuck in the chest (dysphagia): Repeated acid injury over time causes scar tissue to build up in the esophagus, narrowing the tube (stricture). Food has a harder time passing through the tight spot. This can also be a sign of more serious changes like Barrett's esophagus (where the lining starts to change type) or even early cancer.
- Unintended weight loss (losing pounds without trying to diet or exercise more): Weight loss can mean the ulcer or reflux is severe enough to make eating painful, so the person eats less. It can also signal a deeper ulcer eating into tissue, infection with *Helicobacter pylori* (a bacteria that digs into the stomach lining), or a tumor blocking or changing the stomach or esophagus.
- Vomiting blood (bright red or looking like coffee grounds) or passing black, tarry, sticky stools: An ulcer has eroded deep enough to break open a blood vessel, causing bleeding. Bright red blood means active, brisk bleeding. Coffee-ground vomit or black tarry stool (melena) means blood has been sitting in the stomach or intestine long enough for stomach acid to turn it dark. This is a medical emergency.
- Anemia (low red blood cell count) found on blood work, often with feeling tired, pale, or weak: Slow, steady bleeding from an ulcer or inflamed lining leaks small amounts of blood over weeks or months. The body loses iron faster than it can replace it, so red blood cell production drops. Without enough red cells, less oxygen reaches your tissues, making you tired and pale.

Focused exam

- Burning pain behind the breastbone (retrosternal burning) that gets worse when lying down or right after meals: This is the classic signal that stomach acid is splashing backward up into the esophagus. It means the lower esophageal sphincter (LES), the ring of muscle at the bottom of the esophagus that acts like a one-way valve, is not staying closed the way it should. When acid reaches tissue that is not built to handle it, you feel that burn.
- Dull, burning pain in the upper middle belly (epigastric pain), especially between meals or at night, sometimes relieved by eating or antacids: This pattern points to a peptic ulcer — a raw crater or sore in the lining of the stomach or duodenum (the first part of the small intestine). Acid is eating through the protective mucus layer and touching bare tissue underneath. Pain between meals means acid is sitting on the sore with no food to soak it up.
- Regurgitation — sour liquid or bits of food coming back up into the mouth or throat without any effort to vomit: When the LES is weak or opens at the wrong times, stomach contents flow backward. This is proof the valve itself is the problem, not the stomach muscle. It is different from vomiting, which involves the whole stomach squeezing hard.
- A dry cough that will not go away, or new wheezing and trouble breathing at night or when lying flat: Acid from GERD can travel all the way up the esophagus and spill over into the throat and windpipe, irritating the vocal cords and lungs. The body tries to protect the airway by coughing. Many people and even doctors miss this as a sign of GERD because there is no heartburn.
- Positive test for *Helicobacter pylori* (*H. pylori*) bacteria — by urea breath test, stool antigen test, or biopsy during endoscopy — in someone with upper belly pain: *H. pylori* is a spiral-shaped germ that burrows into the stomach lining, weakens the protective mucus, and lets acid burn holes (ulcers). Finding it means you have a treatable cause of peptic ulcer disease. Killing the bacteria stops new ulcers from forming.
- Black, sticky, tar-like stools (melena) or vomit that looks like coffee grounds or contains bright red blood: This means an ulcer has eaten through a blood vessel and the person is bleeding inside. Black stool means the blood has been digested; coffee-ground vomit means old blood from the stomach. Either way, the body is losing blood and this is an emergency.
- Trouble swallowing (dysphagia) or the feeling that food is getting stuck on the way down: Long-term acid damage can scar the esophagus and make it narrow (a stricture), or it can change the lining cells into a pre-cancer form called Barrett esophagus. Either way, this is a red flag that you need to look inside with an endoscope to see what is happening and catch cancer early if it is starting.

Diagnostics · every test answers a question

- Upper endoscopy (esophagogastroduodenoscopy, EGD) → Is there visible damage to the esophagus, stomach, or duodenum? Is there an ulcer, stricture, Barrett esophagus, or cancer?
- Helicobacter pylori testing — urea breath test, stool antigen test, or rapid urease test on biopsy during endoscopy → Is H. pylori bacteria present in the stomach?
- Complete blood count (CBC) → Is the person anemic from chronic slow bleeding?
- Fecal occult blood test (FOBT) or fecal immunochemical test (FIT) → Is there hidden blood in the stool that you cannot see with your eyes?
- Serum gastrin level (if refractory or atypical ulcers) → Is there too much gastrin hormone driving excessive acid production?
- Esophageal pH monitoring (24-hour ambulatory or wireless capsule) → How much acid is actually refluxing into the esophagus over a full day and night?
- Barium swallow (esophagram) with fluoroscopy → Is there a structural problem like a stricture, hiatal hernia, or motility issue blocking or slowing the esophagus?

Treatment logic

- Proton pump inhibitor (PPI) — omeprazole 20–40 mg once daily or equivalent — for 8 weeks in GERD, 4–8 weeks in peptic ulcer disease → PPIs block the hydrogen-potassium pump in stomach cells that makes acid. Turning off that pump drops stomach acid by more than 90%, letting the raw lining in the esophagus or stomach heal and stopping the burn. Healing rates for ulcers reach 90–95% at 8 weeks.
- H. pylori eradication therapy — standard triple therapy (PPI + clarithromycin 500 mg + amoxicillin 1 g, all twice daily for 14 days) or quadruple therapy (PPI + bismuth + tetracycline + metronidazole for 10–14 days) if resistance is likely → Kills the H. pylori bacteria colonizing the stomach. Without the germ eating away the mucus, ulcers heal and almost never come back (recurrence drops from ~70% to < 5%). Current guidelines prefer 14-day regimens because they work better than 7–10 days, especially with rising antibiotic resistance.
- Stop NSAIDs (ibuprofen, naproxen, aspirin at high dose) or switch to acetaminophen; if NSAIDs must continue, add PPI daily for protection → NSAIDs block an enzyme (COX-1) that makes protective mucus and blood flow in the stomach lining. Stopping them removes the injury and lets the lining heal. If stopping is not possible (for example, low-dose aspirin for heart protection), a daily PPI cuts ulcer risk by about 80%.

- Lifestyle changes — lose weight if overweight, raise the head of the bed 6–8 inches, avoid meals within 3 hours of bedtime, stop smoking, limit alcohol, caffeine, chocolate, and fatty foods → Excess belly fat and big meals increase pressure in the abdomen and push stomach contents up. Elevating the head uses gravity to keep acid down at night. Smoking weakens the LES and slows healing. Cutting triggers reduces how often the sphincter relaxes inappropriately.
- Surgical fundoplication (Nissen or partial wrap) or LINX magnetic sphincter augmentation (for GERD refractory to medical therapy) → Fundoplication wraps the top of the stomach around the lower esophagus to tighten the LES and physically stop reflux. LINX places a ring of magnetic beads around the LES to reinforce closure. Both restore the valve function and can eliminate the need for daily PPI in 85–90% of people at 5 years.
- Urgent upper endoscopy (EGD) if alarm features are present — new trouble swallowing (dysphagia), unintended weight loss > 5%, gastrointestinal bleeding, iron-deficiency anemia, persistent vomiting, age > 60 with new-onset symptoms, family history of upper GI cancer → Lets the doctor see inside, take biopsies to check for cancer or Barrett esophagus, stop active bleeding with clips or cautery, and dilate strictures. Early cancer (stage 0–1) has a 5-year survival > 90% if caught, versus < 30% for advanced disease.

Medications

Pantoprazole (Protonix), Omeprazole (Prilosec), Esomeprazole (Nexium) · Proton pump inhibitor (PPI)

Shuts off the tiny pumps inside stomach cells that squirt acid into the stomach, so the stomach makes much less acid overall. That gives a sore or raw spot (ulcer or inflamed esophagus) time to heal without being bathed in acid.

1. You swallow the pill; it dissolves in your small intestine and enters the bloodstream.
2. The drug travels to the parietal cells lining the stomach.
3. It gets trapped and turned on inside the acid-making compartments of those cells.
4. Once active, it permanently binds to and blocks the H^+/K^+ -ATPase pump (the proton pump).
5. That pump can no longer swap potassium for hydrogen ions, so no new acid is made by that pump.
6. Acid output drops by 90–95 % within a few days, letting injured tissue heal.

SAFETY WATCH

Long use (years) may lower magnesium, calcium, and vitamin B₁₂ because stomach acid helps absorb them; your doctor may check levels if you take a PPI for more than a year. · Rare risk of bone fractures with very long-term use (especially hip, wrist, spine) in older adults. · Slightly higher risk of *Clostridioides difficile* (C. diff) diarrhea and some other gut infections because stomach acid normally kills many germs. · Possible kidney problems (acute interstitial nephritis) in rare cases; tell your doctor if urine changes or swelling happens. · Do NOT stop suddenly after months of daily use without talking to your doctor—acid can rebound and feel worse for a few days.

Famotidine (Pepcid), Nizatidine (Axiid) · H₂-receptor antagonist (H₂ blocker)

Blocks one of the main 'on' switches for acid-making cells in the stomach—the histamine₂ receptor—so those cells get fewer signals to pump out acid. Not as powerful as a PPI, but works faster (within an hour) and is good for mild symptoms or nighttime heartburn.

1. Histamine (a messenger chemical released in the stomach) normally docks onto H₂ receptors and tells parietal cells to secrete acid.
2. The H₂ blocker sits on those receptors instead, preventing histamine from binding.
3. Without that histamine signal, the parietal cell makes less acid—typically 50–70 % less.
4. Relief happens within 30–60 minutes and lasts about 8–12 hours.

SAFETY WATCH

Generally very safe for short-term use. · Can lose effectiveness (tolerance) if taken every day for weeks, because the stomach upregulates other pathways; works best as needed or at bedtime. · Rare confusion or dizziness in older adults or people with kidney disease, because the drug is cleared by the kidneys. · May interact with some antifungals (like ketoconazole) by raising stomach pH and reducing their absorption.

Amoxicillin + Clarithromycin (or Metronidazole) + PPI (triple therapy), OR Bismuth + Metronidazole + Tetracycline + PPI (quadruple therapy) · H. pylori eradication regimen (antibiotic combination)

Kills the *Helicobacter pylori* bacteria living in the stomach lining. H. pylori burrows into the mucus layer and damages the protective barrier, which lets acid cause ulcers. Wiping out the bacteria lets the ulcer heal and sharply lowers the chance it comes back.

1. The PPI cuts acid production so antibiotics survive in the stomach and the mucus layer thickens, helping antibiotics penetrate.

2. Amoxicillin punches holes in the bacterial cell wall; clarithromycin (or metronidazole) stops the bacteria from making proteins they need to live.
3. Bismuth (in quadruple therapy) coats the stomach, disrupts bacterial enzymes, and keeps *H. pylori* from sticking to the lining.
4. Tetracycline and metronidazole (quadruple) attack bacterial DNA and protein factories from different angles.
5. After 10–14 days, over 85–90 % of infections are cleared if the patient takes every dose.

SAFETY WATCH

Diarrhea and stomach upset are common; probiotics may help but do not skip antibiotic doses. · Clarithromycin can interact with many drugs (statins, some heart medicines) by blocking liver enzymes; tell your doctor every medicine you take. · Metronidazole causes a severe reaction (flushing, vomiting, fast heart rate) with alcohol; avoid all alcohol during treatment and for 3 days after. · Tetracycline stains developing teeth, so it is NOT used in children under 8 or pregnant women. · Bismuth turns stool black (harmless) and can cause temporary dark discoloration of the tongue. · Rising antibiotic resistance (especially to clarithromycin and metronidazole) means your doctor may order a breath or stool test 4 weeks after treatment to confirm the bacteria are gone.

Sucralfate (Carafate) · Mucosal protectant

Forms a sticky gel that glues itself to raw or ulcerated spots in the stomach or duodenum, creating a physical bandage that shields the sore from acid and pepsin while it heals underneath.

1. Sucralfate is an aluminum salt of sucrose (table sugar) plus sulfate groups.
2. In the acidic environment of the stomach, it becomes very sticky.
3. It binds selectively to proteins in damaged tissue (the ulcer base), forming a protective paste that lasts 6 hours.
4. This barrier keeps acid, bile, and pepsin from attacking the healing tissue.
5. The ulcer heals from below while the coating gradually dissolves and passes in stool.

SAFETY WATCH

Constipation is the most common side effect. · Can bind to other medicines (especially thyroid hormone, some antibiotics, blood thinners) and block their absorption; take sucralfate 2 hours before or after other drugs. · Contains aluminum; use cautiously and short-term in people with severe kidney disease, because aluminum can build up. · Safe in pregnancy (Category B)—often chosen when a PPI is not preferred.

Antacids (calcium carbonate [Tums], aluminum/magnesium hydroxide [Maalox, Mylanta]) · Antacid (buffering agent)

Chemically neutralizes stomach acid that is already sitting in the stomach, raising the pH instantly and giving quick (but short-lived) relief of heartburn or ulcer pain.

1. You chew or swallow the antacid.
2. The base (carbonate or hydroxide) reacts directly with hydrochloric acid (HCl) in the stomach.
3. The reaction produces water, carbon dioxide (you may burp), and a salt (calcium chloride, magnesium chloride, or aluminum chloride).
4. Stomach pH rises from ~1–2 toward 3–4, which inactivates pepsin and soothes burning.
5. Relief is fast (5–15 minutes) but lasts only 30–60 minutes, because the stomach keeps making new acid.

SAFETY WATCH

Calcium carbonate can cause constipation and, if used in large amounts daily, can raise blood calcium (hypercalcemia) and kidney-stone risk. · Aluminum hydroxide causes constipation; magnesium hydroxide causes diarrhea—combination products balance the two. · Chronic high-dose use of aluminum antacids may contribute to weak bones (osteomalacia) or, in kidney disease, aluminum toxicity. · Can interfere with absorption of many drugs (antibiotics, iron, thyroid hormone); separate by 2 hours. · Sodium bicarbonate (baking soda) antacids can overload the body with sodium and cause metabolic alkalosis (the blood turning too alkaline) if overused—not recommended for regular use.

Misoprostol (Cytotec) · Prostaglandin E₁ analog

Replaces protective prostaglandins in the stomach lining that NSAIDs (like ibuprofen, aspirin, naproxen) knock out. Prostaglandins tell the stomach to make thick mucus, pump out bicarbonate (a natural acid buffer), and keep good blood flow to the lining—all of which shield it from acid damage.

1. NSAIDs block an enzyme called COX-1, which the stomach uses to make prostaglandin E₂.
2. Without prostaglandin E₂, mucus production drops, bicarbonate secretion falls, and blood flow to the lining decreases—the stomach becomes vulnerable.
3. Misoprostol mimics natural prostaglandin E₁, docking onto prostaglandin receptors in the stomach.
4. This signals cells to secrete more mucus and bicarbonate and keeps blood vessels open, restoring the protective barrier.
5. It also tells parietal cells to make less acid, adding a second layer of defense.

SAFETY WATCH

Diarrhea and abdominal cramping are very common (up to 30–40 % of people) because prostaglandins also speed gut motility. · Absolutely contraindicated in pregnancy—misoprostol causes uterine contractions and is used (off-label) to induce abortion; women of childbearing age must use reliable contraception. · Must be taken 3–4 times daily with food, which many patients find inconvenient; a PPI is usually preferred for NSAID ulcer prevention unless the patient is already on a PPI and still at high risk. · Does NOT prevent cardiovascular events that NSAIDs may cause; it only protects the stomach lining.

Red flags

- New heartburn or belly pain starting after age 60 (higher risk of cancer)
- Trouble swallowing or feeling like food is sticking in the chest
- Vomiting blood (red or coffee-ground color) or black, tarry stools
- Unintended weight loss (losing 10 pounds or more without trying)
- Anemia (low red blood cell count) or signs of bleeding on blood tests

Nursing priorities

1. Assess for signs of active bleeding: check vital signs for a fast heart rate (tachycardia) (fast heart rate over 100) and low blood pressure (hypotension) (low blood pressure under 90 systolic), examine stool color, and ask about vomiting blood. — Bleeding peptic ulcers can cause hypovolemic shock (dangerous drop in blood volume) from blood loss, requiring immediate fluid resuscitation and possible transfusion to maintain tissue blood flow (perfusion) (blood flow to organs).
2. Administer PPI medication on empty stomach 30 to 60 minutes before breakfast as ordered. — PPIs (proton pump inhibitors like omeprazole) work best when taken before the first meal because they block acid pumps that activate during eating, reducing stomach acid production by over 90 percent.
3. Teach patient to elevate head of bed 6 to 8 inches using blocks under bed legs, not just pillows. — Gravity prevents acid reflux during sleep when lower esophageal sphincter (the valve between esophagus and stomach) relaxes, and proper bed elevation is more effective than pillow props which can increase abdominal pressure.
4. Review all medications and hold NSAIDs, then notify provider about alternatives for pain management. — NSAIDs inhibit prostaglandins (protective chemicals) in the stomach lining, causing direct mucosal injury and ulcer formation, so stopping them allows healing while PPI therapy works.

5. Educate patient to avoid eating within 3 hours of bedtime and identify personal trigger foods to eliminate. — A full stomach increases pressure on the lower esophageal sphincter and stimulates more acid secretion, while lying down with food present promotes reflux of acidic contents into the esophagus.
6. Teach proper PPI adherence and explain that symptom relief takes 3 to 5 days, with full healing requiring 8 weeks. — Patients often stop medication when symptoms improve in days, but mucosal healing (repair of stomach or esophagus lining) takes weeks and incomplete courses lead to ulcer recurrence and complications.
7. If *H. pylori* (a stomach bacteria) testing is ordered, ensure patient stops PPI for 2 weeks before breath or stool test. — PPIs suppress *H. pylori* bacteria temporarily, causing false-negative test results and missed infections that require specific antibiotic therapy for permanent ulcer cure.
8. Monitor for alarm symptoms daily and teach patient to report new trouble swallowing (dysphagia), persistent vomiting, severe pain, or any bleeding immediately. — Complications like gastric outlet obstruction (blocked stomach exit), perforation (hole in stomach wall), or malignancy can develop rapidly and require urgent endoscopy or surgical intervention to prevent death.

Sources

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- American College of Gastroenterology (ACG) Peptic Ulcer Disease Guideline 2017
- American Gastroenterological Association (AGA)
- Cochrane Reviews on *Helicobacter pylori* and peptic ulcer

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