

PUD and GERD

A plain-English guide for you and your family

WHAT IS HAPPENING

Your stomach makes strong acid to break down food. Special cells called parietal cells pump out this acid every day. To keep the stomach from digesting itself, the lining makes a thick layer of mucus (a slimy protective gel) and releases bicarbonate (a base chemical that neutralizes acid, like baking soda does). At the top of your stomach sits a ring of muscle called the lower esophageal sphincter—it works like a one-way door, keeping food and acid down after you swallow. When everything works right, acid stays where it belongs. But two things can go wrong. In GERD (gastroesophageal reflux disease), that muscle door gets weak or lazy, or extra pressure in your belly (from being overweight, pregnant, or having a hiatal hernia where part of the stomach pokes up through the diaphragm) pushes acid backward into the esophagus (the food tube that connects your mouth to your stomach). The esophagus has no mucus shield, so acid burns it and causes heartburn. Over months or years, that burning can scar the lining (esophagitis) or even change the cells into a tougher stomach-like type (Barrett's esophagus), which is a warning flag for future cancer risk. In PUD (peptic ulcer disease), the problem is inside the stomach or the first part of the small intestine (the duodenum). Either *H. pylori* bacteria drill through the mucus layer and damage the lining, or NSAIDs (non-steroidal anti-inflammatory drugs like ibuprofen or aspirin) block the chemicals that build mucus, leaving the lining naked against acid. Acid then eats a crater—an ulcer—into the wall. If it goes deep enough, it hits a blood vessel and bleeds, showing up as black tarry stool or throwing up blood that looks like coffee grounds. If it goes all the way through the wall, stomach contents spill into the belly and cause peritonitis (a dangerous infection). The body tries to cope by making extra mucus, slowing stomach emptying, and tightening blood flow, but these are just stopgaps. The real fix is stopping the acid oversupply (with a proton-pump inhibitor like omeprazole), killing *H. pylori* if it's there (with antibiotics), and stopping NSAIDs. Each symptom—heartburn, belly pain that changes with eating, black stool—points back to the same root problem: acid where it shouldn't be, and tissue that can't handle it.

What you might feel

- Burning behind the breastbone (heartburn) that gets worse when you lie down or bend over

- Gnawing or burning pain in the upper belly, often between meals or at night
- Pain that gets better when you eat food or take antacids
- Nausea or feeling full quickly after eating a small amount
- Bloating or burping more than usual
- Trouble swallowing or food getting stuck
- Unintended weight loss
- Black, tarry stools or vomit that looks like coffee grounds
- New heartburn or belly pain starting after age 60

Call the doctor right away if...

- 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

What the care team will do

- Check hemoglobin and hematocrit levels and look at stool color every shift if the patient reports black stools or vomiting blood
- Hold the next dose of aspirin, ibuprofen, naproxen, or any NSAID and notify the provider
- Give the prescribed PPI 30 to 60 minutes before breakfast every day, not at bedtime
- Teach the patient to raise the head of the bed 6 to 8 inches using blocks under the bed legs, not just pillows
- Document the last meal time and make sure no food is given within 3 hours of bedtime
- Verify that H. pylori testing was done with stool antigen, breath test, or biopsy during endoscopy, and confirm antibiotic therapy is ordered if positive
- 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
- Teach portion control and refer to weight-loss resources if BMI is over 30

Medicines you may be given — and why

Omeprazole (Prilosec), lansoprazole (Prevacid), esomeprazole (Nexium), pantoprazole (Protonix)

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.

Ranitidine (Zantac—withdrawn 2020), famotidine (Pepcid), nizatidine (Axid), cimetidine (Tagamet)

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.

Sucralfate (Carafate)

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.

Aluminum/magnesium hydroxide (Maalox, Mylanta), calcium carbonate (Tums, Roloids)

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.

Amoxicillin + clarithromycin + PPI (or metronidazole instead of amoxicillin if penicillin allergy); or bismuth + metronidazole + tetracycline + PPI

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).

Misoprostol (Cytotec)

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.

Metoclopramide (Reglan)

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.

Where this information comes from

- 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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