in the clinic

Gastroesophageal Reflux Disease

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Gastroesophageal reflux disease (GERD) is one of the most common gastrointestinal disorders in Western industrialized countries. Men and women develop GERD with equal frequency, but complicated GERD occurs more frequently in men and with advanced age. It is typically the result of prolonged exposure of the esophagus to gastric acid due to impaired esophageal motility, defects in the lower esophageal sphincter, and impairments in the antireflux barrier at the gastroesophageal junction. The acid exposure can damage the esophageal mucosa, potentially leading to Barrett's esophagus and esophageal cancer. GERD is a chronic disease, and many patients require lifelong therapy. Treatment helps to reduce symptoms, promote esophageal healing, and reduce the risk for cancer.

Diagnosis

Consider GERD in Patients with the Following Symptoms

- Heartburn or regurgitation
- Wheezing or dyspnea
- Chronic cough
- Chronic hoarseness or sore throat
- Globus
- Throat clearing
- Chest pain
- Halitosis

What symptoms and signs should prompt clinicians to consider GERD?

Typical GERD symptoms include chest discomfort (heartburn) and regurgitation. Symptoms occur most often after meals, especially fatty meals. Lying down, bending, or physical exertion often aggravate symptoms, and antacids provide relief. Patients with classic symptoms rarely require testing to confirm the diagnosis because of the high positive predictive value of classic symptoms (1). When heartburn (89% specificity, 81% positive predictive value) and regurgitation (95% specificity, 57% positive predictive value) occur together, a physician can diagnose GERD with greater than 90% accuracy (2).

GERD can also cause extraesophageal symptoms, including wheezing, chronic cough, shortness of breath, hoarseness, unexplained chest pain, globus (choking sensation), halitosis, and sore throat or a sense of needing to clear one's throat. Up to 80% of patients have at least one extraesophageal symptom. It is worth noting that although these symptoms are associated with GERD, establishing a definitive causal relationship between GERD and extraesophageal symptoms is difficult because GERD may be one of many causes of these symptoms.

When should clinicians consider an empirical therapeutic trial of acid-suppression therapy to support a preliminary diagnosis of GERD?

Performing diagnostic tests for all patients presenting with symptoms that might indicate GERD would be costly and is not necessary to arrive at a sufficiently accurate diagnosis. Response to an empirical trial of acid-suppression therapy is considered a sufficiently sensitive and specific method for establishing a GERD diagnosis among patients with classic symptoms of heartburn or regurgitation. Although proton pump inhibitors (PPIs) are more expensive than H_2-receptor blockers, PPIs are considered the drug of choice for an empirical therapeutic trial because they block acid more effectively than H_2-receptor blockers. An empirical trial typically consists of a double-dose of a PPI (such as omeprazole 20 to 40 mg twice daily) for 1 week or a standard-dose PPI (such as omeprazole 20 to 40 mg once daily) for 2 weeks.

A study that compared 24-hour pH monitoring with a 2-week course of high-dose omeprazole in 35 patients with erosive esophagitis found that the omeprazole test was at least as sensitive as 24-hour pH monitoring in diagnosing GERD (3).

A study randomly assigned 85 patients who had ambulatory pH monitoring and grade 0 or 1 esophagitis by upper endoscopy to either omeprazole 40 mg/d or placebo for 14 days and concluded that a symptomatic response to omeprazole had a sensitivity

1. DeVault KR, Castell DO. Updated guidelines for the diagnosis and treatment of gastroesophageal reflux disease. Am J Gastroenterol. 2005; 100:190-200. [PMID: 15654800]
2. Klauser AG, Schindlbeck NE, Müller-Lissner SA. Symptoms in gastro-oesophageal reflux disease. Lancet. 1990;335:205-8. [PMID: 1967675]
3. Fass R, Ofman JJ, Sampliner RE, et al. The omeprazole test is as sensitive as 24-h oesophageal pH monitoring in diagnosing gastro-oesophageal reflux disease in symptomatic patients with erosive oesophagitis. Aliment Pharmacol Ther. 2000;14:389-96. [PMID: 10759617]
4. Schenk BE, Kuipers EJ, Klinkenberg-Knol EC, et al. Omeprazole as a diagnostic tool in gastroesophageal reflux disease. Am J Gastroenterol. 1997;92:1997-2000. [PMID: 9362179]

and specificity similar to ambulatory 24-hour pH monitoring (4).

According to one meta-analysis of 15 studies that compared the clinical response to PPI with objective measures, such as 24-hour pH monitoring, endoscopy, and symptom questionnaires, testing may be necessary to definitively diagnose GERD in some patients even though many patients with uncomplicated GERD respond to empirical PPI therapy (5).

When should clinicians consider upper endoscopy in evaluating patients with possible GERD?

If patients respond to empirical therapy, endoscopy is not necessary to confirm the diagnosis. Although the specificity of esophagitis on endoscopy is 90% to 100%, approximately 50% to 70% of patients with classic GERD symptoms have no esophagitis on endoscopy (6). If endoscopy is done, then histologic evaluation of seemingly normal squamous mucosa has little power to detect pathologic acid reflux (7). However, the American College of Gastroenterology recommends that clinicians consider upper endoscopy to rule out Barrett's esophagus in patients with chronic symptoms; to evaluate patients who do not respond to empirical therapy; and to investigate symptoms, such as dysphagia or weight loss, that suggest stricture, ulceration, or malignancy (1).

What other diagnoses should clinicians consider in patients with suspected GERD and atypical symptoms?

Clinicians should be aware that, in some patients, the cause of GERD-like symptoms or endoscopic esophagitis is not reflux but rather infection, pill-induced injury, or radiation. In patients who have atypical symptoms of GERD or in those who have not responded to empirical therapy, clinicians should consider alternative gastrointestinal or biliary disease processes (Table 1).

When patients present with chest pain, clinicians should always consider coronary artery disease before concluding that GERD is the cause of the chest pain. Symptoms can be unreliable for differentiating GERD from a cardiac source of chest pain (1). GERD is present in approximately 50% of unexplained chest pain cases after coronary artery disease has been excluded, and although classic symptoms are present in many cases where GERD is the cause of chest pain, they are not always present (8, 9).

Which other laboratory tests should clinicians consider in evaluating patients when the diagnosis of GERD is uncertain?

When patients present with atypical symptoms, testing with esophageal manometry, pH monitoring, and barium swallow may help to differentiate GERD from other diagnoses.

Ambulatory pH monitoring

Ambulatory pH monitoring detects the presence or absence of reflux of acidic gastric contents and is the best way to measure the actual amount of time reflux is present and to correlate symptoms with reflux episodes. However, up to 25% of patients with documented esophagitis may have normal results on pH monitoring (10). Traditionally, pH monitoring is performed with catheter-based probes. A wireless pH capsule probe is a new technique that may be more tolerable and may allow for longer assessment of esophageal pH. Impedence-pH monitoring is another emerging technique that evaluates intraluminal

Warning Symptoms for Stricture, Ulceration, or Malignancy

- Dysphagia or odynophagia
- Bleeding
- Weight loss
- Early satiety
- Choking (coughing, shortness of breath, or hoarseness caused by acid)
- Anorexia
- Frequent vomiting

5. Numans ME, Lau J, de Wit NJ, Bonis PA. Short-term treatment with proton-pump inhibitors as a test for gastroesophageal reflux disease: a meta-analysis of diagnostic test characteristics. Ann Intern Med. 2004;140:518-27. [PMID: 15068979]
6. Tefera L, Fein M, Ritter MP, et al. Can the combination of symptoms and endoscopy confirm the presence of gastroesophageal reflux disease? Am Surg. 1997;63:933-6. [PMID: 9322676]
7. Schindlbeck NE, Wiebecke B, Klauser AG, et al. Diagnostic value of histology in non-erosive gastro-oesophageal reflux disease. Gut. 1996;39:151-4. [PMID: 8977332]
8. Hewson EG, Sinclair JW, Dalton CB, Richter JE. Twenty-four-hour esophageal pH monitoring: the most useful test for evaluating noncardiac chest pain. Am J Med. 1991;90:576-83. [PMID: 2029015]
9. Davies HA, Jones DB, Rhodes J, Newcombe RG. Angina-like esophageal pain: differentiation from cardiac pain by history. J Clin Gastroenterol. 1985;7:477-81. [PMID: 4086742]
10. Martinez SD, Malagon IB, Garewal HS, et al. Non-erosive reflux disease (NERD)—-acid reflux and symptom patterns. Aliment Pharmacol Ther. 2003;17:537-45. [PMID: 12622762]

Table 1. Differential Diagnosis of GERD

Disease	Characteristics	Notes
Pill esophagitis	Presents with dysphagia or odynophagia	History of ingestion of the offending pill (e.g., potassium chloride, quinidine, tetracycline, doxycycline, NSAIDs, alendronate)
Infectious esophagitis	Presents with dysphagia or odynophagia	Often in immunocompromised patients with candidal, cytomegalovirus, or herpes simplex virus esophagitis
Esophageal motor disorders: achalasia, diffuse esophageal spasm, hypertensive or spastic motility disorders (e.g., nutcracker esophagus)	Dysphagia for liquids and solids; also may be associated with chest pain	Nutcracker esophagus may be coincident with GERD; heartburn or chest pain in achalasia not due to reflux but to fermentation of retained esophageal contents or esophageal muscle spasm
Nonulcer dyspepsia	Functional disorder, discomfort in midline of upper abdomen with fullness, bloating, or nausea	Usually does not respond to acid suppression
Eosinophilic esophagitis	Allergic esophagitis; vomiting and abdominal pain that improve with removal of offending food	Eosinophilis seen on esophageal biopsy
Esophageal cancer	Presents with dysphagia and weight loss, often in patients with longstanding GERD	Usually incurable by the time of clinical presentation
Coronary artery disease	Chest pain that may be clinically indistinguishable from chest pain associated with GERD	In patients at high risk for cardiac disease, should rule out cardiac disease before evaluating for GERD
Conditions Associated with GERD		
Pregnancy	Symptoms are experienced by 25%–50% of pregnant women	The frequency and severity of symptoms increase throughout gestation
Hypersecretory states (e.g., the Zollinger-Ellison syndrome)	43% of patients with the Zollinger-Ellison syndrome have endoscopic esophagitis	Patients also may have associated peptic ulceration or diarrhea
Connective tissue disorders (e.g., scleroderma)	Esophagus is involved in up to 90% of patients with scleroderma; often results in severe esophagitis and stricture formation	Characterized by low or absent LES pressure and poor esophageal motor function

GERD = gastroesophageal reflux disease; LES = lower esophageal sphincter; NSAIDs = nonsteroidal anti-inflammatory drugs.

11. Sharma N, Agrawal A, Freeman J, et al. An analysis of persistent symptoms in acid-suppressed patients undergoing impedendance-ph monitoring. Clin Gastroenterol Hepatol. 2008; 6: 521-4. [PMID: 18356117]
12. Johnston BT, Troshinsky MB, Castell JA, et al. Comparison of barium radiology with esophageal pH monitoring in the diagnosis of gastroesophageal reflux disease. Am J Gastroenterol. 1996;91: 1181-5. [PMID: 8651167]
13. O'Connor HJ. Review article: Helicobacter pylori and gastrooesophageal reflux disease-clinical implications and management. Aliment Pharmacol Ther. 1999;13:117-27. [PMID: 10102940]
14. Goldblum JR, Vicari JJ, Falk GW, et al. Inflammation and intestinal metaplasia of the gastric cardia: the role of gastroesophageal reflux and H. pylori infection. Gastroenterology. 1998;114:633-9. [PMID: 9516382]

resistance and pH, so it can be helpful to distinguish nonacid from acid reflux (11). These tests may be helpful in evaluating patients with symptoms that are atypical or refractory to empirical therapy with PPIs.

Barium radiography

Barium radiography, the most sensitive test for detecting esophageal strictures, may be useful for evaluating patients who present with dysphagia. Barium radiography has limited usefulness in most patients, however, and should not be used in routine diagnosis (1). Reflux of barium during radiographic examination is positive in only 25% to 75% of patients with known GERD and is falsely positive in up to 20% of control participants (12).

Esophageal manometry

Esophageal manometry measures muscle pressure in the lower esophagus and has a very limited role in GERD diagnosis. Use of this technique is generally limited to research protocols or to evaluate esophageal function before antireflux surgery. There are no specific manometric findings sensitive and specific for the clinical diagnosis of GERD.

Is there any connection between GERD and *Helicobacter pylori* infection?

There is controversy over the role of *Helicobacter pylori* in GERD (13). Concomitant *H. pylori* gastric infection and GERD may reduce the effects of GERD by causing gastric atrophy and decreased gastric acid production, so eradication of *H. pylori* may worsen GERD by increasing gastric acid production. Furthermore, ammonia produced by *H. pylori* infection could buffer the gastric fluid refluxing into the esophagus, an effect that would be lost after *H. pylori* eradication (14). Conversely, one prospective study demonstrated that eradication of *H. pylori* actually improved the endoscopic appearance of reflux

esophagitis in patients with duodenal ulcer (15).

In theory, patients who are receiving prolonged PPI therapy and who are also infected with *H. pylori* may be at risk for atrophic gastritis, but studies have found no evidence of accelerated development of atrophic gastritis in patients with *H. pylori* who are on long-term omeprazole (16, 17). Currently, *H. pylori* eradication in GERD patients who require long-term PPI therapy is not considered necessary to prevent the development of atrophic gastritis.

Finally, some research suggests that the type of *H. pylori* strain infecting a patient might be relevant to GERD. One study found that patients carrying cagA-positive strains of *H. pylori* may be protected against the complications of GERD, especially Barrett's esophagus and its associated dysplasia and adenocarcinoma (18).

When should clinicians consider gastroenterology consultation during the evaluation of GERD?

Consultation may be helpful when a patient does not respond to an empirical 4- to 8-week trial of acid suppression with a standard-dose PPI. It is also indicated when a patient has pulmonary or otolaryngeal symptoms, such as wheezing, shortness of breath, chronic cough or hoarseness, unexplained chest pain, globus, choking, halitosis, and sore throat, that do not respond to an empirical therapy of at least double-dose PPI for 2 to 3 months. The presence of certain warning signs also warrants further diagnostic evaluation because these symptoms may signal a complication, such as cancer, stricture, or ulceration (1).

15. Ishiki K, Mizuno M, Take S, et al. Helicobacter pylori eradication improves pre-existing reflux esophagitis in patients with duodenal ulcer disease. Clin Gastroenterol Hepatol. 2004;2:474-9. [PMID: 15181615]
16. Lundell L, Miettinen P, Myrvold HE, et al. Lack of effect of acid suppression therapy on gastric atrophy. Nordic Gerd Study Group. Gastroenterology. 1999;117:319-26. [PMID: 10419912]
17. Gillen D, Wirz AA, Neithercut WD, et al. Helicobacter pylori infection potentiates the inhibition of gastric acid secretion by omeprazole. Gut. 1999;44:468-75. [PMID: 10075952]
18. Vaezi MF, Falk GW, Peek RM, et al. CagA-positive strains of Helicobacter pylori may protect against Barrett's esophagus. Am J Gastroenterol. 2000;95:2206-11. [PMID: 11007219]

Diagnosis... Common symptoms of GERD include heartburn and regurgitation, especially when the patient is lying down. Other symptoms include dysphagia, chronic cough or hoarseness, shortness of breath or wheezing, sore throat, throat clearing, globus, and halitosis. Always consider and exclude coronary artery disease in patients with chest pain even when it is suspected to be a symptom of GERD. In most uncomplicated cases, clinicians can accurately diagnose GERD on the basis of symptoms. Relief of classic symptoms with high-dose acid suppression is sufficiently sensitive and specific to confirm the diagnosis. In patients with atypical symptoms or who are unresponsive to empirical therapy, consider alternative disease processes. Upper endoscopy is usually reserved for patients with atypical symptoms or to evaluate for Barrett's esophagus in patients with chronic GERD.

CLINICAL BOTTOM LINE

Treatment

What is the role of dietary modification in the treatment of GERD?

Dietary modifications may reduce GERD symptoms, but they have not been rigorously tested in clinical trials and their benefits are modest at best. In particular, patients may benefit from avoiding certain foods that decrease lower esophageal sphincter (LES) pressure, delay gastric emptying, or provoke reflux symptoms. Counsel patients to avoid large, fatty meals and foods and beverages that contribute to GERD symptoms (see Box).

Dietary modifications that lead to weight loss might also reduce GERD symptoms and complications. However, various studies that examined a possible link between obesity and GERD had inconclusive findings. There is some observational evidence that obesity is associated with an elevated risk for

Foods and Beverages That May Contribute to GERD Symptoms

- Chocolate
- Peppermint
- Onions
- Garlic
- Alcohol
- Carbonated beverages
- Citrus juices
- Tomato products
- Large, fatty meals

Behaviors to Decrease GERD Symptoms and Distal Acid Exposure

- Elevate the head of the bed while sleeping
- Avoid recumbency for 3 hours after meals
- Sleep in the left lateral position
- Stop smoking
- Avoid alcohol

19. Corley DA, Kubo A, Zhao W. Abdominal obesity, ethnicity and gastro-oesophageal reflux symptoms. Gut. 2007;56:756-62. [PMID: 17047097]
20. Lagergren J, Bergström R, Nyrén O. No relation between body mass and gastro-oesophageal reflux symptoms in a Swedish population based study. Gut. 2000;47:26-9. [PMID: 10861260]
21. Australian Cancer Study. Combined effects of obesity, acid reflux and smoking on the risk of adenocarcinomas of the oesophagus. Gut. 2008;57:173-80. [PMID: 17932103]
22. Lagergren J, Bergström R, Adami HO, Nyrén O. Association between medications that relax the lower esophageal sphincter and risk for esophageal adenocarcinoma. Ann Intern Med. 2000; 133:165-75. [PMID: 10906830]
23. Behar J, Sheahan DG, Biancani P, et al. Medical and surgical management of reflux esophagitis. A 38-month report of a prospective clinical trial. N Engl J Med. 1975;293:263-8. [PMID: 237234]
24. Lieberman DA. Medical therapy for chronic reflux esophagitis. Long-term follow-up. Arch Intern Med. 1987;147:1717-20. [PMID: 3116959]
25. Simon TJ, Berlin RG, Gardner AH, et al. Self-Directed Treatment of Intermittent Heartburn: A Randomized, Multicenter, Double-Blind, Placebo-Controlled Evaluation of Antacid and Low Doses of an H(2)-Receptor Antagonist (Famotidine). Am J Ther. 1995;2:304-313. [PMID: 11850668]

adenocarcinoma of the esophagus in patients with GERD.

A cross-sectional study based on data from 80110 members of the Kaiser Permanente multiphasic health check-up cohort found the presence of reflux-type symptoms in 11% of the population, with an association between obesity and an increase in GERD-like symptoms in white male patients but not in other ethnic groups (19).

A population-based study of 820 middle-aged or elderly persons in Sweden in 1995 to 1997 found no association between normal body mass index versus >25 kg/mL and GERD symptoms (odds ratio, 0.99 [95% CI, 0.66 to 1.100]) (20).

A recent Australian study compared almost 800 patients with adenocarcinoma of the esophagus with 1580 adults without cancer and found that obesity in combination with ongoing GERD symptoms increased the risk for adenocarcinoma of the lower esophagus to nearly 17 times the risk in nonobese adults without GERD symptoms (21).

Are behavioral interventions effective in the treatment of GERD?

Behavioral modifications, such as not lying down immediately after eating or elevating the head of the bed can help decrease symptoms of reflux and distal acid exposure (see Box). Although observations suggest that these lifestyle changes decrease reflux symptoms and esophageal acid exposure, the true efficacy of these maneuvers in patients has not been rigorously tested in clinical trials. Alcohol and tobacco use can also aggravate GERD and should be avoided to reduce symptoms.

Which medications cause or exacerbate GERD, and how should clinicians counsel patients regarding the use of these medications?

Certain medications may cause or exacerbate GERD by decreasing LES pressure or decreasing esophageal acid clearance (see Box) (22).

Patients with GERD-related strictures may also need to avoid pills that could lodge proximal to strictures and result in esophagitis, ulcers, and recurrent or refractory strictures. Nonsteroidal anti-inflammatory drugs, alendronate, potassium preparations, quinidine, iron supplements, and multiple antibiotics have been implicated in pill-induced esophagitis.

Which nonprescription medications are effective in the management of GERD?

The goals of drug therapy are elimination of symptoms, healing of existing esophagitis, prevention of complications, and maintenance of remission. Many patients with mild GERD have adequate relief of symptoms with antacids and over-the-counter H_2-receptor antagonists or PPIs. Two older studies that predate availability of over-the-counter H_2-receptor antagonists and PPIs in the United States suggest that effective symptom relief occurs in 20% of patients using over-the-counter agents (Table 2) (23, 24).

Antacids

Antacids are commonly used to temporarily relieve heartburn. They work within the esophageal lumen to rapidly elevate esophageal pH and neutralize esophageal acid within 15 to 30 minutes, typically producing modest relief lasting up to 90 minutes. Although inexpensive and fast-working for relief of individual heartburn episodes, drawbacks of antacids are a relatively brief duration of action and

Drugs That May Cause or Exacerbate GERD

- Theophylline
- Nitrates
- Anticholinergic agents
- Calcium-channel blockers
- α-Adrenergic antagonists
- Prostaglandins
- Sedatives

Table 2. Drug Treatment for GERD

Agent	Mechanism of Action	Benefits	Side Effects	Notes
Antacids	Buffer gastric acid	20% efficacy rate	Diarrhea, constipation	Chewable forms increase saliva, which helps neutralize acid; faster onset of action than an OTC H_2-RA
Alginic acids	Create foamy raft on surface of gastric pool	20% efficacy rate	Diarrhea, constipation	Often combined with antacid
OTC H_2-RAs Ranitidine 75 mg bid, cimetidine 200 mg bid, famotidine 10 mg bid, nizatidine 75 mg bid,	Decrease gastric acid secretion by binding to histamine receptor on parietall cell	Less efficacy than prescription doses	Similar to prescription doses	OTC doses are one half the standard prescription dose
OTC PPIs Omeprazole 20 mg bid	Block gastric acid secretion by binding to proton pump on parietal cell	80%–100% efficacy rate	Similar to prescription doses	Indicated for patients with symptoms at least 2 d/wk. May take 1–4 d before achieving full effect
Prescription H_2-RA Ranitidine 150–300 mg bid, cimetidine 400 mg bid to tid, famotidine 20–40 mg bid, nizatidibe 150–300 mg bid	Decrease gastric acid secretion by binding to histamine receptor on parietal cell	50%–60% efficacy rate	Drug interactions with cimetidine, theophylline, phenytoin, and warfarin	No difference in clinical efficacy among agents when using standard doses; much less effective when erosive esophagitis is present; indicated in mild-to-moderate GERD; full doses needed to provide effective maintenance
Prescription prokinetic agents Metoclopramide 10–20 mg 30 min qac and qhs	Increase LES pressure and improve gastric emptying	Mild symptomatic improvement without improvement in histologic, endoscopic, or pH testing	Drowsiness, tremors, depression, irritability, extrapyramidal side effects (20%–50% incidence)	High incidence of side effects and questionable efficacy limit usefulness; may provide benefit in patients with impaired gastric emptying
Prescription PPIs Omeprazole 20–40 mg qd, esomeprazole 40 mg qd, lansoprazole 30–60 mg qd, pantoprazole 40–80 mg qd, rabeprazole 20–40 mg qd	Block gastric acid secretion by binding to the proton pump in parietal cells	80%–100% efficacy rate	Long-term use associated with increase in serum gastrin, atrophic gastritis in *Helicobacter pylori*–infected patients, decreased vitamin B_{12} absorption	Indicated in moderate-to-severe GERD; should be given before meals for maximum pharmacologic effect. No substantial complications from long-term therapy reported; no clear difference in clinical efficacy among agents when standard doses are used.

bid = twice daily; GERD = gastroesophageal reflux disease; H_2-RA = histamine-2–receptor antagonist; LES = lower esophageal sphincter; OTC = over-the-counter; PPI = proton pump inhibitor; qd = once daily; qac = before every meal; qhs = every night; tid = three times daily.

inadequacy as heartburn prophylaxis. Antacids may be combined with alginic acid, which acts as a barrier on top of stomach acids, preventing contact between the acids and the esophagus and helping to prevent symptoms. Few well-designed clinical trials with antacids exist.

A study that randomly assigned 565 patients with heartburn to as-needed treatment with famotidine 10 mg, famotidine 20 mg, antacid, or placebo demonstrated that as-needed antacids up to twice daily were superior to placebo for relief of spontaneous heartburn (25).

A small study compared various antacid formulations in 20 patients with postprandial heartburn and found that chewable tablets and effervescent bicarbonate had longer durations of action than swallowed tablets (26).

26. Robinson M, Rodriguez-Stanley S, Miner PB, et al. Effects of antacid formulation on postprandial oesophageal acidity in patients with a history of episodic heartburn. Aliment Pharmacol Ther. 2002;16:435-43. [PMID: 11876696]

27. Euler AR, Murdock RH Jr, Wilson TH, et al. Ranitidine is effective therapy for erosive esophagitis. Am J Gastroenterol. 1993;88:520-4. [PMID: 8470632]
28. Kahrilas PJ, Fennerty MB, Joelsson B. High- versus standard-dose ranitidine for control of heartburn in poorly responsive acid reflux disease: a prospective, controlled trial. Am J Gastroenterol. 1999;94:92-7. [PMID: 9934737]
29. Inadomi JM, Jamal R, Murata GH, et al. Step-down management of gastroesophageal reflux disease. Gastroenterology. 2001;121:1095-100. [PMID: 11677201]
30. Dean BB, Gano AD Jr, Knight K, et al. Effectiveness of proton pump inhibitors in nonerosive reflux disease. Clin Gastroenterol Hepatol. 2004;2:656-64. [PMID: 15290657]
31. Lind T, Havelund T, Carlsson R, et al. Heartburn without oesophagitis: efficacy of omeprazole therapy and features determining therapeutic response. Scand J Gastroenterol. 1997;32:974-9. [PMID: 9361168]
32. Richter JE, Peura D, Benjamin SB, et al. Efficacy of omeprazole for the treatment of symptomatic acid reflux disease without esophagitis. Arch Intern Med. 2000;160:1810-6. [PMID: 10871975]
33. Gardner JD, Ciociola AA, Robinson M, et al. Determination of the time of onset of action of ranitidine and famotidine on intra-gastric acidity. Aliment Pharmacol Ther. 2002;16:1317-26. [PMID: 12144582]

H_2-receptor antagonists

H_2-receptor antagonists, which bind to H_2 receptors on gastric parietal cells to reduce gastric acid secretion, are a first-line therapy for uncomplicated GERD with mild or intermittent symptoms. They start reducing gastric acid within 1 to 2 hours of dosing, and effects last up to 9 hours. Drawbacks of H_2-receptor antagonists are the delay in effect and the fact that tolerance may develop. Given in a standard dose, H_2-receptor antagonists provide adequate symptom relief in 50% to 60% of patients with mild-to-moderate GERD and heal endoscopic esophagitis in 48% (1).

One study randomly assigned 328 patients with erosive esophagitis to either ranitidine 300 mg 4 times daily, ranitidine 150 mg 4 times daily, or placebo for up to 12 weeks. Symptom relief and healing of esophagitis was better in both ranitidine groups than with placebo (27).

Another trial in 481 patients found no difference in efficacy between ranitidine 150 mg and 300 mg twice daily in relief of heartburn symptom. This study also found that 59% of patients still had some symptoms after 6 weeks of ranitidine therapy (28).

Proton pump inhibitors

PPIs, which block gastric acid secretion by binding to the proton pump in parietal cells, are advised for patients with GERD symptoms at least twice a week. Typical first-line therapy is a 14-day course of over-the-counter omeprazole, the only PPI with U.S. Food and Drug Administration (FDA) approval for over-the-counter use. PPIs are more effective than H_2-receptor antagonists for acute treatment of severe or erosive esophagitis. H_2-receptor antagonists are ineffective for long-term maintenance of these conditions. PPIs may take up to 4 days to relieve symptoms, but patients do not seem to develop tolerance to PPIs as they can with H_2-receptor antagonists.

When should clinicians consider prescription medications, and which medications are available?

Various PPIs and H_2-receptor antagonists are available by prescription, and some are available over the counter (Table 2). There is debate about whether initial treatment should use a step-down or a step-up approach (Figure 1). The step-down approach involves starting with once- or twice-daily PPI therapy and decreasing to the least potent acid-suppression therapy that controls symptoms. The step-up approach involves initiating therapy with standard or even nonprescription doses of an H_2-receptor antagonist and titrating up to the most potent acid-suppression therapy that controls symptoms. Efficacy studies and cost-effectiveness models have not shown superiority of either approach.

One study involving patients on long-term PPI therapy found that more than one half were able to step down from PPI therapy without increasing symptoms or limiting quality of life. Forty-one of 71 (58%) were asymptomatic 1 year after going off PPI therapy. Twenty-four of 71 (34%) required H_2-receptor antagonists, 5 of 71 (7%) required prokinetic agents, 1 of 71 (1%) required both, and 11 of 71 (15%) remained asymptomatic without medication (29).

Symptomatic medical treatment of reflux esophagitis has improved dramatically since PPIs became available in 1989. PPIs provide rapid symptomatic relief and healing of esophagitis in the highest percentage of patients.

A systematic review of 7 trials that evaluated PPIs in patients with nonerosive reflux disease found that the therapeutic gain of PPIs over placebo for sufficient heartburn control was 30% to 35% (30).

A trial in 509 patients with no esophagitis on endoscopy compared omeprazole 20 mg daily, omeprazole 10 mg daily, and placebo. At 4 weeks, the proportion of patients with complete resolution of heartburn in each group was 46%, 31%, and 13%, respectively (31).

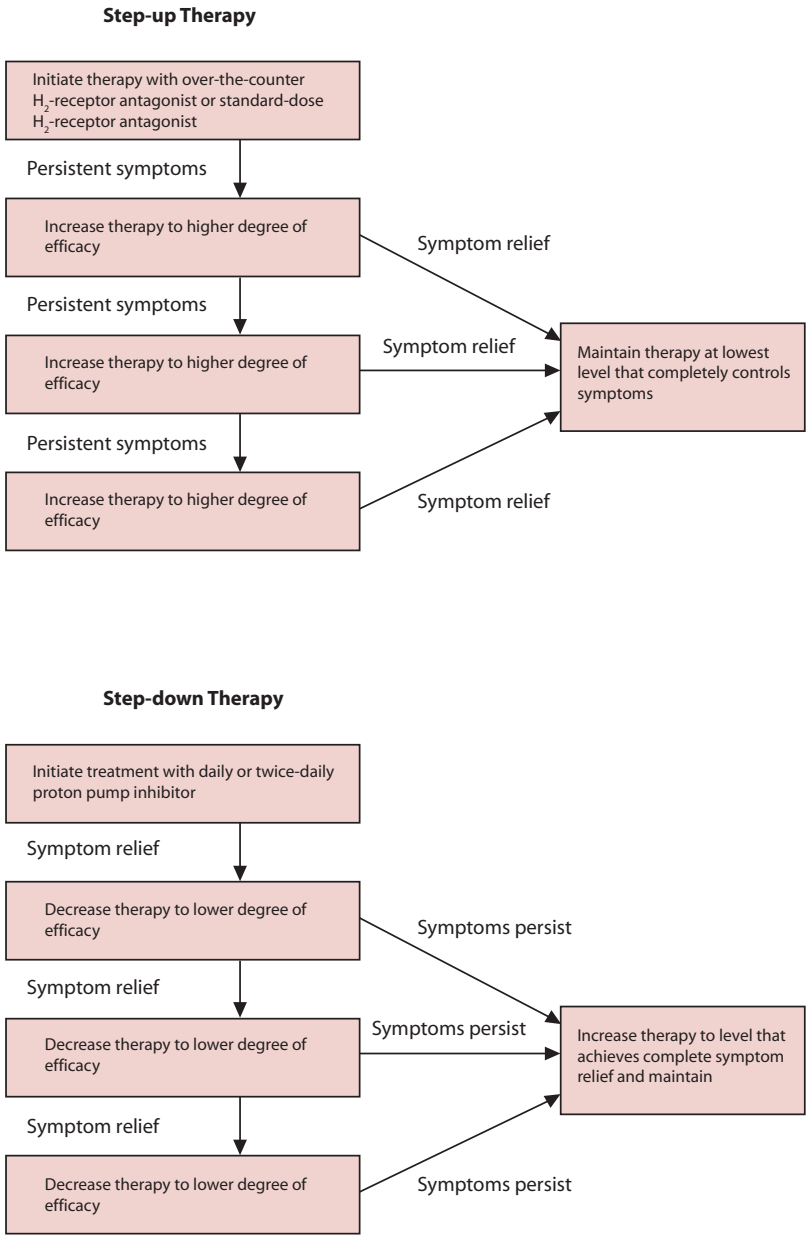

Figure 1. Step-up vs. step-down drug therapy for gastroesophageal relux disease.

A randomized trial in 355 patients with GERD symptoms found that omeprazole 20 mg daily provided superior relief compared with omeprazole 10 mg daily and with placebo (32).

How should clinicians select from among available antireflux medications?

In general, the various H_2-receptor antagonists are equally efficacious in equipotent doses and carry similar adverse effect profiles. However, a few comparative studies of nonprescription H_2-receptor antagonists have indicated that famotidine and ranitidine may have higher potency than cimetidine, and cimetidine and ranitidine may have faster onset of effect on gastric pH than famotidine (33). Research generally also indicates that the various PPIs are equally efficacious in equipotent doses and carry similar adverse effect profiles. For patients with the uncommon form of GERD with

34. Castell DO, Kahrilas PJ, Richter JE, et al. Esomeprazole (40 mg) compared with lansoprazole (30 mg) in the treatment of erosive esophagitis. Am J Gastroenterol. 2002;97:575-83. [PMID: 11922549]
35. Schindlbeck NE, Klauser AG, Berghammer G, et al. Three year follow up of patients with gastrooesophageal reflux disease. Gut. 1992;33:1016-9. [PMID: 1356887]
36. Hetzel DJ, Dent J, Reed WD, et al. Healing and relapse of severe peptic esophagitis after treatment with omeprazole. Gastroenterology. 1988;95:903-12. [PMID: 3044912]
37. Carlsson R, Dent J, Watts R, et al. Gastro-oesophageal reflux disease in primary care: an international study of different treatment strategies with omeprazole. International GORD Study Group. Eur J Gastroenterol Hepatol. 1998;10:119-24. [PMID: 9581986]
38. Vigneri S, Termini R, Leandro G, et al. A comparison of five maintenance therapies for reflux esophagitis. N Engl J Med. 1995;333:1106-10. [PMID: 7565948]
39. Yang YX, Hennessy S, Propert K, et al. Chronic proton pump inhibitor therapy and the risk of colorectal cancer. Gastroenterology. 2007;133:748-54. [PMID: 17678926]
40. Robertson DJ, Larsson H, Friis S, et al. Proton pump inhibitor use and risk of colorectal cancer: a population-based, case-control study. Gastroenterology. 2007;133:755-60. [PMID: 17678921]
41. Yang YX, Lewis JD, Epstein S, Metz DC. Long-term proton pump inhibitor therapy and risk of hip fracture. JAMA. 2006;296:2947-53. [PMID: 17190895]

severe esophagitis, however, esomeprazole may be somewhat more effective than other PPIs (34).

When ordering GERD medications for a patient, clinicians should choose the least-expensive product that is effective for managing symptoms and preventing complications and should prescribe the lowest effective dose for the minimum duration needed.

How long should patients continue pharmacologic therapy for GERD?

Reflux symptoms disappear in only a minority of patients (35), but about 20% of patients with GERD have adequate symptom control with intermittent, nonprescription therapy and lifestyle modification. Although many patients need to remain on long-term GERD therapy to control symptoms, others may be able to reduce dosage or cease treatment once symptoms are controlled and the esophagus has healed. Clinicians may periodically consider trying step-down therapy to a lower-dose PPI or switching from a PPI to an H_2-receptor antagonist.

About 50% to 80% of patients with esophagitis have recurrence after 6 to 12 months of follow-up, regardless of the agent used to achieve healing or symptom control (36, 37). Patients with severe GERD need long-term PPI maintenance therapy to control symptoms and prevent complications. Standard or even high doses of H_2-receptor antagonists are not generally appropriate maintenance therapy for severe GERD (38).

What are the adverse effects of long-term acid-suppression therapy?

Short-term adverse effects with PPIs are uncommon and typically limited to headaches, nausea, constipation, diarrhea, and pruritus. Yet despite evidence from careful follow-up studies, there is ongoing worry that long-term use might cause other adverse effects.

There has been concern about the potential for PPIs to increase the risk for colorectal cancer because the drugs elevate serum gastrin levels, and in vitro studies show that high gastrin levels are associated with increased growth and proliferation of colon cancer cells. However, two reports that examined the potential link between PPIs and increased colorectal cancer risk found no statistically significant overall association between long-term PPI use and colorectal cancer (39, 40).

Research has found possible associations between long-term use of PPIs and bone health, risk for gastroenteritis and other infection, and vitamin B_{12} deficiency.

In an observational study, more than 1 year of PPI therapy was associated with a 44% increased risk for hip fracture among people older than 50 years. The strength of the association with hip fractures increased with both the dosage and the duration of PPI therapy (41).

A nested case–control study performed in 364683 patients on acid-suppressive drugs found higher rates of community-acquired pneumonia among these patients than among those who did not use this type of therapy (2.45 compared with 0.6 per 100 person-years) (42).

An observational study found that current use of PPIs, but not use of H_2-receptor antagonists, was associated with an increased risk for bacterial gastroenteritis (RR, 2.9 [CI, 2.5 to 3.5]) (43).

Another observational study found that the adjusted rate ratio of Clostridium difficile*–associated disease with current use of PPIs was 2.9 (CI, 2.4 to 3.4) and with use of H_2-receptor antagonists was 2.0 (CI, 1.6 to 2.7) (44).*

A study that investigated whether long-term treatment with omeprazole or H_2-receptor antagonists alters vitamin B_{12} levels in patients with the Zollinger–Ellison

syndrome found that B_{12} levels, but not serum folate levels, were substantially lower in patients treated with omeprazole, suggesting that serum vitamin B_{12} levels should be monitored in patients with the Zollinger–Ellison syndrome treated with PPIs (45).

When should clinicians consider surgical therapy for GERD?

Nissen fundoplication is the most common surgical intervention for GERD. This procedure aims to restore the physiology and anatomy of the gastroesophageal junction by wrapping the gastric fundus around the distal esophagus. The FDA has also approved several endoscopic procedures for treatment of GERD, including endoscopic suturing and radiofrequency ablation of the lower esophageal sphincter.

Antireflux surgery is an option for patients who have responded well to PPI therapy but who are concerned about the costs and other consequences of taking daily medication on a long-term basis. Preoperative evaluation before surgery should include documentation of GERD with pH monitoring and esophageal manometry. Patients who have not responded to medical therapy may have symptoms not caused by GERD.

Although surgical therapy is efficacious, a review comparing the efficacy, prevention of complications, safety profile, convenience, and costs of medical or surgical fundoplication therapy for GERD suggested that antireflux surgery had no clear advantage compared with medical therapy, and that medical therapy may be safer and more cost-effective (46). Fundoplication reduces costs associated with PPI use in the short term, but it does not reduce total costs because many patients subsequently return to long-term use of PPIs (47). In a follow-up study conducted 11 to 13 years after antireflux surgery, approximately 60% of the patients were again receiving medical therapy (48).

Postsurgical side effects, such as bloating, flatulence, diarrhea, and dysphagia, may be long-lasting. More serious complications, including esophageal perforation and death, have been reported. Laparoscopic antireflux surgery seems to be equal in effectiveness to open surgery, with greatly decreased morbidity.

Is it necessary to evaluate for Barrett's esophagus periodically?

Barrett's esophagus is premalignant intestinal metaplasia of the mucosa of the lower esophagus that occurs in response to chronic exposure to acidic stomach contents. Barrett's esophagus significantly increases the risk for esophageal adenocarcinoma. The risk for adenocarcinoma from Barrett's esophagus is 30 to 40 times that of the general population, or approximately 0.5% to 1.0% per year (49).

Barrett's esophagus is detected in 8% to 20% of patients with chronic GERD. White race, male gender, chronic duration of reflux symptoms, and positive family history are risk factors for Barrett's esophagus. Older age; white race; male gender; obesity; smoking; use of LES-relaxing drugs; increased frequency, greater severity, and longer duration of reflux symptoms; hiatal hernia; and duration of Barrett's esophagus are risk factors for esophageal adenocarcinoma in a patient with known Barrett's esophagus. Clinical severity of symptoms

Consider surgery as an option for patients with well-documented GERD who require long-term PPI maintenance therapy but show satisfactory relief of symptoms and who:

- Are older than 50 years
- Consider long-term medication a financial burden
- Are noncompliant with drug therapy
- Prefer a single surgical intervention to long-term drug treatment
- Experience prominent symptoms of regurgitation, even with medical control of heartburn symptoms

42. Laheij RJ, Sturkenboom MC, Hassing RJ, et al. Risk of community-acquired pneumonia and use of gastric acid-suppressive drugs. JAMA. 2004;292: 1955-60. [PMID: 15507580]
43. García Rodríguez LA, Ruigómez A, Panés J. Use of acid-suppressing drugs and the risk of bacterial gastroenteritis. Clin Gastroenterol Hepatol. 2007;5:1418-23. [PMID: 18054750]
44. Dial S, Delaney JA, Barkun AN, Suissa S. Use of gastric acid-suppressive agents and the risk of community-acquired Clostridium difficile-associated disease. JAMA. 2005;294: 2989-95. [PMID: 16414946]
45. Termanini B, Gibril F, Sutliff VE, et al. Effect of long-term gastric acid suppressive therapy on serum vitamin B12 levels in patients with Zollinger-Ellison syndrome. Am J Med. 1998;104:422-30. [PMID: 9626024]
46. Spechler SJ. Medical or invasive therapy for GERD: an acidulous analysis. Clin Gastroenterol Hepatol. 2003;1:81-8. [PMID: 15017499]
47. Dire CA, Jones MP, Rulyak SJ, Kahrilas PJ. The economics of laparoscopic Nissen fundoplication. Clin Gastroenterol Hepatol. 2003;1:328-32. [PMID: 15017676]
48. Spechler SJ, Lee E, Ahnen D, et al. Long-term outcome of medical and surgical therapies for gastroesophageal reflux disease: follow-up of a randomized controlled trial. JAMA. 2001;285: 2331-8. [PMID: 11343480]
49. Hogan WJ. Spectrum of supraesophageal complications of gastroesophageal reflux disease. Am J Med. 1997;103:77S-83S. [PMID: 9422629]

50. Lieberman DA, Oehlke M, Helfand M. Risk factors for Barrett's esophagus in community-based practice. GORGE consortium. Gastroenterology Outcomes Research Group in Endoscopy. Am J Gastroenterol. 1997;92:1293-7. [PMID: 9260792]
51. Streitz JM Jr, Andrews CW Jr, Ellis FH Jr. Endoscopic surveillance of Barrett's esophagus. Does it help? J Thorac Cardiovasc Surg. 1993;105:383-7; discussion 387-8. [PMID: 8445916]
52. Peters JH, Clark GW, Ireland AP, et al. Outcome of adenocarcinoma arising in Barrett's esophagus in endoscopically surveyed and nonsurveyed patients. J Thorac Cardiovasc Surg. 1994;108:813-21; discussion 821-2. [PMID: 7967662]

alone is unreliable in distinguishing patients with Barrett's esophagus from those with GERD alone.

One study of 2641 patients undergoing endoscopy found that the risk for Barrett's esophagus in patients with symptoms lasting more than 5 years was 5 times that of patients with symptoms of less than 1 year (50).

Although strong evidence is not available to support a screening recommendation or to define the appropriate timing and interval of screening, consensus is that upper endoscopy should be done in patients with chronic GERD to screen for Barrett's esophagus, dysplastic changes, and early esophageal cancer. At least two studies suggest that endoscopic surveillance of patients with Barrett's esophagus detects carcinoma at an early stage and can improve long-term survival rates (51, 52).

However, the American College of Gastroenterology practice guidelines notes the lack of clear evidence that screening reduces esophageal adenocarcinoma mortality and states that screening in high-risk patients should be individualized (53). The guidelines note that the yield of screening is highest in white men older than 50 years with longstanding heartburn but do not define the specific duration of symptoms after which screening is indicated. Most experts suggest that patients with chronic GERD have endoscopy at least once during their lifetime to screen for Barrett's esophagus, regardless of whether symptoms are controlled. A case-control study found that GERD symptoms lasting longer than 13 years were associated with Barrett's esophagus (54).

How should clinicians manage patients once Barrett's esophagus is present?

Once Barrett's esophagus has been detected, surveillance endoscopy with biopsy should be performed at

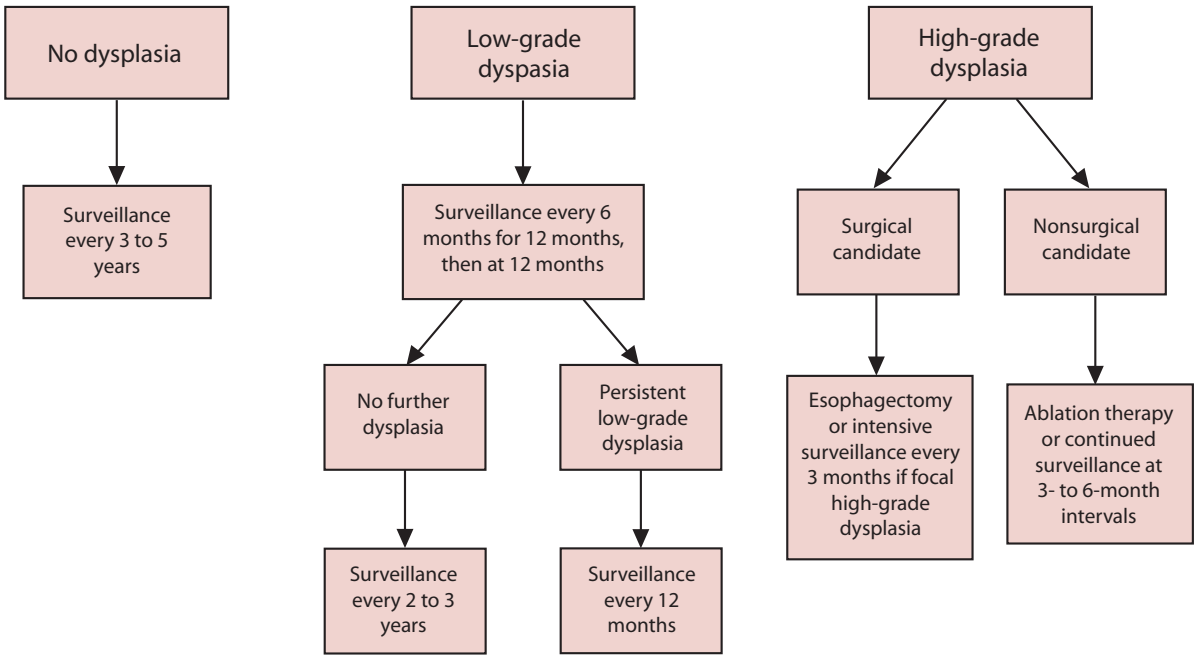

Figure 2. Proposed surveillance and management algorithm for patients with Barrett's esophagus based on grade of dysplasia detected by endoscopic biopsy.

least every 3 years (depending on the grade of dysplasia) to detect neoplastic transformation (53). Because active inflammation can be misinterpreted as dysplasia, mucosal healing should be achieved before biopsies are obtained. Diagnosis of high-grade dysplasia requires repeated endoscopy to exclude concomitant cancer (Figure 2).

Among patients with Barrett's esophagus, acid suppression is especially important because it may play a role in retarding progression of dysplasia (55). Barrett's esophagus alone is not an indication for surgical therapy for GERD (56).

How frequently should clinicians see patients with GERD and what are the components of good follow-up?

GERD is a chronic condition that usually requires ongoing follow-up and maintenance therapy to prevent complications. Clinicians should monitor for symptoms that suggest complications of cancer, stricture, or ulceration; screen for Barrett's esophagus when appropriate; and ensure that medical therapy controls symptoms in the most cost-effective manner.

When should clinicians consider gastroenterology referral for the treatment of a patient with GERD?

Consider consultation with a specialist if patients are refractory to therapy or if atypical symptoms or complications develop. Because most patients' symptoms are controlled with PPI therapy, symptoms that do not respond to PPI therapy may not be caused by GERD. Referral is also advised when evaluating for Barrett's esophagus or for possible surgical intervention for GERD.

53. Wang KK, Sampliner RE. Updated guidelines 2008 for the diagnosis, surveillance, and therapy of Barrett's esophagus. Am J Gastroenterol. 2002; 103: 788-797. [PMID: 12190150]
54. Conio M, Filiberti R, Blanchi S, et al. Risk factors for Barrett's esophagus: a case-control study. Int J Cancer 2002; 97: 225-229.
55. El-Serag HB, Aguirre T, Kuebeler M, Sampliner RE. The length of newly diagnosed Barrett's oesophagus and prior use of acid suppressive therapy. Aliment Pharmacol Ther. 2004;19:1255-60. [PMID: 15191506]
56. Csendes A, Braghetto I, Korn O, Cortés C. Late subjective and objective evaluations of antireflux surgery in patients with reflux esophagitis: analysis of 215 patients. Surgery. 1989;105: 374-82. [PMID: 2784232]

Treatment... Dietary and behavioral modifications may be effective in treatment of GERD. Many patients with mild GERD have adequate relief of symptoms with antacids and over-the-counter H_2-receptor antagonists and PPIs. Prescription medications, particularly PPIs, are indicated for moderate-to-severe GERD. There is ongoing debate about whether initial treatment should use a step-down or a step-up approach. Many patients with moderate-to-severe GERD require indefinite maintenance therapy to control symptoms and prevent complications. There is no clear evidence of serious adverse effects from long-term PPI use. Consider antireflux surgery in patients who have responded well to PPI therapy and who are not interested in long-term medical therapy. Clinicians should provide follow-up to monitor for complications and to ensure that medical maintenance therapy controls symptoms in the most cost-effective manner. Patients with chronic GERD should have endoscopy at least once to screen for Barrett's esophagus.

CLINICAL BOTTOM LINE

Practice Improvement

How do U.S. stakeholders evaluate the quality of care for patients with GERD?

The Center for Medicare & Medicaid Services (CMS) has developed 119 measures of quality of care to use in the 2008 Physician Quality Reporting Initiative (PQRI), an initiative that will financially reward participating physicians

who meet defined quality standards. Of these measures, one involves GERD (see Box). The rationale for this measure is that many patients with GERD remain on medication for years, and experts suspect that not all patients have regular reassessment to determine whether medication is still needed. Research indicates that patients on long-term GERD therapy may be able to have their medications modified on the basis of the presence or absence of symptoms.

What do professional organizations recommend regarding the management of patients with GERD?

In 2005, the American College of Gastroenterology published updated guidelines on diagnosis and treatment of GERD (1). The guidance in this article generally reflects the recommendations in those guidelines. Other GERD treatment guidelines include a 2005 Canadian Association of Gastroenterology consensus conference on the management of GERD in adults (57); a 2006 American Gastrointestinal Association Institute Medical Position Statement on the use of endoscopic therapy for GERD (58); 2007 American College of Gastroenterology practice guidelines on esophageal reflux testing (59); 2001 Society of American Gastrointestinal and Endoscopic Surgeons consensus guidelines on the surgical treatment of GERD (60); and 2008 American College of Gastroenterology guidelines on the diagnosis, surveillance, and management of Barrett's esophagus (53).

Centers for Medicare & Medicaid Services: 2008 Physician Quality Reporting Initiative

Measure #77: Assessment of GERD Symptoms in Patients Receiving Chronic Medication for GERD

Description: Percentage of patients ≥18 years with the diagnosis of GERD who have been prescribed continuous PPI or H_2-receptor antagonist therapy who received an annual assessment of their GERD symptoms after 12 months of therapy.

Numerator: Patients who had an annual assessment of their GERD symptoms after 12 months of therapy.

Denominator: All patients ≥18 years with a diagnosis of GERD who have been prescribed ≥12 months of continuous PPI or H_2-receptor antagonist therapy.

57. Canadian Association of Gastroenterology GERD Consensus Group. Canadian Consensus Conference on the management of gastroesophageal reflux disease in adults - update 2004. Can J Gastroenterol. 2005;19:15-35. [PMID: 15685294]
58. Falk GW, Fennerty MB, Rothstein RI. AGA Institute medical position statement on the use of endoscopic therapy for gastroesophageal reflux disease. Gastroenterology. 2006;131: 1313-4. [PMID: 17030198]
59. Practice Parameters Committee of the American College of Gastroenterology. ACG practice guidelines: esophageal reflux testing. Am J Gastroenterol. 2007;102:668-85. [PMID: 17335450]
60. Society of American Gastrointestinal and Endoscopic Surgeons. Guidelines for Surgical Treatment of Gastroesophageal Reflux Disease (GERD). 2001. Accessed at www.sages.org/publications/publication.php?id=22 on 10 June 2008.

in the clinic

Tool Kit

Gastroesophageal Reflux Disease

PIER Modules

pier.acponline.org

Access the following PIER modules: GERD, Barrett's Esophagus, and Upper Gastrointestinal Endoscopy. PIER modules provide evidence-based, updated information on current diagnosis, treatment, and management, in an electronic format designed for rapid access at the point of care.

Patient Education Resources

www.annals.org/intheclinic/

Access the Patient Information material that appears on the following page for duplication and distribution to patients.

www.acponline.org/patients_families/pdfs/health/heartburn_report.pdf

Access American College of Physicians: ACP Special Report: Understanding and Treating Heartburn

Quality Improvement Tools

pier.acponline.org/qualitym/t004.html

Access the CMS PQRI quality measure for GERD with administrative criteria and background material.

Practice Guidelines

http://www.acg.gi.org/physicians/clinicalupdates.asp#guidelines

Access American College of Gastroenterology practice guidelines

WHAT YOU SHOULD KNOW ABOUT GASTROESOPAHAGEAL REFLUX DISEASE (GERD)

In the Clinic
Annals of Internal Medicine
annals.org

In gastroesophageal reflux disease (GERD), stomach acid washes up into the esophagus. The esophagus is the tube that carries food from the mouth to the stomach. GERD can harm the lining of the esophagus and cause what many people call "heartburn" or "acid indigestion." Some people with GERD may also have a cough, a sore throat, breathing problems, trouble swallowing, or bad breath.

Things that can cause GERD or make it worse:

- Pregnancy
- Smoking
- Alcohol
- Being overweight
- Some foods (fatty or fried foods, chocolate, mint, garlic, onions, citrus fruits or juices, carbonated beverages)
- Lying down after eating

How will the doctor know if problems are caused by GERD?

- Your doctor may give you medicine to make you have less stomach acid. If the medicine helps, your problems were probably from GERD and you probably won't need any tests.
- Sometimes you may need a test to measure acid or pressure or to look at the esophagus lining.

Is there a treatment?

- GERD can be treated by stopping the things that make it worse.
- Taking medicines that block stomach acid can also help.

Is GERD dangerous?

- If GERD is not treated, it can cause bleeding or scars that block the esophagus.
- GERD may make changes in the lining of the esophagus called "Barrett's esophagus." Barrett's esophagus can turn into cancer.
- People who have GERD for many years should get checked for Barrett's esophagus.
- Tell your doctor if you have trouble swallowing, weight loss, vomiting, bleeding, loss of appetite, or chest pain.
- In a few cases, an operation may be needed.

For More Information

Web Sites with Good Information on GERD

American College of Physicians: ACP Special Report: Understanding and Treating Heartburn
http://www.acponline.org/patients_families/pdfs/health/heartburn_report.pdf

National Digestive Diseases Information Clearinghouse: Heartburn, Hiatal Hernia, and GERD
http://digestive.niddk.nih.gov/ddiseases/pubs/gerd/index.htm

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Patient Information

CME Questions

MKSAP
MEDICAL KNOWLEDGE Self-Assessment Program

1. A 56-year-old woman is evaluated because of continuing symptoms due to refractory gastroesophageal reflux that have not improved despite lifestyle modifications and treatment with a twice-daily proton pump inhibitor (omeprazole 40 mg). The patient continues to have occasional substernal chest pain associated with some epigastric burning. She has not had dysphagia, regurgitation, weight loss, or a change in bowel habits. She has no cardiac risk factors.

   Physical examination is normal except for slight overweight. Upper endoscopy is also normal.

   Which of the following is the most appropriate treatment at this time?

   A. Schedule consultation for evaluation for antireflux surgery
   B. Increase the proton pump inhibitor to 3 times daily
   C. Change to a different proton pump inhibitor
   D. Add trazodone to the current regimen
   E. Add ranitidine at bedtime to the current regimen

2. An otherwise-healthy 28-year-old man has a 4-month history of epigastric discomfort and heartburn. Symptoms are usually exacerbated postprandially, especially after eating spicy foods. The patient does not report dysphagia, weight loss, or decreased appetite. He has an active lifestyle and takes no medications. Physical examination is normal except for mild epigastric tenderness. Routine laboratory studies are normal.

   Which of the following is most appropriate at this time?

   A. Upper endoscopy
   B. Esophageal manometry
   C. Ambulatory 24-hour esophageal pH monitoring
   D. Barium swallow
   E. Trial of acid-suppression therapy

3. A 32-year-old woman has a 4-month history of hoarseness and throat clearing. Evaluation by an otolaryngologist disclosed laryngeal inflammation suggestive of gastroesophageal reflux disease, and the patient is referred to you. She is otherwise asymptomatic and does not report heartburn, regurgitation, dysphagia, or weight loss. The patient maintains an active lifestyle and currently takes no medications. Physical examination and routine laboratory studies are normal.

   Which of the following should be done next?

   A. Upper endoscopy
   B. Esophageal manometry
   C. Ambulatory 24-hour esophageal pH monitoring
   D. Barium swallow
   E. Trial of acid-suppression therapy

4. An obese 62-year-old man is evaluated because of heartburn and frequent throat clearing. Gastroesophageal reflux disease (GERD) is diagnosed and therapy with a proton pump inhibitor (omeprazole 40 mg), twice daily, is initiated. He is advised of lifestyle modifications that help prevent GERD.

   After 6 weeks, he is reevaluated because the cough, although somewhat better, persists. He has no postnasal drip. His heartburn has resolved.

   Which of the following is the best next step in this patient's management?

   A. Change his medication to intranasal corticosteroids and antihistamines
   B. Order 24-hour esophageal pH monitoring
   C. Refer him for fundoplication
   D. Continue the proton pump inhibitor therapy and reevaluate him in 6 weeks

5. An 84-year-old woman has a 2-day history of severe substernal chest pain when swallowing. She does not have dysphagia. Two days ago, the patient began taking alendronate for osteoporosis. Other medications are a daily aspirin and an angiotensin-converting enzyme inhibitor. She has a remote history of gastroesophageal reflux disease. Physical examination is normal.

   Which of the following is the most appropriate management at this time?

   A. Schedule upper endoscopy
   B. Schedule a barium swallow
   C. Discontinue alendronate
   D. Begin a proton pump inhibitor
   E. Begin metoclopramide

Questions are largely from the ACP's Medical Knowledge Self-Assessment Program (MKSAP). Go to www.annals.org/intheclinic/ to obtain up to 1.5 CME credits, to view explanations for correct answers, or to purchase the complete MKSAP program.