Acid Diseases of the Esophagus and Stomach

1. Which of the following would be expected to be associated with a decrease in gastric acid secretion?

A. Chronic gastric outlet obstruction
B. Systemic mastocytosis
C. Misoprostol
D. Ingestion of proteins
E. Increased intracranial pressure

The recommended response is C.

In the physiology of gastric acid secretion, parietal cell function is stimulated by histamine, acetylcholine, and gastrin and is inhibited by prostaglandins and somatostatin. Acetylcholine is released from nerve endings as a result of vagal nerve stimulation. In conditions of increased intracranial pressure, there is increased vagal activity. Systemic mastocytosis is associated with increased systemic release of histamine and is associated with gastric acid hypersecretion. Misoprostol is a synthetic, prostaglandin PGE2 analog. Gastric acid secretion is stimulated by the presence of food in the stomach, especially proteins. Gastric distention, which can occur with chronic gastric outlet obstructions, stimulates modest levels of gastric acid secretion through gastrin release via neural reflex pathways.

2. Which of the following conditions is most likely to be associated with decreased serum concentrations of Vitamin B₁₂?

A. Pancreatic exocrine insufficiency  
B. A 10-day course of oral antibiotics  
C. Excess secretion of intrinsic factor  
D. Stimulation of parietal cell function  
E. Multiple endocrine neoplasia type-I (MEN-I) syndrome

The recommended response is A.

Intrinsic factor, a glycoprotein whose primary role is the facilitation of cobalamin (Vitamin B₁₂) absorption, is secreted by the parietal cell under the same stimulatory conditions as is hydrochloric acid. Cobalamin, when liberated from food (protein) by acid and pepsin, initially combines preferentially with R proteins present in saliva. In the alkaline environment of the duodenum where R proteins are hydrolyzed by pancreatic enzymes, cobalamin preferentially binds to intrinsic factor. Failure to absorb cobalamin can occur with intrinsic factor deficiency, pancreatic exocrine insufficiency, small bowel bacterial overgrowth (usually in the presence of achlorhydria), or ileal disease. MEN-I is an autosomal dominant condition that includes hyperparathyroidism, gastrinoma (ZE syndrome) or other islet cell tumors, and pituitary tumors. Thus, the increased parietal cell secretion in ZE syndrome would be expected to be associated with increased intrinsic factor secretion.


Wilcox CM, Alexander LN, Clark WS. Localization of an obstructing esophageal lesion. Is the
3. Mucus protects the gastroduodenal mucosal surface through all of the following mechanisms except:

A. Trapping of bicarbonate ($\text{HCO}_3^-$) in the unstirred layer
B. Increasing gastroduodenal mucosal blood flow
C. Preventing pepsin from injuring epithelial cells
D. Containing protective chemicals such as lipids and trefoil peptides
E. Neutralizing hydrogen ion with negatively charged glycoproteins

The recommended response is B.

Mucus protects the gastroduodenal surface by (1) trapping $\text{HCO}_3^-$ in the unstirred layer, (2) preventing pepsin and gastric lipase from reaching and attacking the lining epithelial cells, (3) lubricating the gastric lining to prevent abrasions from coarse food particles, (4) neutralizing $\text{H}^+$ with negatively charged glycoproteins and peptides of mucus, and (5) trapping ingested bacteria. Mucus also contains protective chemicals that prevent injury to the epithelium, such as lipids (in which $\text{H}^+$ and pepsin are insoluble) and trefoil peptides (which maintain mucosal integrity). Mucus does not affect gastroduodenal mucosal blood flow.


4. Which of the following infectious agents is the least likely cause of gastric ulceration?
A. Cryptosporidium
B. *Helicobacter pylori*
C. Cytomegalovirus
D. *Treponema pallidum*
E. Herpes simplex virus-type 1

The recommended response is A.

Infectious causes of ulcer disease include viral etiologies such as herpes simplex-type 1 and cytomegalovirus (CMV). These should be suspected in immunocompromised or post-transplant patients. However, CMV-induced ulcers have also been observed in immunocompetent patients. The proof that syphilis causes gastric ulcers comes from the demonstration of the organism in the gastric mucosa, appropriate serology, and evidence that lesions regress after antibiotic therapy. Gastric syphilis is caused by *Treponema pallidum*, an organism that can be viewed in gastric biopsies by silver staining and by fluorescent antibody techniques. *Helicobacter pylori* is the most common cause of gastric and duodenal ulcers.


5. A 64-year-old patient with dyspepsia had an upper endoscopy that revealed a gastric ulcer. After
histologic assessment of endoscopic biopsies, adenocarcinoma and H. pylori infection were diagnosed. Endoscopic ultrasound indicated that the gastric adenocarcinoma was limited to the mucosa but regional lymph nodes were suspicious for malignancy. Abdominal CT scanning confirmed suspicious lymph nodes adjacent to the stomach, but there was no evidence of malignancy in other areas of the abdomen. Which of the following is the most appropriate initial treatment?

A. Subtotal gastrectomy
B. Proton pump inhibitor, amoxicillin, and clarithromycin for 10 days
C. Proton pump inhibitor, metronidazole, clarithromycin, and tetracycline for 14 days
D. Radiation therapy
E. Chemotherapy

The recommended response is A.

The prognosis of gastric adenocarcinoma is primarily related to depth of tumor penetration through the gastric wall, irrespective of the extent of nodal involvement. Early gastric cancer is limited to the gastric mucosa or submucosa, as is seen in this case. Advanced gastric cancer extends into the muscular layers of the stomach and has a worse prognosis. The best curative therapy for early gastric cancer is partial or subtotal gastric resection. Chemotherapy and radiation therapy are much less effective than surgical resection. H. pylori therapies are not of benefit in reversing malignancy in patients with established gastric cancer. However, in early gastric cancer patients who have undergone partial gastric resections, H. pylori therapies may reduce the frequency of subsequent cancers. Further, in patients with MALToma, eradication of H. pylori leads to regression in approximately 80% of cases.


6. Besides an octreotide scan, which test has the greatest sensitivity in localizing a gastrinoma?

A. PET scan  
B. Transabdominal ultrasonography  
C. Endoscopic ultrasonography (EUS)  
D. Magnetic resonance imaging (MRI)  
E. CT scan

The recommended response is C.

Gastrinomas are difficult to localize, primarily due to their small size and frequent presentation in multiple locations. The sensitivities of various diagnostic modalities are shown in Table 6. The most sensitive tests are octreotide scanning and endoscopic ultrasonography, which have sensitivities for detection of tumor of approximately 75% and 80% or greater, respectively. Frequently a combination of imaging modalities are used to localize tumors and the studies of greatest value include CT scanning, MRI, endoscopic ultrasonography and octreotide scanning.

7. A patient with dyspeptic symptoms undergoes endoscopy which yields no remarkable findings. Physical exam, routine blood tests, and abdominal ultrasonography are unremarkable. She has not yet been given a trial of any medications. At this point, what is the best initial choice of empiric therapy for this patient?

A. Proton pump inhibitor
B. Alonsetron
C. Ondansetron
D. Tegaserod
E. Sumatriptan

The recommended response is A.

A subset of patients with non-ulcer dyspepsia (NUD) may actually have acid hypersensitivity or GERD, which can present in an atypical fashion. Acid inhibitory therapy with proton pump inhibitors may lead to complete symptom resolution in 25% to 50% of NUD patients. Efficacy of 5-HT3 antagonists (alonsetron, granisetron and ondansetron) or the 5-HT4 antagonist tegaserod, in NUD has not yet been clearly demonstrated. Clinical studies of 5-HT1 agonists (sumatriptan, buspirone) demonstrate improved gastric accommodation in NUD patients. However, due to significant side effects these agents are not currently recommended as first line therapy for NUD.

8. A 38-year-old female has had epigastric pain following meals and at night for the last six weeks. She smokes cigarettes and is a social drinker. She denies use of NSAIDs or aspirin. One week after she begins taking a proton pump inhibitor, upper endoscopy is performed which reveals a clean-based duodenal ulcer in the duodenal bulb. Rapid urease testing of a gastric antral biopsy is negative. Which of the following is the most likely cause of this patient's ulcer?

A. Crohn's disease  
B. *Helicobacter pylori*  
C. Duodenal adenocarcinoma  
D. Cigarette smoking and alcohol  
E. Zollinger-Ellison syndrome

The recommended response is B.

This patient has a duodenal ulcer caused by *Helicobacter pylori*; however, rapid urease testing for *H. pylori* was performed while she was taking a proton pump inhibitor. False negative tests for *H. pylori* may occur in patients recently taking antibiotics, bismuth containing compounds, proton pump inhibitors or possibly H2-receptor antagonists, due to suppression of infection. Patients should not receive proton pump inhibitors for at least 2 weeks before undergoing rapid urease testing, urea breath testing, or stool antigen testing for *H. pylori*. Tobacco, but not moderate amounts of alcohol, is associated with peptic ulcer. However, *H. pylori* and NSAIDs are much more likely causes of peptic ulcers than cigarettes. Duodenal adenocarcinoma is much less common (1 in 1000 duodenal ulcers or less) than gastric adenocarcinoma presenting in gastric ulcers. Zollinger-Ellison syndrome should always be considered in *H. pylori*-negative ulcer patients. However, this patient has no symptoms suggestive of Zollinger-Ellison
syndrome. Crohn's disease may rarely present with duodenal ulcers. However, almost always of these patients will have manifestations of Crohn's disease in the small intestine or colon.


9. Two months ago, a patient was hospitalized with a bleeding gastric ulcer. Testing for \textit{H. pylori} was positive. At discharge, a 10-day course of therapy was prescribed for \textit{H. pylori} eradication. After being asymptomatic for the last 6 weeks, the patient now returns with typical symptoms of his ulcer disease.

At this point, the least helpful test in evaluating whether \textit{H. pylori} was effectively eradicated is:

A. \textit{H. pylori} serology
B. Urea breath test
C. Gastric mucosal histologic test
D. \textit{H. pylori} stool antigen test
E. Gastric mucosal biopsy urease test

The recommended response is A.

A positive antibody test for \textit{H. pylori} is not helpful in determining whether \textit{H. pylori} has been effectively eradicated since antibody tests may remain positive for months after successful eradication of infection. They are, therefore, not recommended for determining \textit{H. pylori} status after treatment. See section on \textit{H. pylori} Diagnosis for further details of testing strategies.

Eckardt VF, Kanzler G, Willems D. Single dilation of symptomatic Schatzki rings. A prospective
10. A 34-year-old white female is hospitalized after a minor episode of hematemesis. She denies recent NSAID use. She has a history of a duodenal ulcer and *H. pylori* infection diagnosed two years ago by endoscopy. She was treated at that time with Pepto-Bismol, metronidazole, tetracycline, and omeprazole. However, the patient only took 4 days of her 14-day prescription. In the current admission, endoscopy reveals a clean-based duodenal ulcer and endoscopic mucosal biopsies were positive during urease testing. What is the most appropriate management?

A. A 14-day course of Pepto-Bismol, metronidazole, tetracycline, and omeprazole with emphasis of the importance of medication compliance
B. 14 days of Pepto-Bismol, metronidazole, amoxicillin, and omeprazole
C. A proton pump inhibitor for 14 days
D. A proton pump inhibitor, clarithromycin, and amoxicillin for 14 days
E. A proton pump inhibitor, clarithromycin, and metronidazole for 14 days

The recommended response is D.

This patient has active duodenal ulceration associated with *H. pylori* infection. Although she has been previously treated for *H. pylori*, she did not complete the prescribed course. Given that her initial regimen contained metronidazole, it can be assumed that her strain of *H. pylori* is now metronidazole resistant. Thus, all regimens that contain metronidazole are not optimal. Furthermore, it would not be appropriate to re-prescribe a regimen that the patient had previously failed to tolerate. Monotherapy with a proton pump inhibitor is not an effective anti-*H. pylori* therapy.

11. A 66-year-old black male with a 6-month history of dyspepsia presents for evaluation. His symptoms previously responded well to antacids and over-the-counter H2-receptor antagonists, but have not been relieved by these treatments in the past two months. He does not have dysphagia or weight loss. Physical examination and blood counts are normal. Which of the following is most appropriate as the next step in the management of this patient?

A. Treatment for *Helicobacter pylori*
B. CT scan
C. Upper endoscopy
D. Gastric motility testing
E. Proton pump inhibitor

The recommended response is C.

An important early decision in the evaluation and management of patients with dyspepsia is to determine whether presenting symptoms and signs are of sufficient concern to suggest the possibility of gastric malignancy or an ulcer complication (e.g., alarm symptoms). These would be new onset of symptoms after age 50 (as in this case), anorexia, dysphagia, gross or occult gastrointestinal bleeding, unexplained anemia, weight loss, significant vomiting or an upper gastrointestinal barium study suspicious for cancer. The presence of these features are indications for early endoscopy.
12. A 52-year-old white male presents for evaluation of sudden onset of abdominal pain and shoulder pain. His past medical history is notable for a history of coronary artery disease, hypertension, gallstones, and osteoarthritis. Medications include an ACE inhibitor, a beta-blocker, aspirin and ibuprofen. Abdominal examination is remarkable for hypoactive bowel sounds, a rigid abdomen and generalized rebound tenderness. Rectal exam is normal and fecal occult blood testing is negative. In evaluation of his shoulder pain, a portable CXR was taken and reveals free air. What is the next most appropriate step in the management of this patient?

A. Consult general surgery
B. Gastric lavage with saline to assess for GI bleeding
C. Abdominal CT scan
D. MRI
E. Helicobacter pylori antibody testing

The recommended response is A.

The chest X-ray is consistent with a perforated viscus. There is a past history of peptic ulcer disease in up to 75% of patients with perforated viscus. The acute onset of severe abdominal pain is usually the first symptom of a perforated duodenal or gastric ulcer. Elderly patients taking NSAIDs are particularly susceptible to complications of peptic ulcer disease such as perforation and most patients with perforated
ulcers will require operations. While an abdominal CT scan is a more sensitive test for the detection of free abdominal air than routine X-rays, in this case, an abdominal CT scan is not necessary to make the diagnosis of a perforated viscus. Since it has already been established that this patient has a perforation, instillation of fluids through a NG tube for gastric lavage would not be prudent and would likely lead to peritoneal contamination. While it would be helpful to know whether *Helicobacter pylori* contributed to pathogenesis of ulceration, the more immediate concern is to repair the perforation. Surgical exploration is the preferred approach for most patients presenting with pneumoperitoneum; however, some patients may be poor surgical candidates because of co-morbid diseases. In such patients, medical therapy may be successful. Such patients may include those with perforation of greater than 24 hours in whom a water-soluble upper GI contrast study reveals the perforation to be sealed off or completely contained. Medical therapy would consist of a nasogastric tube with intermittent suction, intravenous H₂-blockers or proton pump inhibitors and antibiotics.


13. A 40-year-old white male is referred to you because of a history of recurrent duodenal ulcers refractory to proton pump inhibitors. The patient denies NSAID use. Endoscopy reveals a duodenal ulcer. Histologic evaluation of biopsies of the antrum and gastric body are negative for *H. pylori*. Fasting serum gastrin is 1000 pg/ml (normal less than 150 pg/ml). The next most appropriate test is:

A. *H. pylori* serology
B. Urea breath test
C. Secretin test
D. Gastric acid analysis
E. Serum salicylate concentrations

The recommended response is D.

Approximately 90% of Zollinger-Ellison syndrome patients will develop ulcers, usually in the duodenal bulb, but also in the postbulbar duodenum or jejunum. Such ulcers are difficult to heal and may require very high doses of proton pump inhibitors to reduce acid secretion and induce healing. The diagnosis of ZE syndrome is suggested by an elevated serum gastrin concentration in a patient who is not achlorhydric. In patients with ZE syndrome, the gastrin level is almost always above 150 pg/ml. A level of 1000 pg/ml or greater is usually considered diagnostic for ZE syndrome. However, patients with achlorhydria may also have serum gastrin levels of 1000 pg/ml or greater. Therefore, it is necessary to document gastric acid hypersecretion when evaluating very elevated serum gastrin concentrations by gastric secretory studies. The secretin test is used as confirmation of ZE syndrome in patients with equivocal or otherwise undiagnosed hypergastrinemia.

In this case, if the diagnosis of ZE syndrome has not been suggested by the serum gastrin level of 1000 pg/ml, a serum salicylate level would have been useful to evaluate for surreptitious aspirin use in patients with refractory or recurrent ulcers. When comparing tests for H. pylori, histologic evaluations of multiple biopsies from antrum and body are generally more sensitive than serology or urea breath tests. Thus the first two choices would not have provided additional useful diagnostic information.

14. Which of the following mechanisms least describes the contribution of a hiatal hernia to GERD?

A. Loss of augmentation of the antireflux barrier by the crural diaphragm
B. Impaired esophageal acid clearance
C. Transient lower esophageal sphincter relaxation may be elicited more easily
D. Trapping of gastric contents that may then rereflux during transient lower esophageal sphincter relaxation
E. Esophageal shortening

The recommended response is E.

A hiatal hernia may contribute to gastroesophageal reflux by a number of different mechanisms. Gastric contents may be trapped in a hiatal hernia sac and then reflux proximally into the esophagus during swallow-induced relaxation. Large hiatal hernias may widen the diaphragmatic hiatus, which may impair the ability of the crural diaphragm to augment the antireflux barrier. Transient lower esophageal sphincter relaxation may also be elicited more easily in individuals with a hiatal hernia than those without one. Hiatal hernias also impair esophageal clearance by rereflux of retained material from the hernia back into the esophagus. Hiatal hernia size may be the strongest predictor of the severity of esophagitis. Esophageal shortening commonly seen in patients with esophagitis and hiatal hernia is believed to develop as a consequence of GERD.


15. A 50-year-old white female is referred to you by her primary care physician for consideration of upper endoscopy. She describes symptoms of acid regurgitation and classic heartburn occurring almost daily. Treatment with once daily PPI has resulted in incomplete symptom relief. She has now stopped therapy and you recommend an upper endoscopy.

The most likely esophageal finding in this patient will be:

A. No mucosal lesions
B. LA class A esophagitis
C. LA class B esophagitis
D. LA class C esophagitis
E. Barrett's esophagus

The recommended response is A.

It is now clear that up to 70% of patients referred for upper endoscopy in a community setting will have a normal endoscopy. Furthermore, in patients with NERD, the response rate to PPI therapy is less than that observed for individuals with erosive esophagitis. Symptoms are a poor predictor of endoscopic findings, and there is no clear relationship between endoscopic findings and symptom severity. Barrett's esophagus may be found in approximately 6% to 12% of patients with reflux symptoms but again,
symptoms are a poor predictor of Barrett's esophagus. While Barrett's esophagus is more common in men than in women, approximately 25% of individuals with Barrett's esophagus are women.


16. A 35-year-old white male is referred by his general internist to you for endoscopy. The patient has classic heartburn and acid regurgitation. Neither lifestyle measures nor over the counter H₂-receptor antagonists have improved his symptoms. At endoscopy, you see normal esophageal mucosa. There is no hiatal hernia.

What is the best management option at this point?

A. Mucosal biopsy of the esophagus to determine the presence of microscopic esophagitis
B. Mucosal biopsy of the squamocolumnar junction to determine the presence of short-segment Barrett's esophagus
C. Complete the endoscopy and arrange for esophageal impedance testing
D. Complete the endoscopy and arrange for ambulatory pH monitoring
E. Complete the endoscopy and arrange for a treatment trial of a proton pump inhibitor

The recommended response is E.
This patient has nonerosive gastroesophageal reflux disease (NERD). This entity is commonly seen in primary care settings and is characterized by classic GERD symptoms in the absence of endoscopic abnormalities. There is no role for endoscopic biopsy in NERD since there is poor correlation of biopsy results with pH monitoring in adults. Furthermore, there are costs associated with these biopsies. Similarly, a normal appearing squamocolumnar junction should not be biopsied to look for short-segment Barrett's esophagus, since the diagnosis requires both an endoscopic abnormality and intestinal metaplasia. Intestinal metaplasia of a normal appearing gastroesophageal junction is due to intestinal metaplasia of the cardia, which has different implications from intestinal metaplasia of the esophagus. The current role of esophageal impedance testing in GERD remains under investigation, especially for patients refractory to PPI therapy with either classic or extraesophageal symptoms. Prolonged pH monitoring is reasonable to consider if the patient fails a therapeutic trial, but should be deferred until after a therapeutic trial. The best approach to such a patient is an empiric trial of a PPI, since these agents provide superior symptom relief to H$_2$-receptor antagonists.


17. Which of the following statements is true about transient lower esophageal sphincter relaxation (TLESR) in gastroesophageal reflux?
A. It is the dominant mechanism in all patients with GERD
B. It is not seen in normal individuals
C. It is always accompanied by reflux events
D. More frequent TLESR correlates with more severe reflux disease
E. TLESR is typically longer than swallow-induced LES relaxation

The recommended response is E.

While gastroesophageal reflux was once thought to be related primarily to a hypotensive LES, it is clear that TLESR, that is relaxation unrelated to swallowing, is an important mechanism of reflux in normal subjects as well as those with GERD. However, not every TLESR is invariably associated with reflux of gastric contents. It is now apparent that TLESR is not the dominant mechanism of reflux in all GERD patients, but is instead the dominant mechanism in patients with milder degrees of reflux disease whereas anatomic disruption of the gastroesophageal junction associated with a hiatal hernia and a mechanically defective LES are more important in patients with more severe reflux disease. These relaxations are long than deglutitive LES relaxations in duration.


18. A 45-year-old white female comes to seek your opinion about antireflux surgery. She has classic heartburn and acid regurgitation, both of which resolve on twice daily PPI therapy. However, she is concerned about the long-term risks of PPI therapy and is uncomfortable with taking this medication for the rest of her life.

Her preoperative evaluation reveals the following:

- EGD off of PPI therapy: LA class B esophagitis and 2 cm hiatal hernia
- Esophageal manometry: normal peristalsis and amplitude; LES pressure of 9 mm Hg
- 24-hour pH: % time pH <4 of 35% for combined upright and supine position
- Barium esophagram: 3 cm reducible hernia with free reflux

Which of the following statements are true regarding laparoscopic antireflux surgery in this patient if done by an experienced surgeon?

A. Antireflux surgery will offer the patient superior symptom control to her current regimen of PPIs
B. Antireflux surgery will decrease with patient's risk of ever developing esophageal cancer
C. This patient is an ideal candidate for surgery and she can expect a 90% chance of excellent symptom control
D. Surgery permanently eliminates the need for acid suppressive therapy.
E. Surgery provides a permanent therapy to reflux disease

The recommended response is C.
Antireflux surgery is an excellent option for carefully selected patients with well documented GERD. The ideal candidate is the patient with typical symptoms that respond completely to antisecretory therapy such as this patient. Complete relief of GERD symptoms and healing of esophagitis can be anticipated in 90% of patients in experienced hands. Predictors of a successful surgical outcome include surgical experience and patient selection based on typical symptoms previously responsive to medical therapy. While the short term outcome of antireflux surgery is excellent there are both short and long-term problems associated with surgery. Anti-reflux surgery is a complex operation that involves general anesthesia and at least overnight hospitalization, the learning curve for this procedure is steep and it is clear that results from specialized centers are not generalizable to other settings. The 30-day mortality varies from 0.08% to 0.5%. Immediate postoperative complications occur in approximately 4% to 6%. Approximately 25% of patients will experience dysphagia, which decreases to 5% at 6 months. Later postoperative complications are also common, including dysphagia, gas bloat syndrome, increased flatus and diarrhea. These symptoms can at times be incapacitating, and impair quality of life. The long-term failure rate of surgery is approximately 1% annually. Long-term follow-up studies suggest that a large percentage of patients may need acid suppressive therapy despite fundoplication. A randomized controlled Nordic trial that compared open antireflux surgery to PPI therapy found no difference in maintenance of remission at 5 years, if dose titration of PPIs was permitted (Figure 13). Data to date suggest that antireflux surgery offers no clear advantage to medical therapy for the treatment of GERD with respect to healing of esophagitis, prevention of complications including stricture, Barrett's esophagus and esophageal adenocarcinoma, safety, side effects or cost. Thus, it appears that both antireflux surgery and PPI therapy are equivalent options for the treatment of GERD.

19. You are asked for an opinion by the family physician of a 25-year old female with a chronic unexplained cough. A chest X-ray was negative and she has failed to respond to twice daily PPI therapy given for 16 weeks. A dual 24-hour pH test performed by another gastroenterologist revealed no increase in acid exposure in the proximal or distal esophagus while on therapy. She has never had any heartburn or acid regurgitation.

What would be the best next step?

A. Upper endoscopy
B. Barium esophagram

C. Surgical referral for failure of medical therapy

D. Addition of a nocturnal dose of a H2-receptor antagonist

E. Appropriate referral to evaluate for asthma with a methacholine challenge test and sinus disease with a CT of the sinuses

The recommended response is E.

Chronic unexplained cough is one of the extraesophageal manifestations of GERD. Just as in patients with typical symptoms of GERD, there is no diagnostic gold standard for GERD in these patients. Upper endoscopy has a low diagnostic yield in patients with extraesophageal manifestations of GERD and would be of little use in this patient. The same is true for a barium esophagram. Antireflux surgery as an empiric trial is tempting to consider in patients who fail medical therapy. However, unlike the setting of classic GERD where the short-term response rate is approximately 90%, the response for atypical symptoms is closer to 50% unless there is documented acid exposure and a prior response to a PPI. This patient has not had a response to PPI therapy. Nocturnal acid breakthrough, is defined as pH <4 for at least one hour in the overnight period despite twice daily PPI therapy. While a night-time dose of a H2-receptor antagonist may correct nocturnal acid breakthrough when administered acutely, chronic use of a H2-receptor antagonist is associated with tolerance and thus may not reliably decrease intragastric pH when administered on a chronic basis. Furthermore, nocturnal gastric acid breakthrough is a pharmacologic observation that has never been demonstrated to have clinical significance. There is no evidence of reflux in this patient and the failure to respond to PPI therapy predicts a suboptimal response to antireflux surgery.
Patients with a clinical profile highly suggestive of silent GERD as a cause of their cough are characterized by the following findings: 1) normal or clear chest X-ray; 2) no smoking or exposure to environmental irritants; 3) no use of ACE inhibitors; 4) negative methacholine challenge or failure of cough to improve with treatment of asthma; and 5) failure of cough to improve with treatment of postnasal drip syndrome. The most common causes of unexplained cough are asthma, postnasal drip and GERD. Often, these patients may have more than one etiology for their symptoms. A definite diagnosis of cough due to GERD requires that cough nearly or completely be resolved with anti-reflux treatment. This patient failed to respond to an empiric trial of twice daily PPI therapy with documentation of adequate acid suppression. As such, the best management approach in this patient is to exclude asthma with a methacholine challenge test and sinus disease with either a CT scan of the sinuses or administration of empiric therapy for postnasal drip.

DeVault KR. Overview of therapy for extraesophageal manifestations of gastroesophageal reflux disease. Am J Gastroenterol 2000(suppl);95:S39-S44.


Irwin, R. Chronic cough due to gastroesophageal reflux disease. ACCP Evidence-Based Clinical Practice Guidelines. Chest 2006;129:80S-94S.
20. A 40-year-old Hispanic male has just completed a 4-week course of a once daily dose of a proton pump inhibitor for LA Class B esophagitis with complete symptom relief. He asks you what the most appropriate treatment option is for him in the future.

Which of the following statements is true?

A. Continued PPI therapy is the best medical agent to maintain the patient in remission
B. Lifestyle measures alone should be adequate to control his symptoms
C. H₂-receptor antagonists will not control symptoms
D. Step-down therapy with metoclopramide should be tried
E. Immediate referral for radiofrequency ablation treatment

The recommended response is A.

Erosive esophagitis is a chronic disease and relapse is common after discontinuing therapy. Left untreated, the relapse rate for erosive esophagitis is approximately 80%. Maintenance therapy with twice daily doses of H₂-receptor antagonists will result in symptom control in approximately 50% of such patients and as such cannot be dismissed completely. Many patients in a primary care setting can be managed successfully with H₂-receptor antagonists. However, this patient has erosive esophagitis, whereas the majority of GERD patients in a primary care setting have non-erosive reflux disease (NERD). Metoclopramide is the only available prokinetic agent and is best viewed as an adjunctive therapy, primarily for patients with delayed gastric emptying due to its adverse effect profile. Lifestyle measures, while making practical sense for extraesophageal respiratory complications of GERD, are unlikely to be adequate to compensate alone for the pathophysiologic abnormalities encountered in erosive esophagitis. Evidence-based medicine suggests that the benefits of lifestyle measures are
overstated in management of esophageal complications of GERD. A variety of endoscopic techniques are available as alternatives to antisecretory therapy or antireflux surgery. However, some have been withdrawn from the market and the remaining are not supported with convincing long-term efficacy. Continued PPI therapy is superior to other medical options for maintaining such patients in remission.


21. Which statement is true regarding esophageal acid exposure in Barrett’s esophagus?

A. Acid exposure is increased due to gastric acid hypersecretion
B. Acid exposure is increased due to mechanical dysfunction of the LES and impaired esophageal motility
C. Acid exposure is routinely normalized by proton pump inhibitors
D. Adequate symptom control correlate with effective acid control
E. Control of acid exposure decreases cancer risk in Barrett’s esophagus

The recommended response is B.
In Barrett’s esophagus, there is no increase in gastric acid production compared to age-matched controls. Furthermore, while PPIs routinely control GERD symptoms in these patients, up to 40% of patients will continue to have increased esophageal acid exposure despite excellent symptom control. While experimental studies suggest that adequate acid suppression results in a decrease in cellular proliferation in Barrett’s esophagus, there are no data as yet that acid suppression results in a decrease in cancer risk. Barrett’s esophagus is associated with increased esophageal acid exposure due to more profound abnormalities of esophageal motility, LES function and larger hiatal hernias compared to GERD patients without Barrett’s esophagus.


Hirschowitz BI. Gastric acid and pepsin secretion in patients with Barrett’s esophagus and appropriate controls. Dig Dis Sci 1996;41:1384-1391.


22. Based on current data, what is the best estimate of the annual risk for developing adenocarcinoma in patients with Barrett’s esophagus?

A. 100%
B. 50%
C. 25%
D. 5%
E. 0.5%

The recommended response is E.

The risk of developing adenocarcinoma in Barrett’s esophagus patients is 30 to 125 times that encountered in the general population. However, the overall risk of esophageal cancer is low, despite the alarming increase in incidence in esophageal adenocarcinoma in the Western world. Previous studies have overestimated this risk, perhaps due to publication bias. However, this risk is just an estimate as studies published to date are still relatively small. The current estimate of cancer risk in these patients is approximately 0.5% annually.


23. A 45-year-old white male business executive sees you because of classic heartburn and acid regurgitation. An upper endoscopy revealed a displaced squamocolumnar junction. Eight large capacity biopsies were obtained and were read by your pathologist as containing columnar epithelium with pseudogoblet cells that stain negative by Alcian blue staining. You have reviewed the slides with the pathologist and tell the patient which of the following?

A. Biopsies confirm the diagnosis of Barrett’s esophagus and repeat surveillance endoscopy in 1 to 2 years
B. Biopsies confirm the diagnosis of Barrett’s esophagus and repeat surveillance endoscopy in 2 to 3 years
C. Request that the pathologist obtain cytokeratin stains to differentiate the columnar epithelium originating from the stomach or the esophagus

D. Suggest mucosal ablation with bipolar electrocoagulation to eliminate the columnar epithelium

E. Inform the patient that biopsies do not confirm the diagnosis of Barrett’s esophagus, however, since this could represent sampling error you would like to confirm the finding by repeating the biopsy.

The recommended response is E.

This patient represents a common dilemma in Barrett’s esophagus, namely is it present or not? The diagnosis of Barrett’s esophagus is based on two components: displacement of the squamocolumnar junction above the level of the esophagogastric junction as described in this patients and the histologic finding of specialized intestinal metaplasia defined by goblet cells in biopsies obtained from this segment. Some pathologists will still commonly refer to gastric fundic and cardia type epithelium, as found in this patient as “compatible with Barrett’s esophagus”. Thus, while this patient had a displaced squamocolumnar junction, intestinal metaplasia was not encountered. These biopsies demonstrate instead columnar epithelium with pseudogoblet cells characterized by distended gastric surface foveolar-type cells that stain for PAS but do not contain Alcian blue positive acid mucins. However, since the true specialized intestinal metaplasia could have been missed by sampling error, confirmation of the finding by repeat biopsy should be considered. Ablation therapy at present has no role in Barrett’s esophagus without dysplasia. Unfortunately, immunohistochemical cytokeratin staining patterns of biopsies obtained from questionable areas of Barrett’s esophagus do not reliably distinguish between intestinal metaplasia of the cardia and the esophagus.


24. Which of the following statements is true regarding the role of antireflux surgery in Barrett’s esophagus?

A. Antireflux surgery will control symptoms of GERD
B. Antireflux surgery is more effective than medical therapy for the treatment of Barrett’s esophagus
C. Antireflux surgery causes regression of Barrett’s epithelium
D. Antireflux surgery decreases the risk of esophageal cancer
E. Antireflux surgery eliminates the need for surveillance in Barrett's patients

The recommended response is A.

Antireflux surgery effectively alleviates GERD symptoms in Barrett’s esophagus patients. While studies consistently show the development of squamous islands after antireflux surgery, complete regression of Barrett’s epithelium is uncommon and may well represent “pseudoregression” due to surgical repositioning of the esophagus. Some surgical enthusiasts suggest that antireflux surgery decreases the subsequent risk of developing esophageal cancer. However, a large Swedish cohort study found that the risk of developing adenocarcinoma of the esophagus remains elevated after antireflux surgery in GERD patients (Barrett’s status unknown) and was no different for GERD patients not
undergoing antireflux surgery. A recent meta-analysis found a decrease in the risk of adenocarcinoma after antireflux surgery for Barrett’s esophagus. There are no data to support an advantage of antireflux surgery compared to medical therapy for symptoms control in Barrett’s esophagus patients. Thus, while surgery provides an excellent means of symptom control in Barrett’s patients, it does not appear to influence the natural history of Barrett’s esophagus and as such patients remain in need of surveillance endoscopy and biopsy.


25. A 50-year-old white male seeks your opinion about treatment of Barrett’s esophagus. He was diagnosed by you with Barrett’s esophagus 6 years ago at which time a 5 cm segment was found. Subsequently surveillance endoscopy by you at regular scheduled intervals has also revealed intestinal metaplasia without dysplasia on biopsy. The patient has done internet research and wishes to commence either aspirin or selective COX-2 inhibition therapy for chemoprevention. After congratulating the patient for his proactive thoughtful question, what do you recommend?

A. Inform the patients that randomized controlled trials support chemoprevention with aspirin and commence therapy with low-dose aspirin taken daily in conjunction with twice daily PPI therapy
B. Inform the patient that randomized controlled trials support chemoprevention with selective COX-2 inhibitors and commence therapy with any of the selective COX-2 inhibitors taken once daily in conjunction with twice daily PPI therapy

C. Order 24-hour pH monitoring first to document adequate acid suppression prior to commencing therapy with low-dose aspirin

D. Inform the patient that modeling studies support the use of either aspirin or COX-2 inhibitors and commence therapy with either aspirin or a COX-2 inhibitor immediately

E. Inform the patient that studies to date suggest that it is premature to commence chemoprevention with either aspirin or COX-2 inhibitors

The recommended response is E.

This patient presents with a rapidly evolving scenario in Barrett’s esophagus, namely how to counsel proactive patient who has done research into chemoprevention. COX-2 expression increases progressively in Barrett’s metaplasia, dysplasia, and adenocarcinoma, all of which are greater than that encountered in normal esophageal squamous or duodenal columnar epithelium. A number of observational studies suggest a protective association between the use of aspirin or nonsteroidal anti-inflammatory drugs and the risk of developing esophageal adenocarcinoma. Furthermore, greater protection is associated with more frequent use of these compounds. Administration of a selective COX-2 inhibitor for ten days to Barrett’s esophagus patients reduces COX-2 expression. These observations have led to speculation that selective COX-2 inhibitors maybe useful in the chemoprevention of esophageal adenocarcinoma. However, there have been no published randomized controlled clinical trials to address this question, and given the overall low incidence of dysplasia or carcinoma, such a study will be difficult to accomplish. Furthermore, a decision analysis model suggested that such a strategy may not be cost effective in the general population of Barrett’s esophagus patients, but may be
worthy of future consideration in high risk patients, i.e., those with dysplasia, be it low-grade or high-grade. Given the overall low incidence of esophageal cancer in Barrett’s esophagus and potential complications associated with NSAID, it is still premature to administer aspirin or NSAIDs to these patients. The role of 24-hour pH monitoring to titrate PPI dose in Barrett’s esophagus remains uncertain.


Thun MJ. NSAIDs and esophageal cancer: ready for trials but not yet broad clinical application. Gastroenterology 2003;124:246-248 (Editorial)

26. A 45-year-old white male with a known diagnosis of Barrett’s esophagus undergoes routine endoscopic surveillance while on once daily PPI therapy. A 7 cm segment of Barrett’s esophagus is seen with no mucosal irregularities. Four quadrant biopsies obtained at 2 cm intervals return with findings of low-grade dysplasia confirmed by an expert GI pathologist. A repeat endoscopy was done by the same physician within 3 months with the patient on twice daily proton pump inhibitor therapy with identical findings. What would be the most appropriate management for this patient?
A. Immediate surgical referral for esophagectomy
B. Return visit in 4 weeks for endoscopic mucosal resection
C. Return visit in 4 weeks for endoscopic mucosal ablation with photodynamic therapy
D. Return in 12 months for repeat endoscopy with surveillance biopsies
E. Return in 3 years for routine endoscopy with surveillance biopsies

The recommended response is D.

The natural history of low-grade dysplasia is quite variable. In part, this is due to the high degree of interobserver variability in establishing this diagnosis and the variable protocols by which these patients are followed. While the majority of patients with low-grade dysplasia do not progress, a subset of these patients progress on to these more worrisome lesions. Recent work described progression to high-grade dysplasia or adenocarcinoma in 10% to 28%, whereas regression to no dysplasia occurs in approximately 60% to 65%. Current guidelines suggest that after a follow up endoscopy with concentrated biopsies in the region of dysplasia, annual surveillance is recommended until there is no dysplasia. These patients also warrant an increase in acid suppression with PPIs to twice daily in an effort to decrease inflammatory changes that may make interpretation of dysplasia problematic. Unlike low-grade dysplasia in inflammatory bowel disease, surgery is not a consideration given the fact that progression to cancer is relatively uncommon and most patients with low-grade dysplasia regress to no dysplasia. The role of ablation therapy with thermal techniques, photodynamic therapy or endoscopic mucosal resection is uncertain.


27. A 65-year-old white male undergoes screening endoscopy at which time a 1 cm tongue of columnar appearing mucosa is encountered that clearly extends above the squamocolumnar junction. Biopsies from this tongue reveal intestinal metaplasia. Which statement regarding short segment Barrett’s esophagus is true?

A. Cancer risk is not increased.
B. Cancer risk is no different than intestinal metaplasia of the gastric cardia.
C. Surveillance intervals are different than those recommended for long segment Barrett’s esophagus.
D. Intestinal metaplasia of the cardia is histologically distinguishable from intestinal metaplasia in short segment Barrett’s esophagus.
E. Pathophysiologic abnormalities are intermediate between uncomplicated GERD and long segment Barrett’s esophagus.

The recommended response is E.

Patients with short segment Barrett’s esophagus (<3 cm in length) may have a variety of pathophysiologic abnormalities such as those of LES pressure and esophageal acid exposure that are intermediate to those of long segment Barrett’s patients and patients with uncomplicated GERD. As a group, short segment Barrett’s esophagus patients are characterized by an LES pressure lower than
controls, but higher than long segment Barrett’s esophagus and distal acid exposure time is greater than controls, but less than that in long segment Barrett’s esophagus. Furthermore, recent data suggests that the length of Barrett’s esophagus correlates with the duration of acid exposure. Biopsies from a normal esophageal junction may also reveal intestinal metaplasia which is presumed to represent intestinal metaplasia of the cardia. This is histologically indistinguishable from intestinal metaplasia of the esophagus using routine stains. However, the dysplasia risk in short segment Barrett’s esophagus is significantly greater than in intestinal metaplasia of the cardia. As such, surveillance is recommended for patients with short segment Barrett’s esophagus, but not for patients with intestinal metaplasia of the cardia.


28. A 55-year-old man presents with a long-standing history of heartburn for 10 years which has been partially treated with over-the-counter antacids and H-2 antagonists. He is otherwise healthy without any past medical or surgical histories. An endoscopic view of the distal esophagus is shown in Figure 19A. The pathology findings of multiple biopsies are shown in Figure 19B. Which of the following is the most appropriate next step in management in addition to treatment with proton pump inhibitors?

A. Inform the patient that he has Barrett’s esophagus without dysplasia and recommend returning in 1 year for routine EGD with surveillance biopsies.
B. Inform the patient that he has Barrett’s esophagus with low-grade dysplasia and recommend returning for repeat EGD in 3 months for more aggressive biopsies.
C. Inform the patients that he has Barrett’s esophagus with low-grade dysplasia and recommend returning in 1 year for routine EGD with surveillance biopsies.

D. Inform the patient that he has Barrett’s esophagus with high-grade dysplasia and recommend immediate evaluation for surgery, photodynamic therapy, or intensive surveillance.

E. Inform the patient that he has Barrett’s esophagus with high-grade dysplasia and recommend immediate surgical referral for Nissen’s fundoplication.

The recommended response is D.

The endoscopic figure shows long-segment Barrett’s esophagus (BE) with the pathology confirming the presence of intestinal metaplasia with high-grade dysplasia (HGD). The best management of HGD remains highly controversial. Given the uniformly poor survival reported in patients with esophageal adenocarcinoma, the guideline for endoscopic surveillance programs has been developed. The interval is contemplated on the basis of the degree of dysplasia noted on biopsies. Patients without dysplasia are generally followed up with EGD at intervals of 2-3 years after an initial confirmation at 1 year, whereas those with HGD are considered for intervention. Interobserver variability in the interpretation of HGD mandates expert confirmation. If confirmed, the presence of HGD is considered an appropriate criterion for surgical resection because of the high risk of progression and the likelihood of metachronous adenocarcinoma. On the other hand, two other management strategies have been recently proposed and can be considered in the selected group of patients, especially those with the high risk for surgery. These strategies include intensive endoscopic surveillance (every 3 months for the first year, every 6 months for the second year, and yearly thereafter) and endoscopic ablative therapies.

29. A 30-year-old surgical resident presents to GI lab with symptoms of acute dysphagia and inability to swallow saliva. EGD is performed emergently and shows a pill impacted at the proximal esophagus approximately 24 cm from incisor. After the pill dislodges into the stomach, the endoscopic view of esophagus is shown in Figure 20. Which of the following is correct regarding this condition?

A. Female predominance is apparent with the percentage of 75%.
B. Blood eosinophilia is seen in more than 60% of the adult patients.
C. The absence of IgE-related features can exclude the possibility of this condition.
D. Long-standing dysphagia and solid food impaction are the most common presenting symptoms in the adult patients.
E. Proton pump inhibitors are the treatment of choice for the adult patients.

The recommended response is D.

The endoscopic figure shows multiple circumferential lines of the esophageal mucosa, consistent with the diagnosis of eosinophilic esophagitis (EE). The prevalence of EE remains unknown, however it appears to be male predominant with the percentage of 75%. A majority of pediatric patients (51% to 84%) have atopic conditions, including asthma, atopic dermatitis, and food allergy, compared to 29% to 60% in adult patients. Blood eosinophilia is seen in approximately 60% of the pediatric patients, compared to 5% to 50% of the adult patients. The absence of IgE-related features, e.g. negative skin prick tests or a negative radioallergosorbent test does not exclude the possibility of EE. In children, the
predominant feature could be one of gastroesophageal reflux disease (vomiting, dysphagia, and abdominal pain), whereas long-standing dysphagia and solid food impaction are the most common presenting symptoms in the adult patients. Anti-acid therapy is generally ineffective. Topical corticosteroids, leukotriene receptor inhibitors, mast cell stabilizers, and elimination diet are the mainstay of treatment.


30. An otherwise healthy 46-year-old man presents with intermittent episodes of substernal dysphagia only after swallowing solid foods and never with liquids. He denied heartburn, water brash, or regurgitation. An endoscopic view of the distal esophagus is shown in Figure 21. Which of the following is the most appropriate next step in management?

A. Observation
B. 24-hour pH monitoring
C. Esophageal manometry
D. Oral administration with proton pump inhibitors
E. Esophageal dilation with bougienage

The recommended response is E.
The endoscopic figure shows a circumferential mucosal ring at the gastroesophageal junction, consistent with Schatzki ring. It is one of the most common contributing causes of episodic dysphagia, especially with solid foods. Although it has been recognized endoscopically and radiographically for many years, the etiology remains unclear. Chronic injury from gastroesophageal reflux disease and congenital or developmental factors have been proposed. Therapeutic options for patients with symptoms include bougienage dilation, surgical incision, and endoscopic incision. Bougienage is the most widely practiced therapy, although symptoms can recur in approximately 65% of patients and repeated bougienage may require. Endoscopic incision or surgical incision can be considered as an alternative to repeated bougienage in patient with dysphagia caused by recurrent disease.


31. A 48-year-old man undergoes an evaluation for heartburn. An EGD shows a normal Z-line at the gastroesophageal junction at 40 cm from the incisors and mild distal reflux esophagitis. At 25 cm from the incisors, two flat patches of pink mucosa are seen as shown in Figure 22. Which of the following is incorrect regarding this condition?

A. Biopsy shows columnar-lined gastric mucosa
B. It may play a role in the development of esophagitis, web, stricture, ulcer, and perforation
C. Increased risk for adenocarcinoma of the cervical esophagus has been reported
D. Endoscopic surveillance with biopsies is not indicated
E. Chromoendoscopy for diagnosis of intestinal metaplasia is not necessary
The recommended response is C.

The endoscopic figure shows inlet patches, which are areas of heterotopic gastric mucosa located in the cervical esophagus. A frequency of 0.1-10% was observed in several studies when the cervical esophagus was examined carefully during EGD. The diagnosis can be made by the typical endoscopic appearance and confirmed by the presence of columnar-lined gastric mucosa from biopsy. Most inlet patches do not cause any symptoms despite their capability of acid secretion. Several case reports suggest that they may play a role in the development of the esophagitis, web, stricture, ulcer, perforation, and esophagotracheal fistula. Although adenocarcinoma arising in an inlet patch has rarely been reported, an increased risk of adenocarcinoma has never been described and endoscopic surveillance with biopsies is not indicated. Because they do not contain intestinal metaplasia, chromoendoscopy is not necessary.


32. A 30 year-old woman undergoing an EGD to evaluate for dyspepsia for 3 months has an endoscopic view of gastric body in Figure 23. An EUS (image not shown) demonstrates a hypoechoic (dark) lesion with “salt and pepper” like parenchyma in the submucosa (the third echo layer). The differential diagnosis of this lesion could be all of the following except:
A. Lipoma
B. Gastrointestinal stromal tumor
C. Gastric carcinoid tumor
D. Neural sheath tumor (Schwannoma)
E. Ectopic pancreas

The recommended response is A.

Hypoechoic lesions of the gastrointestinal tract consist of a wide variety of tumors, including gastrointestinal stromal tumor (GIST), carcinoid tumor, neural sheath tumor (schwannoma), and ectopic pancreas. Lipomas are classically hyperechoic (bright) on EUS. Endoscopy and EUS alone often cannot distinguish between these lesions and a tissue sample by biopsy, EUS-FNA, EUS-core biopsy, or resection is required to confirm the diagnosis.

The lesion shown in the figure is ectopic pancreas (also known as heterotopic pancreas, aberrant pancreas, or pancreatic rest), a rare submucosal tumor that most commonly consist of cystically dilated exocrine cells. Endocrine pancreatic tissue or a combination of exocrine and endocrine cell types may also be seen. The typical endoscopic finding is a raised area with a central crater or umbilication that corresponds to a draining duct. Although ectopic pancreatic tissue can be found anywhere in the gastrointestinal tract, the gastric antrum, duodenum, and proximal jejunum are the most common locations. Ectopic pancreatic tissue does not usually cause any symptoms, but dyspepsia, bleeding, intussusception, and malignant transformation have been reported. Asymptomatic lesions can be followed expectantly. Surgical resection is preferred to endoscopic resection when malignant transformation is suspected and the muscularis propria is involved.
Lipomas and gastrointestinal stromal tumors (GIST) are typically identified as round masses covered with normal epithelium without umbilication. Lipomas are classically hyperechoic (bright) on EUS. GISTs are considered pre-malignant or malignant and should be excised. They can be diagnosed by EUS-FNA and staining the cells for CD-117 (c-kit). Gastric carcinoid tumors are potentially malignant, especially when they occur sporadically (as opposed to in the setting of achlorhydria, where they are generally benign). Neural sheath tumors are generally benign but can only be diagnosed by biopsy or excision.

When confronted with a hypoechoic lesion in the third echo layer, it is reasonable to excise the lesion with endoscopic mucosal resection after submucosal saline injection. Hypoechoic lesions in the fourth echo layer (muscularis propria) should be sampled with EUS-FNA or excised surgically to determine if they are GISTs.


33. A 65-year-old man with a history of gastroesophageal reflux disease and large hiatal hernia underwent a laparoscopic Nissen fundoplication 5 years ago. His symptoms completely resolved after surgery. He presents with recurrent symptoms of heartburn and regurgitation for 2 months. These symptoms have been partially controlled with oral administration of proton pump inhibitors. The
The endoscopic figure shows the retroflexed view of gastroesophageal junction. There appears to be the absence of anterior and posterior grooves and visible suture material in the gastric fundus suggesting partial disruption of the Nissen fundoplication. Reappearance of gastroesophageal reflux symptoms after a successful antireflux surgery warrants further investigations. The most important investigation is the careful examination of gastroesophageal junction during EGD. Several causes of recurrence can be diagnosed, including a slipped Nissen fundoplication, total or partial disruption of the Nissen fundoplication, and disruption of the crural repair resulting in recurrent hiatal hernia (herniation of the intact fundoplication into the chest cavity). Though EGD is more sensitive than barium contrast radiography, paraesophageal hernia and anatomical relationships among organs may be better appreciated by upper gastrointestinal series. In addition, esophageal pH monitoring study can document the pattern, frequency, and duration of acid reflux, and can help to establish that post-operative symptoms are secondary to acid reflux.

Figures

Figure 13