Update on Dyspepsia: A Case-Oriented Approach

Dyspepsia is a highly prevalent disorder that affects up to 30% of the general population.1-3 Symptoms of dyspepsia include epigastric pain and/or discomfort, early satiety, postprandial fullness, bloating, and nausea. Dyspeptic symptoms may develop due to organic causes (eg, peptic ulcer disease, gastritis, occult acid reflux); however, the vast majority of patients with dyspeptic symptoms are ultimately diagnosed as having functional dyspepsia (FD).3-5

FD is a heterogeneous disorder with symptoms that reflect several underlying pathophysiologic abnormalities.6,7 For many patients, these symptoms are chronic in nature, may reduce their quality of life, and have significant economic consequences. This clinical review will use 4 patient cases to describe the epidemiology, pathophysiology, evaluation, and treatment of patients with dyspeptic symptoms, with an emphasis on FD.
Case 1: “Sophia”

Sophia is a 29-year-old woman sent for evaluation of new abdominal pain. She states that the pain has been present for nearly 6 weeks, and she points to the epigastric region. The pain waxes and wanes in intensity, although it is present nearly every day. She cannot recall any precipitating event.

Sophia's appetite is good and her weight has been stable. There are no symptoms of heartburn, regurgitation, dysphagia, or odynophagia. She works full-time as a teacher and is currently training for a marathon. A review of systems is notable for joint pain associated with running. She does not drink any alcohol and does not smoke cigarettes. Her family history is unremarkable.

Her only medication is a nonsteroidal anti-inflammatory drug (NSAID; ibuprofen 600 mg, by mouth, 3 times per day) for joint pain. A 3-week trial of an over-the-counter, histamine type-2 receptor antagonist (H2RA), taken twice daily, did not alleviate her symptoms. Her physical examination is notable for mild epigastric tenderness without rebound or guarding. A Murphy's sign cannot be elicited on examination; stool is Hemoccult negative.

Laboratory tests are ordered—complete blood count (CBC), lipase, and liver function tests (LFTs)—and Sophia is asked to refrain from all anti-inflammatory agents. The tests all return normal the following day. Sophia calls in for a follow-up 1 week later, stating that her pain has not improved, that she now has some nausea after eating and that not being able to take her NSAID is hindering her training schedule.

Upper endoscopy is scheduled, which reveals multiple shallow, nonbleeding erosions in the antrum. Biopsies from throughout the stomach show evidence of mild inflammation, but no evidence of Helicobacter pylori (H. pylori).

Sophia is started on a daily proton pump inhibitor (PPI), taken 30 minutes before breakfast. She calls back 2 weeks later, saying that her symptoms have essentially resolved. The PPI is stopped 4 weeks later, and Sophia remains symptom-free 2 months later at follow-up while continuing to abstain from NSAIDs.

Summary

This case illustrates that symptoms of dyspepsia are nonspecific and may reflect an organic process (Table 1). As in Sophie's case, or may be functional in nature. Upper endoscopy was performed after symptoms persisted despite stopping the NSAID and treating empirically with an H2RA.

The multiple shallow erosions identified on endoscopy are consistent with NSAID-induced injury. Empiric treatment with a PPI prior to upper endoscopy is an alternative approach; however, earlier endoscopy may lead to the initiation of more appropriate therapy earlier and may reassure the patient.

Epidemiology and Natural History

Dyspepsia is common in the United States, with an estimated prevalence of up to 30% in the general population. Dyspepsia appears to affect men and women equally, and occurs independently of race, religion, and socioeconomic status. Dyspepsia is categorized into 2 large groups of disorders: FD and organic dyspepsia. FD—which is diagnosed only after an appropriate diagnostic evaluation fails to identify an etiology—accounts for approximately two-thirds of all patients with dyspeptic symptoms.

The natural history of dyspepsia has not been well characterized, possibly because patients with FD and organic dyspepsia have been combined in many studies, which also include large numbers of patients with acid reflux. Some studies suggest that FD is a chronic disease for many patients, with approximately 50% of patients remaining symptomatic over a 5-year follow-up period. Contrary to many patients' beliefs, FD is not a risk factor for gastric cancer or peptic ulcer disease.

Organic vs Functional Dyspepsia

The first step in evaluating a patient with dyspepsia is to determine whether or not the symptoms represent an underlying organic process. Common organic causes of dyspepsia are listed in Table 1. Unfortunately, one of the difficulties encountered in evaluating a patient with dyspepsia is that symptoms are nonspecific and cannot accurately differentiate an organic process from a functional disorder (Table 2).

Patients should be questioned about symptoms of heartburn and regurgitation, as acid reflux is a common cause of dyspepsia. Occult acid reflux, however, may be the cause of symptoms in a large number of patients, who may respond to an empirical trial of acid suppression (see “Treatment Options” section). Patients should be questioned about risk factors for peptic ulcer disease and gastritis, and their H. pylori status, if known.

Medications may cause dyspeptic symptoms in some patients, and NSAIDs, as in Sophia's case, are among the worst offenders. Pancreatitis and other pancreatic disorders are uncommon causes of dyspepsia that can usually be identified by a focused history and limited blood work. Patients should be questioned about risk factors for hepatitis and other hepatobiliary disorders as well.

A physical examination should be performed for evidence of liver disease, and to identify organomegaly, ascites, or lymphadenopathy. These findings support an organic cause for the dyspeptic symptoms and warrant a more focused diagnostic evaluation. The most common finding in patients with dyspepsia, however, is epigastric discomfort. Unfortunately, this finding is nonspecific and cannot differentiate an organic process from a functional process.
If the history and physical examination is unrevealing, laboratory tests may provide an answer. A CBC should be ordered; if clinically indicated, serum lipase and LFTs should be ordered as well. It should be noted, however, that no prospective study has identified the clinical utility of serologic tests in the evaluation of patients with dyspeptic symptoms.2,5-7

**Case 2: “Robert”**

Robert is a 23-year-old computer programmer referred for evaluation of upper abdominal discomfort and burning. He states that these symptoms have been present for nearly 1 year, after graduating from college. Symptoms are typically worse later in the day and at night, and are present at least 3 days each week. He admits that his job is stressful and that symptoms are worse with stress. He does not have any symptoms of odynophagia or dysphagia, and denies symptoms of pyrosis, water brash, and regurgitation.

He has gained nearly 25 pounds since graduating from college. Robert attributes this to a lack of exercise, eating a lot of take-out food, and long hours at work. He does not take any medications, and does not smoke cigarettes, although he drinks 2 to 3 beers each night.

Robert has never had an ulcer, nor has he ever had pancreatitis or hepatitis. There is no family history of upper gastrointestinal (GI) malignancy, gallstone disease, hepatitis, or pancreatitis; however, his father has a long history of acid reflux disease. Robert tried over-the-counter simethicone, antacids, and a twice-daily H2RA for several weeks at a time without relief.

His physical examination is remarkable for mild epigastric tenderness without rebound or guarding. There are no signs of chronic liver disease, and there is no evidence of organomegaly, lymphadenopathy, or ascites. A CBC taken 3 months previously for an insurance examination was normal. Given the absence of warning signs on history and examination, the provider believed that Robert’s symptoms were likely due to occult acid reflux.

Robert is asked to start an exercise program to help lose weight and to help reduce stress at work. He is counseled on ways to eat more healthfully, advised to eat a light evening meal, and asked not to drink any caffeine after noon and to cut down his beer intake to just 1 each evening with dinner. He is given a prescription-strength PPI to take 30 minutes before breakfast.

Robert calls back 1 week later, saying that his symptoms of epigastric burning have essentially resolved. Six weeks later, he reports that all symptoms have resolved, and he feels dramatically better. He has lost 10 pounds, is exercising routinely, has stopped all alcohol intake, and is eating a smaller, more healthy dinner each evening. He is told to stop the PPI after a total of 8 weeks and to call back 8 weeks later. When he calls back, he reports that his symptoms have not returned. He continues with the lifestyle recommendations made previously.

### Table 1. Common Organic Causes Of Dyspepsia

- Esophagitis
- Gastric cancer
- Gastritis
- Gastroesophageal reflux disease
- Hepatitis
- Medications (eg, antibiotics, iron, NSAIDs, aspirin-containing products)
- Pancreatitis
- Peptic ulcer disease
- Other hepatobiliary disorders (eg, cholecystitis, choledocholithiasis)

**NSAIDs**, nonsteroidal anti-inflammatory drugs

### Table 2. Common Symptoms Of Dyspepsia

- Belching
- Bloating
- Early satiety
- Postprandial pressure, burning, or fullness
- Postprandial nausea
- Upper abdominal pain or discomfort (burning, pressure)

### Summary

This case highlights the common clinical scenario of occult acid reflux that causes dyspeptic symptoms. The absence of warning signs on history and physical examination enabled the provider to confidently initiate empiric therapy using a PPI. The resolution of symptoms, using the multicomponent approach described, also allowed the provider to safely avoid performing unnecessary and costly tests.
Functional Dyspepsia Defined

The definition of FD keeps changing, leading to some confusion on how best to define a patient with symptoms suggestive of FD. In an effort to incorporate new information about symptom expression and pathophysiology, the Rome criteria were recently revised (Rome III).

FD is now defined as the presence of symptoms thought to originate in the gastroduodenal region in the absence of any organic, systemic, or metabolic disease likely to explain the symptoms (Table 3). The Rome committee also introduced 2 new subcategories for FD—postprandial distress syndrome and epigastric pain syndrome. The usefulness of these new subcategories in clinical practice has not been prospectively studied, however, and time will tell whether the new definition improves our ability to diagnose or treat patients with FD.

Symptoms

Epigastric pain or discomfort is the hallmark symptom in patients with FD (Table 2). The word discomfort is important to emphasize, as many patients will not complain of pain, but rather state that they have a burning feeling, or pressure or fullness in the epigastriac area. Other common symptoms include early satiety, postprandial fullness and nausea, belching, bloating, and nausea. Frustratingly, symptoms of FD do not consistently predict underlying pathophysiology and do not reliably guide therapy.

Etiology and Pathophysiology

The precise etiology of FD is unknown, although limited data suggest that genetics, a prior infection, and environmental factors all play a role in symptom expression (Figure 1). Preliminary studies have demonstrated that patients with the G protein β3 subunit CC genotype are more likely to suffer from dyspepsia. This gene is involved in cell signaling, although the exact mechanisms by which alterations in this gene can produce symptoms of dyspepsia are not known.

Similar to data from studies of irritable bowel syndrome (IBS), Tack and colleagues have demonstrated that a prior gastrointestinal infection may be the cause of FD symptoms in up to 20% of cases. Although H. pylori infection may produce dyspeptic symptoms in a small subset of patients, little data support this pathogen as a cause of symptoms in most FD patients. Finally, psychological factors (eg, stress, anxiety, somatization, depression) may modulate symptom expression in some FD patients.

FD is a heterogeneous disorder, and no single pathophysiologic abnormality can explain the multiple symptoms expressed by FD patients. Research over the past 2 decades has identified several different pathophysiologic processes that may disturb normal gastric motor and sensory function in the upper GI tract of patients with FD (Figure 2).
Abnormal gastric accommodation may account for symptoms of epigastric fullness and pressure in some patients, and is present in approximately 40% of dyspeptic patients. In these patients, increased intragastric pressure may produce symptoms of fullness and early satiety. Antral hypomotility and delayed gastric emptying are present in other patients with FD, with several studies showing that up to 50% of patients with FD have a mild delay in gastric emptying.

Similar to many patients with IBS, patients with FD are characterized by visceral hypersensitivity. Balloon distention studies have demonstrated that up to two-thirds of patients with FD have heightened gastric perception, although this does not correlate with any specific symptom. Other patients with FD may have gastric electrical dysrhythmias, abnormalities of duodenal motor or sensory function, or disordered feedback from the proximal small intestine as the cause of their symptoms.

Case 3: “Maria”

Maria is a 38-year-old, previously healthy woman sent for further evaluation of abdominal discomfort and altered bowel habits. Her symptoms started nearly 9 months previously, after returning from a cruise to Mexico. Several passengers developed nausea, vomiting, and diarrhea after eating at a local restaurant. Maria had significant nausea, but did not vomit. The constant nausea resolved, but she now has symptoms of epigastric discomfort, early satiety, postprandial nausea, and frequent urgent diarrhea associated with lower abdominal discomfort.

Maria’s weight has been stable, and recent blood work—CBC, lipase, LFTs, albumin, and erythrocyte sedimentation rate (ESR)—was all normal. Stool studies on 2 separate occasions have been normal. Her physical examination is notable for mild epigastric tenderness without rebound or guarding, and mild tenderness over the sigmoid colon. Rectal examination is normal, and stool is Hemoccult negative.

A serum IgA and TTG antibody were ordered; these returned normal. Due to a family history of gallstone disease, and occasional sharp episodes of pain in the epigastric and right upper quadrant, an ultrasound of the liver, gallbladder, and pancreas was ordered and returned normal.

Separate 1-month trials of a twice-daily H2RA and a daily over-the-counter PPI did not improve her symptoms. Dietary changes (no lactose, low fructose, low fiber, no caffeine) improved her diarrhea symptoms to some degree, but did not improve her upper GI symptoms. Routine use of loperamide also improved her diarrhea but does not help her upper GI symptoms.

Upper endoscopy is scheduled due to her persistent symptoms and reveals very mild erythema in the antrum and body, but no evidence of esophagitis or ulcer disease. Random biopsies from throughout the stomach show multiple H. pylori organisms.
Maria is treated with a PPI twice daily and 2 separate antibiotics for 10 days. She returns to the clinic 1 month later, stating that her upper GI symptoms have not improved. She is instructed to eat small, frequent, low-fat meals, and is given low-dose desipramine with the diagnosis of FD. A stool antigen test confirms H. pylori eradication. The desipramine dose is gradually titrated upward over the next 6 weeks.

The patient returns for follow-up 8 weeks later, stating that her symptoms have improved and are now tolerable. Her weight has remained stable, and no new symptoms have developed. She has not had any side effects with desipramine, but has noted that she has been able to cut down on her scheduled doses of loperamide.

**Summary**

Maria has developed FD and mild IBS with diarrhea after an infectious illness. Risk factors for this include female gender, stress at the time of the infection, and the absence of vomiting. Persistent symptoms led to several diagnostic studies that were essentially normal. 

H. pylori was identified on upper endoscopy, although this was unlikely to be the cause of her symptoms. She was treated for H. pylori with the knowledge that eradication would decrease her risk for developing an ulcer and gastric cancer in the future, but that it would be unlikely to improve her dyspeptic symptoms. After failing empiric therapy with both an H2RA and a PPI, a tricyclic antidepressant (TCA) was used to treat her functional abdominal pain with improvement in symptoms. As will be discussed later in this review, symptoms of dyspepsia frequently overlap with those of IBS.

**Evaluation**

The cost-effective evaluation of a patient with dyspeptic symptoms has not been well studied. The recommendations made here are based on a review of the literature and clinical experience. Approximately two-thirds of patients sent for evaluation of dyspeptic symptoms will ultimately be diagnosed with FD.

**History**

Taking a careful history in a patient with dyspeptic symptoms serves 3 purposes: It 1) may help to differentiate organic dyspepsia from FD; 2) identifies warning signs that may lead to a more urgent evaluation, including specific diagnostic studies; and 3) documents whether symptoms arise from the upper (not lower) gastrointestinal tract.

Patients should be asked about a history of common disorders that may cause dyspeptic symptoms, including peptic ulcer disease, acid reflux, pancreatitis, hepatitis, and choledocholithiasis. A patient’s H. pylori status should be obtained, if known. The presence of other functional bowel disorders should be determined (eg, IBS, chronic constipation), because these often overlap. Patients should be asked about medication use; this should include over-the-counter agents, and complementary and alternative medications.

Alarm symptoms (eg, odynophagia, dysphagia, recurrent vomiting, unexplained weight loss, gastrointestinal bleeding, anemia, jaundice) should be identified. The presence of these symptoms identifies patients who require urgent or additional investigations. Unfortunately, a study has shown that the presence of alarm features may not reliably discriminate organic disease (ie, ulcer or malignancy) from functional disease. In addition, another study found that a computer model was similar to a clinician in distinguishing organic dyspepsia from FD, and neither was very good. Until these studies are confirmed, however, a careful history is still required, and the presence or absence of warning signs should still be established and documented.

**Physical Examination**

A guided physical examination should be performed in all patients with dyspeptic symptoms. The presence of a palpable mass, ascites, lymphadenopathy, splenomegaly, or evidence of chronic liver disease will likely alter the diagnosis and treatment plan of the patient. In addition, the examination may reassure the patient, although this has not been prospectively studied. Unfortunately, the clinical utility of the physical examination is limited in patients with dyspeptic symptoms. Epigastric tenderness on palpation has been shown to be of little diagnostic value for upper gastrointestinal pathology.

**Laboratory Studies**

No studies have prospectively evaluated the utility of routinely performing serologic tests in patients with dyspeptic symptoms. However, if it has not
**Figure 3. Management Algorithm for Patients With Uninvestigated Dyspepsia**

*H. pylori*, *Helicobacter pylori*; H2RA, histamine type-2-receptor antagonist; PPI, proton pump inhibitor

---

**a** In all patients, management includes avoidance of ulcerogenic agents, patient reassurance, stress reduction, and smoking cessation.

**b** All empiric drug trials should be stopped after 6 to 8 weeks, and endoscopy should be performed if symptoms return or continue. Based on reference 50.
recently been performed, a CBC should be ordered, as the presence of anemia will change the diagnostic evaluation. A serum lipase should be ordered if pancreatitis is being considered, and LFTs should be requested if a hepatobiliary source of pain is considered a possible cause of symptoms.

**Testing for H. pylori**

The American Gastroenterological Association (AGA) recommends that dyspeptic patients under 55 years of age without alarm features undergo testing for *H. pylori*, if the local prevalence of infection is more than 10%. In patients under age 40, either the stool antigen test or the urea breath test should be used, since the serum *H. pylori* test has low diagnostic specificity. A recent systematic review demonstrated that testing and treating for *H. pylori* may reduce the number of upper endoscopy procedures and lead to similar outcomes as with prompt endoscopy. Of note, patients who are positive for *H. pylori* (using the stool antigen test or the urea breath test) and who fail therapy warrant upper endoscopy.

**Endoscopy**

Dyspeptic patients with warning signs, those with onset of new symptoms after age 55, and those without warning signs who fail initial empiric therapy require diagnostic testing. Several studies have shown that upper endoscopy is the best diagnostic test initially in these patients, because endoscopy is more sensitive than barium studies for detecting mucosal abnormalities, may lead to more appropriate therapy earlier, may reassure the patient, and has a low rate of complications (Figure 3).

The utility of upper endoscopy in patients with uninvestigated dyspepsia was confirmed by a recent study of 1,040 primary care patients with dyspeptic symptoms who underwent upper endoscopy. Clinically significant endoscopic findings were present in 58% of patients, with esophagitis being the most common finding (43%), followed by *H. pylori* infection (30%), and peptic ulcer disease (5%).

**Other Diagnostic Studies**

Patients with abnormal liver tests and/or symptoms suggestive of hepatobiliary disease should undergo ultrasonography of the gallbladder. Patients with persistent symptoms of nausea, vomiting, and early satiety may have gastroparesis, and a solid-phase gastric emptying scan is considered the most readily available test to evaluate gastric emptying. At least one-third of patients with FD have a mild delay in gastric emptying, although accelerating gastric emptying may not improve symptoms.

**Overlapping Disorders**

Patients with FD frequently suffer from other functional disorders as well. IBS, acid reflux disease, and chronic constipation may coexist with overlapping symptoms. The presence of multiple overlapping symptoms suggestive of coexisting functional bowel disorders should reassure the clinician that the upper GI symptoms are more likely due to FD, rather than an organic process.

**Patient Concerns, Quality of Life, And Economic Burden**

Patients with FD have significant misconceptions about their disorder. Up to 30% of patients believe that FD increases their risk of developing cancer or ulcer disease. Although FD does not convey any increased risks to long-term health, several studies have demonstrated that FD significantly reduces a patient’s quality of life. In contrast to other functional disorders (eg, IBS), the economic burden of evaluating and treating FD is not as well characterized. A study from the United Kingdom estimated a societal cost of £1 billion each year, primarily due to lost productivity. A recent study, presented at the 2007 American College of Gastroenterology meeting, found that the economic burden of FD in the United States is quite high as well.

**Case 4: “Julia”**

Julia is a 37-year-old woman sent for evaluation of persistent upper abdominal pain. The pain started 2 years previously, although she cannot recall a precipitating event. Symptoms of pressure and fullness in the upper abdomen are present nearly every day, and eating makes her symptoms worse. Julia also reports postprandial nausea and early satiety, in addition to symptoms of upper abdominal bloating, mild regurgitation, and heartburn.

A stool test for *H. pylori* was negative. Results from several sets of blood work over the past 2 years (CBC, lipase, LFTs, electrolytes, and ESR) have been normal. Recent serologic tests for celiac disease were negative. A right upper quadrant ultrasound and an upper endoscopy, including biopsies of the stomach and small intestine, performed over 1 year ago, were normal.

Two separate 6-week trials of an H2RA and then a prescription-strength PPI were not helpful. Julia was placed on a low-dose TCA; although this reduced her discomfort to some degree, it caused significant sedation, and she stopped the medication. Metoclopramide was started due to symptoms of postprandial nausea; however, this worsened her mild yet chronic anxiety.

At the initial interview, Julia states that, despite her daily symptoms, she has gained 15 pounds over the past year. She is very frustrated by her symptoms, and has become depressed. She is worried that her symptoms reflect something serious, and is convinced that she will likely develop stomach cancer because of her symptoms. She states that she has tried acupuncture, magnet therapy, and several herbal supplements, without any benefit.
Julia's physical examination is unremarkable, other than mild epigastric tenderness. Because of the persistent symptoms, repeat blood work is ordered—CBC, amylase, lipase, LFTs, electrolytes, blood urea nitrogen/creatinine, ESR, and thyroid-stimulating hormone—and these tests all return normal.

Julia is worried that she has an ulcer because of the persistent abdominal pain and nausea. After a lengthy discussion about the risks and benefits of a second endoscopy and the very low probability of finding an organic cause for her symptoms, endoscopy is performed, with repeat biopsies of the stomach and small intestine, and the results are normal. After a lengthy discussion regarding the risks, benefits, and utility of further diagnostic testing, a computed tomography scan of the abdomen and pelvis is ordered; this returns normal.

Julia seems somewhat reassured by the normal tests, but is still concerned that her pain and nausea persists. A solid-phase gastric emptying scan is ordered to rule out gastroparesis; this returns normal.

Julia is started on domperidone (10 mg, by mouth, 3 times per day) for the nausea. Paroxetine is started to help with her mild depression and anxiety. Medical options for her functional pain are discussed, but Julia wants to limit the number of medications she takes. She is asked to follow a low-fat diet and eat smaller, more frequent meals. An appointment is made to see a cognitive behavioral therapist to help with coping skills.

Calling in 4 weeks later, Julia says her nausea is better, as is her anxiety. Her pain is still present, although she is less worried that it represents something serious. Both her domperidone and paroxetine doses are increased, and she continues to see a behavioral therapist weekly. At the next follow-up visit 2 months later, the patient reports a 75% improvement in symptoms.

Summary

This case highlights the chronicity of FD symptoms, the difficulty treating these symptoms, and patients' frequent misconceptions about their disorder (ie, Julia's fear that her symptoms represent cancer). A series of diagnostic tests was ordered in stepwise fashion after careful discussion with the patient about the risks, benefits, and utility of diagnostic testing. A solid-phase gastric emptying scan was normal in this patient, although it can be mildly delayed in up to one-third of patients with FD.

Domperidone was used to improve symptoms of nausea, and paroxetine was used to help treat Julia's mild anxiety and depression. Although not tested in formal trials, gabapentin, tramadol, and duloxetine have been used off-label to treat neuropathic pain in patients with FD.

Treatment Options

Despite its prevalence, and the significantly poor quality of life and economic burden that it imposes, FD has yet to have a defined or tested all-purpose algorithm for its treatment. In addition, no medication discussed in this section is approved by the US Food and Drug Administration for the treatment of FD. The following summarizes the literature on treatment options for FD.

Diet

Most patients with FD have symptoms associated with ingestion of food. As such, several dietary recommendations are often made, although no clinical trial has formally evaluated specific
dietary interventions for the treatment of FD. Limited data suggest that dietary fat may increase gastric sensitivity to distention and cause dyspeptic symptoms. If 
Patients with FD often note an improvement by eating low-fat meals, and more frequent, smaller meals.

**ERADICATION OF H. PYLORI**

As noted previously, the AGA recommends testing and treating for *H. pylori* as the first step in the management of younger patients with uncomplicated dyspeptic symptoms. If H2RAs fail to alleviate symptoms, then most providers use a PPI for the treatment of dyspepsia. Short-term risks of this strategy are low, and this may provide relief of symptoms in a small number of people, many of whom may have had silent acid reflux. As a group, PPIs are approximately 10% better than placebo at relieving dyspeptic symptoms with an NNT of 9. However, long-term use of PPIs can be expensive, and emerging data indicate that long-term use of PPIs may place patients at increased risk for *Clostridium difficile* colitis, community-acquired pneumonia, and hip fractures.

**ANTIDEPRESSANTS**

TCAs may relieve symptoms of dyspepsia in patients who have failed H2RAs or prokinetics. Although their precise mechanism is unknown, these drugs may relieve symptoms of visceral hypersensitivity and reduce intragastric pressure. A recent review of available studies found that symptoms of dyspepsia improved with TCAs, although large clinical trials are not available. In general, lower doses of TCAs are used to treat FD than depression. Selective serotonin reuptake inhibitors have not been studied for the treatment of FD.

**HYPNOTHERAPY AND PSYCHOLOGICAL THERAPY**

Hypnotherapy may relieve dyspeptic symptoms in some patients. A recent study found that hypnotherapy was better than medical and supportive therapy at improving quality of life and symptom scores. Although not well studied, psychological therapies, including cognitive behavioral therapy, may relieve symptoms of dyspepsia by reducing coexisting stress and anxiety.

**Conclusion**

Dyspepsia is a highly prevalent disorder seen by all gastroenterologists. Our understanding of the pathophysiology of this disorder remains incomplete, and our ability to adequately treat our patients’ symptoms is hampered by our limited armamentarium. The cases presented in this review, combined with currently available treatment guidelines, should enable health care providers to confidently diagnose patients with dyspepsia. Continued research efforts focusing on the etiology and pathophysiology of this disorder should lead to more effective treatment options in the future.

**References**


AUTHOR DISCLOSURE—In the past 2 years, Dr. Lacy has served as a consultant for Novartis and Takeda; he has received unrestricted investigator-initiated funding from AstraZeneca, Medtronic, and Novartis.

DISCLAIMER—This review is designed to be a summary of information, and represents the opinions of the author. Although detailed, the review is not exhaustive. Readers are strongly urged to consult any relevant primary literature, the complete prescribing information available in the package insert of each drug, and the appropriate clinical protocols. No liability will be assumed for the use of this review, and the absence of typographical errors is not guaranteed. Copyright © 2008, McMahon Publishing, 545 West 45th Street, 8th Floor, New York, NY 10036. Printed in the USA. All rights reserved, including right of reproduction, in whole or in part, in any form.
Patient Guide to Dyspepsia

Dyspepsia is a common condition that many people experience at some point in their life. The most common symptom of dyspepsia is upper abdominal discomfort, which may feel like an ache, pressure, pain, or burning sensation in the upper middle part of the abdomen. Other symptoms include feeling full after eating only a modest-sized meal and nausea after eating. When symptoms occur and there is no obvious cause such as an ulcer, then the condition is called functional or non-ulcer dyspepsia.

What causes dyspepsia?
For some people, dyspepsia can be caused by what and how they eat, such as eating too much, eating too fast, swallowing too much air when eating, eating high-fat foods, or eating during stressful situations. Some medications can also irritate the stomach. Being tired or stressed, smoking, or drinking too much alcohol or caffeinated beverages can trigger dyspepsia or make it worse.

Dyspepsia can also be caused by a stomach ulcer or by acid reflux disease, a condition in which stomach acid backs up into the tube connecting the mouth to the stomach (esophagus). Rarely, dyspepsia is caused by stomach cancer. Your doctor may do some tests to find out if you have these conditions.

How is dyspepsia treated?
Treatment of dyspepsia depends on what your doctors find out is causing it. Some medicines can cause dyspepsia; your doctor may ask you to stop these medications. If you have a stomach ulcer, it can usually be treated with medication. You may need to take an antibiotic if you have a bacterial infection. If you have functional dyspepsia, your physician may prescribe medications that affect stomach muscle and nerve function. Remember to take medicines exactly the way your doctor tells you. If you take an antibiotic, make sure to take every pill until you are finished, even if you already feel better.

Do my symptoms put me at risk for a serious disease?
Although it is possible that your symptoms are caused by something serious like an ulcer or cancer, your doctor will test to find whether this is the case. However, the symptoms you experience regularly because of functional dyspepsia will not put you at higher risk for developing an ulcer or cancer.

Q & A

Common Symptoms of Dyspepsia

- Belching
- Bloating
- Burning sensation, discomfort in upper abdomen or lower chest
- Early feeling of fullness when eating
- Gas
- Heartburn
- Nausea
- Upset stomach

Resources

- Mayo Clinic: www.mayoclinic.org/dyspepsia
- International Foundation for Functional Gastrointestinal Disorders: www.aboutgimotility.org/site/about-gi-motility