



Approach to the evaluation of dysphagia in adults

AUTHOR: Ronnie Fass, MD SECTION EDITOR: Mark Feldman, MD, MACP, AGAF, FACG DEPUTY EDITOR: Kristen M Robson, MD, MBA, FACG

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Sep 2023.** This topic last updated: **Oct 04, 2023.**

INTRODUCTION

Dysphagia is an alarm symptom that warrants prompt evaluation to define the exact cause and initiate appropriate therapy. It may be due to a structural or functional abnormality that interferes with the passage of solids or liquids from the oral cavity to the stomach. Patients' complaints range from the inability to initiate a swallow to the sensation of solids or liquids being hindered during their passage through the esophagus into the stomach.

This topic will review the evaluation of patients with esophageal dysphagia. Our recommendations are largely consistent with guidelines developed by several professional societies [1-4].

The pathogenesis, diagnosis, and evaluation of patients with oropharyngeal dysphagia are discussed separately. (See "Oropharyngeal dysphagia: Etiology and pathogenesis" and "Oropharyngeal dysphagia: Clinical features, diagnosis, and management".)

DEFINITIONS

The terms dysphagia, odynophagia, and globus are defined as follows:

- Dysphagia is a subjective sensation of difficulty or abnormality of swallowing.
- Odynophagia is pain with swallowing.

• Globus sensation is a nonpainful sensation of a lump, tightness, foreign body or retained food bolus in the pharyngeal or cervical area [5]. Globus, on the other hand, is a functional esophageal disorder that is characterized by globus sensation but without an underlying structural abnormality, gastroesophageal reflux disease, eosinophilic esophagitis, or a major esophageal motility disorder [6]. (See "Globus sensation".)

AGE OF ONSET

Dysphagia in older adults should **not** be attributed to normal aging. Aging alone causes mild esophageal motility abnormalities, which are rarely symptomatic [7].

In a study of over 31,000 adults who completed an online survey, 16 percent of individuals reported dysphagia at a mean age of 46.5 years [8]. Among those with dysphagia, 16 percent reported severe symptoms.

ACUTE DYSPHAGIA

The acute onset of inability to swallow solids and/or liquids, including secretions, suggests impaction of food or foreign body in the esophagus and requires immediate attention.

Food impaction is the most common cause for acute onset of dysphagia in adults. (See "Ingested foreign bodies and food impactions in adults".)

Incidence and epidemiology — The estimated annual incidence of esophageal food impaction is 25 per 100,000 persons per year and with a higher incidence in males compared with females (1.5:1) [9,10]. The incidence increases with age, especially after the seventh decade.

Clinical presentation — Patients usually develop symptoms after ingesting meat (most commonly beef, chicken, and turkey), which completely obstructs the esophageal lumen, resulting in apprehension and expectoration of saliva.

Management — Administration of glucagon intravenously can be initially attempted to relax the lower esophageal sphincter and promote passage of the food bolus [11]. The food impaction can be removed during upper endoscopy using grasping devices (either en bloc or piecemeal, depending upon the consistency of the bolus), or it can be gently pushed into the stomach using the endoscope [9,12]. Management of food impaction is discussed in detail separately. (See "Ingested foreign bodies and food impactions in adults", section on 'Food bolus'.)

EVALUATION OF NONACUTE DYSPHAGIA

Distinguishing oropharyngeal from esophageal dysphagia — The first step in evaluating patients with nonacute dysphagia is to determine if the symptoms are due to oropharyngeal or esophageal dysphagia based on the patient's answers to the questions in the following table (table 1) [13]:

- **Oropharyngeal dysphagia** Oropharyngeal or transfer dysphagia is characterized by these features:
 - Patients have difficulty initiating a swallow or report food getting stuck immediately after a swallow.
 - Patients may point toward the cervical region as the site of their symptoms.
 - Swallowing may be accompanied by nasopharyngeal regurgitation, aspiration, and a sensation of residual food remaining in the pharynx.
 - Oral dysfunction can lead to drooling, food spillage, sialorrhea, piecemeal swallows, and dysarthria.
 - Pharyngeal dysfunction can lead to coughing or choking during food consumption, and dysphonia.

The evaluation of patients with oropharyngeal dysphagia is discussed in detail separately (table 2). (See "Oropharyngeal dysphagia: Etiology and pathogenesis" and "Oropharyngeal dysphagia: Clinical features, diagnosis, and management".)

- **Esophageal dysphagia** Patients with esophageal dysphagia commonly report the following:
 - Difficulty swallowing several seconds after initiating a swallow, and
 - A sensation that foods and/or liquids are being obstructed or delayed in their passage from the upper esophagus to the stomach.

Patients may point to the suprasternal notch or to an area behind the lower part of the sternum as the site of obstruction. While retrosternal dysphagia usually corresponds with the location of the lesion, suprasternal dysphagia is commonly referred from below [14]. Esophageal dysphagia arises from within the body of the esophagus, the lower

esophageal sphincter, or cardia. A large number of conditions are associated with esophageal dysphagia (table 3).

Characterizing the symptoms — Dysphagia should be characterized according to types of food that produce symptoms, the time course (ie, progressive or intermittent), severity, and associated symptoms (algorithm 1):

- Solid, liquid, or both? A critical component of the medical history is determining the types of food that produce symptoms (ie, solids, liquids, or both). Dysphagia to both solids and liquids from the onset of symptoms is probably due to a functional disorder of the esophagus. Dysphagia to solids only is usually present when the esophageal lumen is narrowed to 13 mm or less (eg, by a stricture, ring, web, or extrinsic compression).
- Progressive or intermittent? It is important to ask if the symptoms are intermittent or are gradually progressive. Progressive dysphagia, beginning with dysphagia to solids followed by dysphagia to liquids, is usually caused by a peptic stricture or obstructing lesion [15]. Symptoms of peptic stricture are slowly and gradually progressive, whereas those due to a malignancy progress more rapidly [16].

Intermittent dysphagia may be related to a lower esophageal ring or web. Patients with motility disorders may also exhibit progressive dysphagia (eg, achalasia) or may exhibit intermittent or nonprogressive dysphagia (eg, distal esophageal spasm).

Psychologic factors may influence the expression and severity of dysphagia symptoms. In a study including 236 patients with dysphagia who completed psychologic and symptombased questionnaires and were evaluated with esophageal manometry, esophageal hypervigilance and visceral anxiety were the strongest predictors of dysphagia severity (eg, number of food impactions or emergency department visits) [17]. The correlation was observed in patients with and without major esophageal motility disorders (eg, achalasia). While psychologic factors have been identified as predictors of disease severity for patients with other gastrointestinal disorders (eg, gastroesophageal reflux disease, irritable bowel syndrome), this study has suggested that anxiety and hypervigilance may also contribute to patients' perception of dysphagia severity [6,18,19]. (See "Pathophysiology of irritable bowel syndrome", section on 'Psychosocial dysfunction'.)

Associated symptoms — Associated symptoms or findings can help to narrow the differential diagnosis. These may include:

- Heartburn
- Weight loss

- Hematemesis
- Anemia
- Regurgitation of food particles, and
- Respiratory symptoms

As an example, chronic heartburn in a patient with dysphagia may be a clue to complications of gastroesophageal reflux disease, such as erosive esophagitis, peptic stricture, or adenocarcinoma of the esophagus. Patients with peptic stricture usually have a history of heartburn and regurgitation and later weight loss, while patients with esophageal cancer tend to be older males with significant, rapid weight loss [2]. (See "Complications of gastroesophageal reflux in adults".)

Approach to the evaluation of dysphagia in adults - UpToDate

SYMPTOM-BASED DIFFERENTIAL DIAGNOSIS

Solids only with progressive symptoms

Esophageal stricture — Dysphagia to solids that is gradually progressive is suggestive of an esophageal stricture, which may be related to acid reflux, radiation therapy, or eosinophilic esophagus.

Peptic stricture — Peptic stricture is a complication of gastroesophageal reflux disease (GERD) and results from the healing process of severe erosive esophagitis. This benign esophageal stricture is usually found in close proximity to the esophagogastric junction (EGJ). The development of peptic stricture among patients with reflux has been associated with older age, male sex, and longer duration of reflux symptoms [20]. Peptic strictures have been observed in a number of other conditions that lead to increased esophageal acid exposure (eg, systemic sclerosis, Zollinger-Ellison syndrome, nasogastric tube placement, and after Heller myotomy or peroral esophageal myotomy for achalasia). The management of benign esophageal strictures is discussed separately. (See "Endoscopic interventions for nonmalignant esophageal strictures in adults".)

Less common causes of stricture — Patients undergoing radiation therapy for thoracic or head and neck tumors are at risk for developing esophagitis and esophageal strictures. In the acute setting, patients may develop esophagitis resulting in dysphagia and odynophagia. In some patients, chronic ischemia and fibrosis lead to chronic radiation esophagitis, which may present as esophageal ulcerations or strictures in the proximal esophagus [21]. (See "Overview of gastrointestinal toxicity of radiation therapy", section on 'Esophagitis'.) Benign esophageal strictures may also be related to caustic ingestions, postsurgical resection for esophageal or laryngeal cancer, drug-induced stricture, or eosinophilic esophagitis. (See 'Eosinophilic esophagitis' below and "Caustic esophageal injury in adults", section on 'Esophageal strictures'.)

Carcinoma — Cancer of the esophagus or gastric cardia is associated with rapidly progressive dysphagia, initially for solids and later for liquids. In addition, patients may have chest pain, odynophagia, anemia, anorexia, and significant weight loss.

An achalasia-like syndrome (pseudoachalasia) has been described in patients with adenocarcinoma of the cardia due to microscopic infiltration of the myenteric plexus or the vagus nerve [22]. Certain features increase the likelihood that a patient has pseudoachalasia due to malignancy [23]. These include short duration of symptoms (ie, less than six months), presentation after age 60, excessive weight loss in relation to the duration of symptoms, and difficult passage of the endoscope through the gastroesophageal junction. In such cases, endoscopic ultrasonography with fine-needle aspiration (EUS-FNA) should be performed to diagnose an underlying malignancy. (See "Achalasia: Pathogenesis, clinical manifestations, and diagnosis" and "Epidemiology and pathobiology of esophageal cancer".)

Solids only with intermittent symptoms — Dysphagia to solid foods only that is intermittent in nature may be caused by eosinophilic esophagitis, esophageal ring or web, or a vascular anomaly.

Eosinophilic esophagitis — Up to 15 percent of patients being evaluated for dysphagia with endoscopy are found to have eosinophilic esophagitis [24-26]. Endoscopic findings associated with eosinophilic esophagitis include:

- Stacked circular rings ("feline" esophagus) (picture 1)
- Strictures (particularly proximal strictures) (image 1)
- Linear furrows
- Whitish papules (picture 1) (representing eosinophil microabscesses)
- Small caliber esophagus (see "Clinical manifestations and diagnosis of eosinophilic esophagitis (EoE)", section on 'Endoscopy')

Individual endoscopic features suggestive of eosinophilic esophagitis have low sensitivity ranging from 15 to 48 percent but high specificity ranging from 90 to 95 percent [27]. The diagnosis of eosinophilic esophagitis is established by upper endoscopy and esophageal biopsy, which demonstrates an increased number of eosinophils (>15 per high power field). (See "Clinical manifestations and diagnosis of eosinophilic esophagitis (EoE)", section on 'Histology'.)

Esophageal webs and rings — Patients with esophageal rings and webs have intermittent dysphagia for solids. Esophageal webs have been described in association with iron deficiency (ie, the Plummer-Vinson or Patterson-Kelly syndrome) in which case anemia, koilonychia, or other manifestations of iron deficiency may be present (image 2) [28]. (See "Causes and diagnosis of iron deficiency and iron deficiency anemia in adults", section on 'Clinical manifestations'.)

A detailed discussion of esophageal rings and webs is presented separately. (See "Esophageal rings and webs".)

Esophageal webs and rings can partially or completely compromise the esophageal lumen [29]. They can be solitary or multiple.

- An esophageal web is a thin mucosal fold that protrudes into the esophageal lumen and is covered with squamous epithelium. Webs most commonly occur anteriorly in the cervical esophagus, causing focal narrowing in the postcricoid area (image 3).
- Esophageal rings are typically mucosal structures but in rare cases are muscular. Schatzki rings are found at the gastroesophageal junction. These types of rings are smooth, thin (<4 mm in axial length), and covered with squamous mucosa above and columnar epithelium below (picture 2 and image 4) [30].

An esophageal web/ring is diagnosed on barium esophagram and/or upper endoscopy and appears as a focal, thick constriction of variable luminal diameter [31]. Rings are usually found at or a few centimeters above the squamocolumnar junction. Endoscopy is less sensitive than the barium esophagram in detecting esophageal rings and a ring may be missed unless the lower esophagus is widely distended [32].

Cardiovascular abnormalities — Vascular anomalies can cause dysphagia by compressing the esophagus but are rare [33]. Some of the aberrant vessels form complete rings, while others form incomplete rings around the esophagus [34]. (See "Vascular rings and slings".)

- Complete vascular ring anomalies include a double aortic arch, right aortic arch with retroesophageal left subclavian artery and left ligamentum arteriosum, and right aortic arch with mirror-image branching and left ligamentum arteriosum [34].
- Dysphagia lusoria is rare and is due to an aberrant right subclavian artery that passes dorsally between the esophagus and the spine [33,35]. Extrinsic compression of the esophagus may be noted on barium esophagram, and the diagnosis can be established by endoscopic ultrasonography or computed tomography (CT) scan.

• In older adults, severe atherosclerosis or a large aneurysm of the thoracic aorta can result in impingement on the esophagus and produce dysphagia ("dysphagia aortica").

When due to congenital causes, symptoms usually develop during childhood, but they may also develop in adults. Most patients with an aberrant subclavian artery are symptom-free throughout their lives [36]. However, coughing, dysphagia, thoracic pain, or even Horner's syndrome may develop at an older age [37]. If symptoms are intractable, surgical intervention may be necessary. (See "Vascular rings and slings", section on 'Treatment'.)

Liquids alone or with solid dysphagia — Dysphagia to liquids alone or to solids and liquids may be related to either an esophageal motility disorder such as achalasia, EGJ outflow obstruction, absent contractility, or to a functional disorder.

Achalasia — Primary achalasia is a disease of unknown etiology in which there is a loss of normal peristalsis in the distal esophagus and a failure of lower esophageal sphincter (LES) relaxation with swallowing.

Achalasia is an uncommon disorder that can occur at any age but is usually diagnosed in patients between 25 and 60 years. Males and females are affected with equal frequency. Progressively worsening dysphagia for solids (91 percent) and liquids (85 percent) and regurgitation of bland, undigested food or saliva are the most frequent symptoms in patients with achalasia. Other symptoms include chest pain, heartburn, and difficulty belching.

Upper endoscopy, barium esophagram (especially timed barium esophagram) and functional lumen imaging probe (FLIP) are complementary tests to manometry in the diagnosis of achalasia [38]. Findings on barium esophagram that are suggestive of achalasia include a dilated esophagus that terminates in a beak-like narrowing (ie, "bird-beak" appearance), aperistalsis, and poor emptying of barium from the esophagus (image 5 and image 6). However, barium esophagram may be nondiagnostic in up to one-third of patients [39].

Upper endoscopy is performed to exclude pseudoachalasia, and those patients without evidence of mechanical obstruction can then undergo esophageal manometry to confirm the diagnosis. Manometric findings of achalasia include lack of normal peristalsis in the distal twothirds of the esophagus and incomplete LES relaxation resulting in elevated integrated relaxation pressure (IRP). (See "Achalasia: Pathogenesis, clinical manifestations, and diagnosis".)

We use FLIP for evaluating patients with suspected achalasia but inconclusive findings on esophageal manometry [40]. (See "Functional lumen imaging probe (FLIP) for adults with esophageal disorders".) **Disorders of peristalsis** — If upper endoscopy with esophageal biopsies is normal in a patient with dysphagia to solids and/or liquids, further evaluation with esophageal manometry and/or barium esophagram should be obtained (see "High resolution manometry"):

 Distal esophageal spasm and hypercontractile esophagus – Esophageal manometry is obtained to establish the diagnosis of these spastic esophageal motility disorders. The specific manometric criteria to diagnose distal esophageal spasm and hypercontractile esophagus are discussed separately. (See "Distal esophageal spasm and hypercontractile esophagus".)

Distal esophageal spasm (DES) and hypercontractile esophagus can cause intermittent, nonprogressive dysphagia to solids and liquids. Patients may also report associated chest pain [41]. In patients with DES, the barium esophagram may show severe non-peristaltic contractions, which may produce striking abnormalities in the barium column. These findings have resulted in descriptions such as "rosary bead" or "corkscrew" esophagus (image 7 and image 8). However, radiographic studies may be normal among patients with DES or be abnormal in patients with normal manometry testing; as a result, barium esophagram is neither sensitive nor specific in this setting.

- **EGJ outflow obstruction** This disorder is defined as failure or incomplete opening of the EGJ with normal, hypercontractile, or hypocontractile peristalsis. The underlying cause may be incompletely expressed achalasia or mechanical obstruction, and symptoms include continuous or intermittent dysphagia for solids and liquids [40].
- Ineffective esophageal motility By high-resolution esophageal manometry, ineffective motility is defined as >70 percent ineffective swallows or >50 percent failed peristalsis [40]. The manometric diagnosis of ineffective esophageal motility does not always correlate with symptoms or impaired esophageal function. In one study, only 30 percent of patients with ineffective esophageal motility reported dysphagia. Moreover, studies using esophageal intraluminal impedance testing have shown that up to 68 percent of liquid and 59 percent of viscous swallows in such patients showed normal bolus transit [42].
- Absent contractility High-resolution esophageal manometry demonstrates lack of esophageal body peristalsis (ie, 100 percent failed swallows), which may be idiopathic or can be seen in patients with systemic disorders (eg, systemic sclerosis or mixed connected tissue syndrome). Absent contractility can lead to persistent or intermittent dysphagia for both solids and liquids. (See 'Systemic sclerosis (scleroderma)' below.)

Systemic sclerosis (scleroderma) — Patients with systemic sclerosis often have a history of heartburn and progressive dysphagia to both solids and liquids secondary to the underlying

motility abnormality or the presence of erosive esophagitis complicated by peptic stricture, which may occur in up to 50 percent of these patients [43]. The diagnosis of systemic sclerosis is suggested by the presence of skin thickening and hardening (sclerosis) that is not confined to one area (ie, not localized scleroderma). The diagnosis is supported by the presence of extracutaneous features and characteristic serum autoantibodies. Endoscopy may show erosive esophagitis, Barrett's esophagus, or a peptic stricture resulting from acid reflux. (See 'Esophageal stricture' above and "Clinical manifestations and diagnosis of systemic sclerosis (scleroderma) in adults", section on 'Evaluation for suspected systemic sclerosis' and "High resolution manometry", section on 'Absent contractility'.)

Esophageal involvement is present in up to 90 percent of patients with systemic sclerosis [44,45]. Scleroderma primarily involves the smooth muscle layer of the gut wall, resulting in atrophy and sclerosis of the distal two-thirds of the esophagus [44]. Absent peristalsis in the distal two-thirds of the esophagus and poor bolus transit may be seen on esophageal manometry and impedance, as well as low or absent lower esophageal sphincter pressure [46]. The proximal esophagus (striated muscle) is spared and exhibits normal motility. (See "Gastrointestinal manifestations of systemic sclerosis (scleroderma)" and "High resolution manometry", section on 'Absent contractility'.)

Functional dysphagia — According to the Rome IV criteria, functional dysphagia is defined by the following:

- A sense of solid and/or liquid food lodging, sticking, or passing abnormally through the esophagus.
- No evidence that an esophageal mucosal or structural abnormality is the cause of the symptom.
- No evidence that GERD or eosinophilic esophagitis is the cause of the symptom.
- Absence of a major esophageal motor disorder (achalasia, EGJ outflow obstruction, distal esophageal spasm, hypercontractile esophagus, ineffective esophageal motility, and absent peristalsis) [6].

All criteria must be fulfilled for the past three months with symptom onset at least six months prior to the diagnosis and with a frequency of at least once a week.

Symptoms of dysphagia may be intermittent or present after each meal. Patients should be reassured and instructed to avoid precipitating factors and chew well. In our experience, symptoms may improve with time. In patients with severe symptoms, despite these measures,

a trial of a smooth muscle relaxant, such as a calcium channel blocker or tricyclic antidepressant, can be offered. This approach is similar to the initial treatment of distal esophageal spasm, which is discussed separately. (See "Distal esophageal spasm and hypercontractile esophagus", section on 'Management'.)

Empiric dilation with a mechanical (push-type or Bougie) dilator can be offered, but symptom response is variable. (See "Endoscopic interventions for nonmalignant esophageal strictures in adults".)

Odynophagia and dysphagia — Both infectious esophagitis and medication-induced esophagitis commonly present with dysphagia accompanied by painful swallowing.

Infectious esophagitis — Patients with infectious esophagitis, especially due to herpes simplex virus, usually present with odynophagia and/or dysphagia [47,48]. Other causes of infectious esophagitis include cytomegalovirus and *Candida* species. Although *Candida* species are the most common fungal cause of esophagitis, other fungal infections including cryptococcosis, histoplasmosis, blastomycosis, and aspergillosis have rarely been described [49]. Other pathogens, such as mycobacteria, occasionally cause esophagitis in immunosuppressed patients [50]. (See "Herpes simplex virus infection of the esophagus" and "Esophageal candidiasis in adults", section on 'Epidemiology'.)

Medication-induced esophagitis — Medications in pill form may become lodged in the esophagus for a prolonged period and then cause direct esophageal mucosal injury. Symptoms may include dysphagia, odynophagia, and/or retrosternal pain. Patients often have a history of swallowing a pill without water, commonly at bedtime. The diagnosis and management of medication-induced esophagitis is discussed separately. (See "Pill esophagitis".)

Less common causes — Dysphagia and painful swallowing may be reported by patients with reflux esophagitis or esophageal Crohn disease [51].

OTHER CAUSES OF NONSPECIFIC DYSPHAGIA

Lymphocytic esophagitis — Lymphocytic esophagitis is characterized by the presence of a dense peripapillary lymphocytic infiltrate and peripapillary spongiosis involving the lower two-thirds of the esophageal epithelium and the absence of significant neutrophilic or eosinophilic infiltrates [52]. While lymphocytic esophagitis is being increasingly recognized on histopathology in adults and has been associated with dysphagia, it is unclear if it is a distinct clinical entity and its etiology is unknown [53-56].

In one retrospective study of 129,252 adults who had undergone an upper endoscopy, 0.1 percent had lymphocytic esophagitis on biopsy [53]. As compared with patients with normal esophageal biopsies, patients with lymphocytic esophagitis were significantly more likely to be older (63 versus 55 years), to have presented with dysphagia (53 versus 33 percent), and were significantly less likely to have gastroesophageal reflux disease (GERD; 19 versus 38 percent).

Sjögren's disease — Approximately three-quarters of patients with Sjögren's disease have associated dysphagia [57,58]. Defective peristalsis has been demonstrated in one-third or more of patients with primary Sjögren's disease [59]. Xerostomia appears to exacerbate swallowing discomfort but does not appear to correlate with dysphagia [57]. The diagnosis of Sjögren's disease is discussed separately.

- (See "Clinical manifestations of Sjögren's disease: Exocrine gland disease".)
- (See "Clinical manifestations of Sjögren's disease: Extraglandular disease", section on 'Gastrointestinal tract'.)
- (See "Diagnosis and classification of Sjögren's disease", section on 'Diagnosis'.)

APPROACH TO DIAGNOSTIC TESTING

The approach to diagnostic testing to determine the etiology of esophageal dysphagia is based upon the medical history (algorithm 2).

Pre-endoscopy barium esophagram — We perform a barium contrast esophagram (barium swallow) as the **initial test** (prior to upper endoscopy) in patients with the following:

- History/clinical features of proximal esophageal lesion (eg, surgery for laryngeal or esophageal cancer, Zenker's diverticulum, or radiation therapy).
- Known complex (tortuous) stricture (eg, postcaustic injury or radiation therapy) [1].

In these patients, the blind intubation of the proximal esophagus during upper endoscopy may be associated with the risk of perforation due to upper esophageal pathology. However, it is important to note that performing a barium esophagram prior to an upper endoscopy in such patients has not been demonstrated to decrease the rate of endoscopic complications or improve outcomes [60].

Delayed esophageal clearance of barium in patients over 90 years old was originally referred to as "presbyesophagus"; however, we avoid this term because it might imply that changes in esophageal motility are a normal consequence of aging and do not require further evaluation [61]. **Method** — Patients should be instructed to drink barium in the prone-oblique position; maximal distension of the esophagogastric junction (EGJ) is achieved by having the patient swallow barium rapidly in association with a variety of respiratory maneuvers [62]. In addition, asking the patient to swallow 13 mm barium tablet or a solid bolus, such as a marshmallow or bread, may be helpful for demonstrating subtle lesions in patients with persistent or intermittent solid food dysphagia [63,64]. A timed barium esophagram is useful for evaluating patients with achalasia before and after treatment, patients with suspected achalasia but inconclusive initial testing, or patients with esophagogastric junction outflow obstruction [2].

Upper endoscopy — Patients with esophageal dysphagia should be referred for an upper endoscopy to determine the underlying cause, exclude malignancy, and perform therapy (eg, dilation of an esophageal ring) if needed [3,65]. (See "Overview of upper gastrointestinal endoscopy (esophagogastroduodenoscopy)".)

In a study of over 1600 patients with dysphagia who underwent upper endoscopy, the diagnostic yield was 54 percent and risk factors for having major pathology included male sex, heartburn, and odynophagia [65].

Postendoscopy barium esophagram — We obtain a barium esophagram after a negative upper endoscopy in patients in whom a mechanical obstruction is still suspected, as subtle lower esophageal rings or extrinsic esophageal compression can be missed by an upper endoscopy [66]. The test is enhanced by the use of a barium tablet.

Esophageal manometry — Esophageal manometry should be performed in patients with dysphagia in whom upper endoscopy is unrevealing and/or an esophageal motility disorder is suspected. Although certain motility disorders (eg, achalasia) can be strongly suspected based upon their characteristic radiographic appearance when in advanced stages (image 6 and

image 5), confirmation with an esophageal manometry study is required to confirm the diagnosis [1,66].

The Chicago classification diagnostic algorithm categorizes the esophageal motility disorders such as achalasia and distal esophageal spasm [40]. The diagnosis of achalasia, gastrointestinal motility testing, and high resolution esophageal manometry are discussed separately:

- (See "Overview of gastrointestinal motility testing".)
- (See "High resolution manometry".)
- (See "Achalasia: Pathogenesis, clinical manifestations, and diagnosis".)

Functional lumen imaging probe (FLIP) — FLIP is a balloon-assisted technique for assessing distensibility and esophageal contractions [67]. FLIP is used as complementary testing in

patients with inconclusive achalasia or EGJ outflow obstruction. The test can also be used to assess patients with achalasia following endoscopic or surgical intervention.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Dysphagia".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Dysphagia (The Basics)" and "Patient education: Upper endoscopy (The Basics)" and "Patient education: Esophageal stricture (The Basics)" and "Patient education: Esophagitis (The Basics)")
- Beyond the Basics topic (see "Patient education: Upper endoscopy (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Definitions** The terms dysphagia and odynophagia are defined as follows (see 'Definitions' above):
 - Dysphagia is a subjective sensation of difficulty or abnormality of swallowing.
 - Odynophagia is pain with swallowing.

- Acute dysphagia The acute onset of inability to swallow solids and/or liquids, including secretions, suggests impaction of food or foreign body in the esophagus and requires immediate attention. Food impaction is the most common cause for acute onset of dysphagia in adults. (See 'Acute dysphagia' above.)
- Nonacute dysphagia Dysphagia can be classified as oropharyngeal dysphagia or esophageal dysphagia. Oropharyngeal or transfer dysphagia is characterized by difficulty initiating a swallow or by the sensation of food getting stuck immediately after a swallow. Swallowing may be accompanied by coughing, choking, nasopharyngeal regurgitation, aspiration, and a sensation of residual food remaining in the pharynx. Esophageal dysphagia is characterized by difficulty swallowing several seconds after initiating a swallow and a sensation of food getting stuck, or slowly passing through, the esophagus. (See 'Evaluation of nonacute dysphagia' above.)

Esophageal dysphagia should be characterized according to types of food that produce symptoms (ie, solids, liquids, or both), the time course (ie, progressive or intermittent), severity, and associated symptoms (ie, weight loss, heartburn, or regurgitation)

- (algorithm 1). (See 'Characterizing the symptoms' above.)
- **Diagnostic evaluation** The approach to diagnostic testing to determine the cause of esophageal dysphagia is based upon the medical history (algorithm 2):
 - Barium esophagram We perform a barium contrast esophagram as the initial test (prior to upper endoscopy) in patients with the following (see 'Pre-endoscopy barium esophagram' above):
 - History/clinical features of proximal esophageal lesion (eg, surgery for laryngeal or esophageal cancer, Zenker's diverticulum, or radiation therapy).
 - Known complex (tortuous) stricture (eg, postcaustic injury or radiation therapy).
 - Upper endoscopy Patients with esophageal dysphagia should be referred for an upper endoscopy to determine the underlying cause, exclude malignancy, and perform therapy (eg, dilation of an esophageal ring) if needed. (See 'Upper endoscopy' above.)
 - Esophageal manometry Esophageal manometry should be performed in patients with dysphagia in whom upper endoscopy is unrevealing and/or an esophageal motility disorder is suspected. Although certain motility disorders (eg, achalasia) can be strongly suspected based upon their characteristic radiographic appearance when in

advanced stages, confirmation with an esophageal manometry study is required to establish the diagnosis. (See 'Esophageal manometry' above.)

• Functional lumen imaging probe (FLIP) may be used as complementary testing in patients with a suspected but unconfirmed diagnosis of achalasia or with esophagogastric junction outflow obstruction.

Use of UpToDate is subject to the Terms of Use.

REFERENCES

- Spechler SJ. American gastroenterological association medical position statement on treatment of patients with dysphagia caused by benign disorders of the distal esophagus. Gastroenterology 1999; 117:229.
- Malagelada JR, Bazzoli F, Boeckxstaens G, et al. World gastroenterology organisation global guidelines: dysphagia--global guidelines and cascades update September 2014. J Clin Gastroenterol 2015; 49:370.
- **3.** ASGE Standards of Practice Committee, Pasha SF, Acosta RD, et al. The role of endoscopy in the evaluation and management of dysphagia. Gastrointest Endosc 2014; 79:191.
- 4. Gyawali CP, Carlson DA, Chen JW, et al. ACG Clinical Guidelines: Clinical Use of Esophageal Physiologic Testing. Am J Gastroenterol 2020; 115:1412.
- 5. Tawil J, Fass R. Globus: Current Concepts and Dilemmas. J Clin Gastroenterol 2018; 52:845.
- 6. Aziz Q, Fass R, Gyawali CP, et al. Functional Esophageal Disorders. Gastroenterology 2016; 150:1368.
- 7. Shamburek RD, Farrar JT. Disorders of the digestive system in the elderly. N Engl J Med 1990; 322:438.
- 8. Adkins C, Takakura W, Spiegel BMR, et al. Prevalence and Characteristics of Dysphagia Based on a Population-Based Survey. Clin Gastroenterol Hepatol 2020; 18:1970.
- 9. Gretarsdottir HM, Jonasson JG, Björnsson ES. Etiology and management of esophageal food impaction: a population based study. Scand J Gastroenterol 2015; 50:513.
- Sperry SL, Crockett SD, Miller CB, et al. Esophageal foreign-body impactions: epidemiology, time trends, and the impact of the increasing prevalence of eosinophilic esophagitis. Gastrointest Endosc 2011; 74:985.
- 11. Colon V, Grade A, Pulliam G, et al. Effect of doses of glucagon used to treat food impaction on esophageal motor function of normal subjects. Dysphagia 1999; 14:27.

- 12. Vicari JJ, Johanson JF, Frakes JT. Outcomes of acute esophageal food impaction: success of the push technique. Gastrointest Endosc 2001; 53:178.
- 13. Trate DM, Parkman HP, Fisher RS. Dysphagia. Evaluation, diagnosis, and treatment. Prim Care 1996; 23:417.
- 14. Wilcox CM, Alexander LN, Clark WS. Localization of an obstructing esophageal lesion. Is the patient accurate? Dig Dis Sci 1995; 40:2192.
- 15. Gasiorowska A, Fass R. Current approach to dysphagia. Gastroenterol Hepatol 2009; 5:269.
- 16. Marks RD, Richter JE. Peptic strictures of the esophagus. Am J Gastroenterol 1993; 88:1160.
- 17. Carlson DA, Gyawali CP, Roman S, et al. Esophageal Hypervigilance and Visceral Anxiety Are Contributors to Symptom Severity Among Patients Evaluated With High-Resolution Esophageal Manometry. Am J Gastroenterol 2020; 115:367.
- 18. Yadlapati R, Tye M, Keefer L, et al. Psychosocial Distress and Quality of Life Impairment Are Associated With Symptom Severity in PPI Non-Responders With Normal Impedance-pH Profiles. Am J Gastroenterol 2018; 113:31.
- 19. Kessing BF, Bredenoord AJ, Saleh CM, Smout AJ. Effects of anxiety and depression in patients with gastroesophageal reflux disease. Clin Gastroenterol Hepatol 2015; 13:1089.
- 20. El-Serag HB, Sonnenberg A. Association of esophagitis and esophageal strictures with diseases treated with nonsteroidal anti-inflammatory drugs. Am J Gastroenterol 1997; 92:52.
- 21. Dhir V, Vege SS, Mohandas KM, Desai DC. Dilation of proximal esophageal strictures following therapy for head and neck cancer: experience with Savary Gilliard dilators. J Surg Oncol 1996; 63:187.
- 22. DiBaise JK, Quigley EM. Tumor-related dysmotility: gastrointestinal dysmotility syndromes associated with tumors. Dig Dis Sci 1998; 43:1369.
- 23. Tracey JP, Traube M. Difficulties in the diagnosis of pseudoachalasia. Am J Gastroenterol 1994; 89:2014.
- 24. Prasad GA, Talley NJ, Romero Y, et al. Prevalence and predictive factors of eosinophilic esophagitis in patients presenting with dysphagia: a prospective study. Am J Gastroenterol 2007; 102:2627.
- 25. Mackenzie SH, Go M, Chadwick B, et al. Eosinophilic oesophagitis in patients presenting with dysphagia--a prospective analysis. Aliment Pharmacol Ther 2008; 28:1140.
- 26. Kidambi T, Toto E, Ho N, et al. Temporal trends in the relative prevalence of dysphagia etiologies from 1999-2009. World J Gastroenterol 2012; 18:4335.

- 27. Kim HP, Vance RB, Shaheen NJ, Dellon ES. The prevalence and diagnostic utility of endoscopic features of eosinophilic esophagitis: a meta-analysis. Clin Gastroenterol Hepatol 2012; 10:988.
- 28. Hoffman RM, Jaffe PE. Plummer-Vinson syndrome. A case report and literature review. Arch Intern Med 1995; 155:2008.
- 29. Tobin RW. Esophageal rings, webs, and diverticula. J Clin Gastroenterol 1998; 27:285.
- **30.** SCHATZKI R. THE LOWER ESOPHAGEAL RING. LONG TERM FOLLOW-UP OF SYMPTOMATIC AND ASYMPTOMATIC RINGS. Am J Roentgenol Radium Ther Nucl Med 1963; 90:805.
- 31. Hirano I, Gilliam J, Goyal RK. Clinical and manometric features of the lower esophageal muscular ring. Am J Gastroenterol 2000; 95:43.
- 32. Ott DJ, Gelfand DW, Lane TG, Wu WC. Radiologic detection and spectrum of appearances of peptic esophageal strictures. J Clin Gastroenterol 1982; 4:11.
- 33. De Luca L, Bergman JJ, Tytgat GN, Fockens P. EUS imaging of the arteria lusoria: case series and review. Gastrointest Endosc 2000; 52:670.
- 34. Bennett JR, Castell DO. Overview and symptom assessment. In: The Esophagus, Castell DO, Richter JE (Eds), Lippincott, Williams & Wilkins, Philadelphia 1999. p.33.
- **35**. Febrero B, Ríos A, Rodríguez JM, Parrilla P. Dysphagia lusoria as a differential diagnosis in intermittent dysphagia. Gastroenterol Hepatol 2017; 40:354.
- 36. Molz G, Burri B. Aberrant subclavian artery (arteria lusoria): sex differences in the prevalence of various forms of the malformation. Evaluation of 1378 observations. Virchows Arch A Pathol Anat Histol 1978; 380:303.
- 37. Janssen M, Baggen MG, Veen HF, et al. Dysphagia lusoria: clinical aspects, manometric findings, diagnosis, and therapy. Am J Gastroenterol 2000; 95:1411.
- 38. Vaezi MF, Pandolfino JE, Yadlapati RH, et al. ACG Clinical Guidelines: Diagnosis and Management of Achalasia. Am J Gastroenterol 2020; 115:1393.
- 39. Howard PJ, Maher L, Pryde A, et al. Five year prospective study of the incidence, clinical features, and diagnosis of achalasia in Edinburgh. Gut 1992; 33:1011.
- 40. Yadlapati R, Kahrilas PJ, Fox MR, et al. Esophageal motility disorders on high-resolution manometry: Chicago classification version 4.0©. Neurogastroenterol Motil 2021; 33:e14058.
- 41. Clouse RE. Spastic disorders of the esophagus. Gastroenterologist 1997; 5:112.
- **42.** Tutuian R, Castell DO. Clarification of the esophageal function defect in patients with manometric ineffective esophageal motility: studies using combined impedance-

manometry. Clin Gastroenterol Hepatol 2004; 2:230.

- **43**. Zamost BJ, Hirschberg J, Ippoliti AF, et al. Esophagitis in scleroderma. Prevalence and risk factors. Gastroenterology 1987; 92:421.
- 44. Rose S, Young MA, Reynolds JC. Gastrointestinal manifestations of scleroderma. Gastroenterol Clin North Am 1998; 27:563.
- 45. Stampfl D, Denuna S, Varga J, et al. Relations between dysmotility and acid exposure in scleroderma (SSc). Am J Gastroenterol 1990; 85:1226.
- 46. Yarze JC, Varga J, Stampfl D, et al. Esophageal function in systemic sclerosis: a prospective evaluation of motility and acid reflux in 36 patients. Am J Gastroenterol 1993; 88:870.
- 47. Canalejo Castrillero E, García Durán F, Cabello N, García Martínez J. Herpes esophagitis in healthy adults and adolescents: report of 3 cases and review of the literature. Medicine (Baltimore) 2010; 89:204.
- 48. Généreau T, Lortholary O, Bouchaud O, et al. Herpes simplex esophagitis in patients with AIDS: report of 34 cases. The Cooperative Study Group on Herpetic Esophagitis in HIV Infection. Clin Infect Dis 1996; 22:926.
- 49. Sutton FM, Graham DY, Goodgame RW. Infectious esophagitis. Gastrointest Endosc Clin N Am 1994; 4:713.
- 50. O'Rourke A. Infective oesophagitis: epidemiology, cause, diagnosis and treatment options. Curr Opin Otolaryngol Head Neck Surg 2015; 23:459.
- 51. Rudolph I, Goldstein F, DiMarino AJ Jr. Crohn's disease of the esophagus: Three cases and a literature review. Can J Gastroenterol 2001; 15:117.
- 52. Rubio CA, Sjödahl K, Lagergren J. Lymphocytic esophagitis: a histologic subset of chronic esophagitis. Am J Clin Pathol 2006; 125:432.
- 53. Haque S, Genta RM. Lymphocytic oesophagitis: clinicopathological aspects of an emerging condition. Gut 2012; 61:1108.
- 54. Ronkainen J, Walker MM, Aro P, et al. Lymphocytic oesophagitis, a condition in search of a disease? Gut 2012; 61:1776.
- 55. Cohen S, Saxena A, Waljee AK, et al. Lymphocytic esophagitis: a diagnosis of increasing frequency. J Clin Gastroenterol 2012; 46:828.
- 56. Yousef M, Chela H, Ertugrul H, et al. Lymphocytic Esophagitis: A Case Series of Esophageal Disease with Increasing Frequency. Recent Adv Inflamm Allergy Drug Discov 2023; 17:79.
- 57. Anselmino M, Zaninotto G, Costantini M, et al. Esophageal motor function in primary Sjögren's syndrome: correlation with dysphagia and xerostomia. Dig Dis Sci 1997; 42:113.

- 58. Kjellén G, Fransson SG, Lindström F, et al. Esophageal function, radiography, and dysphagia in Sjögren's syndrome. Dig Dis Sci 1986; 31:225.
- 59. Palma R, Freire A, Freitas J, et al. Esophageal motility disorders in patients with Sjögren's syndrome. Dig Dis Sci 1994; 39:758.
- 60. American Gastroenterological Association medical position statement on management of oropharyngeal dysphagia. Gastroenterology 1999; 116:452.
- 61. SOERGEL KH, ZBORALSKE FF, AMBERG JR. PRESBYESOPHAGUS: ESOPHAGEAL MOTILITY IN NONAGENARIANS. J Clin Invest 1964; 43:1472.
- 62. Chen YM, Ott DJ, Gelfand DW, Munitz HA. Multiphasic examination of the esophagogastric region for strictures, rings, and hiatal hernia: evaluation of the individual techniques. Gastrointest Radiol 1985; 10:311.
- 63. Somers S, Stevenson GW, Thompson G. Comparison of endoscopy and barium swallow with marshmallow in dysphagia. Can Assoc Radiol J 1986; 37:73.
- 64. Gallo SH, McClave SA, Makk LJ, Looney SW. Standardization of clinical criteria required for use of the 12.5 millimeter barium tablet in evaluating esophageal lumenal patency. Gastrointest Endosc 1996; 44:181.
- 65. Varadarajulu S, Eloubeidi MA, Patel RS, et al. The yield and the predictors of esophageal pathology when upper endoscopy is used for the initial evaluation of dysphagia. Gastrointest Endosc 2005; 61:804.
- 66. Ott DJ. Radiographic techniques and efficacy in evaluating esophageal dysphagia. Dysphagia 1990; 5:192.
- 67. Chen JW, Khan A, Chokshi RV, et al. Interrater Reliability of Functional Lumen Imaging Probe Panometry and High-Resolution Manometry for the Assessment of Esophageal Motility Disorders. Am J Gastroenterol 2023; 118:1334.

Topic 2241 Version 36.0

GRAPHICS

Questions to ask patients with dysphagia

Do you have problems initiating a swallow or do you feel food getting stuck a few seconds after swallowing? (Helps distinguish oropharyngeal from esophageal dysphagia.)

Do you cough or choke or is food coming back through your nose after swallowing? (Coughing, choking, or nasal regurgitation suggests aspiration and oropharyngeal dysphagia.)

Do you have problem swallowing solids, liquids, or both? (Liquids, not solids, suggests a motility disorder; solids progressing to liquids suggests a benign or malignant obstruction.)

How long have you had problems swallowing and have your symptoms progressed, remained stable, or are they intermittent? (Rapidly progressive dysphagia is concerning for malignancy.)

Could you point to where you feel food is getting stuck? (Ability to localize source of dysphagia is unreliable; best with oropharyngeal dysphagia.)

Do you have other symptoms such as loss of appetite, weight loss, nausea, vomiting, regurgitation of food particles, heartburn, vomiting fresh or old blood, pain during swallowing, or chest pain?

Do you have medical problems such as diabetes mellitus, scleroderma, Sjögren's syndrome, overlap syndrome, AIDS, neuromuscular disorders (stroke, Parkinson's, myasthenia gravis, muscular dystrophy, multiple sclerosis), cancer, Chagas' disease or others?

Have you had surgery on your larynx, esophagus, stomach, or spine?

Have you received radiation therapy in the past?

What medications are you using now (ask specifically about potassium chloride, alendronate, ferrous sulfate, quinidine, ascorbic acid, tetracycline, aspirin and NSAIDs)? (Pill esophagitis can cause dysphagia.)

AIDs: acquired immune deficiency syndrome; NSAIDs: nonsteroidal anti-inflammatory drugs.

Graphic 68343 Version 7.0

Representative causes of oropharyngeal dysphagia

Iatrogenic	Neurological
Medication side effects (chemotherapy, neuroleptics, etc)	Brainstem tumors
	Head trauma
Postsurgical muscular or neurogenic	Stroke
Radiation	Cerebral palsy
Corrosive (pill injury, intentional)	Guillain-Barré syndrome
Infectious	Huntington disease
Mucositis (herpes, cytomegalovirus, Candida, etc)	Multiple sclerosis
Diphtheria	Polio
Botulism	Postpolio syndrome
Lyme disease	Tardive dyskinesia
Syphilis	Metabolic encephalopathies
	Amyotrophic lateral sclerosis
Metabolic	Parkinson disease
Amyloidosis	Dementia
Cushing's syndrome	Structural
Thyrotoxicosis	Cricopharyngeal bar
Wilson disease	Zenker's diverticulum
Myopathic	Cervical webs
Connective tissue disease (overlap syndrome)	Oropharyngeal tumors
Dermatomyositis	Osteophytes and skeletal abnormalities
Myasthenia gravis	Congenital (cleft palate, diverticula, pouches,
Myotonic dystrophy	etc)
Oculopharyngeal dystrophy	
Polymyositis	
Sarcoidosis	
Paraneoplastic syndromes	

Adapted from: Cook IJ, Kahrilas PJ. AGA: Technical review: Management of oropharyngeal dysphagia. Gastroenterology 1999; 116:455.

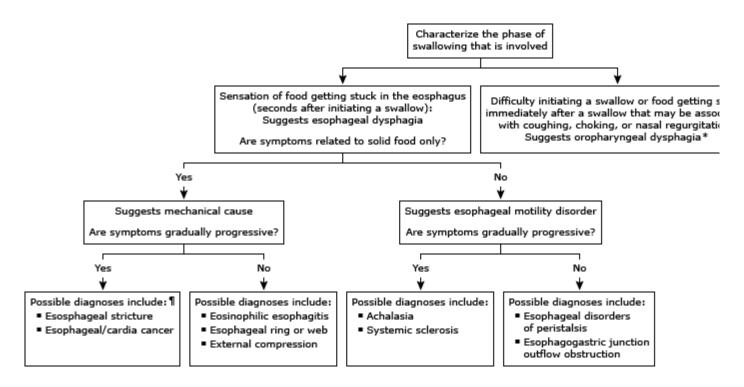
Graphic 52707 Version 7.0

Causes of esophageal dysphagia

Intrinsic	
Benign tumors	
Caustic esophagitis/stricture	
Diverticula	
Malignancy	
Peptic stricture	
Eosinophilic esophagitis	
Infectious esophagitis	
Pill esophagitis	
Postsurgery (laryngeal, esophageal, gas	tric)
Radiation esophagitis/stricture	
Rings and webs	
Lymphocytic esophagitis	
Extrinsic	
Aberrant subclavian artery	
Cervical osteophytes	
Enlarged aorta	
Enlarged left atrium	
Mediastinal mass (lymphadenopathy, lu	ng cancer, etc)
Postsurgery (laryngeal, spinal)	
Motility disorders	
Achalasia	
Chagas disease	
Primary motility disorders	
Secondary motility disorders	
Functional	

Graphic 80528 Version 5.0

Differential diagnosis of nonacute dysphagia in adults

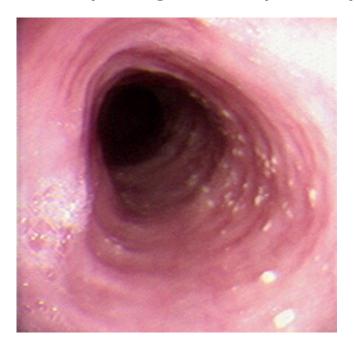


* Refer to UpToDate content on evaluation and management of oropharyngeal dysphagia.

¶ Patients with esophageal stricture may report chronic heartburn, whereas patients with esophageal cance tend to be older and often have rapid weight loss.

Graphic 70866 Version 8.0

Endoscopic image of eosinophilic esophagitis

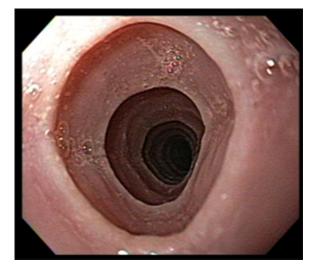


This upper endoscopy in a 36-year-old man with dysphagia showed multiple rings in the proximal to mid esophagus, giving it the appearance of a trachea. Small whitish papules are also visible representing eosinophilic abscesses on histology. The patient's symptoms responded to swallowed (ie, topical) fluticasone.

Courtesy of Eric D Libby, MD.

Graphic 51432 Version 4.0

Endoscopic image of the esophagus showing multiple ring-like strictures in a patient with eosinophilic esophagitis

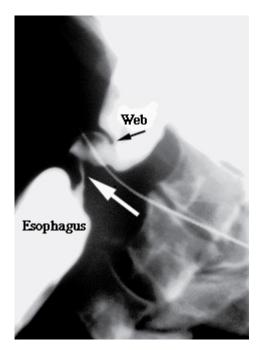


This upper endoscopy in a patient with eosinophilic esophagitis showed multiple ring-like strictures in the proximal and midesophagus.

Courtesy of Andres Gelrud, MD and Anthony Lembo, MD.

Graphic 50566 Version 3.0

Barium swallow in a patient with Plummer Vinson syndrome

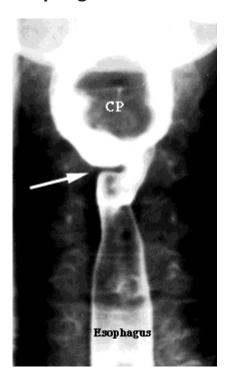


This barium swallow study obtained in a 53-year-old female with dysphagia and anemia demonstrates an upper esophageal web (black arrow) immediately above a tight stricture of the esophagus (white arrow).

Courtesy of Jonathan Kruskal, MD, PhD.

Graphic 81131 Version 3.0

Esophageal web on barium swallow

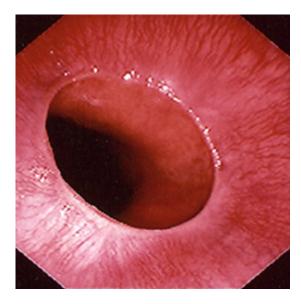


This modified barium swallow, obtained in a 45-year-old man with dysphagia, demonstrates an asymmetric esophageal web arising from the right side of the upper esophagus (arrow).

Courtesy of Jonathan Kruskal, MD, PhD.

Graphic 76514 Version 4.0

Esophageal (Schatzki) ring

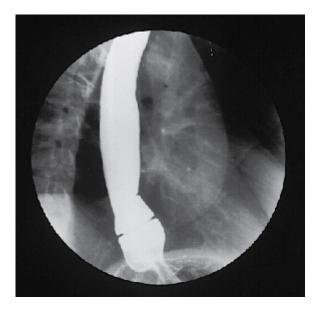


Endoscopic view of an esophageal (Schatzki) ring, which often cannot be well visualized unless the lower esophagus is widely distended. The ring appears as a thin membrane with a concentric smooth contour that projects into the lumen.

Courtesy of James B McGee, MD.

Graphic 55092 Version 3.0

Esophageal (Schatzki) ring seen on barium swallow



Esophageal (Schatzki) ring at the gastroesophageal junction visualized on a barium swallow.

Courtesy of Peter J Kahrilas, MD.

Graphic 68185 Version 4.0

Achalasia



Barium esophagram showing a dilated esophagus and bird's beak appearance typical of achalasia. Retained food is also visible.

Courtesy of Ram Dickman, MD.

Graphic 53672 Version 4.0

Dilation of the esophagus in a patient with achalasia (barium esophagram)

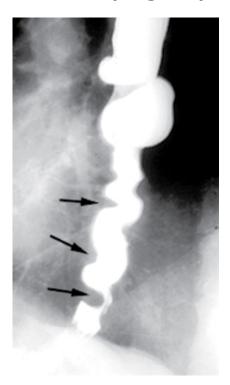


Barium esophagram in a 62-year-old man demonstrates a dilated, barium-filled esophagus with a region of persistent narrowing (arrow) at the gastroesophageal junction, producing the so-called bird's beak appearance. Achalasia was confirmed with manometry and the patient underwent successful dilation of the esophagus.

Courtesy of Jonathan Kruskal, MD.

Graphic 54252 Version 4.0

Diffuse esophageal spasm

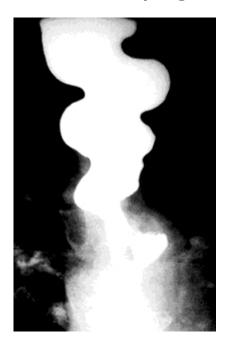


This barium swallow in an older man with noncardiogenic chest pain shows multiple areas of spasm (arrows) throughout the length of the esophagus. This spasm was accentuated by stasis within the esophageal lumen and esophagitis.

Courtesy of Jonathan Kruskal, MD, PhD.

Graphic 69821 Version 6.0

Corkscrew esophagus

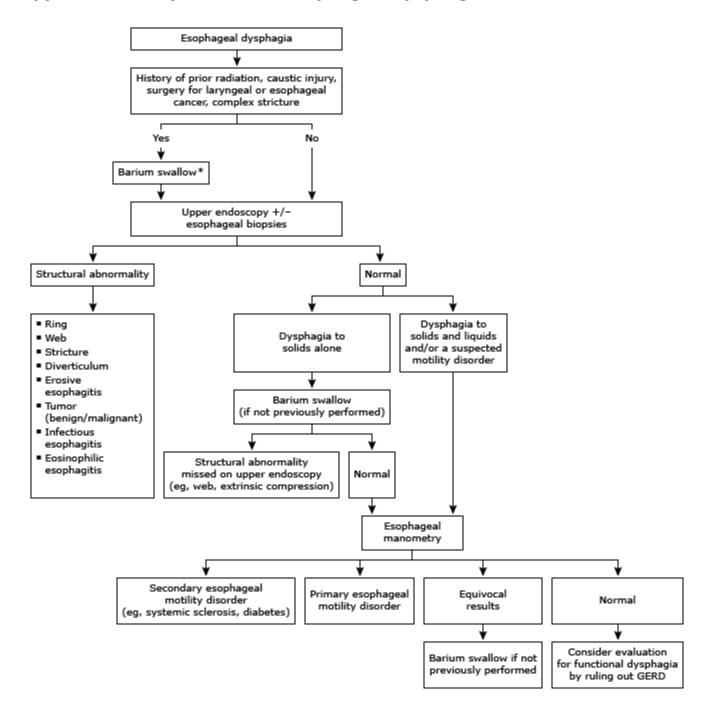


Esophagram performed in a 72-year-old man with intractable retrosternal pain and reflux shows marked spasm throughout the length of the esophagus, which produces a corkscrew-like appearance.

Courtesy of Jonathan Kruskal, MD, PhD.

Graphic 81244 Version 5.0

Approach to the patient with esophageal dysphagia



GERD: gastroesophageal reflux disease.

* Performing a barium swallow prior to an upper endoscopy is controversial.

Graphic 81108 Version 4.0

Contributor Disclosures

Ronnie Fass, MD Equity Ownership/Stock Options: Ginger-Health [GERD]. Consultant/Advisory Boards: Celexio [GERD]; Dexcal [GERD]; Evoke [Gastroparesis]; GERDCare [GERD]; Medtronics [Esophageal manometry]; Phantom [GERD]; Takeda [GERD]; Veritas [GERD]. Speaker's Bureau: Adcock-Ingram [GERD]; AstraZeneca [GERD]; Carnot [GERD]; Eisai [GERD]; Johnson & Johnson [GERD]; Laborie [Esophageal manometry]; Medicamenta [GERD]; Takeda [GERD]. All of the relevant financial relationships listed have been mitigated. **Mark Feldman, MD, MACP, AGAF, FACG** No relevant financial relationship(s) with ineligible companies to disclose. **Kristen M Robson, MD, MBA, FACG** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy

 \rightarrow