Dysphagia Revisited: Common and Unusual Causes[1]

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Abbreviations: CMV = cytomegalovirus, DES = diffuse esophageal spasm, EIP = esophageal intramural pseudodiverticulosis, GEJ = gastroesophageal junction, GERD = gastroesophageal reflux disease, HIV = human immunodeficiency virus, LPO = left posterior oblique, RAO = right anterior oblique

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- List the imaging techniques that may be used in the evaluation of patients who present with dysphagia.
- Discuss the spectrum of common and uncommon or unexpected causes of dysphagia.
- Describe the imaging findings of disease entities that may produce oropharyngeal or substernal dysphagia.

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Dysphagia is a common clinical problem whose prevalence is increasing with the aging population in the United States. The term *dysphagia* is commonly used to describe subjective awareness of swallowing difficulty during the passage of a bolus from the mouth to the stomach or the perception of obstruction during swallowing. Dysphagia may be further classified as oropharyngeal or substernal, depending on the location of this sensation. It can be due to benign or malignant structural lesions, esophageal motility abnormalities, oropharyngeal dysfunction (including aspiration), neuromuscular disorders, or postsurgical changes and is also associated with gastroesophageal reflux disease. Pathologic conditions of the oral cavity, pharynx, esophagus, and proximal stomach can manifest with dysphagia. Imaging remains the preferred method for evaluating patients with dysphagia, and dysphagia is an increasingly encountered indication for radiologic evaluation. Fluoroscopic studies, including the modified barium swallow and esophagography in particular, are often used in the assessment of patients with dysphagia, and the techniques used for these studies should be tailored to the patient's needs. Fluoroscopic studies can be used to evaluate the esophagus for structural abnormalities (eg, webs, diverticula, strictures, masses) and to assess function (eg, the swallowing mechanism and esophageal motility). Knowledge of the imaging spectrum of disease entities that may cause dysphagia and thorough radiologic assessment with a tailored approach may help avoid misdiagnosis.

Introduction

Dysphagia can be caused by functional or structural abnormalities of the oral cavity, pharynx, esophagus, and/or gastric cardia. It is important to distinguish between oropharyngeal and substernal esophageal dysphagia, since potential causes, radiologic evaluation, and treatment may differ (1).

Dysphagia is defined by medical dictionaries as difficulty in swallowing. The term *dysphagia* is commonly used to describe a symptom that manifests as *(a)* subjective awareness of swallowing difficulty during the passage of a liquid or solid bolus from the mouth to the stomach, or *(b)* the perception of obstruction during swallowing. Patients with dysphagia may experience the sensation of food getting stuck in the throat or chest, coughing or choking with swallowing, discomfort, or the ability to "sense" the act of swallowing. In addition, patients with dysphagia may experience a voice change, frequent throat clearing, otalgia, weight loss, dehydration, and/or pneumonia (2). Patients reporting "swallowing problems" could be experiencing dysphagia, odynophagia, globus sensation, and/or heartburn.

TEACHING POINTS

- The prevalence of neuromuscular disorders and structural abnormalities (ie, Zenker diverticulum) also increases with age, and it is important to look for a cause of dysphagia in elderly patients.
- Prevention of aspiration requires superior and anterior elevation of the larynx, closure of the larynx, and epiglottic tilt, and these mechanisms should be carefully evaluated at videofluoroscopy.
- Cough is an important potential indicator of aspiration due to oropharyngeal dysphagia. However, aspiration can be silent in up to 55% of patients.
- Rather than the tapered beaklike narrowing seen in achalasia, luminal narrowing of the distal esophagus and GEJ in pseudoachalasia tends to appear irregular with lobulated margins, since the cause of this narrowing is primary or metastatic tumor.
- In the setting of a hiatal hernia and gastroesophageal reflux, a focal stricture in the midesophagus is highly suggestive of Barrett esophagus.

Dysphagia is a relatively common and increasingly prevalent clinical problem. An estimated 10 million Americans are evaluated for dysphagia each year (2–5). In addition, because of the aging population in the United States, an increasing number of patients are presenting for radiologic evaluation of dysphagia. Dysphagia is associated with increased morbidity, mortality, financial cost, and health care burden.

Dysphagia has a prevalence of nearly 22% in the adult primary care population and of 13.5% in the general population (4). Patients in a primary care setting who have dysphagia are more likely to be women than men (80% versus 20%) and are more likely to be older than patients who do not have dysphagia (4). Dysphagia is also more common in patients with gastroesophageal reflux disease (GERD) than in those without GERD (4,6). Other medical conditions that may predispose patients to dysphagia include stroke, Alzheimer disease, multiple sclerosis, amyotrophic lateral sclerosis, and Parkinson disease, and the prevalence of many of these conditions increases with patient age (2,3,7,8). Untreated dysphagia can lead to dehydration, malnutrition, aspiration, pneumonia, and death (4,5,8).

Persons over 65 years of age account for two-thirds of individuals with dysphagia (9). Furthermore, this component of the population is rapidly expanding: Over the past decade, the number of persons over 65 years of age increased by 28%, whereas the rest of the population increased by only 0.8% (3). Dysphagia occurs in up to 50% of nursing home residents, and the elderly have an increased risk of complications from dysphagia (including aspiration pneumonia) compared with younger individuals (3,4,7,8).

Oropharyngeal dysphagia is an underrecognized cause of nutritional and respiratory complications in elderly patients (3,8). The aging process is associated with measurable changes in muscular and nervous system function; however, aging itself does not cause dysphagia (8). The prevalence of neuromuscular disorders and structural abnormalities (ie, Zenker diverticulum) also increases with age, and it is important to look for a cause of dysphagia in elderly patients (1,8). Overall, elderly patients with dysphagia have a poor outcome and a high mortality rate (3).

In this article, we discuss dysphagia in terms of techniques for imaging evaluation, common and uncommon causes (eg, swallowing dysfunction, motility abnormalities, diverticular disease, webs and rings, extrinsic processes, vascular phenomena, infection, strictures, neoplasms, foreign bodies, and postoperative changes), and relevant imaging findings.

Oropharyngeal versus Substernal Dysphagia

Oropharyngeal Dysphagia

Oropharyngeal dysphagia represents a clinical situation in which a patient symptomatically localizes a sensation of blockage or discomfort in the throat. Patients may complain of a "lump" or "sticking" in the throat. Oropharyngeal dysphagia may be caused by disease affecting the pharynx, esophagus, or gastric cardia (10,11). It should be noted that abnormalities of the mid- to distal esophagus or gastric cardia can result in referred dysphagia to the pharyngeal region (11). However, oropharyngeal dysphagia is more common in patients with a history of neurologic disorders including stroke, head and neck surgery, or radiation therapy (1). Oropharyngeal dysphagia can lead to various medical complications, including respiratory problems, malnutrition, dehydration, bronchial spasms, and airway obstruction (12).

Oropharyngeal symptoms may result from *(a)* laryngeal penetration or aspiration, *(b)* soft-palate insufficiency, and/or *(c)* an abnormal oral phase of swallowing. Recognition of the different patterns of symptoms associated with these entities may aid in diagnosis and treatment: Patients with laryngeal penetration or aspiration may present with coughing or choking upon swallowing, those with soft-palate insufficiency may exhibit a nasal voice or nasal regurgitation, and those with an abnormal oral phase of swallowing may report food dribbling and difficulty in chewing (13). Oropharyngeal dysphagia can be functional (eg, due to aspiration or penetration) or structural (eg, due to Zenker diverticulum, tumor, webs, extrinsic masses, or cervical spine disease). Cricopharyngeal dysfunction can also produce oropharyngeal dysphagia.

Substernal Dysphagia

Substernal dysphagia represents a clinical situation in which a patient symptomatically localizes a sensation of discomfort or blockage between the thoracic inlet and the xiphoid process (ie, in the region of the thoracic esophagus). Other symptoms may include odynophagia, chest pain, and heartburn. Substernal dysphagia may be functional (due to an esophageal motility abnormality) or structural (eg, due to ring stricture, an extrinsic mass, or a tumor) and may be due to or associated with GERD. The sensation of dysphagia in the substernal region is typically caused by disease in the esophagus or proximal stomach. Pharyngeal abnormalities do not usually produce referred symptoms distally (1,10,11).

Imaging Evaluation of Dysphagia

Despite an overall decline in the number of gastrointestinal fluoroscopic procedures, the number of esophagographic and barium swallow studies has actually increased (14,15). Although computed tomography (CT) and magnetic resonance (MR) imaging can be used to evaluate dysphagia (16,17), fluoroscopy remains the primary and preferred modality. In fact, a barium study remains the test most commonly requested by primary care physicians, gastroenterologists, and radiologists for the evaluation of dysphagia (1,10,18,19). Barium studies can help assess function as well as structural problems, sometimes better than endoscopy (1,10).

Esophagography provides anatomic and functional information about the pharynx, esophagus, gastroesophageal junction (GEJ), and gastric cardia, including evaluation of esophageal motility and assessment for gastroesophageal reflux. A *modified barium swallow*, often performed in conjunction with a swallowing therapist, provides information about swallowing function, response to therapeutic measures, and necessary dietary requirements. The modified barium swallow remains the standard of reference for evaluating the swallowing mechanism (2,12,20).

A detailed imaging assessment of patients with dysphagia includes evaluation of the pharynx, esophagus, and gastric cardia. The study should be tailored to the patient's needs (eg, studies for oropharyngeal dysphagia may require a different approach than studies for substernal dysphagia) (10,13). For example, a patient who complains of coughing upon swallowing may need to undergo an evaluation of swallowing function before inspection of the thoracic esophagus and evaluation for gastroesophageal reflux are performed.

In the evaluation of the pharynx and cervical esophagus, videofluoroscopy should be used to assess swallowing function and to assess specifically for aspiration or penetration. Ideally, videofluoroscopic images are obtained in the lateral and anteroposterior projections for detailed evaluation of swallowing function and oropharyngeal motility. Double-contrast images of the pharynx are also acquired in the lateral and anteroposterior projections to evaluate for structural abnormalities (21). Oblique imaging may be performed as necessary. Imaging of the cervical esophagus for assessment of structural abnormalities is best accomplished with rapid-sequence imaging—ideally in the lateral and anteroposterior projections—because the bolus passes quickly through this region. Full-column images of the cervical esophagus can typically be obtained with rapid-sequence imaging at two frames per second as the bolus passes.

The thoracic esophagus is optimally evaluated with double-contrast images acquired with the patient in the upright left posterior oblique (LPO) position in conjunction with single-contrast images acquired primarily with the patient in the prone right anterior oblique (RAO) position (10,22). Esophageal motility is best evaluated at fluoroscopy with the patient in the prone RAO position. Fluoroscopy should be used to assess for gastroesophageal reflux. A 12.5-mm barium tablet may be administered to detect more subtle areas of esophageal narrowing (22) and is useful for determining the luminal caliber of an esophageal stricture. The tablet should be swallowed with a small amount of water or water mixed with dilute barium, and passage should be observed at fluoroscopy. If the tablet becomes lodged in a particular location and underlying disease was not observed earlier in this region, the patient should first swallow a small amount of additional water. If the tablet remains lodged, a more detailed assessment should be performed with additional projections and further administration of barium. The gastric cardia is best imaged using a double-contrast technique with the patient in the recumbent right lateral position (10).

Oropharyngeal Functional Abnormalities

Functional causes of oropharyngeal dysphagia may include laryngeal penetration, aspiration, and cricopharyngeal dysfunction. Contrast material seen entering the larynx at fluoroscopy indicates penetration. Aspiration is diagnosed when the ingested bolus extends inferiorly through the true vocal cords into the proximal trachea during videofluoroscopic evaluation of the swallow (Fig 1) (12). Prevention of aspiration requires superior and anterior elevation of the larynx, closure of the larynx, and epiglottic tilt, and these mechanisms should be carefully evaluated at videofluoroscopy (23). Aspiration may occur before, during, or after the swallow and is the most threatening symptom of oropharyngeal dysphagia. Aspiration pneumonia occurs in four to eight of every 1000 inpatients in the United States (12,24), and 50,000 U.S. patients die each year from complications of aspiration (3,8,12).

Aspiration and the Elderly

Elderly patients with oropharyngeal dysphagia have a poor outcome, particularly due to aspiration. In elderly nursing home residents with oropharyngeal dysphagia, aspiration pneumonia occurs in 50% during the first year, with a mortality rate of over 45% (3). Overall, the mortality rate from aspiration pneumonia ranges from 20% to 65% in patients over 65 years of age (3,8,12). The most common cause of oropharyngeal dysphagia in the elderly is stroke, with a high morbidity, mortality, and cost (8). Dysphagia occurs in 29%–64% of stroke patients, and 20% of these patients die of aspiration pneumonia within 1 year after the stroke (4).

Silent Aspiration

Cough is an important potential indicator of aspiration due to oropharyngeal dysphagia (12). However, aspiration can be silent in up to 55% of patients (20). Silent aspiration is defined as passage of ingested contents through and below the true vocal cords without producing a reflexive cough or some other overt sign that aspiration has occurred (20). Consequently, aspiration risk and extent are underestimated at clinical bedside examination, thereby increasing the importance of barium studies—specifically, the modified barium swallow. Patients with silent aspiration have an increased risk of complications of aspiration, including aspiration pneumonia, due to lack of recognition of the problem and diminished ability to protect the airway compared with patients who exhibit nonsilent aspiration (ie, cough reflex) (12,20).

Cricopharyngeal Dysfunction

Cricopharyngeal dysfunction may also cause oropharyngeal dysphagia. The cricopharyngeus muscle is the main component of the upper esophageal sphincter and is normally closed between swallows. This muscle must relax and open completely for passage of the bolus, which requires elevation of the larynx, pharyngeal constrictors, and a bolus. Cricopharyngeal dysfunction may consist of incomplete relaxation and/or early closure of the cricopharyngeus muscle (upper esophageal sphincter). This can result in proximal dilatation and trapping of contents, with secondary problems of penetration and/or aspiration. Prominence of the cricopharyngeus muscle can be seen as a posterior indentation on barium studies (Fig 2). This finding can be incidental or can produce dysphagia (8,13). A cricopharyngeal bar is likely contributing to the patient's symptoms if the indentation from the cricopharyngeal muscle takes up more than 50% of the expected luminal diameter with maximal distention and/or there is proximal dilatation leading to this indentation. Prominence of the cricopharyngeus muscle may be associated with pharyngeal constrictor muscle weakness, failure of laryngeal elevation, and/or a Zenker diverticulum. Cricopharyngeal prominence can also occur as a response to esophageal disease (eg, GERD, esophageal spasm) (13).

Figure 1. Aspiration. Anteroposterior double-contrast image of the pharynx shows pooling of contrast material in the pyriform sinuses and valleculae. Contrast material is also seen pooling in the larynx (arrows) and extending inferiorly below the level of the vocal cords into the trachea, consistent with aspiration.

Esophageal Motility Abnormalities

Patients with esophageal motility abnormalities most often present with slowly progressive dysphagia to liquids and solids, occasionally with associated weight loss (18). Esophageal motility can readily be assessed at fluoroscopy, ideally with the patient in the prone RAO position (10). Two to five individual swallows should be observed at fluoroscopy as the bolus passes. An individual swallow should be evaluated, since a second or double swallow will interfere with primary peristalsis and limit evaluation of esophageal motility. Patients may be instructed to take one swallow and then open the mouth, since it is not possible to swallow with the mouth open.

Specific motility disorders may include diffuse esophageal spasm (DES), achalasia, and sclero-

Figure 2. Incomplete relaxation of the cricopharyngeus muscle. Lateral fluoroscopic spot image acquired during swallowing shows incomplete relaxation of the cricopharyngeus muscle (upper esophageal sphincter), which appears as a posterior indentation (black arrow). Note also the trace penetration with contrast material in the larynx (white arrow).

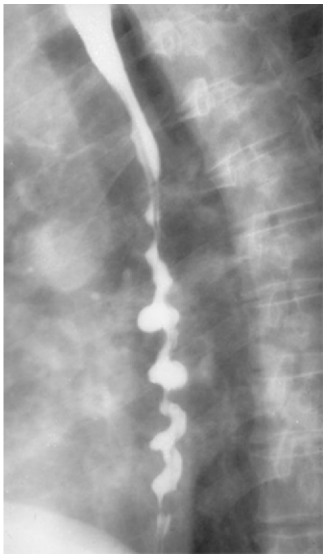


Figure 3. DES. Lateral spot image obtained during esophagography shows the thoracic esophagus with a corkscrew appearance due to multiple spontaneous and uncoordinated contractions obliterating the esophageal lumen.

derma. However, esophageal motility disorders may not fall into any of these categories and may occur in association with GERD. Findings of nonspecific motility abnormalities may include numerous tertiary contractions, poor propagation of the primary peristaltic wave, abnormal esophageal emptying, and stasis (18). Esophageal manometry may be necessary for the definitive diagnosis of a motility disorder. Manometry can help evaluate the esophageal motor pattern, contraction amplitude, and lower esophageal sphincter pressure and function.

Diffuse Esophageal Spasm

DES is a motility abnormality in which there are multiple spontaneous and uncoordinated esophageal contractions. Patients may present with intermittent chest pain and/or dysphagia. DES can occur at any age but is more common in patients over 50 years of age. It may be intermittent and occurs primarily in the distal two-thirds of the esophagus. Uncoordinated contractions can obliterate the esophageal lumen and result in a classic corkscrew appearance at esophagography (Fig 3).

Achalasia

Achalasia is a motility abnormality that is characterized by the absence of primary esophageal peristalsis with failure of the lower esophageal sphincter to relax. There is often associated increased lower esophageal sphincter pressure (18). Increased intraluminal pressure results in a dilated esophagus. Esophageal dilatation is seen at esophagography in conjunction with tapered beaklike narrowing distally (Fig 4). However, it is important to be aware of the possibility of pseudoachalasia. In pseudoachalasia, the esophagus is dilated, with narrowing of the distal esophagus and GEJ. Rather than the tapered beaklike narrowing seen in achalasia, luminal narrowing of the distal esophagus and GEJ in pseudoachalasia tends to appear irregular with lobulated margins, since the cause of this narrowing is primary or metastatic tumor (Fig 5). Patients with pseudoachalasia present with a more abrupt onset of symptoms, as opposed to the long-standing history of dysphagia seen in patients with achalasia. In addition, patients with pseudoachalasia tend to present with weight loss due to underlying malignancy, whereas weight loss is uncommon in patients with achalasia. If pseudoachalasia is suspected, CT may be helpful in evaluating disease extent and assessing for distant metastases.

Scleroderma

Scleroderma is a smooth muscle disorder that is characterized by diminished or absent peristalsis in approximately the distal two-thirds of the esophagus (corresponding to the region of smooth muscle in the esophagus). An incompetent lower esophageal sphincter leads to a patulous GEJ, and there tends to be esophageal dilatation (Fig 6). Associated gastroesophageal reflux and esophageal stasis may lead to the development of a peptic stricture (22).

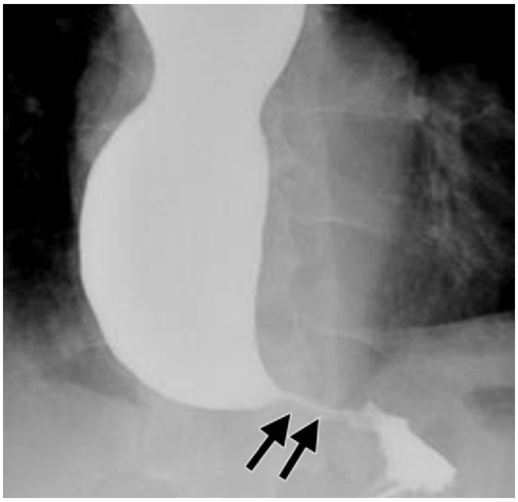


Figure 4. Achalasia. Anteroposterior upright image shows a dilated thoracic esophagus with pooling of contrast material, fluid, and debris. Tapered beaklike narrowing is seen distally (arrows). Fluoroscopy demonstrated the absence of primary esophageal peristalsis.

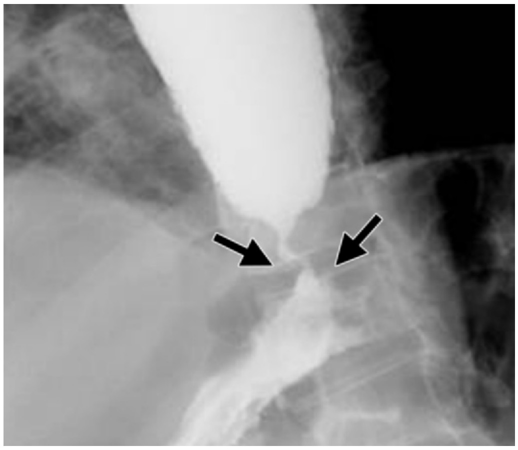


Figure 5. Pseudoachalasia. Right posterior oblique image shows a dilated esophagus with narrowing of the distal esophagus and GEJ. However, rather than the tapered beaklike narrowing seen in achalasia, the margins are characterized by lobulations extending into the proximal stomach (arrows), representing metastatic tumor infiltration.

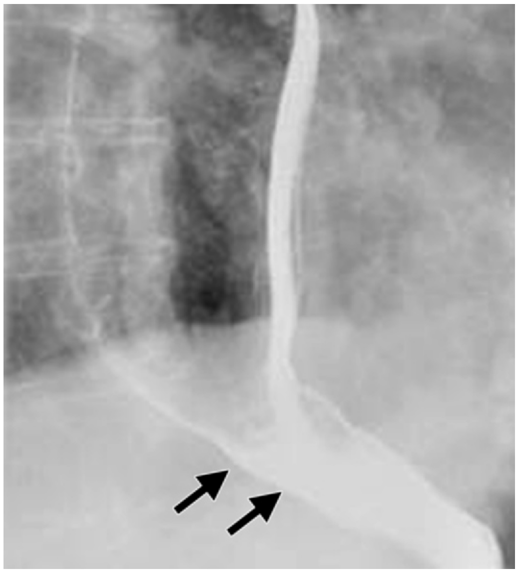


Figure 6. Scleroderma. Double-contrast upright LPO image shows esophageal dilatation with a patulous distal esophagus and GEJ (arrows). Fluoroscopy demonstrated severely diminished primary peristalsis in the distal two-thirds of the thoracic esophagus, with normal peristalsis in the upper thoracic esophagus.

Structural Abnormalities

Diverticula

The most common pharyngoesophageal diverticula include pharyngeal, Zenker, Killian-Jamieson, and epiphrenic diverticula. Diverticula arising from the pharynx or proximal esophagus are associated with an increased risk of aspiration, particularly overflow aspiration of the contents of the diverticulum after the swallow is completed.

Pharyngeal diverticula are more often unilateral than bilateral. Whereas a pharyngeal pouch represents a transient protrusion, a pharyngeal diverticulum is a persistent protrusion, persisting on barium studies after the bolus has passed. Pharyngeal diverticula arise at the thyrohyoid membrane and are seen along the anterolateral pharynx (Fig 7), typically due to chronically increased intrapharyngeal pressures as may occur in horn players or glass blowers (13,19).

Zenker diverticula are the most common diverticular cause of dysphagia (19). A Zenker diverticulum is an acquired mucosal herniation arising in the hypopharynx just proximal to the cricopharyngeus muscle. It arises posteriorly at the level of relative muscular weakness known as Killian dehiscence (Fig 8) (8). Although it arises in the midline, a large Zenker diverticulum can be seen to extend laterally when viewed in the anteroposterior projection (13). A large Zenker diverticulum may further contribute to dysphagia due to associated mass effect and compression of the cervical esophagus. Zenker diverticula are associated with cricopharyngeal dysfunction and GERD.

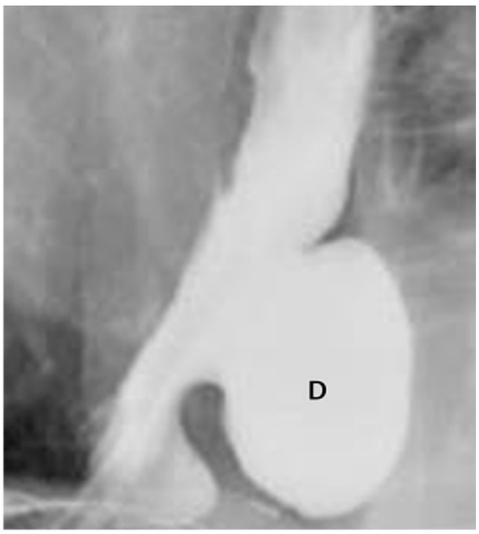
Killian-Jamieson diverticula are located more inferiorly than Zenker diverticula, at the level of the Killian-Jamieson space. A Killian-Jamieson diverticulum occurs below the level of the cricopharyngeus muscle, arising from the lateral and anterior wall of the cervical esophagus (Fig 9). These diverticula are less common than Zenker diverticula and tend to be less symptomatic (19).

7.

8.

Figures 7, 8. **(7)** Lateral pharyngeal diverticulum. Anteroposterior image obtained during swallowing shows a focal outpouching along the lateral pharynx (arrow). This finding persisted after the swallow and was located slightly anteriorly on the lateral images (not shown), indicative of a lateral pharyngeal diverticulum. **(8)** Zenker diverticulum. Slightly oblique lateral image obtained during swallowing shows a large diverticulum arising posteriorly (arrow) at the level of the cricopharyngeus muscle. The diverticulum extends inferiorly to exert mass effect on the cervical esophagus and was seen in the midline in the anteroposterior projection.

9.



10.

Figures 9, 10. **(9)** Killian-Jamieson diverticulum. Slightly oblique lateral image obtained during swallowing shows a diverticulum arising anteriorly from the cervical esophagus (arrow) below the level of the cricopharyngeus muscle. The diverticulum was seen extending laterally and to the left of midline in the anteroposterior projection. **(10)** Epiphrenic diverticulum. Single-contrast prone RAO image shows a large epiphrenic diverticulum *(D)* arising from the distal thoracic esophagus. Associated esophageal dysmotility with numerous tertiary contractions was noted at fluoroscopy.

Epiphrenic diverticula occur in the distal third of the thoracic esophagus. They represent pulsion diverticula and are often associated with motility abnormalities (Fig 10).

Patients with symptomatic pharyngoesophageal diverticula may be treated with endoscopic or surgical diverticulectomy or diverticuloplexy (diverticular suspension). A simultaneous myotomy may be performed as well (eg, cricopharyngeal myotomy for a Zenker diverticulum and distal esophagomyotomy for an epiphrenic diverticulum). Endoscopic diverticulotomy may be successful in some patients.

Webs and Rings

Esophageal webs are thin mucosal folds that typically appear as a shelf-like filling defect 1–2 mm in height in the cervical esophagus or lower hypopharynx. Cervical esophageal webs are most often located anteriorly (Fig 11) but may be circumferential (25). Although cervical esophageal webs may be an incidental finding, they can result in proximal dilatation and varying degrees of obstruction. Esophageal webs can be multiple, and maximal luminal distention is often necessary to make the diagnosis. A constricting proximal web can limit the ability to diagnose an additional more distal web. Esophageal webs are associated with other conditions such as benign mucous membrane pemphigoid, epidermolysis bullosa dystrophica, and GERD (26).

Schatzki ring is the most common type of esophageal ring. It appears as a short, smooth, concentric ring in the distal esophagus (B ring) measuring 1–3 mm in height and typically associated with a hiatal hernia (Fig 12) (22). Esophageal rings are best identified and characterized with maximal luminal distention, achieved by acquiring images with the patient drinking barium while in the prone RAO position (10,27).

Patients with a symptomatic esophageal web or ring may respond to dietary changes. If conservative treatment fails, endoscopic dilation may be helpful.

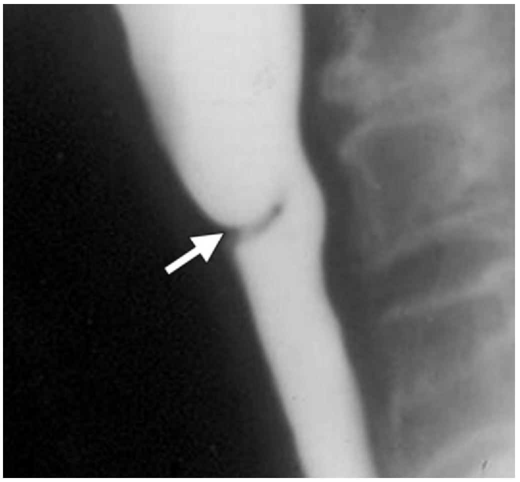


Figure 11. Anterior cervical esophageal web. Lateral image obtained during swallowing shows a thin, shelf-like mucosal fold (arrow) in the anterior cervical esophagus. Mild proximal dilatation is also noted.

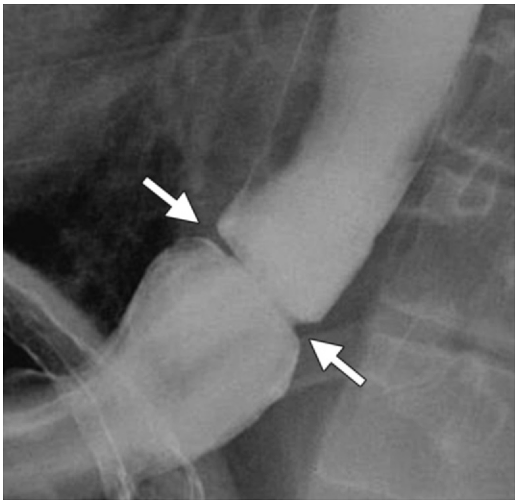


Figure 12. Schatzki ring. Prone RAO image acquired as the patient was drinking barium shows a short, smooth concentric ring in the distal esophagus (arrows) just above an associated small hiatal hernia. Note the maximal luminal distention above and below the ring.

Extrinsic Compression of the Pharynx or Esophagus

Extrinsic compression of the esophagus may result in dysphagia. Extrinsic compression can be due to mass effect from adjacent tumors, masses or adenopathy, cardiovascular processes, or musculoskeletal abnormalities. If mass effect on the esophagus is seen at esophagography, cross-sectional imaging may be necessary for a more definitive diagnosis.

Extrinsic Masses.—Mass effect due to thyroid disease is most often seen at the level of the cervical esophagus. However, superior mediastinal extension of a thyroid mass can result in mass effect on the upper thoracic esophagus at esophagography. Rapid-sequence imaging with the patient in the anteroposterior position best depicts this mass effect (Fig 13). However, mild narrowing may occur as a normal finding at the level of the thoracic inlet and should not be mistaken for thyroid disease. Thyroid ultrasonography (US) or neck CT may help confirm thyroid disease and help determine its extent. Extrinsic compression of the esophagus may also be seen due to lung cancer and metastatic adenopathy (Fig 14), and there may be associated secondary invasion of the esophagus. Chest CT is helpful in determining disease extent.

Cardiovascular Compression.—Vascular compression of the esophagus may be due to anatomic variants such as an aberrant right subclavian artery (Fig 15) or to cardiovascular disease processes (Fig 16) including but not limited to aortic aneurysms, dissection, and cardiomegaly. The appearance of mass effect from cardiomegaly or an aberrant right subclavian artery at esophagography may be essentially diagnostic; however, other cardiovascular processes may require chest CT or MR angiography for a more definitive diagnosis (Fig 16b).

Musculoskeletal Processes.—The esophagus may also be compressed due to musculoskeletal processes, most often osteophytes (28). The

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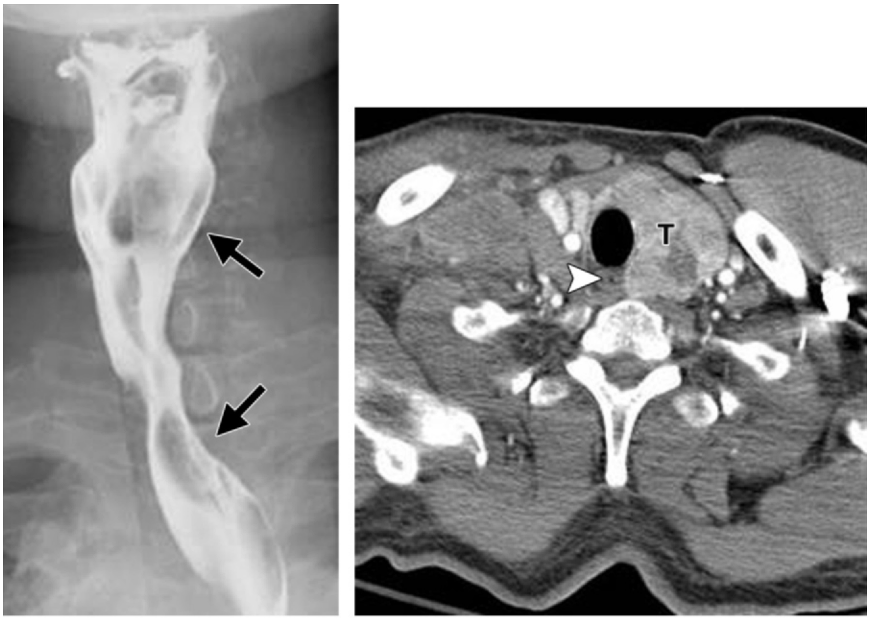
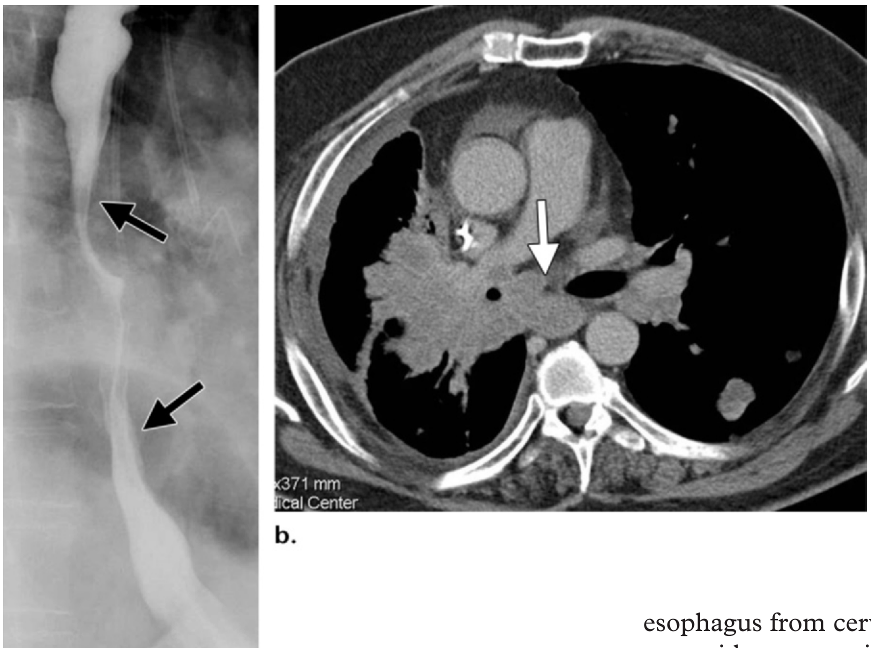


a. b.

Figure 13. Mass effect due to thyroid disease. **(a)** Anteroposterior image obtained during swallowing shows extrinsic mass effect (arrows) from a large mass in the left side of the neck, which displaces the cervical esophagus to the right. A transient right lateral pharyngeal pouch is also noted. **(b)** Contrast-enhanced CT image shows a large left thyroid goiter *(T)* displacing the trachea and esophagus (arrowhead) to the right of midline.



a. b.

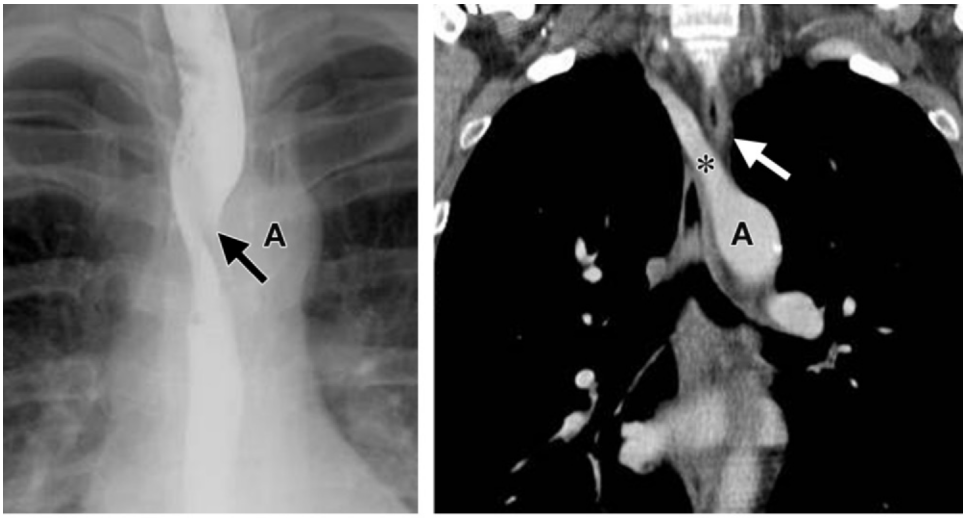
Figure 14. Lung cancer involving the esophagus. **(a)** Single-contrast LPO image shows mass effect throughout the midthoracic esophagus (arrows). **(b)** Contrast-enhanced CT image shows a large lung cancer invading the mediastinum, with severe compression of the esophagus at this level (arrow).

esophagus is most likely to be compressed by osteophytes at the level of C5-C6, where the lumen is fixed to the cricoid cartilage, and in the upper thoracic esophagus, where it is attached to the diaphragm (29). However, large osteophytes can cause mechanical blockage or induce a localized inflammatory reaction affecting the esophagus elsewhere, thereby producing dysphagia. Although 20%–30% of the general population have hypertrophic spurs along the anterior cervical spine, only about 6% of this group report having dysphagia (30,31). Mass effect on the cervical esophagus from cervical osteophytes is best seen on rapid-sequence images obtained during swallowing with the patient in the straight lateral position (Fig 17). Lateral oblique and anteroposterior images may fail to reveal the finding.

Esophageal Infections

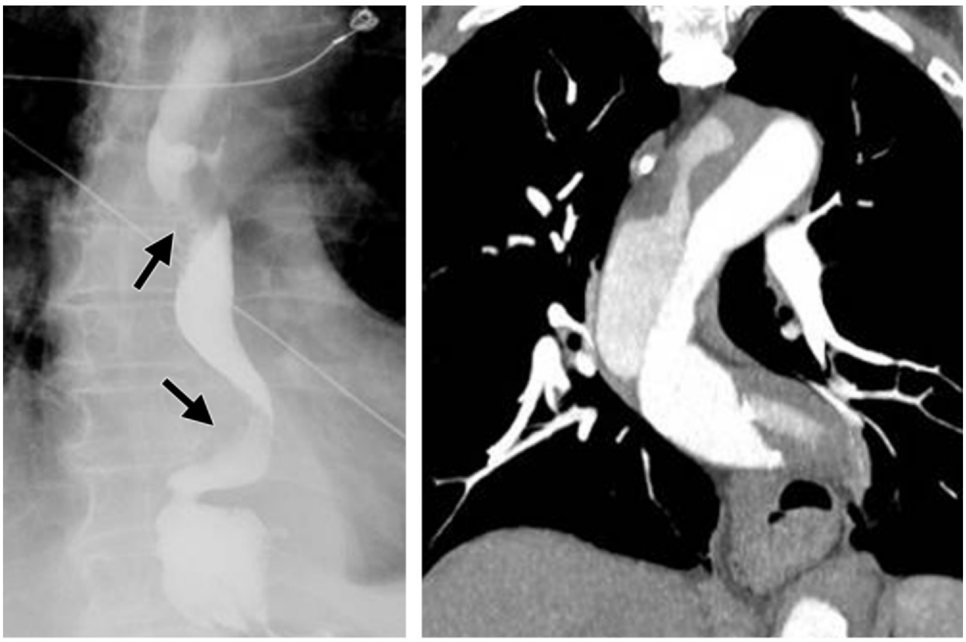
Patients with esophageal infections such as candidiasis, herpesvirus, human immunodeficiency virus (HIV), and cytomegalovirus (CMV) may present with dysphagia as well as odynophagia. Severe esophageal infection can result in subsequent development of strictures.

Candidiasis is the most common cause of infectious esophagitis, especially in immunocompromised patients. At double-contrast esophagography, candidiasis appears as plaques or



a. b.

Figure 15. Aberrant right subclavian artery. **(a)** Single-contrast anteroposterior image shows obliquely oriented mass effect on the thoracic esophagus (arrow) just at and above a left-sided aortic arch *(A)*. Mass effect was noted posteriorly in the lateral projection. **(b)** Contrast-enhanced CT image shows an aberrant right subclavian artery (*) arising from the left-sided aortic arch *(A)* and crossing the esophagus (arrow) as it extends obliquely to the right.



a. b.

Figure 16. Dissecting thoracic aortic aneurysm. **(a)** Single-contrast anteroposterior image shows multifocal mass effect on the thoracic esophagus (arrows). **(b)** Coronal contrast-enhanced maximum-intensity-projection CT image reveals that the mass effect is due to a dilated thoracic aorta with aortic dissection.

nodules that are longitudinally oriented within the esophagus, with normal intervening mucosa (Fig 18) (32). In patients with AIDS, candidiasis may have a classic "shaggy" appearance with pronounced irregularity of the luminal contour at esophagography.

Herpesvirus is the most common cause of viral esophagitis and most often appears at double-contrast imaging as small ulcers, typically less than 1 cm in size (Fig 19) (32,33). Herpes ulcers are most often multiple and superficial. Herpesvirus esophagitis can occur in otherwise healthy immunocompetent patients.

HIV and CMV ulcers are typically larger than herpesvirus ulcers. HIV and CMV infections cause large flat ulcers, most often greater than 1 cm in size. It is not possible to distinguish CMV ulcers from HIV ulcers at esophagography, and

Figure 17. Cervical osteophytes. Lateral image obtained during swallowing shows extrinsic mass effect on the pharynx and cervical esophagus from confluent osteophytes in the anterior cervical spine (arrows).

Figure 18. Candidiasis. Double-contrast upright LPO image shows plaques and nodules due to candidal infection that are longitudinally oriented within the thoracic esophagus.

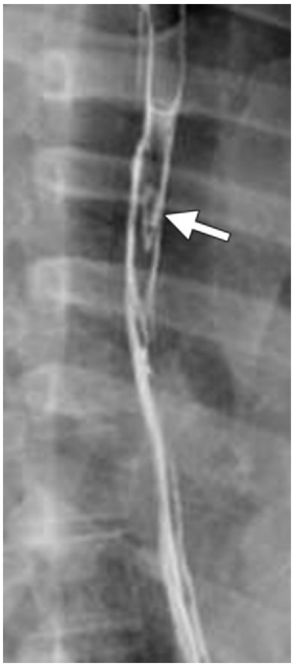


Figure 19. Herpes virus esophagitis. Double-contrast LPO image shows a small superficial ulcer (arrow) due to herpes infection in the mid- to upper esophagus.

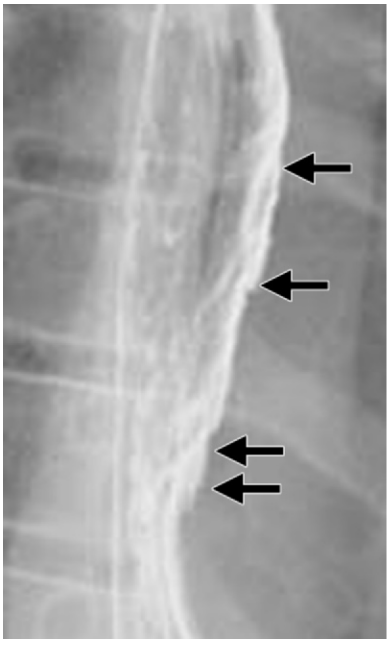


Figure 20. CMV esophagitis in an immunocompromised patient. Double-contrast LPO image shows several large flat ulcers due to CMV infection in the thoracic esophagus. The margins of two of the ulcers are seen in profile (arrows). HIV ulcers would appear similar, and endoscopy is necessary for accurate diagnosis and treatment.

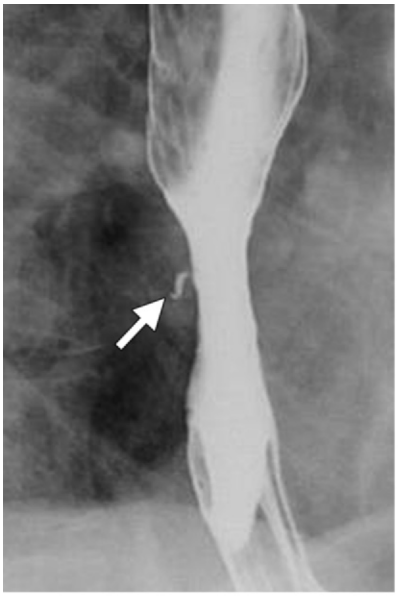
endoscopy is necessary for accurate diagnosis and treatment (Fig 20) (34,35).

Esophageal Strictures

Benign esophageal strictures are a leading cause of dysphagia, and dysphagia is the most common presenting complaint of patients with these strictures (22). Patients with benign strictures tend to have a history of long-standing, intermittent, or nonprogressive dysphagia. On the other hand, patients with malignant strictures present with more recent onset of rapidly progressive dysphagia and associated weight loss (1). Multiple processes may cause benign esophageal strictures, and, although there is overlap among possible diagnoses, the differential diagnosis may be narrowed based on the location of the esophageal stricture. In some cases, endoscopy with brushings or biopsy may be necessary to establish a definitive diagnosis and exclude malignancy. Symptomatic benign esophageal strictures may be successfully treated with endoscopic dilation; however, refractory or severe strictures may require stent placement or surgical esophageal reconstruction.

Distal Esophageal Strictures.—The most common stricture found in the distal esophagus is a GERD-induced peptic stricture. In fact, GERD is the leading cause of esophageal strictures

RadioGraphics



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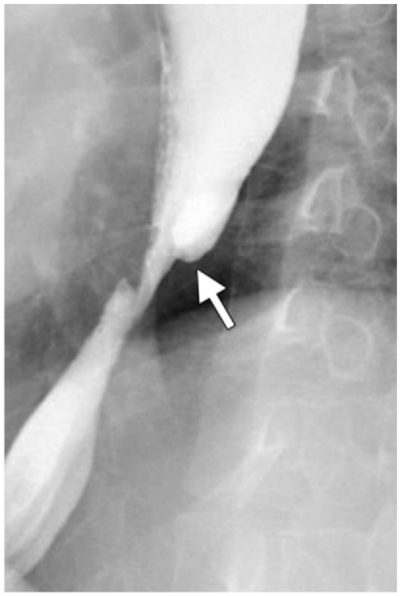


22.

Figures 21, 22. **(21)** Peptic stricture with esophageal intramural pseudodiverticulosis (EIP). Double-contrast upright LPO image shows smooth, tapered narrowing in the distal esophagus above a small hiatal hernia with associated EIP, a finding that appears as a tiny outpouching of contrast material (arrow). Mild nodularity of the distal esophageal mucosa due to reflux esophagitis is noted slightly more superiorly. **(22)** Peptic stricture with sacculation. Single-contrast prone RAO image shows a focal peptic stricture with narrowing in the distal esophagus above a small hiatal hernia. Note the associated sacculation due to scarring along the proximal aspect of the stricture (arrow).

overall (22). Peptic strictures most often occur in the distal esophagus and typically appear at esophagography as smooth, tapered narrowing that extends for a distance of 1–4 cm (Fig 21) (36). Peptic strictures are usually concentric but may appear asymmetric. Scarring can lead to sacculation (Fig 22), and there may be associated EIP (Fig 21) (22). Other entities that may cause distal esophageal strictures include scleroderma, an indwelling nasogastric tube, Zollinger-Ellison syndrome, and alkaline reflux esophagitis.

Upper to Midesophageal Strictures.—If a stricture is identified in the mid- to upper esophagus, Barrett esophagus should be considered (22). This condition occurs most often as a consequence of long-standing GERD and reflux esophagitis and represents acquired progressive columnar metaplasia of the squamous epithelium in the distal esophagus (37,38). Barrett esophagus is a premalignant condition with an associated increased risk of adenocarcinoma. Most Barrett strictures appear as distal esophageal peptic-type strictures. However, a stricture in the mid- to upper esophagus—a less common finding—is more specific for Barrett esophagus (Fig 23) (22,39). In the setting of a hiatal hernia and gastroesophageal reflux, a focal stricture in the midesophagus is highly suggestive of Barrett esophagus (22). If Barrett esophagus is suspected, endoscopy and biopsy should be performed. If the diagnosis of Barrett esophagus is confirmed, periodic endoscopic surveillance with biopsies should be performed to detect and treat dysplastic changes before the development of invasive carcinoma. Other

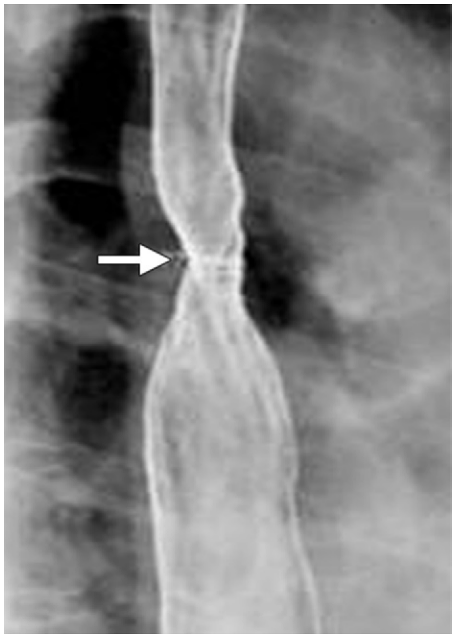
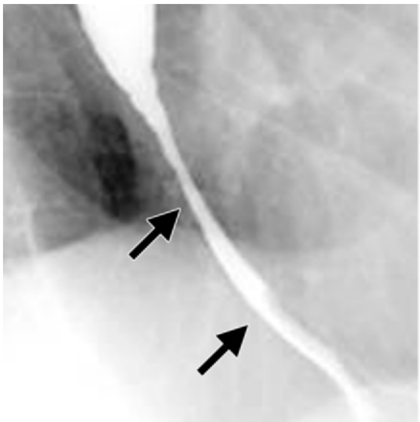


Figure 23. Barrett esophagus. Double-contrast upright LPO image shows a focal stricture due to Barrett esophagus in the midesophagus (arrow). Note the mildly nodular appearance of the more inferior esophageal mucosa due to reflux esophagitis. The GEJ has a patulous appearance.

possible causes of strictures in the mid- to upper esophagus include mediastinal radiation therapy, caustic ingestion (Fig 24), medication-induced stricture (Fig 25), congenital esophageal stenosis, and skin disorders.

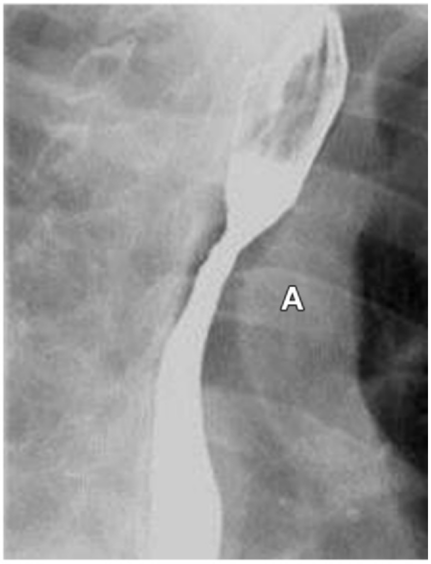
Other Causes of Strictures.—Additional causes of benign esophageal strictures include EIP, Crohn disease, graft-versus-host disease, candidiasis,



24a.

Figures 24, 25. **(24)** Caustic ingestion in a patient who had ingested lye 1 year earlier. **(24a)** Single-contrast LPO image shows a long stricture with tapered margins in the mid- to distal esophagus (arrows). **(24b)** Single-contrast supine image shows a focal stricture with tapered margins in the gastric antrum (arrows). **(25)** Pill-induced esophagitis complicated by an upper esophageal stricture. Initial double-contrast esophagography showed three small ulcers at the level of the aortic arch due to pill-induced esophagitis. LPO image from an esophagographic study performed 3 months later shows subsequent development of a stricture with tapered margins at the same level. *A* = aortic arch.

24b.



25.

Behçet disease, prior sclerotherapy, and eosinophilic esophagitis (22).

EIP represents dilated excretory ducts of deep mucous glands, appearing as flask-shaped outpouchings or tiny collections of contrast material (1–4 mm in size) that seem to float outside the esophagus (Fig 26). EIP may occur as a consequence of scarring and is associated with strictures in up to 90% of patients (22). Bridging of pseudodiverticula may result in intramural tracks.

Patients with Crohn disease may present with dysphagia caused by the development of benign strictures. Benign strictures can occur in the pharynx (Fig 27) and/or esophagus, albeit typically only in the setting of involvement by Crohn disease elsewhere (eg, within the small bowel or colon).

Idiopathic eosinophilic esophagitis is an increasingly common inflammatory condition that may lead to stricture formation. It is characterized pathologically by eosinophilic infiltration of the esophagus, a condition that is thought to occur as an inflammatory response to food allergens. Patients may present with long-standing dysphagia and recurrent food impactions and may also have a history of allergies, asthma, and/or peripheral eosinophilia. Idiopathic eosinophilic esophagitis may cause segmental or diffuse esophageal strictures that can be diagnosed with esophagography. Strictures occur most often in the upper to midthoracic esophagus and may be associated with distinctive ringlike indentations ("ringed esophagus"). Alternatively, there may be a diffusely small-caliber esophagus (40). However, endoscopy should be performed for a definitive diagnosis. Treatment options may include steroid therapy and dietary changes. When esophageal strictures are present, endoscopic dilation may be helpful.

Esophageal Cancer

Patients with esophageal carcinoma tend to present with recent onset and rapidly progressive dysphagia with associated weight loss. Historically, the most common pathologic type of esophageal cancer was squamous cell carcinoma.

Figure 26. EIP with mild benign focal stricture. Double-contrast LPO image shows multiple tiny flask-shaped outpouchings of EIP associated with a mild stricture in the upper esophagus (arrows). The outpouchings appear to float outside the esophagus and are also seen more inferiorly within the esophagus.

Figure 27. Pharyngeal Crohn disease with aspiration in a patient with known ileocolic Crohn disease. Lateral image obtained during swallowing shows lobulated filling defects within a distorted pharynx and a focal stricture at the pharyngoesophageal junction due to Crohn disease. Aspirated contrast material is seen in the trachea (arrows).

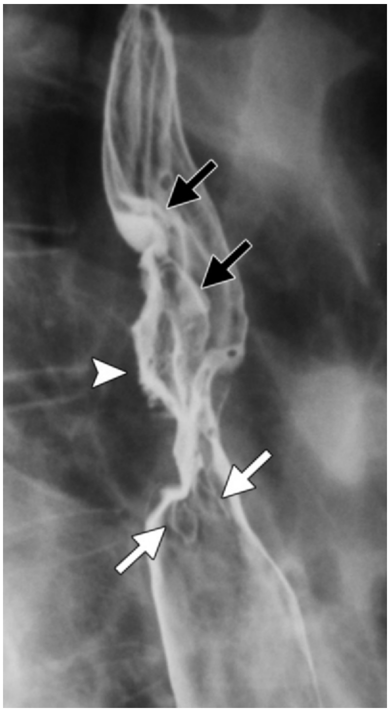


Figure 28. Primary esophageal cancer. Double-contrast LPO image shows a large mass in the mid- to upper esophagus with polypoid (white arrows), ulcerative (arrowhead), and varicoid (black arrows) components. Pathologic analysis revealed esophageal squamous cell carcinoma.

In recent years, however, there has been an overwhelming increase in the prevalence of esophageal adenocarcinoma (41–43). Adenocarcinoma is most often found in the distal esophagus and may arise in Barrett esophagus or as gastric cancer invading the distal esophagus. However, oropharyngeal cancers remain predominantly squamous cell carcinoma (21). Regardless of pathologic type, esophageal cancer can appear infiltrative, ulcerative, polypoid, and/or varicoid at esophagography (Fig 28) (44–46). Invasive esophageal cancer may result in sinus tracks, fistulous communication with the airway or mediastinum, and/or invasion of adjacent structures (47). Chest and abdominal CT and/or positron emission tomography (PET)/CT should be performed for assessment for possible resectability and staging of disease extent, including evaluation for lymphadenopathy and distant metastases.

Patients with achalasia have a substantially greater risk of developing primary esophageal cancer (nine to 28 times that of persons without achalasia) (48). Because of the long-standing history of dysphagia in patients with achalasia, secondary cancers tend to manifest late in the disease course, appearing as a large, bulky mass within a markedly dilated esophagus. As a consequence, patients have a poor prognosis (48,49). In the absence of underlying achalasia, an extensive, bulky intraluminal mass may suggest a diagnosis of esophageal sarcoma (Fig 29). A gastrointestinal stromal tumor arising from the stomach may appear as a large, bulky mass in the vicinity of the GEJ (Fig 30), and patients may present with dysphagia.

Other Masses

Patients with an impacted foreign body or food bolus in the esophagus may present with acute onset of dysphagia. Water-soluble contrast material should be used for esophagography if this is a concern, since there is an increased risk of perforation with increased duration of impaction. The impacted food or object can be seen as a focal mass or filling defect in the esophagus and may be expanding the lumen (Fig 31). Obstruction may be present. At esophagography, contrast material may be seen filling the interstices of an impacted food bolus or within a foreign body, thereby helping differentiate such a process from neoplasm.

Other disease entities that can appear masslike within the esophagus in patients who present with dysphagia include leiomyoma, duplication cyst, and, rarely, esophageal hematoma. Leiomyomas represent more than one-half of all benign

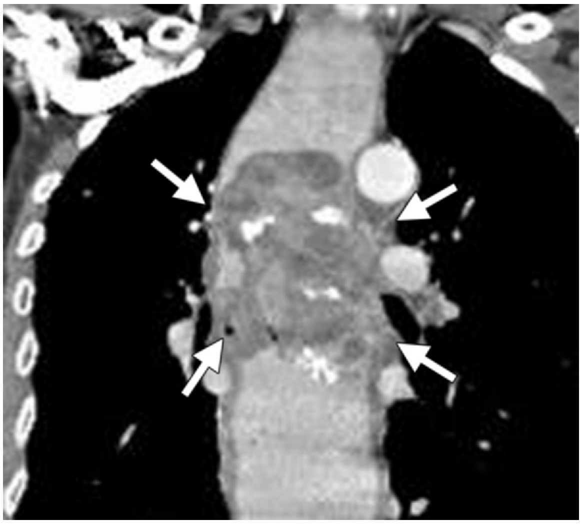


Figure 29. Esophageal sarcoma. Coronal contrast-enhanced multiplanar reformatted chest CT image shows dilatation of the esophagus by a large, bulky soft-tissue mass (arrows) that contains calcifications and expands the lumen. Pathologic analysis revealed esophageal sarcoma.

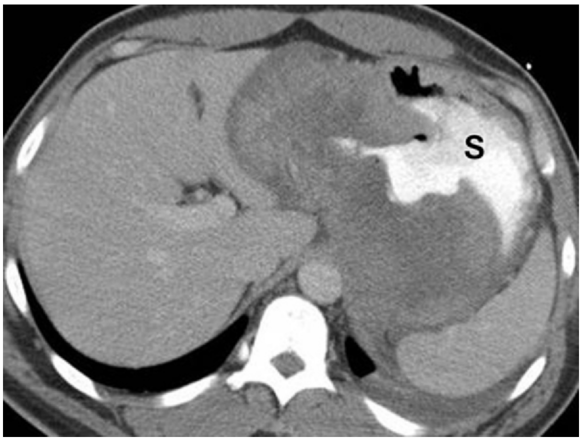


Figure 30. Gastrointestinal stromal tumor arising from the stomach. Contrast-enhanced CT image shows a large, bulky ulcerative mass arising from the stomach *(S)* and extending into the vicinity of the GEJ, findings that are consistent with a malignant gastrointestinal stromal tumor.

esophageal tumors and tend to be solitary submucosal masses ranging from 2 to 8 cm in size (Fig 32) (43,50,51). A duplication cyst occurs in the esophagus in 20% of cases (52). Esophageal duplication cysts may be cystic or tubular and generally do not communicate with the esophageal lumen (Fig 33).

Postoperative Dysphagia

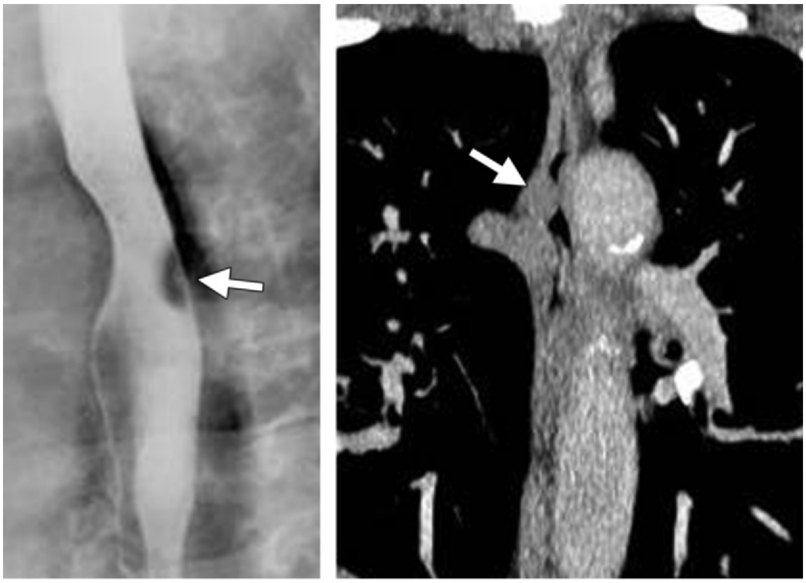
Patients may present with dysphagia following surgery involving the esophagus, pharynx, larynx, stomach, or adjacent structures. Possible causes of dysphagia in the postoperative setting include aspiration (due to recent intubation or to postoperative changes involving the pharyngolaryngeal region), leak, fistula, strictures, malfunction of

Figure 31. Impacted food bolus. Lateral image obtained during swallowing shows a lobulated filling defect (arrows) below the cricopharyngeus muscle in the cervical esophagus, a finding that is consistent with an impacted food bolus. Note the presence of contrast material in the interstices of the "mass."

a surgical device (eg, cervical fusion plate, gastric band), obstruction, indwelling foreign body, motility abnormalities, or other postoperative complications.

Dysphagia following laryngectomy can occur due to leak and/or fistula in the early postoperative course (Fig 34) or due to stricture in the more delayed postoperative course (Fig 35). Pharyngocutaneous fistulas occur in 6%–21% of patients following laryngectomy, typically in the early postoperative course (Fig 34) (53). Delayed development of fistula could indicate recurrent tumor (53). Following anterior cervical fusion or other neck surgery, dysphagia may occur in the early postoperative course due to edema and/or hematoma, with soft-tissue swelling and displacement of the pharynx and/or cervical esophagus. In the delayed postoperative course, dysphagia may be due to hardware malfunction, fibrosis, or esophageal stricture. Leak and strictures can also occur following esophagectomy.

It is important to remember that patients with a history of surgery involving the GEJ and proximal stomach may also present with dysphagia in the early or late postoperative course. Following fundoplication, dysphagia can occur due to *(a)* a tight wrap with associated esophageal dilatation, or *(b)* partial or complete disruption of the fundoplication wrap with a recurrent hiatal hernia (54). Bariatric surgery is a popular treatment option for morbidly obese patients, and alterations made



a. b.

Figure 32. Benign esophageal leiomyoma. **(a)** Single-contrast prone RAO image shows a small filling defect (arrow) protruding into the lumen of the esophagus near the level of the aortic arch. The lesion's obtuse borders suggest that it is submucosal in nature. **(b)** Coronal contrast-enhanced multiplanar reformatted CT image shows a corresponding small soft-tissue mass, also with obtuse margins, at the same level (arrow) and protruding into the esophageal lumen. Pathologic analysis revealed a benign leiomyoma.

Figure 33. Esophageal duplication cyst. Single-contrast prone RAO image shows a prominent filling defect (arrow) protruding into the lumen of the distal esophagus. The lesion has obtuse margins and does not communicate with the esophageal lumen. Endoscopic US revealed an esophageal duplication cyst.

Figure 34. Pharyngocutaneous fistula following laryngectomy. Lateral image obtained during swallowing of water-soluble contrast material shows extravasation of contrast material from the anterior aspect of the inferior pharynx, a finding that is consistent with early postoperative leak. Extraluminal contrast material (arrows) tracks anterior and inferior to a tracheostomy toward the skin surface, a finding that is consistent with fistula.

to the gastrointestinal tract—in particular, the stomach—can result in complications producing dysphagia. Following gastric band placement, dysphagia can occur due to a tight stoma with pouch obstruction, band erosion, or band slippage with fundic herniation (55). Following Roux-en-Y gastric bypass surgery, patients may present with dysphagia due to *(a)* pouch dilatation with gastrojejunal stomal obstruction, or *(b)* more downstream processes, including obstruction at or related to the jejunojejunal anastomosis (56). In addition, esophageal motility disorders are not uncommon following bariatric procedures. In these patients, the surgical anatomy should be evaluated before routine esophagography is performed.

Conclusion

Dysphagia is a common clinical problem, and imaging is essential for evaluating patients who have difficulty swallowing. Barium studies remain the preferred method for evaluating patients with dysphagia, allowing assessment of function and morphology. A wide spectrum of oral, pharyngeal, esophageal, and proximal gastric diseases may manifest with dysphagia. An accurate clinical and surgical history may help guide the radiologic examination. Thorough radiologic assessment and a knowledge of the imaging spectrum of the possible causes of dysphagia may help avoid misdiagnosis.

Figure 35. Delayed stricture in a patient who had undergone laryngectomy 6 months earlier. Lateral image obtained during swallowing shows a focal benign stricture with tapered margins in the neopharynx (arrow).

References

1. Kuo P, Holloway RH, Nguyen NQ. Current and future techniques in the evaluation of dysphagia. J Gastroenterol Hepatol 2012;27(5):873–881.
2. Domenech E, Kelly J. Swallowing disorders. Med Clin North Am 1999;83(1):97–113, ix.
3. Rofes L, Arreola V, Romea M, et al. Pathophysiology of oropharyngeal dysphagia in the frail elderly. Neurogastroenterol Motil 2010;22(8):851–858, e230.
4. Wilkins T, Gillies RA, Thomas AM, Wagner PJ. The prevalence of dysphagia in primary care patients: a HamesNet Research Network study. J Am Board Fam Med 2007;20(2):144–150.
5. Ekberg O, Hamdy S, Woisard V, Wuttge-Hannig A, Ortega P. Social and psychological burden of dysphagia: its impact on diagnosis and treatment. Dysphagia 2002;17(2):139–146.
6. Locke GR 3rd, Talley NJ, Fett SL, Zinsmeister AR, Melton LJ 3rd. Prevalence and clinical spectrum of gastroesophageal reflux: a population-based study in Olmsted County, Minnesota. Gastroenterology 1997;112(5):1448–1456.
7. Marik PE, Kaplan D. Aspiration pneumonia and dysphagia in the elderly. Chest 2003;124(1):328–336.
8. Cook IJ. Oropharyngeal dysphagia. Gastroenterol Clin North Am 2009;38(3):411–431.
9. Schindler JS, Kelly JH. Swallowing disorders in the elderly. Laryngoscope 2002;112(4):589–602.
10. Levine MS, Rubesin SE. Radiologic investigation of dysphagia. AJR Am J Roentgenol 1990;154(6):1157–1163.
11. Wilcox CM, Alexander LN, Clark WS. Localization of an obstructing esophageal lesion: is the patient accurate? Dig Dis Sci 1995;40(10):2192–2196.
12. Smith Hammond C. Cough and aspiration of food and liquids due to oral pharyngeal dysphagia. Lung 2008;186(suppl 1):S35–S40.
13. Rubesin SE. Oral and pharyngeal dysphagia. Gastroenterol Clin North Am 1995;24(2):331–352.
14. DiSantis DJ. Gastrointestinal fluoroscopy: what are we still doing? AJR Am J Roentgenol 2008;191(5):1480–1482.
15. Gelfand DW, Ott DJ, Chen YM. Decreasing numbers of gastrointestinal studies: report of data from 69 radiologic practices. AJR Am J Roentgenol 1987;148(6):1133–1136.
16. Hartl DM, Kolb F, Bretagne E, Marandas P, Sigal R. Cine magnetic resonance imaging with single-shot fast spin echo for evaluation of dysphagia and aspiration. Dysphagia 2006;21(3):156–162.
17. Fujii N, Inamoto Y, Saitoh E, et al. Evaluation of swallowing using 320-detector-row multislice CT. I. Single- and multiphase volume scanning for three-dimensional morphological and kinematic analysis. Dysphagia 2011;26(2):99–107.
18. Katz PO, Castell DO. Esophageal motility disorders. Am J Med Sci 1985;290(2):61–69.
19. Grant PD, Morgan DE, Scholz FJ, Canon CL. Pharyngeal dysphagia: what the radiologist needs to know. Curr Probl Diagn Radiol 2009;38(1):17–32.
20. Garon BR, Sierzant T, Ormiston C. Silent aspiration: results of 2,000 video fluoroscopic evaluations. J Neurosci Nurs 2009;41(4):178–185.
21. Tao TY, Menias CO, Herman TE, McAlister WH, Balfe DM. Easier to swallow: pictorial review of structural findings of the pharynx at barium pharyngography. RadioGraphics 2013;33(7):e189–e208.
22. Luedtke P, Levine MS, Rubesin SE, Weinstein DS, Laufer I. Radiologic diagnosis of benign esophageal strictures: a pattern approach. RadioGraphics 2003;23(4):897–909.
23. Jones B. Normal and abnormal swallowing: imaging in diagnosis and therapy. 2nd ed. New York, NY: Springer, 2003.
24. Pikus L, Levine MS, Yang YX, et al. Videofluoroscopic studies of swallowing dysfunction and the relative risk of pneumonia. AJR Am J Roentgenol 2003;180(6):1613–1616.
25. Clements JL Jr, Cox GW, Torres WE, Weens HS. Cervical esophageal webs: a roentgenanatomic correlation—observations on the pharyngoesophagus. Am J Roentgenol Radium Ther Nucl Med 1974;121(2):221–231.
26. Nosher JL, Campbell WL, Seaman WB. The clinical significance of cervical esophageal and hypopharyngeal webs. Radiology 1975;117(1):45–47.
27. Ott DJ, Chen YM, Wu WC, Gelfand DW, Munitz HA. Radiographic and endoscopic sensitivity in detecting lower esophageal mucosal ring. AJR Am J Roentgenol 1986;147(2):261–265.
28. Strasser G, Schima W, Schober E, Pokieser P, Kaider A, Denk DM. Cervical osteophytes impinging on the pharynx: importance of size and concurrent disorders for development of aspiration. AJR Am J Roentgenol 2000;174(2):449–453.
29. Yutan E, Daras M, Koppel BS. Dysphagia due to cervical osteophytes. Clin Imaging 2001;25(4):262–264.
30. Bone RC, Nahum AM, Harris AS. Evaluation and correction of dysphagia-producing cervical osteophytosis. Laryngoscope 1974;84(11):2045–2050.
31. Hilding DA, Tachdjian MO. Dysphagia and hypertrophic spurring of the cervical spine. N Engl J Med 1960;263:11–14.
32. Levine MS, Woldenberg R, Herlinger H, Laufer I. Opportunistic esophagitis in AIDS: radiographic diagnosis. Radiology 1987;165(3):815–820.
33. Wilcox CM, Schwartz DA, Clark WS. Esophageal ulceration in human immunodeficiency virus infection: causes, response to therapy, and long-term outcome. Ann Intern Med 1995;123(2):143–149.

34. Sor S, Levine MS, Kowalski TE, Laufer I, Rubesin SE, Herlinger H. Giant ulcers of the esophagus in patients with human immunodeficiency virus: clinical, radiographic, and pathologic findings. Radiology 1995;194(2):447–451.
35. Werneck-Silva AL, Prado IB. Role of upper endoscopy in diagnosing opportunistic infections in human immunodeficiency virus-infected patients. World J Gastroenterol 2009;15(9):1050–1056.
36. Ott DJ, Gelfand DW, Lane TG, Wu WC. Radiologic detection and spectrum of appearances of peptic esophageal strictures. J Clin Gastroenterol 1982;4(1):11–15.
37. Agha FP. Barrett carcinoma of the esophagus: clinical and radiographic analysis of 34 cases. AJR Am J Roentgenol 1985;145(1):41–46.
38. Spechler SJ, Goyal RK. Barrett's esophagus. N Engl J Med 1986;315(6):362–371.
39. Levine MS, Herman JB, Furth EE. Barrett's esophagus and esophageal adenocarcinoma: the scope of the problem. Abdom Imaging 1995;20(4):291–298.
40. White SB, Levine MS, Rubesin SE, Spencer GS, Katzka DA, Laufer I. The small-caliber esophagus: radiographic sign of idiopathic eosinophilic esophagitis. Radiology 2010; 256(1):127–134.
41. Blot WJ, Devesa SS, Fraumeni JF Jr. Continuing climb in rates of esophageal adenocarcinoma: an update. JAMA 1993;270(11):1320.
42. Devesa SS, Blot WJ, Fraumeni JF Jr. Changing patterns in the incidence of esophageal and gastric carcinoma in the United States. Cancer 1998;83(10):2049–2053.
43. Lewis RB, Mehrotra AK, Rodriguez P, Levine MS. Esophageal neoplasms: radiologic-pathologic correlation. RadioGraphics 2013;33(4):1083–1108.
44. Goldstein HM, Zornoza J, Hopens T. Intrinsic diseases of the adult esophagus: benign and malignant tumors. Semin Roentgenol 1981;16(3):183–197.
45. Levine MS, Chu P, Furth EE, Rubesin SE, Laufer I, Herlinger H. Carcinoma of the esophagus and esophagogastric junction: sensitivity of radiographic diagnosis. AJR Am J Roentgenol 1997;168(6):1423–1426.
46. Wiot JW, Felson B. Cancer of the gastrointestinal tract: radiographic differential diagnosis. JAMA 1973;226(13): 1548–1552.
47. Levine MS, Halvorsen RA. Carcinoma of the esophagus. In: Gore RM, Levine MS, eds. Textbook of gastrointestinal radiology. 3rd ed. Philadelphia, Pa: Saunders, 2008; 417–446.
48. Carter R, Brewer LA 3rd. Achalasia and esophageal carcinoma: studies in early diagnosis for improved surgical management. Am J Surg 1975;130(2):114–120.
49. Hankins JR, McLaughlin JS. The association of carcinoma of the esophagus with achalasia. J Thorac Cardiovasc Surg 1975;69(3):355–360.
50. Plachta A. Benign tumors of the esophagus: review of literature and report of 99 cases. Am J Gastroenterol 1962;38:639–652.
51. Seremetis MG, Lyons WS, deGuzman VC, Peabody JW Jr. Leiomyomata of the esophagus: an analysis of 838 cases. Cancer 1976;38(5):2166–2177.
52. Macpherson RI. Gastrointestinal tract duplications: clinical, pathologic, etiologic, and radiologic considerations. RadioGraphics 1993;13(5):1063–1080.
53. Hier M, Black MJ, Lafond G. Pharyngo-cutaneous fistulas after total laryngectomy: incidence, etiology and outcome analysis. J Otolaryngol 1993;22(3):164–166.
54. Canon CL, Morgan DE, Einstein DM, Herts BR, Hawn MT, Johnson LF. Surgical approach to gastroesophageal reflux disease: what the radiologist needs to know. RadioGraphics 2005;25(6):1485–1499.
55. Carucci LR, Turner MA, Szucs RA. Adjustable laparoscopic gastric banding for morbid obesity: imaging assessment and complications. Radiol Clin North Am 2007;45 (2):261–274.
56. Carucci LR, Turner MA. Imaging after bariatric surgery for morbid obesity: Roux-en-Y gastric bypass and laparoscopic adjustable gastric banding. Semin Roentgenol 2009; 44(4):283–296.