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Published online before print
10.1148/radiol.2372050199
Radiology 2005; 237:414–427

Abbreviations:
AIDS = acquired immunodeficiency syndrome
CMV = cytomegalovirus
HIV = human immunodeficiency virus
LPO = left posterior oblique
RAO = right anterior oblique

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Diseases of the Esophagus: Diagnosis with Esophagography[1]

The barium esophagram is a valuable diagnostic test for evaluating structural and functional abnormalities of the esophagus. The study is usually performed as a multiphasic examination that includes upright double-contrast views with a high-density barium suspension, prone single-contrast views with a low-density barium suspension, and, not infrequently, mucosal-relief views with either density of barium suspension. The double-contrast phase optimizes the ability to detect inflammatory or neoplastic diseases, whereas the single-contrast phase optimizes the ability to detect hiatal hernias and lower esophageal rings or strictures. Fluoroscopic examination of the esophagus is also important for assessing motility disorders such as achalasia and diffuse esophageal spasm. This article is a review of gastroesophageal reflux disease, other types of esophagitis, benign and malignant esophageal tumors, varices, lower esophageal rings, diverticula, and esophageal motility disorders, all of which can be diagnosed with the aid of esophagography.

Despite the current focus on cross-sectional imaging studies such as those of computed tomography (CT) and magnetic resonance (MR) imaging, the barium examination continues to be the primary radiologic modality for the evaluation of patients with dysphagia, reflux symptoms, or other clinical findings of esophageal disease. Double-contrast esophagrams are particularly useful for the detection of reflux disease and its complications, infectious esophagitis, esophageal carcinoma, or other structural lesions of the esophagus. The fluoroscopic portion of the examination is also useful for the assessment of esophageal motility and detection of esophageal motility disorders such as achalasia and diffuse esophageal spasm. The purpose of this article is to review gastroesophageal reflux disease, other types of esophagitis, benign and malignant esophageal tumors, varices, lower esophageal rings, diverticula, and esophageal motility disorders, all of which can be diagnosed with the aid of esophagography.

TECHNIQUE

Barium studies of the esophagus are usually performed as multiphasic examinations that include upright double-contrast views with a high-density barium suspension, prone single-contrast views with a low-density barium suspension, and, not infrequently, mucosal-relief views with either density of barium suspension (1). The patient first ingests an effervescent agent and then rapidly gulps high-density barium in the upright left posterior oblique (LPO) position (patient positions are described with respect to the fluoroscopic table top) to obtain double-contrast views of the esophagus, with the esophagus thrown off the spine to avoid confusion with overlapping bone structures. The normal distended esophagus has a thin white luminal contour in profile and a smooth homogeneous appearance en face (Fig 1). Collapsed or partially collapsed views (ie, mucosal-relief views) show the normal longitudinal folds as thin, straight, delicate structures no more than 1–2 mm in width. After having the patient turn a 360° circle to optimally coat the gastric fundus with barium, he or she is placed in a recumbent right-side-down position for double-contrast views of the gastric cardia and fundus. The cardia is often recognized by the presence of three or four stellate folds that radiate to a central point at the gastroesophageal junction, also known as the cardiac rosette (Fig 2) (2).

ESSENTIALS

- *Findings of reflux include fine nodularity or granularity of the mucosa, erosions or ulcers, thickened longitudinal folds, inflammatory esophagogastric polyps, and scarring with strictures, sacculations, or fixed transverse folds.*
- *Esophagography can be used to classify patients with reflux symptoms as being at high, moderate, or low risk for Barrett esophagus, on the basis of specific radiologic criteria.*
- *Candida esophagitis is characterized on esophagrams by plaques or a "shaggy" esophagus, whereas herpes esophagitis is characterized by multiple small ulcers, and human immunodeficiency virus and cytomegalovirus are characterized by one or more giant flat ulcers.*
- *On esophagrams, early esophageal cancers manifest as small polypoid or plaquelike lesions or superficial spreading lesions, whereas advanced esophageal cancers manifest as infiltrating, polypoid, ulcerative, or varicoid lesions.*
- *At fluoroscopy, achalasia is associated with absent peristalsis and beaklike narrowing of the distal esophagus due to incomplete opening of the lower esophageal sphincter, whereas diffuse esophageal spasm is associated with intermittent weakening or absence of primary peristalsis with multiple, repetitive, nonperistaltic contractions, sometimes producing a corkscrew esophageal appearance.*

The patient is then placed in a prone right anterior oblique (RAO) position and is asked to take discrete swallows of low-density barium to evaluate esophageal motility. Esophageal dysmotility is considered to be present when abnormal peristalsis is detected for two or more of five separate swallows (3). The patient next gulps the low-density barium suspension to optimally distend the esophagus to show rings, strictures, or hiatal hernias that could be missed on upright double-contrast views. Finally, the patient is turned from a supine to RPO and right-lateral positions to assess for spontaneous gastroesophageal reflux or for reflux induced by a Valsalva maneuver to increase intraabdominal pressure.

GASTROESOPHAGEAL REFLUX DISEASE

The purpose of barium studies in patients with reflux symptoms is not simply to document the presence of a hiatal hernia or gastroesophageal reflux but to detect the morphologic sequelae of reflux, including reflux esophagitis, peptic strictures, Barrett esophagus, and esophageal adenocarcinoma. These conditions are discussed separately in the following sections.

Reflux Esophagitis

Reflux esophagitis is by far the most common inflammatory disease involving the esophagus. Double-contrast esophagrams have been shown to have a sensitivity approaching 90% for the detection of reflux esophagitis because of the ability to reveal mucosal abnormalities that cannot be visualized on single-contrast studies (4,5). Most cases that are missed occur in patients with milder forms of esophagitis, which manifest at endoscopy as mucosal erythema and friability. The single most common sign of reflux esophagitis on double-contrast esophagrams is a finely nodular or granular appearance with poorly defined radiolucencies that fade peripherally due to edema and inflammation of the mucosa (Fig 3) (6,7). This nodularity or granularity almost always extends proximally from the gastroesophageal junction as a continuous area of disease.

Barium studies may also reveal shallow ulcers and erosions in the distal esophagus. The ulcers can have a punctate, linear, or stellate configuration and are often associated with a surrounding halo of edematous mucosa, radiating folds, or sacculation of the adjacent wall (Fig 4) (8). Some patients have a solitary ulcer at or near the gastroesophageal junction, often on the posterior wall of the distal esophagus, presumably as a result of prolonged exposure to refluxed acid that pools posteriorly when patients sleep in the supine position (9). Other patients have more widespread ulceration of the distal third or half of the thoracic esophagus, but this ulceration always extends proximally from the gastroesophageal junction. Thus, the presence of ulcers that are confined to the upper or middle parts of the esophagus should suggest another cause for the patient's disease.

Reflux esophagitis may also manifest as thickened longitudinal folds as a result of edema and inflammation that extend into the submucosa. However, thickened folds should be recognized as a nonspecific finding of esophagitis from a host of causes. Other patients with chronic reflux esophagitis may have a single enlarged fold that arises at the gastric cardia and extends upward into the distal esophagus as a smooth, polypoid protuberance, also known as an inflammatory esophagogastric polyp (Fig 5) (10,11). This lesion is a chronically inflamed fold rather than a true neoplasm, so it is also known as an inflammatory pseudopolyp (10,11). Because these lesions have no malignant potential, endoscopy is not warranted when barium studies reveal typical findings of an inflammatory esophagogastric polyp in the distal esophagus. If these lesions are unusually large or lobulated, however, endoscopy and biopsy may be required to rule out a tumor in the distal esophagus.

Scarring and Strictures

Localized scarring from reflux esophagitis may manifest on barium studies as flattening, puckering, or sacculation of the adjacent esophageal wall, often associated with folds radiating toward the site of scarring. Further scarring can lead to the development of a circumferential stricture (ie, "peptic" stricture) in the distal esophagus, almost always located above a hiatal hernia (12). Such strictures often appear as concentric segments of smooth tapered narrowing (Fig 6), but asymmetric scarring can lead to asymmetric narrowing with focal sacculation or ballooning of the esophageal wall between areas of fibrosis (Fig 7) (8). Other peptic strictures may manifest as short ringlike areas of narrowing (Fig 8) that could be mistaken for Schatzki rings in patients with dysphagia (8). When there is marked irregularity, flattening, or nodularity of one or more walls of the stricture, endoscopy and biopsy may be required to rule out a malignant stricture as the cause of these findings.

Scarring from reflux esophagitis can also lead to longitudinal shortening of the esophagus and the development of fixed transverse folds, producing a "step-ladder" appearance as a result of pooling of barium between the folds (see Fig 7) (13). These folds should be differentiated from the thin transverse striations (ie, "feline" esophagus) often seen for brief intervals at fluoroscopy due to transient contraction of the longitudinally oriented muscularis mucosae in patients with reflux (Fig 9) (14,15).

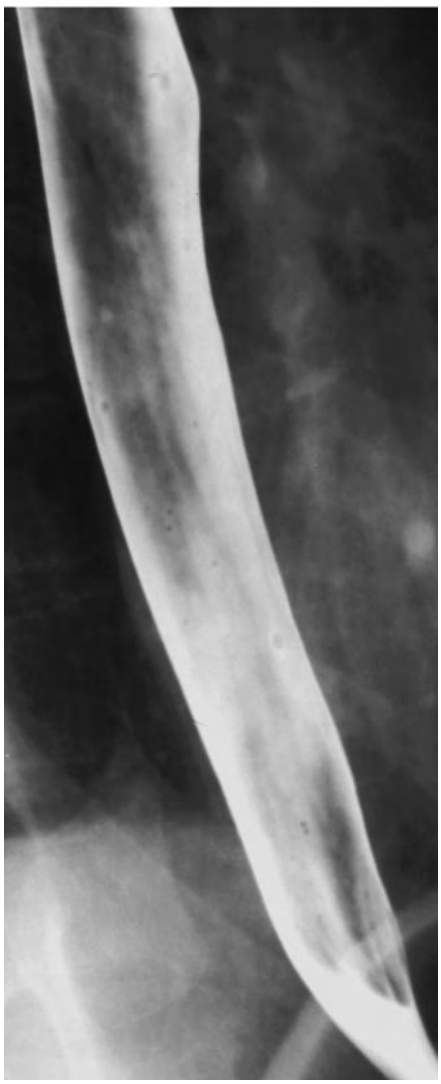


Figure 1. Upright LPO spot image from double-contrast esophagography shows normal esophagus with smooth homogeneous appearance en face.

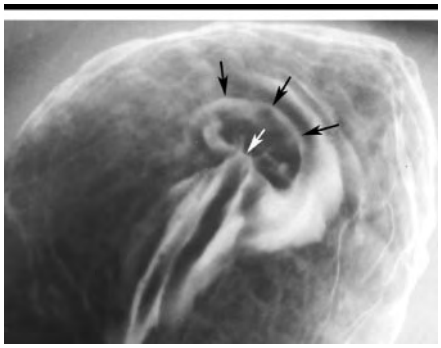


Figure 2. Recumbent right-side-down, lateral spot image of normal gastric cardia shows stellate folds radiating to central point (white arrow) at gastroesophageal junction. Note hooding fold (black arrows) above cardia.

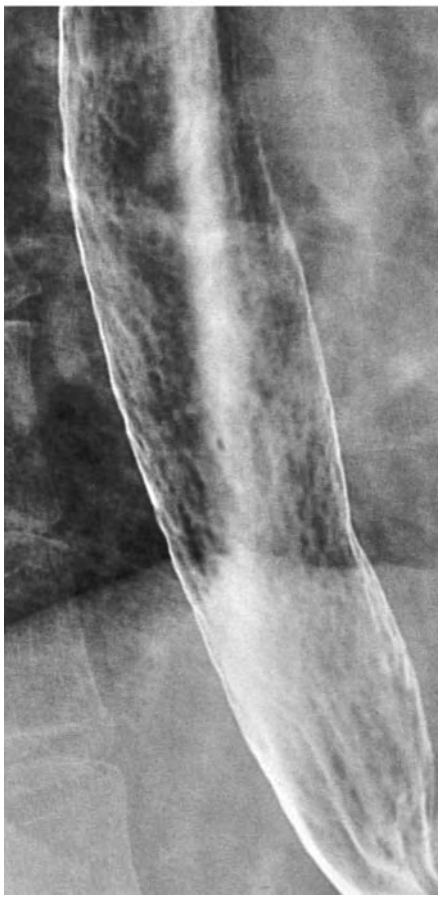


Figure 3. Upright LPO spot image from double-contrast esophagography shows reflux esophagitis with extensive granularity of lower esophagus due to edema and inflammation of mucosa.

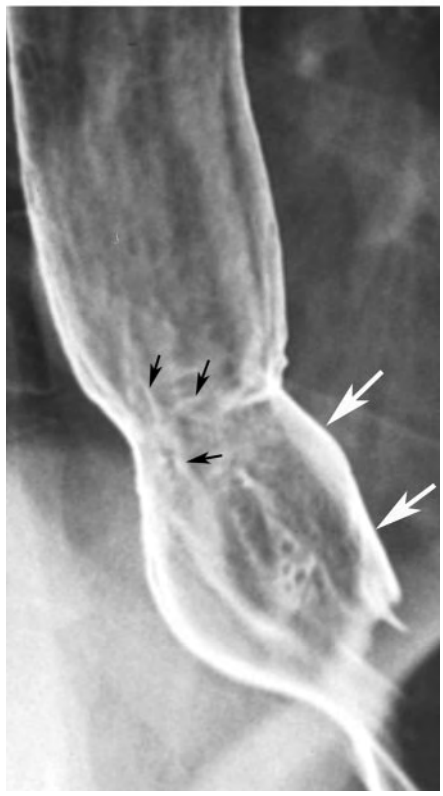


Figure 4. Upright LPO spot image from double-contrast esophagography shows reflux esophagitis with small linear ulcers (black arrows) in distal esophagus just above hiatal hernia (white arrows).

Barrett Esophagus

Barrett esophagus is characterized by progressive columnar metaplasia of the distal esophagus caused by chronic gastroesophageal reflux and reflux esophagitis. Barrett esophagus is thought to develop in about 10% of all patients with reflux esophagitis (16). Double-contrast esophagrams can be used to classify, on the basis of specific radiologic criteria, patients with reflux symptoms as being at high, moderate, or low risk for Barrett esophagus (17). Patients are classified at high risk when a barium study reveals a midesophageal stricture (Fig 10) or ulcer or a reticular pattern of the mucosa (usually associated with a hiatal hernia and/or gastroesophageal reflux) (17). In such cases, endoscopy and biopsy should be performed to help obtain a definitive diagnosis. Although a reticular mucosal pattern has been found in only 5%–10% of all patients with Barrett esophagus (18,19), this finding has been recognized as a highly specific sign of Barrett esophagus, particularly if the pattern is adjacent to the distal aspect of a midesophageal stricture (see Fig 10) (18). The reticular pattern is characterized by tiny barium-filled grooves or crevices resembling the areae gastricae in the stomach.

Patients are classified at moderate risk for Barrett esophagus when a barium study reveals esophagitis or peptic strictures in the distal esophagus (17). These radiographic findings reflect chronic inflammatory disease and scarring; the decision to perform endoscopy in this group should be based on the severity of symptoms, the age, and the overall health of the patient.

Finally, patients are classified at low risk for Barrett esophagus when barium studies reveal no structural abnormalities (regardless of the presence or absence of reflux or a hiatal hernia). The majority of patients are found to be in the low-risk category, and the prevalence of Barrett esophagus is so small in this group that such individuals can be treated empirically for their reflux symptoms without the need for endoscopy (17).

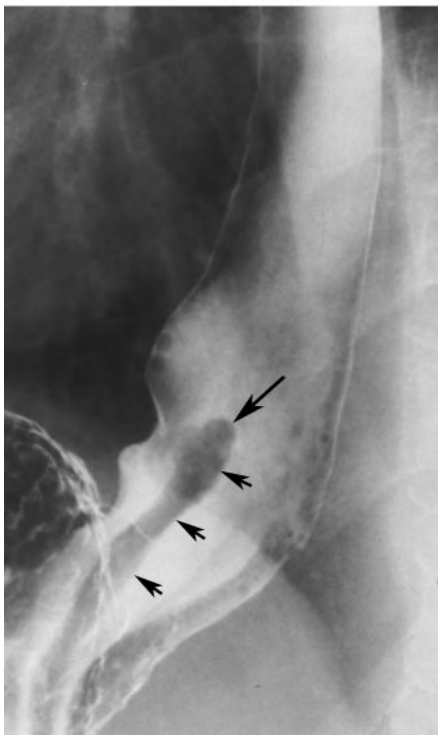


Figure 5. Prone RAO spot image from single-contrast esophagography shows inflammatory esophagogastric polyp as enlarged fold (small arrows) extending upward into distal esophagus, where it terminates as a smooth polypoid protuberance (large arrow). Inflammatory polyps are thought to be a sign of chronic reflux esophagitis.

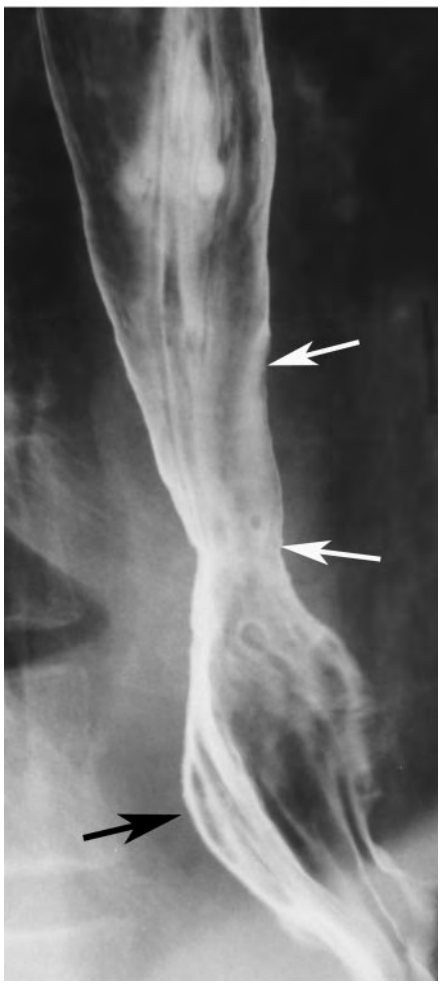


Figure 6. Upright LPO spot image from double-contrast esophagography shows smooth tapered segment of concentric narrowing (white arrows) due to peptic stricture in distal esophagus above a hiatal hernia (black arrow).

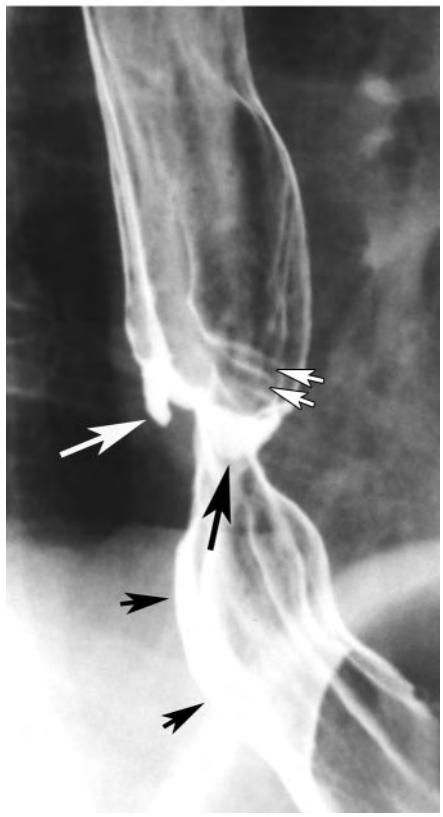


Figure 7. Upright LPO spot image from double-contrast esophagography shows asymmetric peptic stricture in distal esophagus above hiatal hernia (small black arrows), with sacculation of wall both en face (large black arrow) and in profile (large white arrow). Note fixed transverse folds (small white arrows) from associated longitudinal scarring of distal esophagus above the stricture.

INFECTIOUS ESOPHAGITIS

Candida Esophagitis

Candida albicans is the most common cause of infectious esophagitis. It usually occurs as an opportunistic infection in immunocompromised patients, particularly those with acquired immunodeficiency syndrome (AIDS), but *Candida* esophagitis can also result from local esophageal stasis caused by severe esophageal motility disorders such as achalasia and scleroderma (20). Only about 50% of patients with *Candida* esophagitis are found to have thrush, so the absence of oropharyngeal disease in no way excludes this diagnosis.

Candida esophagitis usually manifests on double-contrast studies as discrete plaquelike lesions that are seen as linear or irregular filling defects that tend to be oriented longitudinally and are separated by normal mucosa (Fig 11) (21). Double-contrast esophagrams have been found to have a sensitivity of 90% for the detection of *Candida* esophagitis (21,22), primarily because of the ability to show these plaques.

Glycogenic acanthosis is another common condition affecting elderly people. In glycogenic acanthosis, cytoplasmic glycogen accumulates in the squamous epithelial cell lining of the esophagus, which causes focal plaquelike thickening of the mucosa (23,24). Glycogenic acanthosis may manifest on double-contrast studies as multiple small nodules or plaques (Fig 12) (23,24). The major consideration in the differential diagnosis is *Candida* esophagitis. However, the nodules of glycogenic acanthosis have a more rounded appearance, whereas the plaques of *Candida* esophagitis tend to have a more linear configuration. Also, candidiasis usually occurs in immunocompromised patients with odynophagia, whereas glycogenic acanthosis occurs in older individuals with no esophageal symptoms. It therefore is usually possible to differentiate these conditions on the basis of the clinical and radiographic findings.

During the past 2 decades, a much more fulminant form of candidiasis has been encountered in patients with AIDS, who may present with a grossly irregular or "shaggy" esophagus caused by innumerable coalescent pseudomembranes and plaques, with trapping of barium between the lesions (Fig 13) (25). Other patients with achalasia or scleroderma may develop a "foamy" esophagus, with innumerable tiny bubbles layering out in the barium column; this phenomenon presumably results from the yeast form of fungal infection (26). When typical findings of *Candida* esophagitis are encountered on double-contrast esophagrams, patients with these findings can be treated with antifungal agents such as fluconazole, without the need for endoscopy.

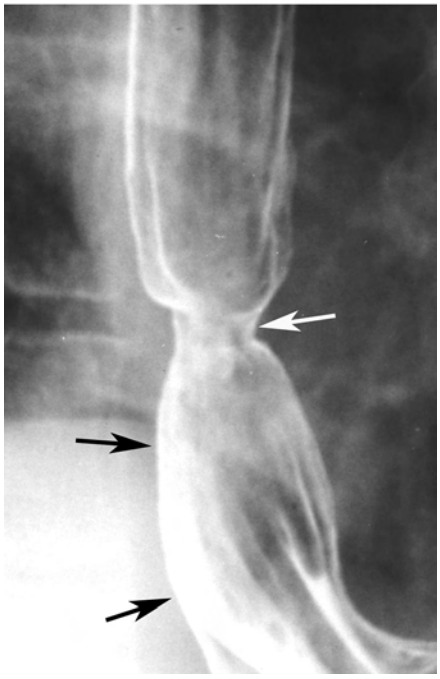


Figure 8. Upright LPO spot image from double-contrast esophagography shows short ringlike peptic stricture (white arrow) in distal esophagus above a hiatal hernia (black arrows). Although this stricture could be mistaken for a Schatzki ring, it has a longer vertical height than does a true Schatzki ring.

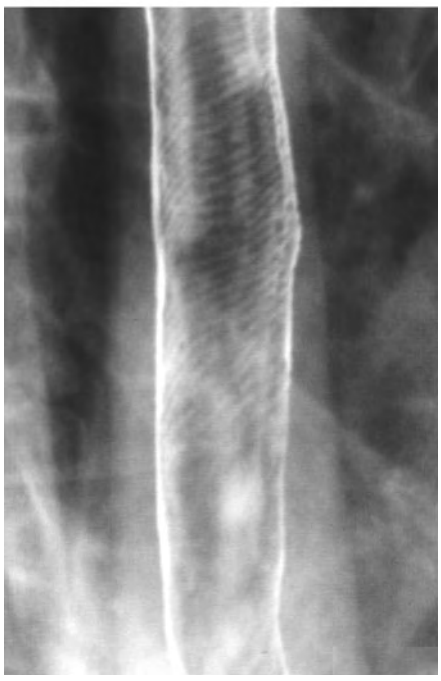


Figure 9. Upright LPO spot image from double-contrast esophagography shows thin transverse striations due to transient contraction of longitudinally oriented muscularis mucosae in patient with "feline" esophagus. This finding is often associated with gastroesophageal reflux.

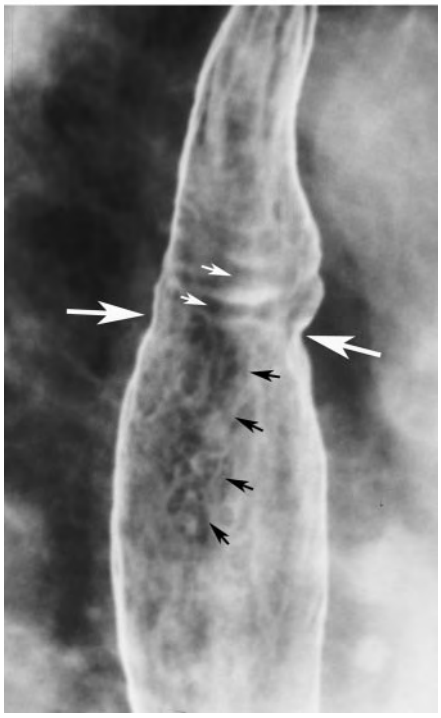


Figure 10. Upright LPO spot image from double-contrast esophagography shows mild ringlike constriction (large white arrows) in midesophagus in a patient with Barrett esophagus. Also note distinctive reticular pattern (black arrows) extending distally from the stricture, a classic radiographic sign of Barrett esophagus. This patient also has fixed transverse folds (small white arrows) just above the stricture due to longitudinal scarring in this region.

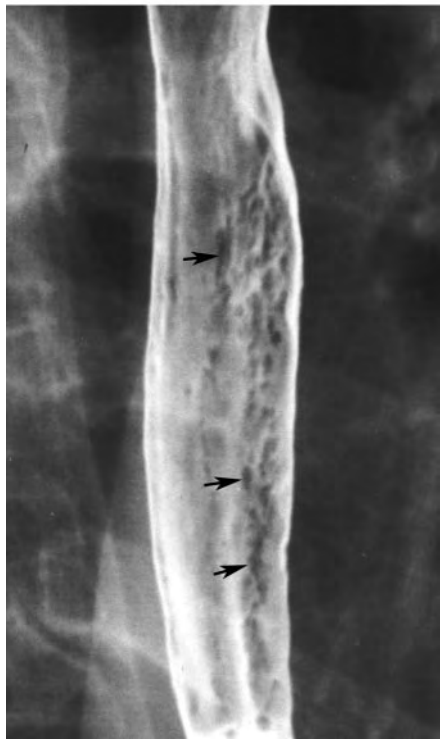


Figure 11. Upright LPO spot image from double-contrast esophagography shows multiple discrete plaquelike lesions (arrows) in the esophagus in a patient with *Candida* esophagitis. Note how plaques have a linear configuration and are separated by normal intervening mucosa.

Herpes Esophagitis

The herpes simplex virus is another frequent cause of infectious esophagitis (27). Most patients with this virus are immunocompromised, but herpes esophagitis may occasionally develop as an acute self-limited disease in immunocompetent individuals (28). Herpes esophagitis initially leads to the development of small mucosal vesicles that rupture to form discrete punched-out ulcers. As a result, herpes esophagitis may manifest on double-contrast studies as small superficial ulcers against a background of normal mucosa (27,29). The ulcers can have a punctate, stellate, or ringlike configuration and are often surrounded by radiolucent mounds of edema (Fig 14). In the appropriate clinical setting, the presence of small discrete ulcers without plaques should be highly suggestive of herpes esophagitis, because ulceration in candidiasis almost always occurs against a background of diffuse plaque formation. As the disease progresses, however, herpes esophagitis may manifest as a combination of ulcers and plaques that mimic *Candida* esophagitis (27).

Herpes esophagitis in otherwise healthy patients manifests as innumerable tiny ulcers clustered together in the midesophagus below the level of the left main bronchus (28). The ulcers are even smaller than those in immunocompromised patients with herpes esophagitis, presumably because these individuals have an intact immune system that prevents the ulcers from enlarging.

Cytomegalovirus Esophagitis

Cytomegalovirus (CMV) is another cause of infectious esophagitis that occurs primarily in patients with AIDS. CMV esophagitis may manifest on double-contrast studies as multiple small ulcers or, even more commonly, as one or more giant flat ulcers that are several centimeters or more in length (30). The ulcers may have an ovoid or diamond-shaped configuration and are often surrounded by a thin radiolucent rim of edematous mucosa. Because herpetic ulcers rarely become this large, the pres-

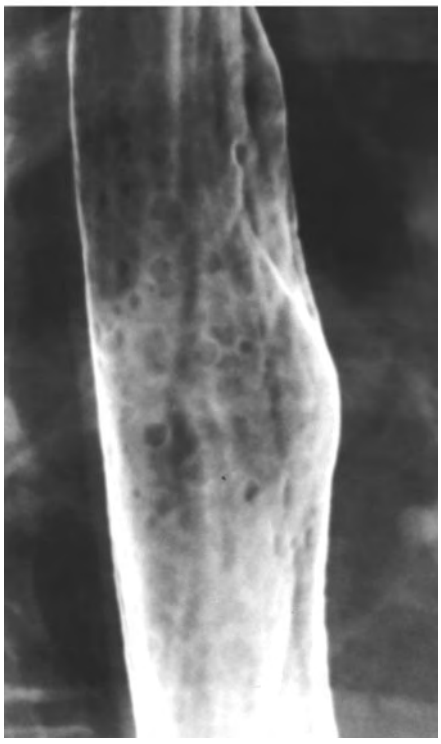


Figure 12. Upright LPO spot image from double-contrast esophagography shows multiple small plaques and nodules in midesophagus in an elderly patient with glycogenic acanthosis. Note how nodules have a more rounded appearance than the fungal plaques in Figure 11.

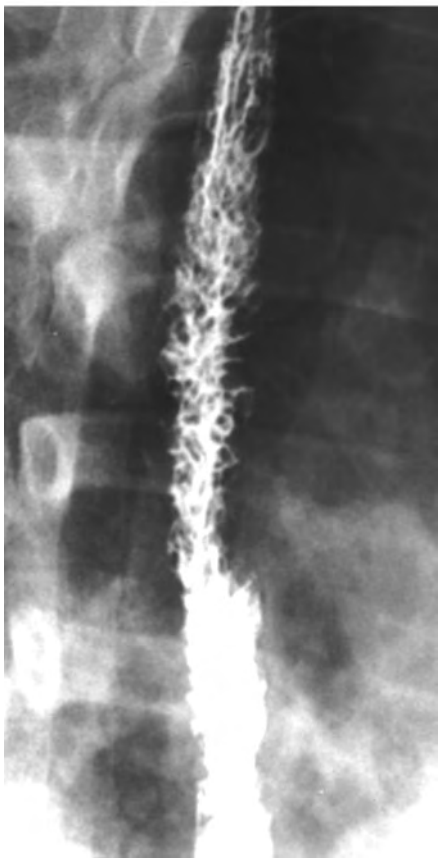


Figure 13. Upright LPO spot image from double-contrast esophagography shows shaggy esophagus of fulminant *Candida* esophagitis in a patient with AIDS. This shaggy contour results from innumerable pseudomembranes and plaques, with trapping of barium between lesions.

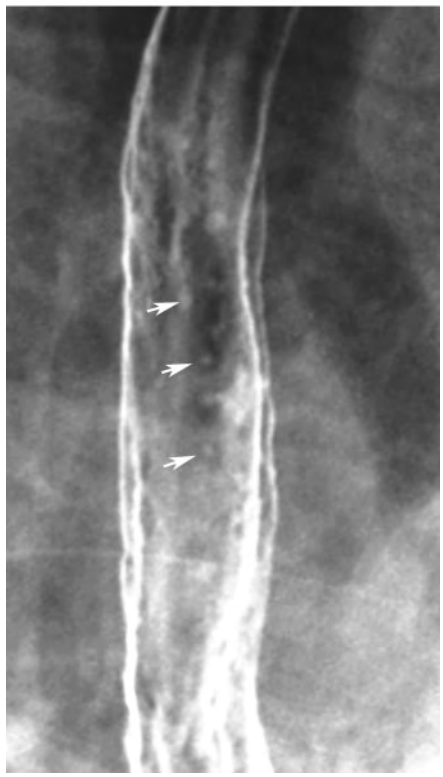


Figure 14. Upright LPO spot image from double-contrast esophagography shows multiple small superficial ulcers (arrows) in midesophagus of a patient with herpes esophagitis.

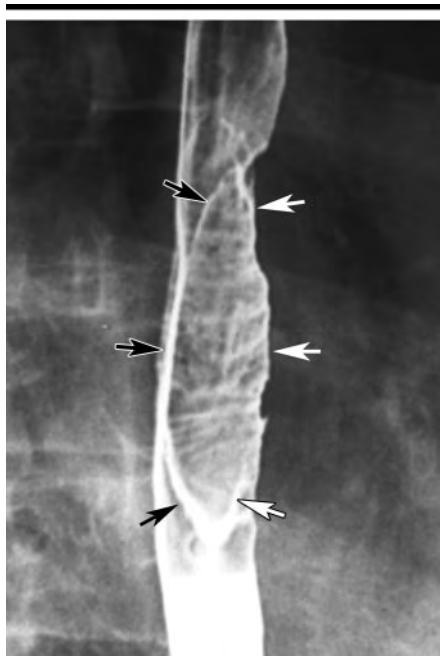


Figure 15. Upright LPO spot image from double-contrast esophagography shows giant diamond-shaped HIV ulcer (arrows) in the midesophagus in HIV-positive patient with odynophagia. Although CMV esophagitis can produce identical findings, endoscopic brushings and biopsy specimens revealed no evidence of CMV.

ence of one or more giant ulcers should suggest the possibility of CMV esophagitis in patients with AIDS. However, the differential diagnosis also includes giant human immunodeficiency virus (HIV) ulcers in the esophagus (see next section). Because CMV esophagitis is treated with relatively toxic antiviral agents such as ganciclovir, endoscopy (with biopsy specimens, brushings, or cultures from the esophagus) is required to confirm the presence of CMV infection before these patients are treated.

HIV Esophagitis

HIV infection of the esophagus can lead to the development of giant esophageal ulcers indistinguishable from those caused by CMV esophagitis. Double-contrast esophagrams typically reveal one or more large ovoid or diamond-shaped ulcers surrounded by a radiolucent rim of edema (Fig 15), sometimes associated with a cluster of small satellite ulcers (31,32). Occasionally, these individuals may have associated palatal ulcers or a characteristic maculopapular rash on the upper body (31,32). The diagnosis is established by obtaining endoscopic biopsy specimens, brushings, or cultures to rule out CMV esophagitis as the cause of the ulcers. Unlike CMV ulcers, HIV-related esophageal ulcers usually heal markedly after treatment with oral steroids (31,32). Thus, endoscopy is required in HIV-positive patients with giant esophageal ulcers, to differentiate esophagitis caused by HIV and CMV so that appropriate therapy can be instituted.

DRUG-INDUCED ESOPHAGITIS

Tetracycline and doxycycline are the two agents most commonly responsible for drug-induced esophagitis in the United States, but other causative agents include potassium chloride, quinidine, aspirin or other nonsteroidal antiinflammatory drugs, and alendronate sodium (33–35).

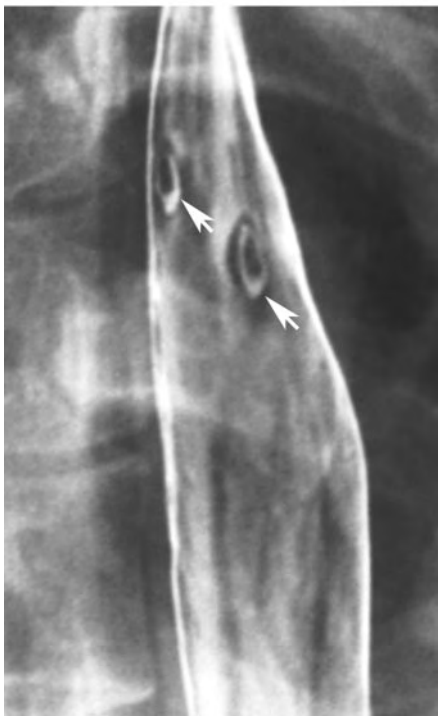


Figure 16. Upright LPO spot image from double-contrast esophagography shows two small superficial ulcers as ring shadows (arrows) in midesophagus. This patient had drug-induced esophagitis after taking doxycycline.

Affected individuals typically ingest the medication with little or no water immediately before going to bed. The capsule or pill then usually becomes lodged in the midesophagus, where it is compressed by the adjacent aortic arch or left main bronchus. Prolonged contact of the esophageal mucosa with these medications presumably causes an irritant contact esophagitis. Affected individuals may present with severe odynophagia, but marked clinical improvement usually occurs after withdrawal of the offending agent.

The radiographic findings in drug-induced esophagitis depend on the nature of the offending medication. Tetracycline and doxycycline are associated with the development of small shallow ulcers in the upper or middle part of the esophagus and are indistinguishable from those in herpes esophagitis (Fig 16) (36,37). Because of their superficial nature, these ulcers almost always heal without scarring or strictures. In contrast, potassium chloride, quinidine, and nonsteroidal antiinflammatory drugs may cause more severe esophageal injury leading to the development of larger ulcers and possible stricture formation (38–40). Alendronate sodium (an inhibitor of osteoclast-mediated bone resorption, used to prevent osteoporosis in postmenopausal women) may cause severe esophagitis with extensive ulceration and strictures that are usually confined to the distal esophagus (41).

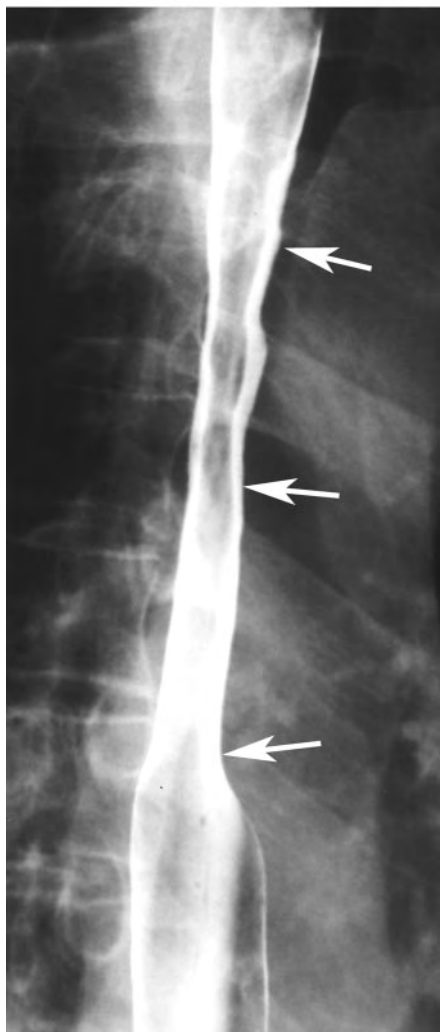


Figure 17. Upright LPO spot image from double-contrast esophagography shows radiation stricture as a long segment of smooth tapered narrowing (arrows) in upper thoracic esophagus. This patient had undergone mediastinal irradiation.

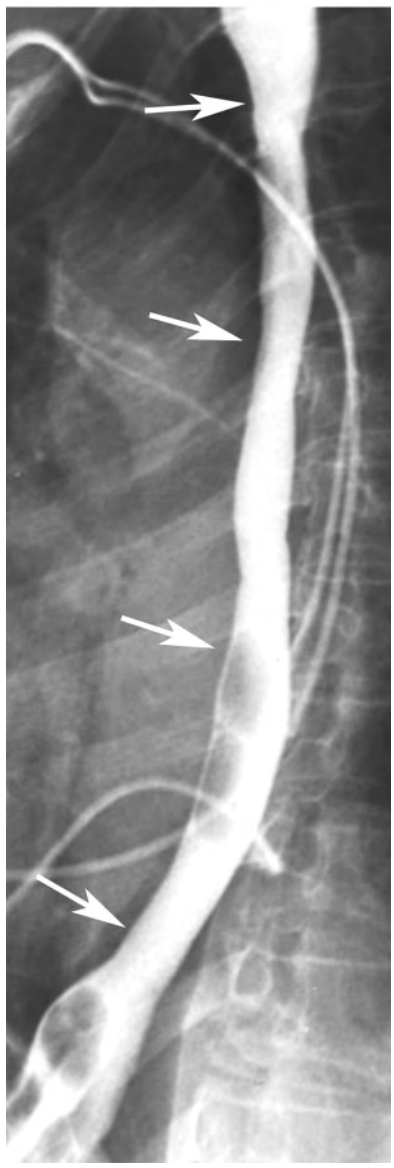


Figure 18. Prone RAO spot image from single-contrast esophagography shows long smooth stricture (arrows) in the thoracic esophagus caused by lye ingestion. Also note cardiac pacemaker wires.

RADIATION-INDUCED ESOPHAGITIS

A radiation dose of 5000 cGy or more to the mediastinum may cause severe injury to the esophagus. Acute radiation-induced esophagitis usually occurs 2–4 weeks after the initiation of radiation therapy (42). The mucosa typically has a granular appearance because of edema and inflammation of the irradiated segment (42). Ulceration and decreased luminal distensibility are other frequent findings (42). The extent of disease conforms to the margins of the radiation portal. Most cases of acute radiation esophagitis are self-limited, but some patients may have progressive dysphagia due to the development of radiation strictures 4–8 months after completion of radiation therapy (43). Such strictures typically appear as smooth tapered areas of concentric narrowing within a preexisting radiation portal (Fig 17).

CAUSTIC ESOPHAGITIS

Whether accidental or intentional, ingestion of lye or other caustic agents causes marked esophagitis, eventually leading to extensive stricture formation. Liquid lye causes liquefactive necrosis, resulting in the most severe form of caustic injury to the esophagus (44). Endoscopy is usually performed as the primary diagnostic test to help assess the extent and severity of esophageal injury after ingestion of a caustic agent. If esophageal perforation is suspected, however, a radiographic study with water-soluble contrast material should be performed to document the presence of a leak. Such studies may also reveal marked edema, spasm, and ulceration of the affected esophagus. As the esophagitis heals, follow-up barium studies have a major role in helping determine the length of developing strictures, which typically involve a longer segment of the esophagus (Fig 18) than does scarring from other types of esophagitis (44). Patients with chronic lye strictures also have an increased risk of developing squamous cell carcinoma of the esophagus (45), so a new area of mucosal irregularity or nodularity within a preexisting lye stricture on a barium study should raise concern about the possibility of a superimposed carcinoma.

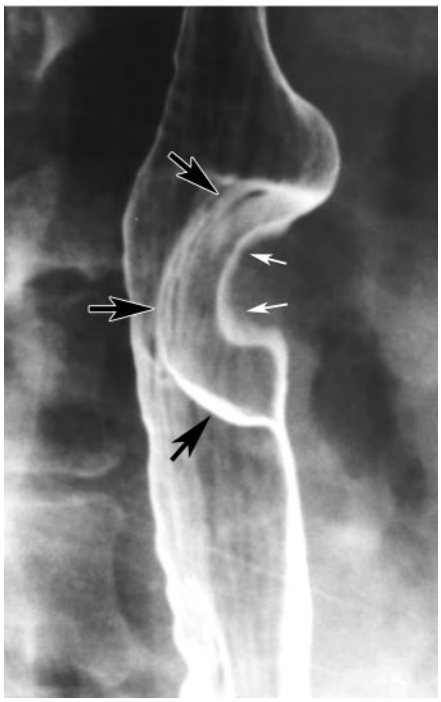


Figure 19. Upright LPO spot image from double-contrast esophagography shows leiomyoma as a smooth submucosal mass (black arrows) in the midesophagus. Note how lesion forms slightly obtuse angles with adjacent esophageal wall. Also note kissing artifact (white arrows) due to apposition of opposing esophageal walls.

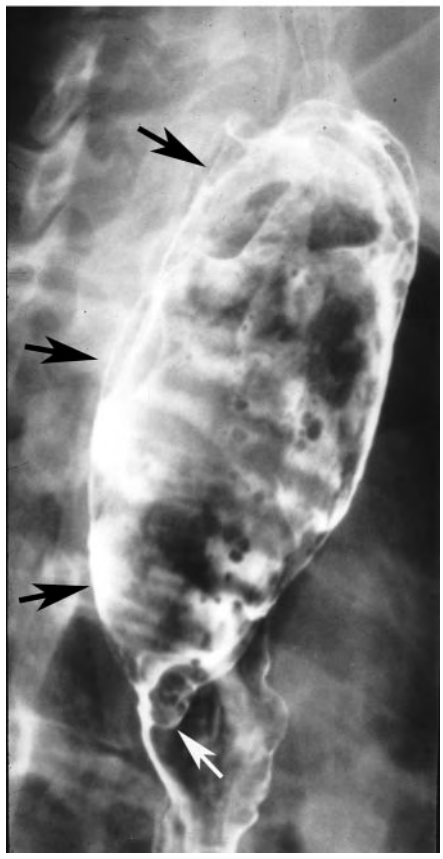


Figure 20. Upright LPO spot image from double-contrast esophagography shows giant fibrovascular polyp as smooth sausage-shaped mass (black arrows) expanding the lumen of the upper thoracic esophagus. Note polypoid distal tip (white arrow) of polyp. Fibrovascular polyps usually arise from the region of the cricopharyngeus muscle. (Reprinted, with permission, from reference 50.)

OTHER ESOPHAGITIDES

Alkaline reflux esophagitis is caused by reflux of bile or pancreatic secretions into the esophagus after partial or total gastrectomy (46). This form of esophagitis is characterized on barium studies by mucosal nodularity or ulceration or, in severe disease, by distal esophageal strictures that often progress rapidly in length and severity over a short period of time (46). The risk of developing alkaline reflux esophagitis can be decreased by performing a Roux-en-Y reconstruction to minimize reflux of alkaline secretions into the esophagus after partial or total gastrectomy.

Nasogastric intubation is an uncommon cause of esophagitis and stricture formation in the distal esophagus. It has been postulated that the strictures result from severe reflux esophagitis caused by constant reflux of acid around the tube into the distal esophagus (44). Affected individuals sometimes develop marked esophageal strictures that show rapid progression in length and severity on serial barium studies (44).

Other uncommon causes of esophagitis include Crohn disease, tuberculosis, eosinophilic esophagitis, chronic graft-versus-host disease, Behçet syndrome, and, rarely, skin disorders involving the esophagus, such as epidermolysis bullosa dystrophica and benign mucous membrane pemphigoid (44).

BENIGN TUMORS

Benign tumors of the esophagus compose only about 20% of all esophageal neoplasms (47). The majority are detected as incidental findings in asymptomatic patients. Squamous papillomas are the most common benign mucosal tumor in the esophagus. These lesions usually appear on double-contrast esophagrams as small sessile polyps with a smooth or slightly lobulated contour (48). Some papillomas may be difficult to distinguish from small esophageal cancers on the basis of the radiographic findings, so endoscopy is required for a definitive diagnosis.

In contrast, leiomyomas are the most common benign submucosal tumor in the esophagus. Affected individuals are usually asymptomatic but may occasionally present with dysphagia, depending on the size of the tumor and how much it encroaches on the lumen. Leiomyomas usually manifest on esophagrams as a smooth submucosal mass etched in white, which forms a right or slightly obtuse angle with the adjacent esophageal wall when viewed in profile (Fig 19) (49). As a result, these lesions may be indistinguishable from other mesenchymal tumors such as fibromas, neurofibromas, and hemangiomas (except that leiomyomas are more likely on empirical grounds).

Fibrovascular polyps are rare benign tumors consisting of varying amounts of fibrovascular and adipose tissue covered by squamous epithelium (50). Fibrovascular polyps usually arise near the level of the cricopharyngeus muscle, gradually elongating over a period of years as they are dragged inferiorly by means of esoph-

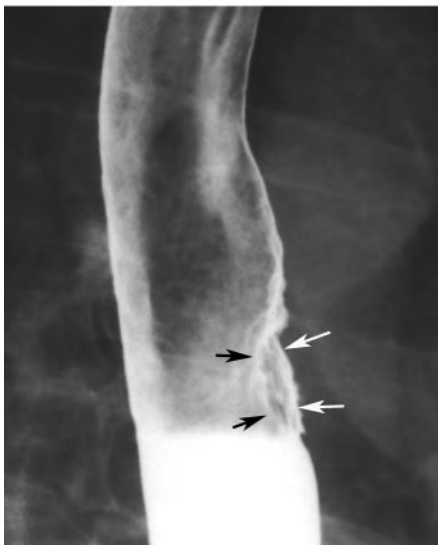


Figure 21. Upright LPO spot image from double-contrast esophagography shows early esophageal cancer in profile as plaquelike lesion (black arrows) with flat central ulcer (white arrows) on left lateral wall of the midesophagus.

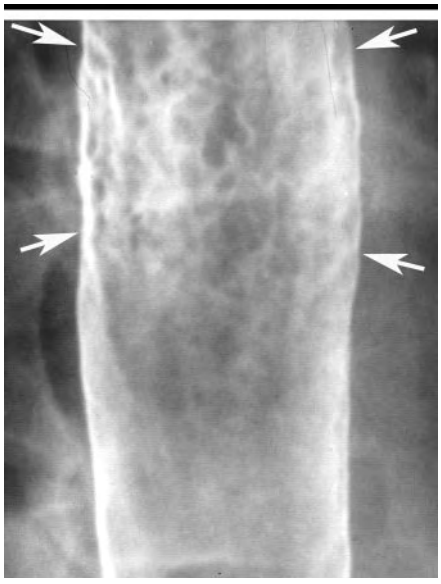


Figure 22. Upright LPO spot image from double-contrast esophagography shows superficial spreading carcinoma as focal area of mucosal nodularity (arrows) in midesophagus, without a discrete mass. Note how nodules merge with one another, producing a confluent area of disease.

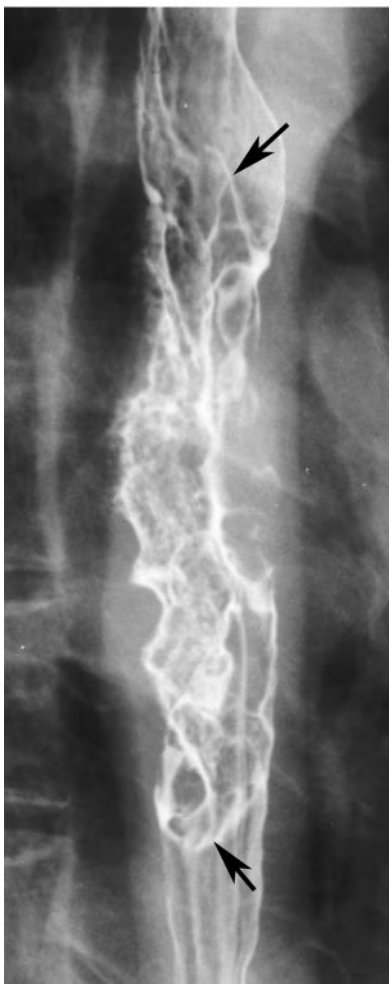


Figure 23. Upright LPO spot image from double-contrast esophagography shows advanced infiltrating carcinoma as irregular area of narrowing, with mucosal nodularity, ulceration, and shelflike margins (arrows) in midesophagus.

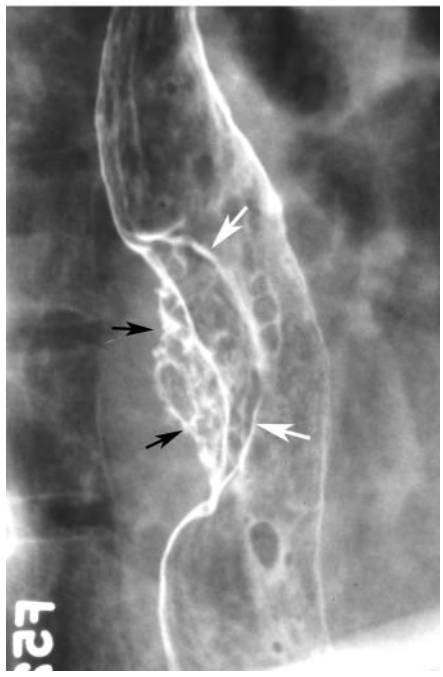


Figure 24. Upright LPO spot image from double-contrast esophagography shows advanced esophageal carcinoma as polypoid mass (white arrows) with a large area of ulceration (black arrows) on the right posterolateral wall of the distal esophagus (small rounded defects abutting mass are air bubbles).

ageal peristalsis. In rare cases, affected individuals may have a spectacular clinical presentation, with regurgitation of a fleshy mass into the pharynx or mouth or even asphyxia and sudden death if the regurgitated polyp occludes the larynx (50). Fibrovascular polyps typically appear on barium studies as smooth, expansile, sausage-shaped masses that expand the lumen of the upper and middle portions of the esophagus (Fig 20) (50). Lesions composed predominantly of adipose tissue may appear as fat-attenuation lesions on CT images, whereas lesions containing greater amounts of fibrovascular tissue may have a heterogeneous appearance with areas of fat juxtaposed with areas of soft-tissue attenuation (50).

MALIGNANT TUMORS

Esophageal Carcinoma

Esophageal carcinoma constitutes about 1% of all cancers in the United States and 7% of all gastrointestinal tumors (51). Early dissemination of tumor occurs because the esophagus lacks a serosa, so there is no anatomic barrier to prevent these cancers from spreading rapidly into the mediastinum. Patients with esophageal carcinoma usually present with dysphagia, but this is a late finding that develops after the tumor has invaded the esophageal wall, periesophageal lymphatics, or other mediastinal structures. As a result, most patients have advanced lesions at the time of diagnosis, with a 5-year survival rate of less than 10% (52). Histologically, 50%–70% of these tumors are squamous cell carcinomas, and the remaining 30%–50% are adenocarcinomas (53).

Squamous cell carcinoma most commonly develops in patients with a long-standing history of alcohol and/or tobacco consumption, whereas adenocarcinoma virtually always arises against a background of Barrett mucosa in the esophagus. The reported prevalence of adenocarcinoma in patients with Barrett esophagus is about 10% (54). Because these tumors develop by means of a sequence of progressively severe epithelial dysplasia, many investigators advocate endoscopic surveillance of patients with known Barrett esophagus to detect dysplastic or early carcinomatous changes before the development of an advanced adenocarcinoma (54).

Double-contrast esophagography has a sensitivity of greater than 95% in the detection of esophageal cancer (55), a number comparable to the endoscopic sensitivity of 95%–100% when brushings and biopsy specimens are obtained (53). Early esophageal cancers are usually small protruded lesions less than 3.5 cm in size. These tumors may manifest on double-contrast studies as plaquelike lesions (often containing flat central ulcers) (Fig 21), sessile polyps with a smooth or slightly lobulated contour, or focal irregularities of the esophageal wall (56). Early adenocarcinomas may also manifest as a localized area of wall flattening or irregularity within a preexisting peptic stricture (53). Superficial spreading carcinoma is another form of early esophageal cancer characterized by poorly defined nodules or plaques that merge with one another, producing a confluent area of disease (Fig 22) (56,57).

Advanced esophageal carcinomas usually appear on barium studies as infiltrating, polypoid, ulcerative, or, less commonly, varicoid lesions (53). Infiltrating carcinomas manifest as irregular luminal narrowing with mucosal nodularity or ulceration and abrupt shelflike borders (Fig 23). Polypoid carcinomas appear as lobulated intraluminal masses or as polypoid ulcerated masses (Fig 24). Primary ulcerative carcinomas are seen as giant meniscoid ulcers surrounded by a radiolucent rind of tumor (58). Finally, varicoid carcinomas are those in which submucosal spread of tumor produces thickened, tortuous, longitudinal defects (Fig 25) that mimic the appearance of varices (59). Varicoid tumors have a fixed configuration, however, whereas varices change in size and shape at fluoroscopy. Also, varices rarely cause dysphagia because they are soft and compressible. Thus, it is usually possible to differentiate these conditions on the basis of the clinical and radiographic findings.

Squamous cell carcinoma and adenocarcinoma of the esophagus cannot be reliably differentiated on barium studies. Nevertheless, squamous cell carcinoma tends to involve the upper or middle part of the esophagus, whereas adenocarcinoma is located mainly in the distal esophagus. Unlike squamous cell carcinoma, adenocarcinoma also has a marked tendency to invade the gastric cardia or fundus and composes as many as 50% of all malignant tumors that involve the gastroesophageal junction (60,61).

Esophageal carcinomas can metastasize to other parts of the esophagus via a rich network of intramural lymphatic channels. These satellite metastases may appear as polypoid, plaquelike, or ulcerated lesions separated from the primary tumor by normal mucosa (53). Tumor also spreads subdiaphragmatically to the proximal stomach via submucosal lymphatics. Metastases to the gastric cardia and fundus may appear as large submucosal masses that often contain central ulcers (62).

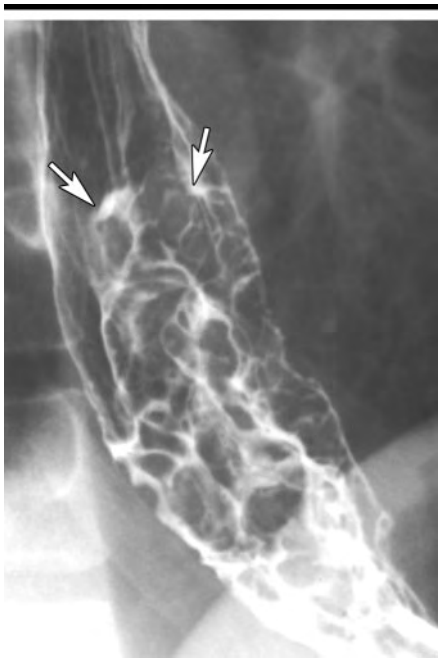


Figure 25. Upright LPO spot image from double-contrast esophagography shows varicoid carcinoma as lobulated submucosal lesion in the distal esophagus that could be mistaken for varices. However, this lesion had a fixed appearance at fluoroscopy and an abrupt proximal demarcation (arrows) from normal esophagus.

Appropriate treatment strategies for esophageal carcinoma depend on accurate staging of the tumor with the aid of CT, MR imaging, or endoscopic sonography. Tumor staging is beyond the scope of this review.

Other Malignant Tumors

Non-Hodgkin lymphoma and, rarely, Hodgkin lymphoma may involve the esophagus. Esophageal lymphoma may manifest on barium studies as submucosal masses, polypoid lesions, enlarged folds, or strictures (63). Spindle cell carcinoma (formerly known as carcinosarcoma) is another rare tumor; it is characterized by a bulky polypoid intraluminal mass that expands the lumen of the esophagus without causing obstruction (64). Other rare malignant tumors that involve the esophagus include leiomyosarcoma, malignant melanoma, and Kaposi sarcoma.

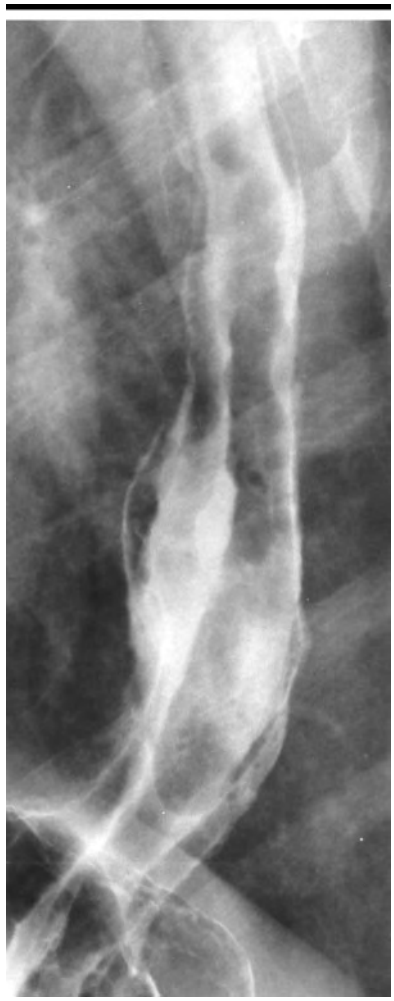


Figure 26. Prone RAO spot image from single-contrast esophagography shows uphill esophageal varices as serpiginous defects in lower esophagus. This patient had portal hypertension.

VARICES

Uphill Varices

Uphill varices are usually caused by portal hypertension with hepatofugal flow through dilated esophageal collateral vessels to the superior vena cava. Uphill varices may cause marked upper gastrointestinal tract bleeding. Varices appear on barium studies as serpentine longitudinal filling defects in the distal half of the thoracic portion of the esophagus (Fig 26) (65). They are best seen on mucosal-relief views of the collapsed esophagus while the patient is in a prone RAO position, with use of a high-density barium suspension or barium paste to in-

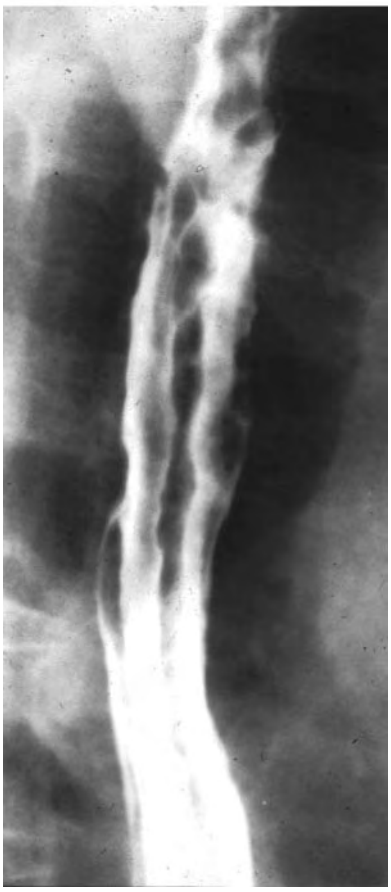
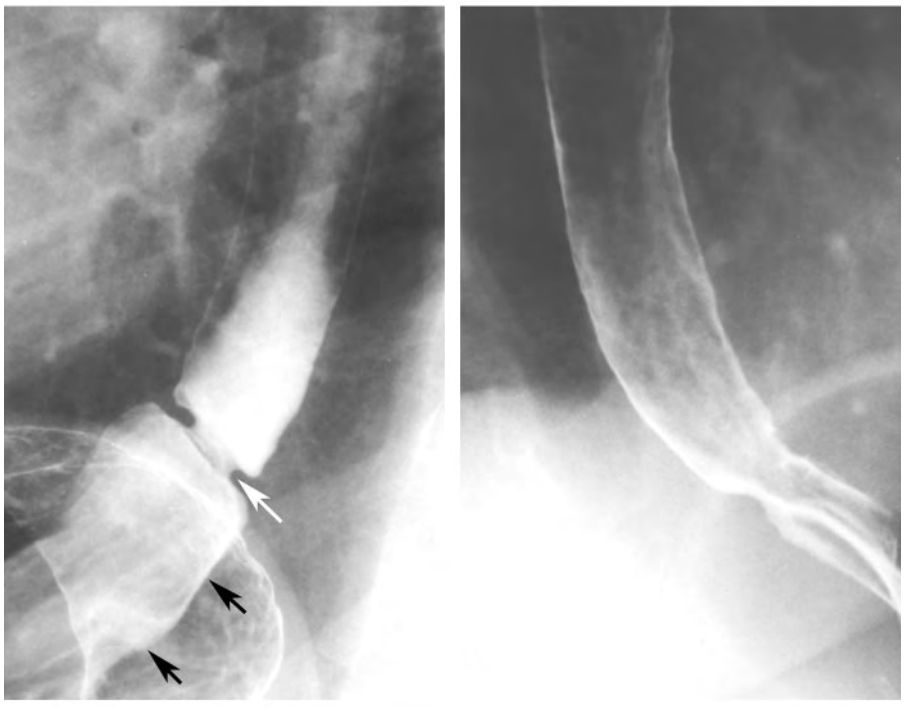


Figure 27. Upright LPO spot image from double-contrast esophagography shows downhill esophageal varices as serpiginous defects in midesophagus above the level of the carina. This patient had superior vena cava syndrome.



a. **b.**

Figure 28. Schatzki ring. **(a)** Prone RAO spot image from single-contrast phase of esophagography shows Schatzki ring as smooth, symmetric, ringlike constriction (white arrow) in distal esophagus directly above a hiatal hernia (black arrows). **(b)** Upright LPO spot image from double-contrast phase of same examination shows mild narrowing of distal esophagus without demonstration of the ring because of inadequate distention of this region.

crease mucosal adherence (65). The differential diagnosis for varices includes varicoid carcinoma and esophagitis with thickened folds.

Downhill Varices

Downhill varices are caused by superior vena cava obstruction with downward flow via dilated esophageal collateral vessels to the portal venous system and the inferior vena cava. Causes of downhill varices include central catheter–related thrombosis of the superior vena cava, bronchogenic carcinoma or other metastatic tumors involving the mediastinum, lymphoma, substernal goiter, mediastinal radiation, and sclerosing mediastinitis (65). Most patients with downhill varices have superior vena cava syndrome at the time of clinical presentation. Such varices typically appear as serpentine longitudinal filling defects that, unlike uphill esophageal varices, are confined to the upper or middle part of the esophagus (Fig 27). Venography, CT, or MR imaging may be performed to confirm the presence of superior vena cava obstruction and to help determine the underlying cause.

LOWER ESOPHAGEAL RINGS

Lower esophageal rings are a frequent finding on esophagrams, but only a small percentage cause symptoms. The term *Schatzki ring* is reserved for lower esophageal rings in patients who present with dysphagia. Such rings are almost always located at the gastroesophageal junction. Histologically, the superior surface of the ring is lined by squamous epithelium, and the inferior surface is lined by columnar epithelium. The exact pathogenesis of Schatzki rings is uncertain, but some rings are thought to develop as a result of scarring from reflux esophagitis.

Lower esophageal rings appear on barium studies as smooth-surfaced ringlike constrictions 2–3-mm in height at the gastroesophageal junction, almost always located above a hiatal hernia (Fig 28a) (66). The rings can be missed if the distal esophagus is not adequately distended at fluoroscopy (Fig 28b), so it is important to obtain prone views of the esophagus during continuous drinking of a low-density barium suspension (67). Conversely, rings can also be missed if the hiatal hernia is overdistended, resulting in overlap of the distal esophagus and hernia that obscure the ring (68). Rings with a maximum luminal diameter of more than 20 mm rarely cause dysphagia, whereas rings with a maximum diameter of less than 13 mm almost always cause dysphagia (66). Studies have shown that esophagography is a sensitive technique for the detection of symptomatic lower esophageal rings, sometimes revealing rings that are missed at endoscopy (67).

DIVERTICULA

Pulsion Diverticula

Pulsion diverticula tend to be located in the distal esophagus and are often associated with fluoroscopic or manometric evidence of esophageal dysmotility (69). These diverticula are usually detected as incidental findings in patients with no esophageal symptoms. However,

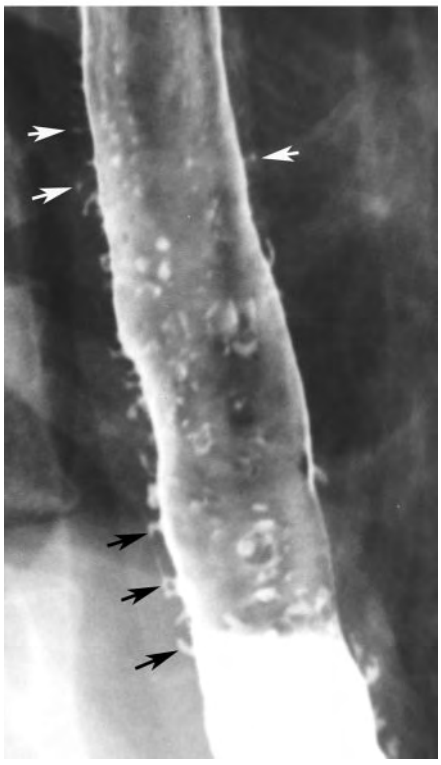


Figure 29. Upright LPO spot image from double-contrast esophagography shows multiple esophageal intramural pseudodiverticula as flask-shaped outpouchings (black arrows) in longitudinal rows parallel to the long axis of the esophagus. Note how some pseudodiverticula (white arrows) appear to be floating outside the esophagus without apparent communication with the lumen.

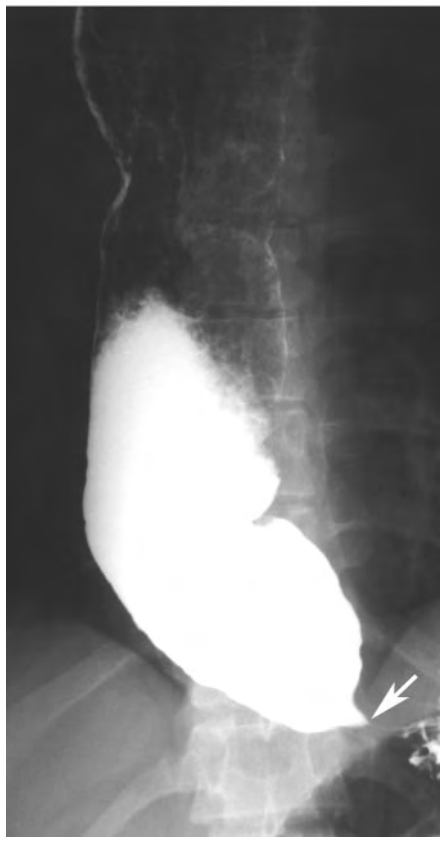


Figure 30. Upright frontal spot image from double-contrast esophagography shows typical findings of primary achalasia, with dilated aperistaltic esophagus and tapered beaklike narrowing (arrow) of distal esophagus due to incomplete opening of the lower esophageal sphincter. This image was obtained in a middle-aged patient with long-standing dysphagia.

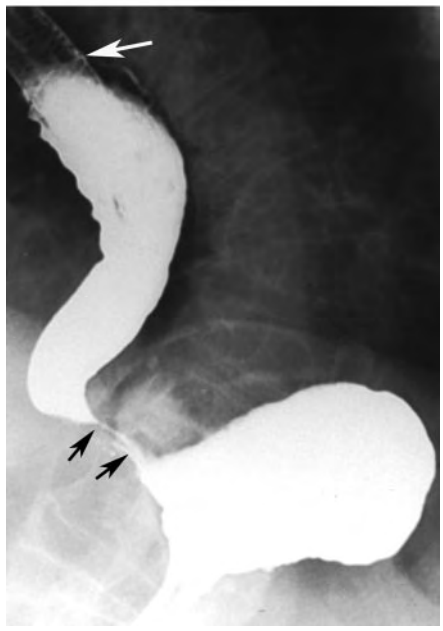


Figure 31. Prone RAO spot image from single-contrast esophagography shows beaklike narrowing (black arrows) of distal esophagus caused by secondary achalasia. Note greater length of narrowed segment than is usually seen in primary achalasia (see Fig 30). Also note metallic esophageal stent (white arrow) for palliation of esophageal carcinoma, which presumably had spread to gastroesophageal junction via periesophageal lymphatics. This image was obtained in an elderly patient with recent onset of dysphagia and weight loss.

a large epiphrenic diverticulum adjacent to the gastroesophageal junction may fill with debris, causing dysphagia, regurgitation, or aspiration (70). Pulsion diverticula appear on barium studies as rounded wide-necked outpouchings that fail to empty when the esophagus collapses (69).

Traction Diverticula

Traction diverticula occur in the midesophagus and are usually caused by scarring from tuberculosis or histoplasmosis in perihilar lymph nodes (69). Because traction diverticula contain all layers of the esophageal wall, they maintain their elastic recoil and empty their contents when the esophagus collapses at fluoroscopy. Traction diverticula often have a triangular or tented appearance resulting from traction on the diverticulum by the fibrotic process in the adjacent mediastinum (69).

Esophageal Intramural Pseudodiverticula

Esophageal intramural pseudodiverticula consist of dilated excretory ducts of deep mucous glands in the esophagus. The pseudodiverticula typically appear on esophagrams as flask-shaped outpouchings in rows parallel to the longitudinal axis of the esophagus (Fig 29) (71). When viewed en face on double-contrast esophagrams, the pseudodiverticula can be mistaken for tiny ulcers. When viewed in profile, however, they often appear to be floating outside the wall of the esophagus, without apparent communication with the lumen (see Fig 29) (72). Many patients have an isolated cluster of pseudodiverticula in the distal esophagus in the region of a peptic stricture (72). In such cases, the pseudodiverticula probably occur as a sequela of scarring from reflux esophagitis. Less frequently, the pseudodiverticula have a diffuse distribution and are associated with high esophageal strictures (71), or they occur as an isolated finding. When strictures are present, these patients may present with dysphagia, but the pseudodiverticula themselves rarely cause symptoms.

ESOPHAGEAL MOTILITY DISORDERS

Achalasia

Primary achalasia is an idiopathic condition involving the myenteric plexus of the esophagus, whereas secondary achalasia is caused by other underlying conditions, most commonly a malignant tumor involving the gastroesophageal junction (especially carcinoma of the gastric cardia) (73). Primary achalasia is characterized by absence of primary peristalsis in the esophagus and incomplete relaxation of the lower esophageal sphincter, which manifests on barium studies as a tapered beaklike narrowing of the distal esophagus adjacent to the gastroesophageal junction (Fig 30) (73). In advanced disease, the esophagus can be-

come massively dilated and tortuous distally (ie, "sigmoid" esophagus). Because of the slow progression of symptoms, affected individuals typically have long-standing dysphagia when they seek medical attention.

Secondary achalasia is also characterized by absent peristalsis in the esophagus and beaklike narrowing near the gastroesophageal junction (74). In secondary achalasia caused by tumor at the gastroesophageal junction, however, the length of the narrowed segment is often greater than that in primary achalasia because of spread of tumor into the distal esophagus (Fig 31) (74). The narrowed segment may also be asymmetric, nodular, or ulcerated because of tumor infiltrating this region. In some cases, barium studies may reveal other signs of malignancy at the cardia, with distortion or obliteration of the normal cardiac rosette (74). The clinical history also is important, because patients with primary achalasia almost always have long-standing dysphagia, whereas patients with secondary achalasia are usually older (over age 60 years) with recent onset of dysphagia (less than 6 months) and weight loss (75).

Other Esophageal Motility Disorders

Symptomatic diffuse esophageal spasm may manifest on barium studies as intermittently weakened or absent primary peristalsis with repetitive, lumen-obliterating, nonperistaltic contractions that produce a classic "corkscrew" esophagus (76). More commonly, however, these patients have multiple nonperistaltic contractions of mild to moderate severity without a corkscrew esophagus (77). Older patients may have intermittent weakening of peristalsis in the distal esophagus and multiple nonperistaltic contractions in the absence of esophageal symptoms, a relatively common manifestation of aging known as *presbyesophagus* (78).

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