A clinical/pattern approach for barium esophagography

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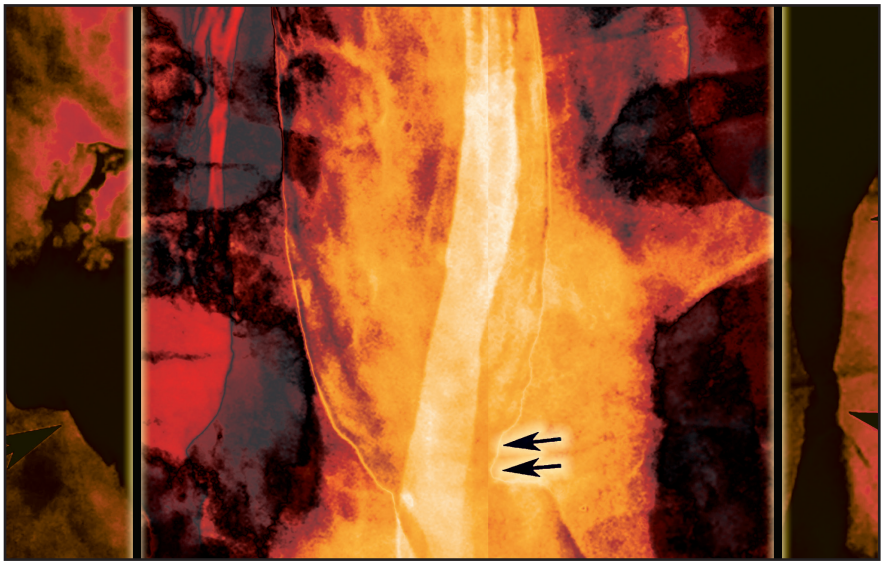
Despite technologic advances in gastrointestinal (GI) radiology, barium esophagography remains an indispensable technique for detecting a variety of morphologic abnormalities in the esophagus, including nodules or plaques, ulcers, strictures, and rings. These abnormalities may be associated with radiographic findings that strongly suggest the underlying cause of disease. Not infrequently, however, the correct diagnosis is established only by combining the radiographic findings with the clinical history and presentation. This article therefore presents a pattern approach for esophagography based on the radiographic and clinical findings.



Technique

Double-contrast esophagography is performed as a biphasic examination that includes both double- and single-contrast views of the esophagus.[1] After ingesting an effervescent agent, the patient continuously swallows high-density barium in the upright, left posterior oblique position for double-contrast views of the esophagus. A double-contrast view of the gastric cardia is also obtained in a recumbent, right side down position. The patient is then placed in a prone, right anterior oblique position and asked to take discrete swallows of low-density barium to evaluate esophageal motility. Finally, the patient continuously swallows low-density barium in the prone position to optimally distend the esophagus. The double-contrast phase of the study optimizes detection of mucosal disease, while the single-contrast phase optimizes detection of narrowing due to strictures or rings in the esophagus.

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Nodules or plaques

Reflux esophagitis

Reflux esophagitis, the most common inflammatory condition involving the esophagus, is most often manifested on double-contrast studies by a finely nodular or granular appearance caused by edema and inflammation of the mucosa. The granularity is characterized by poorly defined radiolucencies in the distal esophagus extending proximally from the gastroesophageal junction as a continuous area of disease (Figure 1).[1-3] This finding is relatively sensitive and specific for reflux esophagitis, especially in patients with reflux symptoms

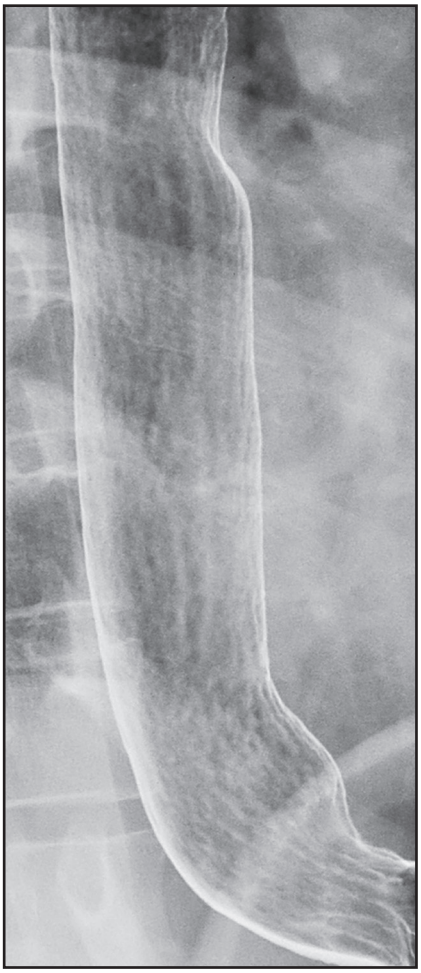

FIGURE 1. *Reflux esophagitis with a finely nodular or granular appearance of the mucosa. Note how this granularity extends proximally from the gastroesophageal junction as a continuous area of disease.*

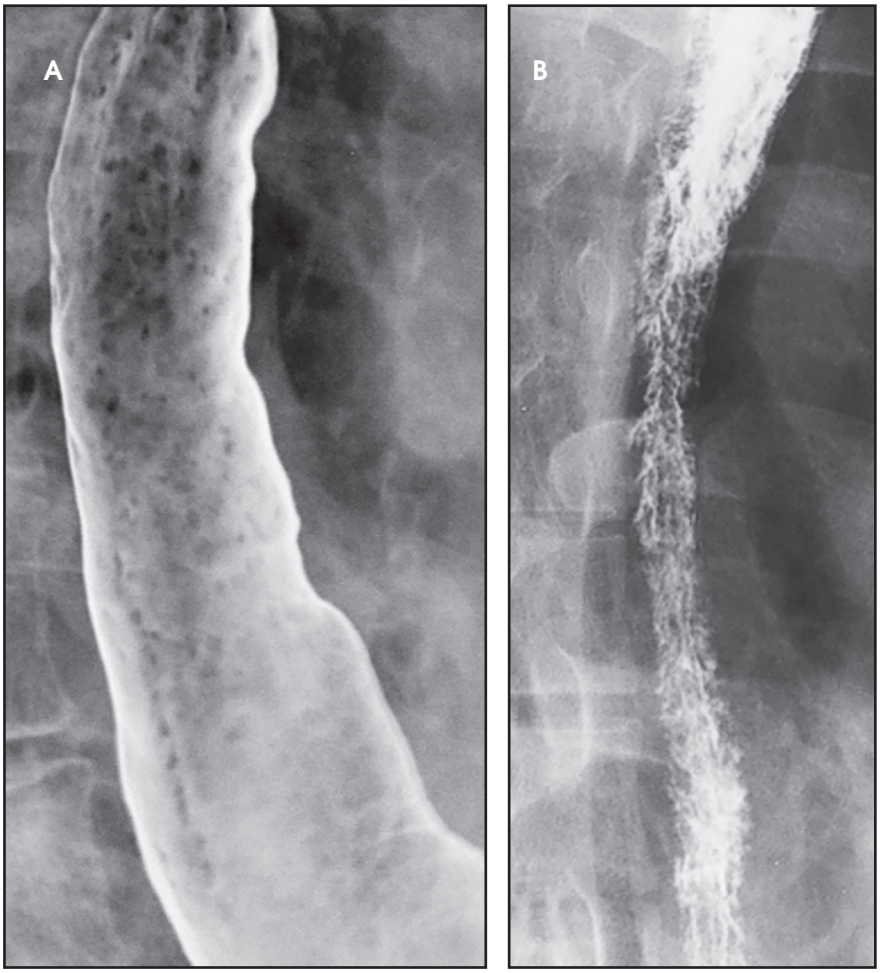

FIGURE 2. Candida esophagitis. *(A) This patient has multiple small, discrete plaquelike lesions separated by normal intervening mucosa in the mid and lower esophagus. (B) Another patient with AIDS has a grossly irregular or shaggy esophagus due to multiple plaques and pseudomembranes with trapping of barium between these lesions.*

such as heartburn, acid regurgitation, coughing and, less frequently, dysphagia or a globus sensation.[3]

Candida esophagitis

Candida albicans, the most common cause of infectious esophagitis, usually occurs as an opportunistic infection in patients who are immunocompromised from diabetes, malignancy, chemotherapy, AIDS or other causes.[4,5] Less frequently, *Candida* esophagitis results from stasis caused by esophageal motility disorders such as scleroderma and achalasia that allow the fungal organism to overgrow and colonize the esophagus.[6] Affected individuals typically present with acute dysphagia (difficulty swallowing) or, even more commonly, odynophagia (pain on swallowing). In some cases, the pain may be so severe that affected individuals are unable to swallow their saliva. However, only about 50% of patients have associated thrush, so the absence of oropharyngeal disease in no way excludes this diagnosis.[5]

Candida esophagitis is usually manifested on double-contrast studies by multiple discrete, plaquelike defects separated by normal intervening mucosa (Figure 2A).[4,5] The plaques tend to involve the upper and/or midesophagus and have a linear or irregular configuration.[4,5] Patients with AIDS may develop a more fulminant form of candidiasis, with trapping of barium between innumerable plaques and pseudomembranes, producing a so-called *shaggy* esophagus (Figure 2B).[5] This finding, which is virtually diagnostic of advanced *Candida* esophagitis, is not often seen in modern medical practice because of more effective therapy for HIV-positive patients.

Glycogenic acanthosis

Glycogenic acanthosis is a common degenerative condition characterized by accumulation of cytoplasmic glycogen in the squamous epithelium of the esophagus. This condition is manifested on double-contrast studies by small, rounded nodules and plaques, most often in the midesophagus (Figure 3).[7] Glycogenic acanthosis may closely resemble *Candida* esophagitis, but the

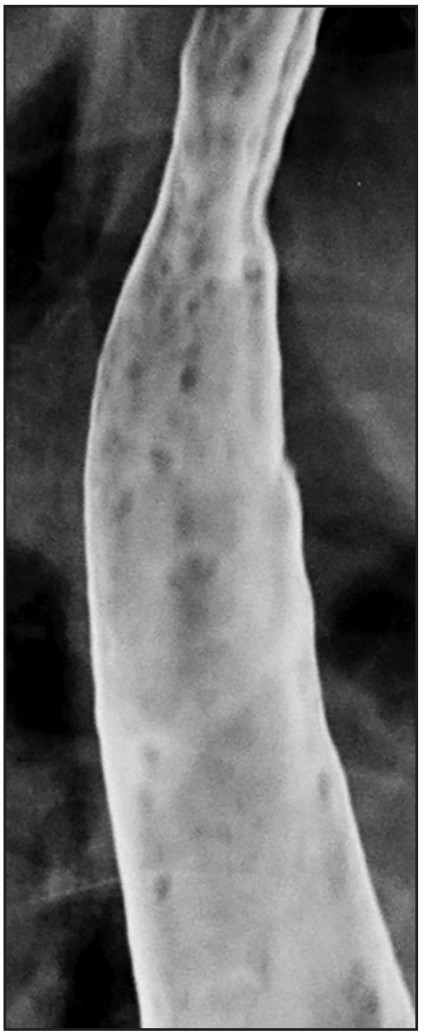

FIGURE 3. *Glycogenic acanthosis with scattered small, rounded nodules and plaques in the midesophagus. While* Candida esophagitis *could produce similar findings, the clinical history is extremely helpful for differentiating these conditions.*

FIGURE 4. *Reflux esophagitis with tiny areas of ulceration and several shallow, linear ulcers (arrows) in the distal esophagus.*

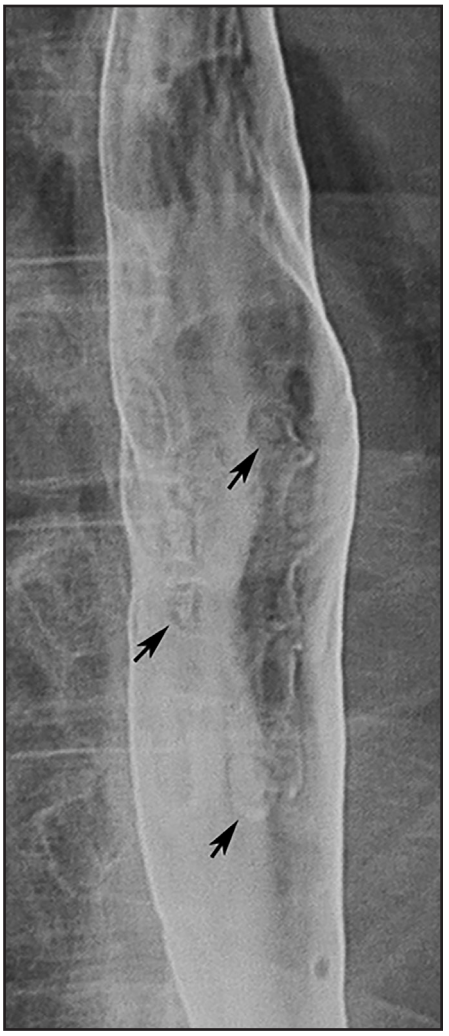

FIGURE 5. *Herpes esophagitis with multiple small, discrete ulcers surrounded by radiolucent halos of edematous mucosa (arrows) in the midesophagus. In an immunocompromised patient with odynophagia, these findings should be highly suggestive of herpes esophagitis.*

plaques of candidiasis tend to have a linear or irregular appearance, whereas the nodules of glycogenic acanthosis are more rounded. Furthermore, *Candida* esophagitis occurs in immunocompromised patients with odynophagia, whereas glycogenic acanthosis develops in elderly patients who are not immunocompromised and have no esophageal symptoms. Thus, it is almost always possible to differentiate these conditions on the basis of the clinical findings.

Superficial spreading carcinoma

Superficial spreading carcinoma (SSC) is an unusual form of esophageal cancer in which tumor is confined to the mucosa or submucosa, regardless of the presence or absence of lymph node metastases.[8] SSC is typically manifested on double-contrast studies by a cluster of poorly defined nodules or plaques that merge with one another, producing a confluent area of disease.[1,8,9] SSC can usually be differentiated from *Candida* esophagitis and glycogenic acanthosis, in which the plaques and nodules have discrete borders and are separated by normal intervening mucosa. When SSC is suspected on the basis of the radiographic findings, endoscopy and biopsy should be performed for a definitive diagnosis, so these patients can be treated before they develop more advanced disease.

Small ulcers

Reflux esophagitis

Reflux esophagitis is the most common inflammatory condition involving the esophagus. While many patients with reflux esophagitis have a finely nodular or granular mucosa (see earlier section), more advanced disease may be manifested by multiple small, shallow ulcers and erosions in the distal esophagus. The ulcers often have a punctate or

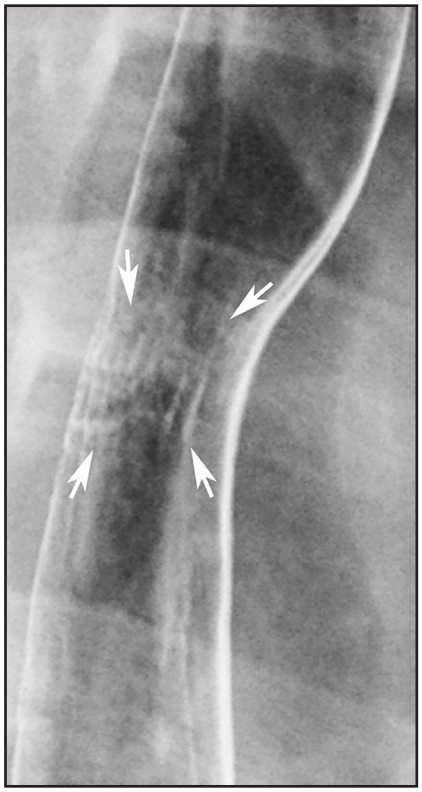

FIGURE 6. *Drug-induced esophagitis with a cluster of shallow, linear ulcers (between arrows) in the midesophagus at the level of the aortic arch. This patient developed odynophagia after taking doxycycline.*

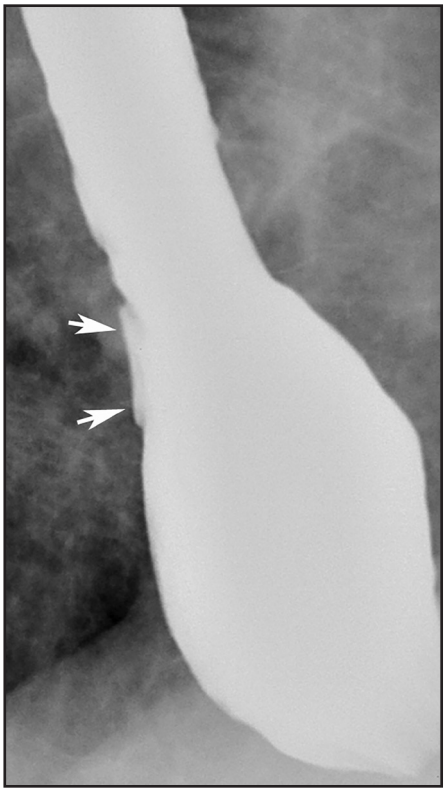

FIGURE 7. *HIV esophagitis with a giant (>1 cm in diameter), flat ulcer (arrows) on the right lateral wall of the distal esophagus. Note the thin, faint radiolucent rim of edema abutting the ulcer crater. The patient's symptoms resolved after treatment with steroids.*

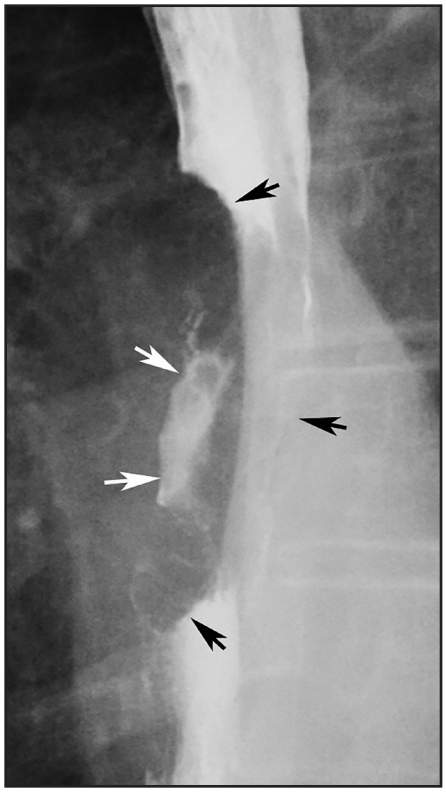

FIGURE 8. *Ulcerated squamous cell carcinoma of the midesophagus with a large ulcer (white arrows) surrounded by a thick, radiolucent mass of tumor (black arrows). This polypoid, ulcerated carcinoma has a very different appearance than the giant HIV ulcer shown in Figure 7.*

linear configuration and may be associated with radiating folds or surrounding halos of edematous mucosa (Figure 4).[1] The ulcers nearly always develop at or adjacent to the gastroesophageal junction, extending proximally a variable distance as a continuous area of disease.[1] Ulceration that spares the distal esophagus should therefore suggest another cause of disease. Less frequently, reflux esophagitis may be manifested by a single dominant ulcer at or abutting the gastroesophageal junction. These so-called *marginal ulcers* are usually located on the posterior wall of the distal esophagus, most likely because of prolonged exposure to refluxed acid that pools by gravity in the posterior esophagus when the patient sleeps in a supine position.[10]

Herpes esophagitis

The herpes simplex virus type 1 is the second most common cause of infectious esophagitis in immunocompromised patients.[5] This condition is usually manifested on double-contrast studies by multiple small ulcers in the upper or midesophagus, often surrounded by radiolucent mounds of edema (Figure 5).[11,12] Most patients present with odynophagia, but herpetic lesions are not commonly found in the oropharynx, so it is difficult to differentiate herpes from *Candida* esophagitis on the basis of the clinical findings.

Herpes esophagitis may occasionally develop as an acute, self-limited disease in otherwise healthy patients. Affected individuals present with a flulike syndrome consisting of fever, headaches, myalgias, and upper respiratory symptoms for a period of 7-10 days prior to the sudden onset of severe odynophagia.[13] Double-contrast studies typically reveal multiple tiny ulcers that are even smaller than those in immunocompromised patients with herpes esophagitis, presumably because they have an intact immune system that prevents the ulcers from enlarging.[13]

Drug-induced esophagitis

Patients on oral medications, particularly antibiotics such as tetracycline and doxycycline and non-steroidal anti-inflammatory drugs (NSAIDs), may develop a focal contact esophagitis. These individuals typically present with acute odynophagia and have a history of ingesting the offending medication with little or no water immediately before going to bed. As a result, the capsules or tablets may lodge in the midesophagus, where it is compressed by the aortic arch or left main bronchus. Double-contrast studies usually reveal multiple small ulcers in the midesophagus (Figure 6).[1,14] This condition may be difficult to differentiate from herpes esophagitis on barium studies, but the clinical history usually suggests the correct diagnosis.

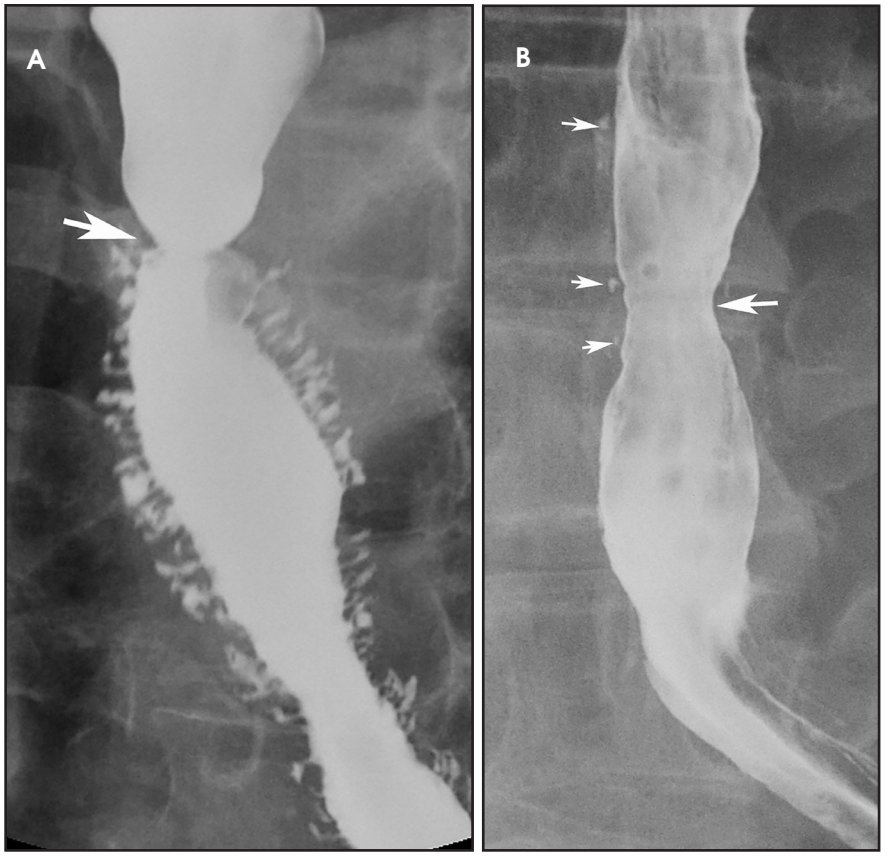

FIGURE 9. *Esophageal intramural pseudodiverticulosis. (A) The pseudodiverticula have a characteristic appearance, with multiple thin, flask-shaped outpouchings in longitudinal rows parallel to the long axis of the esophagus. Also note a short, tight stricture (arrow) in the upper esophagus above the pseudodiverticula. (B) Another patient has a smooth, tapered segment of narrowing secondary to a peptic stricture (large arrow) in the distal esophagus. There also are tiny pseudodiverticula (small arrows) at and just above the stricture. Note how the pseudodiverticula seem to be floating outside the wall of the esophagus, whereas true ulcers viewed in profile are almost always seen to communicate directly with the lumen.*

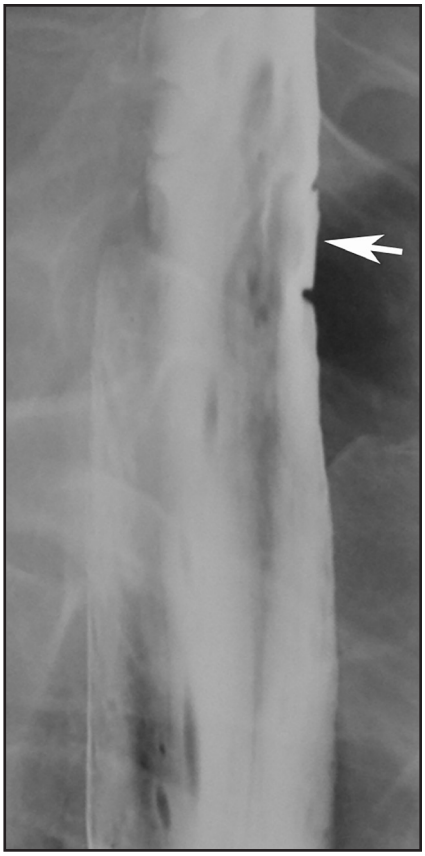

FIGURE 10. *Ectopic gastric mucosa manifested by a broad, shallow depression (arrow) on the left lateral wall of the upper thoracic esophagus near the thoracic inlet. Note the shallow indentations at its superior and inferior margins. Ectopic gastric mucosa is more commonly found on the right lateral wall of the upper esophagus.*

Crohn's disease

Crohn's disease involving the esophagus is occasionally manifested by small, superficial ulcers indistinguishable from aphthoid ulcers in the small bowel or colon in patients with this disease.[15,16] Because esophageal Crohn's disease is almost always associated with ileocolic disease, this diagnosis should only be considered in patients with known Crohn's disease elsewhere in the GI tract.

Large ulcers

CMV esophagitis

CMV esophagitis may be manifested by one or more giant, flat ulcers that are indistinguishable from human immunodeficiency virus (HIV) ulcers in the esophagus (see next section).[1] Affected individuals typically present with odynophagia and are found to have AIDS. Because of the potential toxicity of antiviral agents such as gancyclovir, endoscopic biopsies or cultures are required to confirm the presence of CMV before instituting treatment. Although better therapy for patients with the HIV virus has reduced the number of patients with AIDS, CMV esophagitis may occasionally develop in patients who receive steroids or bone marrow transplants.[17-19]

HIV esophagitis

Patients with HIV may develop one or more giant esophageal ulcers that are caused directly by the HIV virus itself, as confirmed on electron microscopy of biopsy specimens showing viral particles with morphologic features of HIV.[20] These ulcers are usually located in the lower or midesophagus, appearing on barium studies as giant (greater than 1 cm), ovoid or diamond-shaped craters surrounded by a thin, radiolucent rim of edema (Figure 7).[21,22] These ulcers are indistinguishable from giant CMV ulcers in the esophagus, but most HIV ulcers heal rapidly on treatment with steroids,[21,22] whereas CMV ulcers require treatment with antiviral agents. Endoscopy and biopsy are therefore required to differentiate these infections before instituting therapy.

Drug-induced esophagitis

Unlike tetracycline- or doxycycline-induced esophagitis, which is manifested by small, shallow ulcers, esophagitis caused by potassium chloride, quinidine,

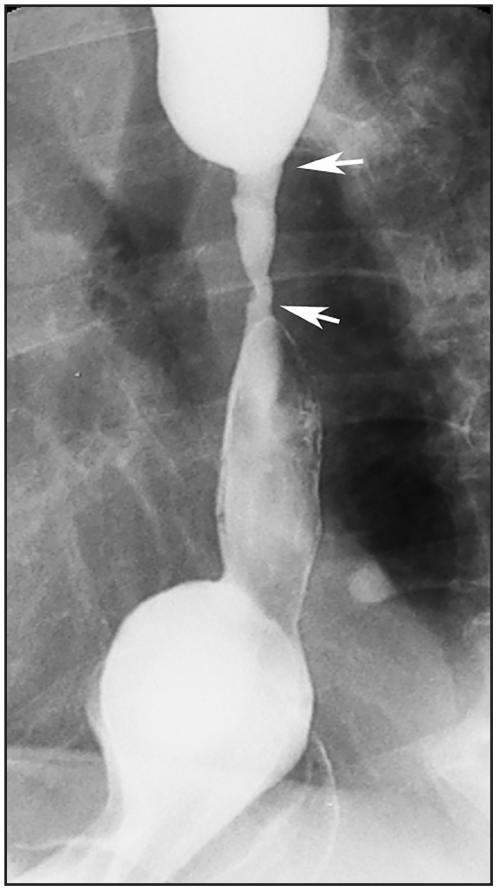

FIGURE 11. *Barrett's esophagus with an esophageal stricture. This stricture (arrows) is located at a considerable distance from the gastroesophageal junction (above a hiatal hernia), whereas uncomplicated peptic strictures in the absence of Barrett's esophagus are usually more distal. In the presence of a hiatal hernia and reflux, a stricture in this location should be highly suggestive of Barrett's esophagus.*

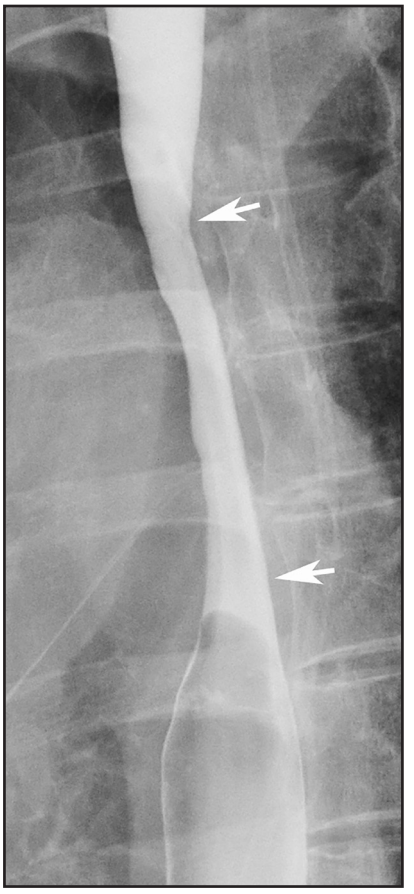

FIGURE 12. *Radiation stricture manifested by a long segment of concentric narrowing with a smooth contour and tapered margins (arrows) in the midesophagus. This patient had received mediastinal irradiation for bronchogenic carcinoma.*

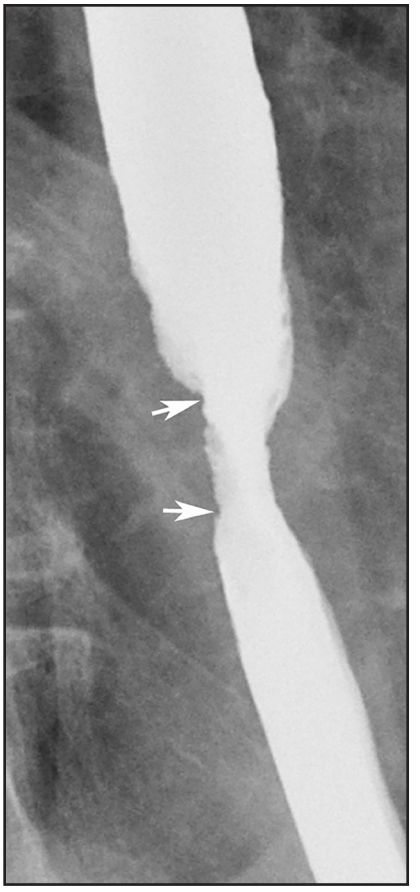

FIGURE 13. *Infiltrating squamous cell carcinoma manifested by a short segment of eccentric narrowing with an irregular contour and abrupt proximal and distal margins (arrows) in the midesophagus. This patient presented with recent onset of dysphagia and weight loss.*

NSAIDs, and alendronate sometimes leads to the development of giant esophageal ulcers.[23-25] The correct diagnosis is usually suggested by the clinical history.

Barrett's esophagus

Barrett's esophagus is an acquired condition in which there is progressive columnar metaplasia of the distal esophagus secondary to long-standing reflux disease. Barrett's esophagus is occasionally manifested by a single large ulcer within the columnar epithelium, occurring at a discrete distance from the gastroesophageal junction. Although uncommon, this finding should be highly suggestive of Barrett's esophagus in a patient with a hiatal hernia and gastroesophageal reflux.[1,14,26]

Esophageal carcinoma

Necrotic esophageal carcinomas may be manifested by a large ulcerated mass. In such cases, barium studies may reveal a giant meniscoid or ovoid ulcer surrounded by a thick, irregular mass of tumor (Figure 8). In contrast, giant benign ulcers (e.g., CMV and HIV ulcers) have a smooth, thin rim of surrounding edema, producing a different radiographic appearance (see Figure 7).

Mimics of ulceration

Esophageal intramural pseudodiverticulosis

Esophageal intramural pseudodiverticula are dilated excretory ducts of deep mucous glands in the esophagus. Barium studies usually reveal multiple tiny, flask-shaped outpouchings in longitudinal rows parallel to the long axis of the esophagus (Figure 9A).[27] When viewed en face, the pseudodiverticula can easily be mistaken for tiny ulcers. When viewed in profile, however, these structures often seem to be "floating" outside the esophagus, whereas true ulcers communicate directly with the lumen. Some pseudodiverticula have a diffuse distribution and are associated with high strictures (see Figure 9A), but pseudodiverticula more commonly occur in the distal esophagus in patients with peptic strictures (Figure 9B).[27,28] While the pathogenesis is uncertain, it has been postulated that pseudodiverticula develop as a result of glandular dilatation

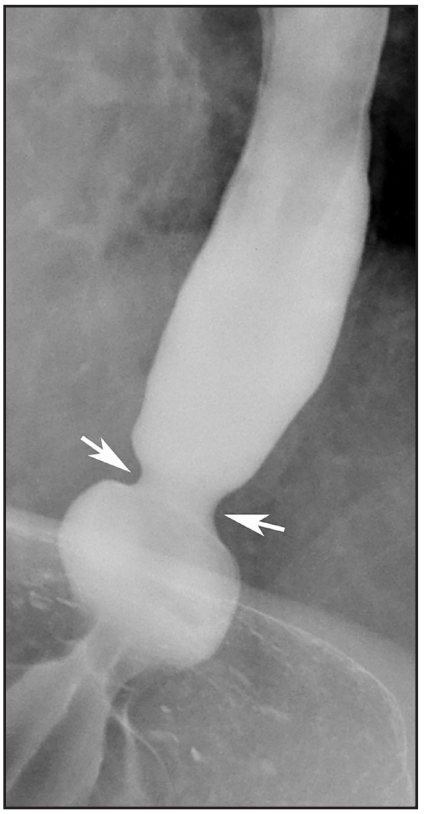

FIGURE 14. *Ringlike peptic stricture. This patient has a short segment of narrowing (arrows) in the distal esophagus above a hiatal hernia. This stricture could be mistaken for a Schatzki ring, but has a greater vertical height than a true lower esophageal ring.*

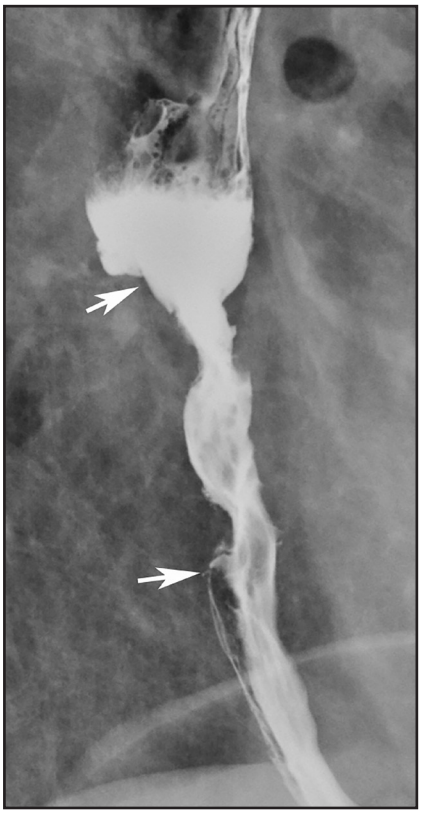

FIGURE 15. *Infiltrating adenocarcinoma in Barrett's esophagus. There is a long segment of irregular narrowing with shelflike proximal and distal margins (arrows) in the distal esophagus.*

from chronic inflammation. Occasionally, they may also be found in patients with alcoholism, diabetes, or *Candida* esophagitis.[29]

Ectopic gastric mucosa

Ectopic gastric mucosa is a common congenital anomaly unrelated to Barrett's esophagus. It typically is discovered as an incidental finding on the right lateral or, less commonly, the left lateral wall of the upper esophagus at or near the thoracic inlet and is manifested on barium studies by a broad, flat depression, with shallow indentations at its superior and inferior margins (Figure 10).[30] While this finding could be mistaken for a flat ulcer, its characteristic appearance and location should suggest the correct diagnosis. The vast majority of patients with ectopic gastric mucosa in the esophagus are asymptomatic.

Upper and midesophageal strictures

Barrett's esophagus

While most strictures in Barrett's esophagus are located in the distal esophagus, some patients may develop ringlike or tapered strictures in the midesophagus (Figure 11).[26] Because uncomplicated peptic strictures are almost always located within several centimeters of the gastroesophageal junction, a midesophageal stricture should be highly suggestive of Barrett's esophagus in a patient with a hiatal hernia and reflux.[26]

Mediastinal irradiation

Patients who receive high doses of external beam radiation to the mediastinum may develop radiation strictures within 4 to 8 months after completion of therapy. These strictures typically appear as long segments of smooth, tapered narrowing in the upper or midesophagus within a preexisting radiation portal (Figure 12).[31]

Esophageal carcinoma

Malignant tumors in the upper or midesophagus are usually squamous cell carcinomas. While benign strictures typically have a smooth contour and tapered margins (Figures 11 and 12), malignant strictures have a more irregular contour and abrupt, shelflike margins, often associated with mucosal nodularity and ulceration (Figure 13).[1,31] The history is also important, as patients with benign strictures have more long-standing dysphagia and little or no weight loss, whereas patients with malignant strictures have recent onset of progressive dysphagia and substantial weight loss. Thus, infiltrating carcinomas can usually be differentiated from benign strictures on the basis of the clinical and radiographic findings.

Distal esophageal strictures

Peptic stricture

Most benign strictures in the distal esophagus are caused by scarring from reflux esophagitis.[28,31] These reflux-induced or so-called *peptic* strictures most commonly appear as discrete (1-4 cm in length) segments of smooth, tapered narrowing, almost always above a hiatal hernia (Figure 9B). Not infrequently, however, peptic strictures are short (less than 1 cm in length), ring-like constrictions at or near the gastroesophageal junction . Such strictures can be mistaken for Schatzki rings (Figure 14),[28,31] though they tend to be more asymmetric and have a slightly greater height than true rings. Conversely, nasogastric intubation, Zollinger-Ellison syndrome, and alkaline reflux esophagitis may result in rapidly progressive reflux-type strictures involving a much longer segment of the distal esophagus than most peptic strictures.[28] Peptic strictures that have an unequivocally benign radiographic appearance are virtually always found to be benign, but strictures that are nodular, irregular, or asymmetric should be evaluated by endoscopy and biopsy to rule out malignant tumor.

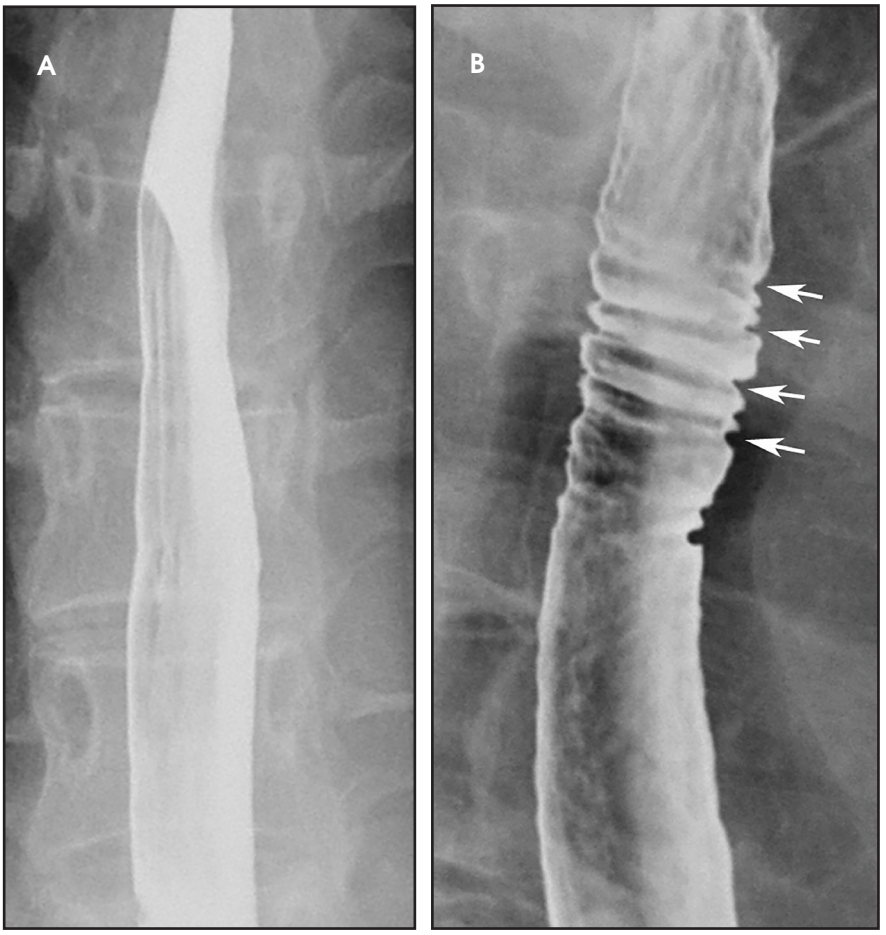

FIGURE 16. *Eosinophilic esophagitis. (A) This patient has a small-caliber esophagus. Note how there is loss of distensibility of the entire thoracic esophagus, which has a smooth contour without a discrete stricture. (B) Another patient has a ringed esophagus, with multiple discrete ringlike indentations (arrows) in the midesophagus. In a young man with long-standing dysphagia and an atopic history or asthma, a small-caliber or ringed esophagus should be highly suggestive of eosinophilic esophagitis.*

Adenocarcinoma

Esophageal adenocarcinomas arise in areas of preexisting columnar metaplasia within Barrett's esophagus and therefore tend to be located in the distal esophagus. Advanced adenocarcinomas are usually infiltrating lesions that narrow the lumen and, unlike squamous cell carcinomas, have a marked tendency to invade the gastric cardia and fundus.[1] These lesions typically appear on barium studies as irregular areas of luminal narrowing with shelflike margins (Figure 15). Occasionally, however, an early adenocarcinoma may be recognized by nodularity or irregularity within a preexisting peptic stricture.[1] Endoscopy and biopsy are required to rule out malignant tumor in these patients.

Diffuse esophageal narrowing

Eosinophilic esophagitis

Eosinophilic esophagitis is an inflammatory condition, usually occurring in children or young adults (especially men) with long-standing dysphagia and recurrent food impactions. Affected individuals often have an atopic history, asthma, and/or peripheral eosinophilia.[32] Some patients have smooth, long-segment narrowing or diffuse loss of distensibility of the entire thoracic esophagus (without a discrete stricture), producing a so-called *small-caliber esophagus* (Figure 16A).[33] Other patients have multiple distinctive ringlike indentations (sometimes associated with a focal stricture or diffuse esophageal narrowing), producing a so-called

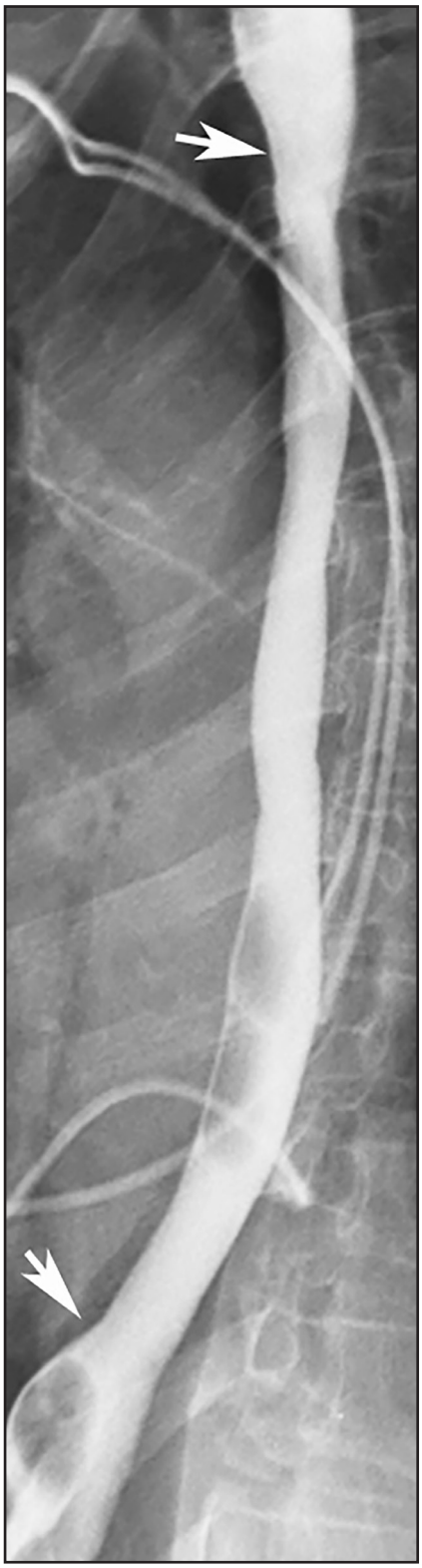

FIGURE 17. *Lye stricture with a long stricture extending from just below the thoracic inlet (upper arrow) to just above the gastroesophageal junction (lower arrow). Other benign strictures are rarely associated with such extensive esophageal narrowing.*

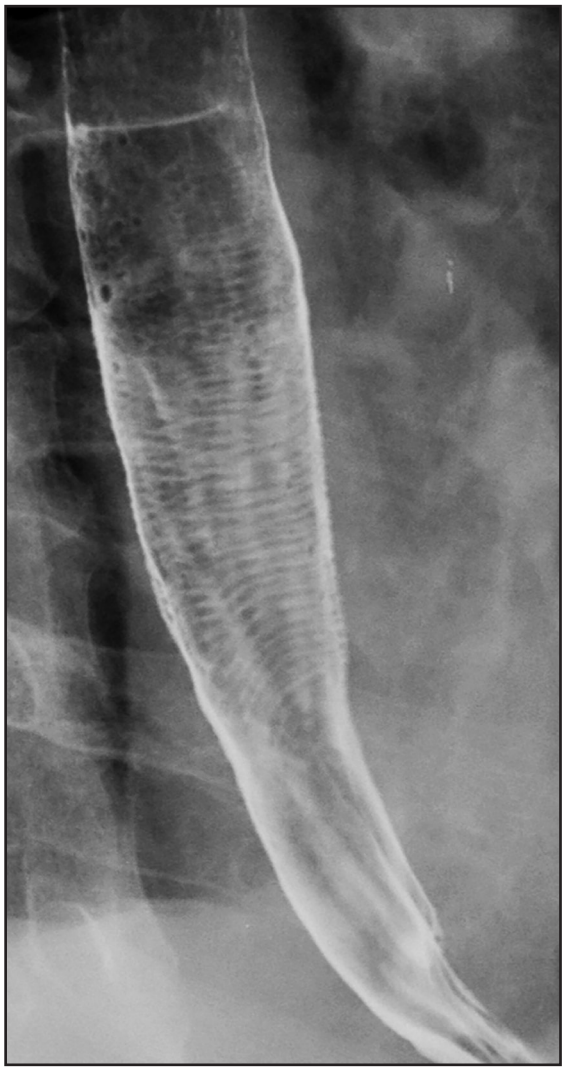

FIGURE 18. *Feline esophagus with fine transverse folds seen as multiple closely spaced, horizontal striations in the lower thoracic esophagus. This was a transient finding at fluoroscopy in a patient with marked gastroesophageal reflux.*

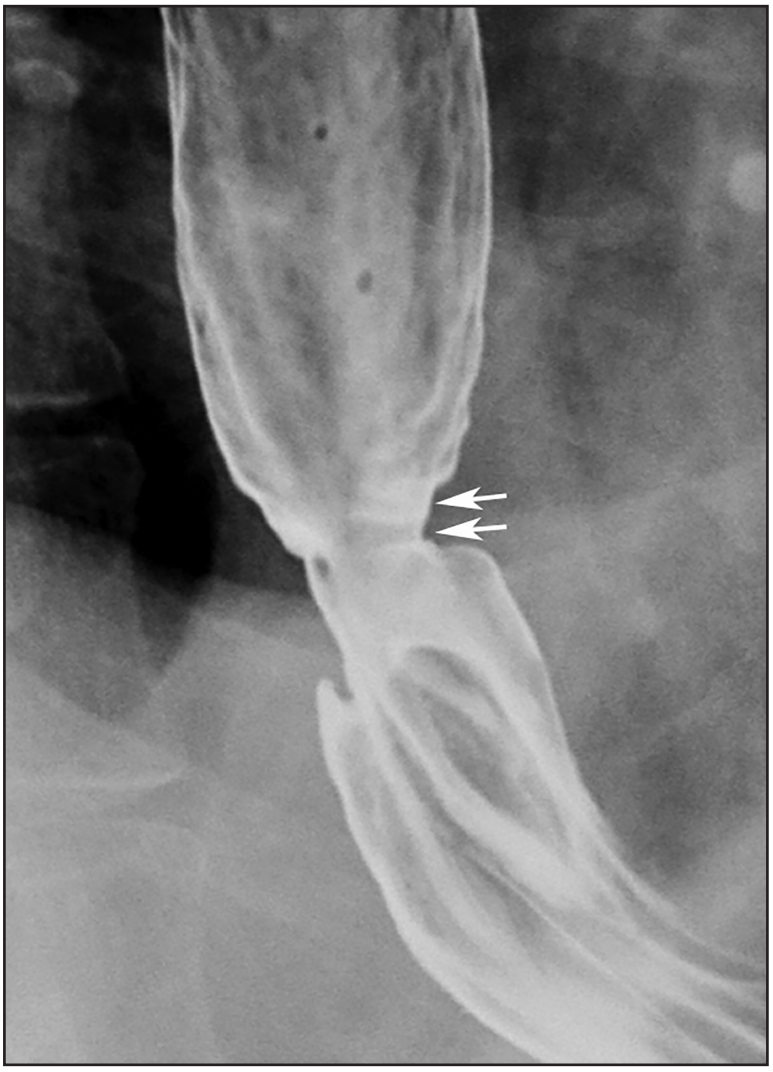

FIGURE 19. *Peptic stricture with fixed transverse folds. This patient has a short, eccentric, but smooth stricture in the distal esophagus with trapping of barium between fixed transverse folds (arrows) in the region of the stricture due to associated longitudinal scarring from reflux esophagitis. The folds are few in number, do not extend across the circumference of the esophagus, and are associated with a peptic stricture, enabling differentiating from the feline esophagus shown in Figure 18.*

ringed esophagus (Figure 16B).[34] In a young man with long-standing dysphagia and an atopic history, a small-caliber or ringed esophagus should be highly suggestive of eosinophilic esophagitis.

Caustic ingestion

Ingestion of a strong acid or base may cause severe esophagitis, leading to stricture formation within 1-3 months. Lye strictures are manifested by segmental narrowing of the upper or mid-esophagus or, in advanced cases, by diffuse, marked narrowing of nearly the entire thoracic esophagus (Figure 17).[28,31] The small-caliber esophagus of eosinophilic esophagitis may produce similar findings (see Figure 16A), but is not usually associated with as much narrowing and irregularity as a severe lye stricture. In problematic cases, the correct diagnosis can almost always be made from the clinical history.

Esophageal rings

Feline esophagus

The feline esophagus is manifested on barium studies by closely spaced, thin, horizontal striations extending across the circumference of the esophagus (Figure 18).[35] While the feline esophagus may be discovered as an incidental finding, it is nearly always associated with gastroesophageal reflux and is usually observed during actual reflux episodes.[35] The characteristic appearance and transient nature of the feline esophagus enable differentiation from other types of esophageal rings.

Fixed transverse folds

Double-contrast barium studies may occasionally reveal fixed transverse folds in the distal esophagus, with

trapping of barium between the folds, producing a characteristic *stepladder* appearance (Figure 19).[36] The folds are usually 2 to 5 mm in width and do not extend fully across the esophagus.[36] They almost always develop in the region of a peptic stricture and most likely result from longitudinal scarring and shortening of the esophagus due to chronic reflux esophagitis (see Figure 19).[36] Unlike the feline esophagus and nonperistaltic esophageal contractions, which are transient in nature, these transverse folds are seen as a persistent finding on esophagography.[36]

Ringed esophagus of eosinophilic esophagitis

As discussed earlier, patients with eosinophilic esophagitis may develop distinctive ringlike indentations (sometimes associated with a focal stricture or diffuse esophageal narrowing), producing a ringed esophagus (see Figure 16).[34] While the pathogenesis of the rings is unknown, this appearance should be highly suggestive of eosinophilic esophagitis, particularly in young men with long-standing dysphagia, recurrent food impactions, and a history of allergies or asthma.

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