10.1 Nonneoplastic Conditions

10.1.1 Gastroesophageal Reflux Disease

Early reflux esophagitis typically is characterized on double-contrast studies by granularity of the distal esophagus secondary to mucosal edema and inflammation [1]. Shallow ulcers appear as punctate, linear, or serpiginous barium collections, sometimes associated with radiating folds [1]. This ulceration almost always extends proximally from the gastroesophageal junction as a continuous area of disease, so ulcers that are confined to the upper or midesophagus should suggest another cause for the patient’s disease. Scarring from reflux esophagitis can lead to the development of reflux-induced (peptic) strictures, typically seen as smooth, tapered areas of distal esophageal narrowing or as asymmetric ring-like strictures above a hiatal hernia [1].

10.1.2 Barrett’s Esophagus

Barrett’s esophagus is a premalignant condition in which the usual squamous epithelium in the distal esophagus is replaced by metaplastic columnar epithelium secondary to chronic reflux esophagitis. This condition develops in 10% of patients with reflux esophagitis and 20–40% with peptic strictures [1]. Barrett’s esophagus sometimes can be recognized on barium studies by the development of a tapered or ring-like stricture in the midesophagus [1]. Other patients may develop a distinctive reticular pattern of the mucosa [2]. In the presence of a hiatal hernia and reflux, a high stricture or reticular pattern should be strongly suggestive of Barrett’s esophagus. Another recently reported finding of Barrett’s esophagus is a high Z line at the level of an elevated squamo-columnar mucosal junction above long-segment columnar metaplasia [3].

10.1.3 Infectious Esophagitis

Candida esophagitis typically is manifested on double-contrast barium studies by multiple discrete linear or irregular plaquelike defects separated by normal mucosa [4]. Patients with AIDS occasionally may develop a more fulminating form of candidiasis characterized by a grossly irregular or shaggy esophagus secondary to trapping of barium between innumerable plaques and pseudomembranes [5].
In an immunocompromised patient with dysphagia or odynophagia, these findings should be highly suggestive of *Candida* esophagitis. In contrast, herpes esophagitis usually is characterized by multiple small, discrete ulcers in the upper or midesophagus without associated plaque formation (Fig. 10.1) [6].

Patients with AIDS occasionally may develop CMV esophagitis, manifested by giant ulcers greater than 1 cm in size [7]. Because herpetic ulcers rarely become this large, giant ulcers should suggest CMV in patients with AIDS. Human immunodeficiency virus (HIV) has also been recognized as a cause of giant esophageal ulcers indistinguishable from those in CMV [8]. Unlike CMV ulcers, however, HIV ulcers may undergo rapid healing on treatment with oral steroids, so endoscopy is required to differentiate these infections.

10.1.4 Eosinophilic Esophagitis

Eosinophilic esophagitis (EoE) has been recognized as a chronic inflammatory disease causing dysphagia and recurrent food impactions in young men, who often have a history of allergies, asthma, or peripheral eosinophilia. EoE may be manifested on esophagography by segmental strictures, discrete ringlike indentations (the so-called ringed esophagus) [9], or a small-caliber esophagus with smooth, long-segment narrowing of virtually the entire thoracic esophagus (Fig. 10.2) [10]. Recently, lichen planus involving the esophagus has been recognized as another cause of a small-caliber esophagus indistinguishable from that in EoE [11]. Unlike EoE, however, lichen planus typically occurs in elderly women with a skin rash.
and is not associated with allergies, asthma, or peripheral eosinophilia [11].

10.1.5 Drug-Induced Esophagitis

Drug-induced esophagitis is caused by various medications, including tetracycline, doxycycline, potassium chloride, quinidine, alendronate, and nonsteroidal anti-inflammatory drugs (NSAIDs) [12]. Affected individuals often ingest the medication with little or no water immediately before going to bed, so the medication lodges in the midesophagus, causing a focal contact esophagitis. Double-contrast studies may reveal a focal cluster of small, discrete ulcers in the midesophagus indistinguishable from those in herpes esophagitis [12], but the correct diagnosis usually is suggested by the clinical history.

10.1.6 Erosive Gastritis

Erosive gastritis is characterized on double-contrast studies by multiple punctate or slitlike collections of barium surrounded by radiolucent halos of edematous mucosa [13]. The erosions tend to be located in the gastric antrum and often are aligned on the crests of the folds. NSAIDs are by far the most common cause of erosive gastritis, accounting for more than 50% of cases. Occasionally, however, NSAID-induced erosive gastritis may be manifested by multiple distinctive linear or serpiginous erosions that tend to be clustered in the gastric antrum or body, often near the greater curvature [14]. These erosions are thought to result from localized mucosal injury as the dissolving NSAID capsules collect by gravity in the most dependent portion of the stomach. Whatever the explanation, detection of this finding should lead to careful questioning about recent NSAID use, and if confirmed, the offending medication should be withdrawn.

10.1.7 *Helicobacter Pylori* Gastritis

*Helicobacter pylori* gastritis can be diagnosed on barium studies by thickened folds in the antrum, body, or, less commonly, fundus of the stomach [15]. Other patients with *H. pylori* may have a polypoid form of gastritis characterized by grossly thickened, lobulated folds, mimicking the appearance of Ménétrier’s disease, lymphoma, or even a submucosally infiltrating carcinoma [15]. In these cases, endoscopy and biopsy are required for a definitive diagnosis.

10.1.8 Gastric Ulcers

Benign gastric ulcers classically appear en face as round or ovoid barium collections with a smooth, surrounding mound of edema and/or thin, straight folds radiating to the edge of the ulcer crater [16]. When viewed in profile, benign ulcers project beyond the gastric wall and are often associated with an ulcer mound or collar. In contrast, malignant ulcers appear en face as irregular craters in a discrete mass with nodularity or clubbing of adjoining folds [16]. When viewed in profile, malignant ulcers project inside the lumen within a mass that forms acute angles with the wall rather than the obtuse angles expected for a benign mound of edema [16].

Most benign ulcers are located on the lesser curvature or posterior wall of the gastric antrum of the body [16]. Occasionally, however, benign ulcers may occur on the greater curvature of the distal antrum, and nearly all of these ulcers are found to be caused by NSAIDs [16]. As NSAID-induced greater curvature ulcers enlarge, they can penetrate inferiorly via the gastrocolic ligament into the transverse colon, producing a gastrocolic fistula [17]. In today’s pill-oriented society, these NSAID-induced ulcers are the most common cause of gastrocolic fistulas.

10.2 Neoplastic Conditions

10.2.1 Esophageal Cancer

On double-contrast barium studies, early squamous cell carcinomas of the esophagus present as small sessile polypoid lesions with smooth or slightly lobulated contours; plaque-like lesions that often have flat, central ulcers that are best visualized in profile; or as superficial spreading lesions with a nodular appearance of the mucosa without a discrete mass. Advanced squamous cell carcinomas may appear infiltrative, ulcerative (Fig. 10.3), polypoid, or, less commonly, varicoid. Advanced carcinomas most commonly have an infiltrative appearance because they tend to grow circumferentially in the esophageal wall [18].

Early adenocarcinomas of the esophagus arising from Barrett’s mucosa can manifest as small sessile polyps, plaque-like lesions, or superficial spreading lesions that cause focal nodularity of the mucosa without a discrete mass. These early cancers can also cause focal irregularity, flattening, or nodularity in a pre-existing peptic stricture. Accordingly, early endoscopy and biopsy are necessary to exclude adenocarcinoma whenever any of these suspicious features develop in a region of a peptic stricture. Advanced adenocarcinoma of the esophagus can appear infiltrating, polypoid, ulcerative, or, less commonly, varicoid on barium studies [18].
Squamous cell carcinomas of the esophagus tend to be located in the upper or midthoracic esophagus and only rarely invade the adjacent stomach by contiguous spread. Adenocarcinomas of the esophagus develop primarily in the distal thoracic esophagus and have a marked tendency to invade the gastric cardia and fundus (Fig. 10.4). It may not always be possible to differentiate gastric adenocarcinoma of the gastric cardia invading the distal esophagus from a Barrett’s-related cancer arising from the distal esophagus [18].

Since a multimodality approach is presently used to treat esophageal cancer, a precise histologic diagnosis and highly accurate tumor staging are prerequisites for selecting the most suitable treatment options. This requires histologic classification of the tumor type, the exclusion of distant organ metastases, localization of the primary tumor in relation to the tracheobronchial tree, and determination of the penetration of the primary tumor through the esophageal wall into the surrounding tissues [19].

CT is useful in distinguishing between patients with early cancer (T1 and T2) who need further evaluation with EUS and those with T3 and T4 disease. For local staging of esophageal cancer using CT, 3 mm is considered the upper limit of normal mural thickness. The following comprise radiologic T stage criteria for esophageal cancer:

**T1:** focal or circumferential wall thickening of >3 and ≤10 mm and/or intense enhancement of the esophageal wall, without stenosis, and the outer borders of the tumor are smooth.

**T2:** focal, polypoid, or diffuse circumferential thickening of the esophageal wall >10 and ≤15 mm and the outer borders of the tumor are either smooth or show stranding for less than one-third of the tumor extension.

**T3:** tumor appears symmetric or asymmetric, and markedly diffuse or circumferential wall thickening of ≥15 mm, with mild-to-severe stenosis, and marked stranding for over one-third of the tumor extension, or extensive blurring of the outer border.

**T4:** tumor shows invasion into one of the adjacent structures: pericardium, the diaphragm, the pleura (T4a), the tracheobronchial tree, or the aorta and spine (T4b) [20].

Lymphatic spread is found in 74–88% of patients with esophageal carcinoma. Accurate lymph node assessment for
metastatic spread is challenging, even with PET/MDCT. Diagnostic accuracy is improved if morphology, including size and shape, contrast enhancement pattern, and tracer uptake of lymph nodes are considered. N staging is as follows: N0, no regional lymph node metastasis; N1, 1–2 positive regional lymph nodes; N2, 3–6 positive regional lymph nodes; and N3, ≥7 positive regional lymph nodes [21].

Hematogenous metastases from esophageal carcinoma may involve the liver, lungs, adrenal glands, kidneys, bones, and brain. Lymph node involvement outside the periesophageal location is considered M1 disease [21].

### 10.2.2 Gastric Cancer

On double-contrast barium studies, Type I early gastric cancers typically appear as small protruded lesions in the stomach. Type II early gastric cancers are relatively flat lesions with elevated, superficial, or protruded components. These lesions may present as plaque-like elevations, mucosal nodularity, shallow ulcers, or some combination of these findings. Occasionally, these lesions can be quite extensive, involving a large portion of the surface area of the stomach without invading beyond the submucosa. Type III early gastric cancers manifest as shallow ulcer craters with nodularity of the adjacent mucosa and clubbing or fusion of radiating folds due to infiltration of the adjoining folds by tumor. Advanced gastric carcinomas usually appear on barium studies as polypoid, ulcerative, or infiltrative lesions [22].

As with esophageal carcinoma, precise histologic diagnosis and highly accurate tumor staging of gastric cancer are prerequisites for selecting the most suitable treatment options. In early advanced gastric cancers, the outer border of the stomach is smooth. The probability of transmural extension of the tumor (T3) is directly correlated with mural thickness. In transmural extension, the serosal contour becomes blurred, and strand-like areas of increased attenuation may be seen extending into the perigastric fat. Tumor spread frequently occurs via ligamentous and peritoneal reflections to adjacent organs (T4). The liver may be invaded via the gastrohepatic ligament, the pancreas via the lesser sac, the spleen via the gastroplenic ligament (Fig. 10.5), and the transverse colon via the gastrocolic ligament. The distal esophagus is directly involved by carcinoma of the cardia in about 60% of patients, whereas the duodenum is involved by carcinoma of the antrum in 13–18% of patients [22–24].

Lymphatic spread is found in 74–88% of patients with gastric carcinoma because of the abundant lymphatic vessels in the stomach. The frequency of lymphatic metastases is related to the size and depth of penetration of the tumor. N staging depends on the number of positive perigastric lymph nodes (N1 1–5, N2 6–15, and N3 >15 affected lymph nodes) [23, 24].

Hematogenous metastases from gastric carcinoma most commonly involve the liver because the stomach is drained by the portal vein. Less common sites include the lungs, adrenal glands, kidneys, bones, and brain. Lymph node involvement outside the perigastric location is considered M1 disease. Advanced cancers can develop peritoneal and ovarian (Krukenberg) metastases [24].

![Fig. 10.5 Adenocarcinoma of the stomach invading the gastroplenic ligament and splenic hilum (arrows). This gastric neoplasm shows ulceration on the axial (a) and coronal (b) reformatted image](image-url)
10.2.3 Gastric Lymphoma

Lymphoma involves the stomach more frequently than any other portion of the gastrointestinal tract. Primary gastric lymphomas are confined to the stomach and regional lymph nodes (about 35% of gastrointestinal lymphomas) and are predominantly non-Hodgkin lymphomas of B-cell origin. Lymphoma of mucosa-associated lymphoid tissue (MALT) is a distinct type of extranodal lymphoma that is characterized by a relatively indolent clinical course and has a much better prognosis than gastric carcinoma, with overall 5-year survival rates of 50–60%. Although the clinical symptoms in high-grade lymphoma and MALT lymphoma may be similar, they differ in several aspects [25, 26].

High-grade B-cell lymphoma has a relatively aggressive course as opposed to the more indolent and favorable outcome of MALT lymphoma. In high-grade gastric lymphomas, the extent of disease is usually greater at presentation, with involvement of adjacent organs and perigastric lymph nodes [25].

Lymphomas may involve any portion of the stomach. Transpyloric spread of tumor into the duodenum occurs in about 30% of patients. Despite extensive lymphomatous infiltration, the stomach usually remains pliable and distensible without significant luminal narrowing. Early gastric lymphoma is confined to the mucosa and submucosa, with an average size of only 3.5 cm at diagnosis. Gastric lymphomas are usually advanced lesions with a mean diameter of 10 cm. Most cases involve the antrum and body, although the entire stomach can be involved [24].

There are four gross pathologic types of gastric lymphoma [22]. Infiltrative gastric lymphomas manifest as focal or diffuse enlargement of gastric folds due to submucosal spread of tumor. One or more ulcerated lesions characterize ulcerative gastric lymphoma. Polypoïd gastric lymphomas are characterized by intraluminal masses that may simulate polypoid carcinomas. Multiple submucosal nodules ranging in size between several millimeters and several centimeters characterize nodular gastric lymphoma.

CT is the primary imaging modality for pretreatment evaluation of abdominal lymphoma. In patients with suspected gastric lymphoma, CT provides depiction of gastric involvement and staging of generalized lymphoma in the abdomen and chest. Furthermore, CT may aid in early diagnosis of disease progression in patients undergoing therapy and follow-up for low-grade MALT lymphoma, which may progress to high-grade B-cell lymphoma. The CT appearances of lymphoma and gastric carcinoma may be very similar.

10.2.4 Gastrointestinal Stromal Tumor (GIST)

GISTs have recently been recognized as the most common mesenchymal neoplasm of the gastrointestinal tract. Many but not all mesenchymal tumors previously diagnosed as leiomyomas, leiomyoblastomas, and leiomyosarcomas are now considered GISTs on the basis of specific immunohistochemical criteria- c-kit positivity. The malignant variety of GISTs represents only about 3% of all malignant gastrointestinal tumors. Approximately 60–70% are found in the stomach. It is known that 10–30% of GISTs are malignant, and the risk of malignancy increases with extragastric location, diameter greater than 5 cm, and extension into adjacent organ. Before and during surgery, in the absence of metastases, it is difficult to distinguish benign and malignant lesions. As with leiomyomas and leiomyosarcomas, intramural endogastric (Fig. 10.6) and exogastric lesions can be distinguished [27, 28] on cross-sectional imaging studies.

10.2.5 Carcinoid Tumors

Carcinoid tumors of the stomach are rare and have three subtypes, which have unique endoscopic and radiographic appearances. Type 1 gastric carcinoid tumors are associated with enterochromaffin-like cell hyperplasia, hypergastrinemia, and chronic atrophic gastritis, with or without pernicious anemia. Type 1 tumors generally represent benign disease. Nodal and hepatic metastases are very rare [29, 30]. Type 2 tumors are the least common type, representing 5–10% of gastric carcinoid tumors. They are seen in the hypergastrinemic state of Zollinger-Ellison syndrome in association with multiple endocrine neoplasia (MEN) type 1. Approximately 30% of patients with MEN 1 have gastric carcinoid tumors. Type 2 tumors also arise from enterochromaffin-like cells in the setting of hyperplasia. These tumors are multicentric and variable in size but are prone to developing local lymph node metastases. The appearance of these tumors on CT and radiographs of the upper gastrointestinal tract can be striking because there are multiple masses in the setting of diffuse gastric wall thickening [29, 30]. Type 3 gastric carcinoid tumors are sporadic tumors and are not associated with a hypergastrinemic state. They represent about 13% of gastric carcinoid tumors. Unlike type 1 and 2 tumors, type 3 tumors are large, solitary tumors that may show ulceration and are more likely to be invasive with distant metastases. The likelihood of metastasis is dependent on tumor size. Carcinoid syndrome may be seen in patients with hepatic metastases [29, 30].

When gastric carcinoid tumors are suspected, contrast material and water enhanced multidetector CT should be used to detect small mucosal masses. The discovery of polyps in a patient with chronic atrophic gastritis should alert the radiologist to the possibility of type 1 gastric carcinoid tumors. CT is necessary to properly assess type 2 (MEN 1 associated) and type 3 (sporadic) gastric carcinoid tumors, given the increased predisposition for nodal and hepatic metastases [29, 30].
Key Points

- In the presence of a hiatal hernia and reflux, a high esophageal stricture or reticular pattern should be strongly suggestive of Barrett’s esophagus.
- Benign gastric ulcers classically appear en face as round or ovoid barium collections with a smooth, surrounding mound of edema and/or thin, straight folds radiating to the edge of the ulcer crater.
- Highly accurate tumor staging is a prerequisite for the selection of the most appropriate therapy of patients with upper gastrointestinal tract malignancies.
- Endoscopic ultrasound is more accurate for T staging than CT and MR in early upper gastrointestinal tract malignancies.
- PET/CT is recommended for staging locally advanced esophageal carcinoma.

Fig. 10.6 Gastric gastrointestinal stromal tumor. An ulcerating intramural mass is identified in the greater curvature aspect of the proximal gastric body. (a) Double-contrast upper GI series. (b) Shaded surface display from a CT gastrography study. (c) Endoluminal view from a CT gastrography study. (d) Upper GI endoscopic view of the lesion.
References

1. Levine MS. Gastroesophageal reflux disease. In: Gore RM, Levine MS, editors. Textbook of gastrointestinal radiology. 4th ed. Philadelphia, PA: Elsevier; 2015. p. 291–311.
2. Levine MS, Kressel HY, Caroline DF, et al. Barrett esophagus: reticular pattern of the mucosa. Radiology. 1982;147:663–7.
3. Levine MS, Ahmad NA, Rubesin SE. Elevated Z line: a new sign of Barrett’s esophagus on double-contrast barium esophagograms. Clin Imaging. 2015;39:1103–4.
4. Levine MS, Macones AJ, Laufer I. Candida esophagitis: accuracy of radiographic diagnosis. Radiology. 1985;154:581–7.
5. Levine MS, Wolfenberg R, Herlinger H, et al. Opportunistic esophagitis in AIDS: radiographic diagnosis. Radiology. 1977;165:815–20.
6. Levine MS, Loevner LA, Saul SH, et al. Herpes esophagitis: sensitivity of double-contrast esophagography. AJR. 1988;151:57–62.
7. Balthazar EJ, Megibow AJ, Hulnick DH. Cytomegalovirus esophagitis and gastritis in AIDS. AJR. 1985;144:1201–4.
8. Sor S, Levine MS, Kowalski TE, et al. Giant ulcers of the esophagus in patients with human immunodeficiency virus: clinical, radiographic, and pathologic findings. Radiology. 1995;194:447–51.
9. Zimmerman SL, Levine MS, Rubesin SE, et al. Idiopathic eosinophilic esophagitis in adults: the ringed esophagus. Radiology. 2005;236:159–65.
10. White SB, Levine MS, Rubesin SE, et al. The small-caliber esophagus: radiographic sign of idiopathic eosinophilic esophagitis. Radiology. 2010;256:127–34.
11. Rauschecker AM, Levine MS, Whitson MJ, et al. Esophageal lichen planus: clinical and radiographic findings in eight patients. AJR. 2017;208:1–6.
12. Levine MS. Other esophagitis. In: Gore RM, Levine MS, editors. Textbook of gastrointestinal radiology. 4th ed. Philadelphia, PA: Elsevier; 2015. p. 326–49.
13. Levine MS. Inflammatory conditions of the stomach and duodenum. In: Gore RM, Levine MS, editors. Textbook of gastrointestinal radiology. 4th ed. Philadelphia, PA: Elsevier; 2015. p. 467–95.
14. Levine MS, Peptic ulcers. In: Gore RM, Levine MS, editors. Textbook of gastrointestinal radiology. 4th ed. Philadelphia, PA: Elsevier; 2015. p. 467–95.
15. Levine MS, Kelly MR, Laufer I, et al. Gastrocolic fistulas: the increasing role of aspirin. Radiology. 1993;187:359–61.
16. Levine MS, Halvorsen RA. Carcinoma of the esophagus. In: Gore RM, Levine MS, editors. Textbook of gastrointestinal radiology. 4th ed. Philadelphia, PA: Elsevier; 2015. p. 366–93.
17. Shah MA. Future directions in improving outcomes for patients with gastric and esophageal cancer. Hematol Oncol Clin North Am. 2017;31(3):545–52.
18. Mehta K, Bianco V, Awais O, Luketich JD, Pennathur A. Minimally invasive staging of esophageal cancer. Ann Cardiothorac Surg. 2017;6(2):110–8.
19. Goel R, Subramanian RM, Wachsmann JW. PET/computed tomography scanning and precision medicine: esophageal cancer. PET Clin. 2017;12(4):373–91.
20. Levine MS, Megibow AJ, Kochman ML. Carcinoma of the stomach and duodenum. In: Gore RM, Levine MS, editors. Textbook of gastrointestinal radiology. 4th ed. Philadelphia, PA: Elsevier; 2015. p. 546–70.
21. Hayes T, Smyth E, Riddell A, Allum W. Staging in esophageal and gastric cancers. Hematol Oncol Clin North Am. 2017;31(3):427–40.
22. Levine MS, Verstandig A, Laufer I. Serpiginous gastric erosions of radiographic presentation of gastrointestinal stromal tumors on imaging. Abdom Radiol. 2017;42(5):1350–64.
23. Shah MA. Future directions in improving outcomes for patients with gastric and esophageal cancer. Hematol Oncol Clin North Am. 2017;31(3):545–52.
24. Goel R, Subramanian RM, Wachsmann JW. PET/computed tomography scanning and precision medicine: esophageal cancer. PET Clin. 2017;12(4):373–91.
25. Ikoma N, Badgwell BD, Mansfield PF. Multimodality treatment of gastric lymphoma. Surg Clin North Am. 2017;97(2):405–20.
26. Ma Z, Fang M, Huang Y, He L, et al. CT-based radiomics signature for differentiating Borrmann type IV gastric cancer from primary gastric lymphoma. Eur J Radiol. 2017;91:142–7.
27. Keung EZ, Raut CP. Management of gastrointestinal stromal tumors. Surg Clin North Am. 2017;97(2):437–52.
28. Scena D, Baboura L, Copelan A, Shirkhoda A, Sokhandon F. Getting the GIST: a pictorial review of the various patterns of presentation of gastrointestinal stromal tumors on imaging. Abdom Radiol. 2017;42(5):1350–64.
29. Corey B, Chen H. Neuroendocrine tumors of the stomach. Surg Clin North Am. 2017;97(2):333–43.
30. Lin YM, Chiu NC, Li AF, Liu CA, Chou YH, Chiou YY. Unusual gastric tumors and tumor-like lesions: radiological with pathological correlation and literature review. World J Gastroenterol. 2017;23(14):2493–504.