Esophageal Neoplasms
Radiologic-Pathologic Correlation

John P. Lichtenberger III, MD[a,*], Merissa N. Zeman, MD[b], Adam R. Dulberger, MD, Capt, USAF, MC[c], Sadiq Alqutub, MD[d], Brett W. Carter, MD[e], Maria A. Manning, MD[f,g]

KEYWORDS

• Esophageal neoplasms • Esophagus • Radiologic-pathologic correlation • Computed tomography • Magnetic resonance imaging • FDG PET/CT

KEY POINTS

- The radiologic-pathologic correlation of esophageal neoplasms is an important skill for clinical imagers, informing both diagnosis and anticipated clinical management.
- The epidemiology and management of esophageal carcinomas are changing, and clinical imagers will have increased specificity and clinical relevancy if they can put these tumors into an appropriate clinical context.
- Rare malignancies and benign esophageal neoplasms have distinct imaging appearances because of their underlying histology.

INTRODUCTION

Esophageal cancer is the seventh most common cancer worldwide, with more than 572,000 cases, resulting in more than 508,000 deaths in 2018.[1] In the United States in 2020, the American Cancer Society estimates there will be more than 18,400 new cases of esophageal cancer diagnosed, resulting in more than 16,000 deaths.[2] Although esophageal squamous cell carcinoma (ESCC) accounts for approximately 90% of cases worldwide, largely attributable to cigarette smoking and alcohol consumption, there has been a shift toward esophageal adenocarcinomas (EACs), especially in North America and Europe.[3] In the United States, the prevalence of EAC has now surpassed that of ESCC, as the prevalence of gastroesophageal reflux disease (GERD) and prevalence of obesity have increased.[4–6]

At autopsy, benign tumors represent only 20% of esophageal neoplasms.[7] Most are small lesions that cause no symptoms; however, dysphagia, bleeding, or other symptoms can occur, in which case endoscopic or surgical removal may be necessary. Of these, leiomyomas occur most frequently, accounting for more than 50% of all benign neoplasms, and occur nearly twice as often in men.[8,9] Tumor-like conditions, such as fibrovascular polyps, also occur in the esophagus and have a distinct clinical presentation and imaging appearance.

This article examines the imaging appearances of esophageal neoplasms, with an emphasis on the pathologic basis of those manifestations. Although accurate diagnosis of esophageal neoplasms typically begins with imaging, the pathologic findings play a key role in determining treatment and

[a] The George Washington University Medical Faculty Associates, 900 23rd Street Northwest, Suite G 2092, Washington, DC 20037, USA; [b] Department of Radiology, The George Washington University Hospital, 900 23rd Street Northwest, Suite G 2092, Washington, DC 20037, USA; [c] Department of Radiology, David Grant Medical Center, Travis Air Force Base, 101 Bodin Cir, Fairfield, CA 94533, USA; [d] Department of Pathology, The George Washington University Hospital, 900 23rd Street Northwest, Suite G 2092, Washington, DC 20037, USA; [e] Department of Thoracic Imaging, MD Anderson Cancer Center, 1515 Holcombe Boulevard, Unit 1478, Houston, TX 77030, USA; [f] American Institute for Radiologic Pathology, American College of Radiology, 1100 Wayne Avenue, Suite 1020, Silver Spring, MD 20910, USA; [g] MedStar Georgetown University Hospital, Washington, DC, USA
* Corresponding author.
E-mail address: jlichtenberger@mfa.gwu.edu

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prognosis, and imagers should be equipped to discuss this radiologic-pathologic relationship as part of the care team.

ESOPHAGEAL SQUAMOUS CELL CARCINOMA

Clinical Features

ESCC is a malignant epithelial tumor of the esophagus with squamous cell differentiation. The etiology of ESCC is multifactorial and has been shown to be strongly population-dependent.[6] In the United States, the 2 most significant risk factors are tobacco use and excessive alcohol consumption, which together have a synergistic effect. Certain medical conditions, including Fanconi anemia, lye strictures, Plummer-Vinson syndrome, Zenker diverticulum, tylosis, achalasia, and prior therapeutic radiation to the chest, also place patients at higher risk for ESCC.[10]

ESCC has a male predominance and is 5 times more common among African Americans than whites.[10] The median age of presentation is within the seventh decade of life. Patients tend to be asymptomatic early in the disease, but, by the time of presentation, 80% to 90% of patients endorse progressive dysphagia (Hofstetter, 2019).[10] Weight loss results from decreased oral intake and is associated with worse clinical outcomes.[10] Other symptoms include odynophagia, emesis, cough, chest pain, and anemia.

Local tumor extension can manifest as hoarseness secondary to recurrent laryngeal nerve injury or, occasionally, as an esophagorespiratory fistula. Rarely, ESCC patients may demonstrate hypercalcemia secondary to tumoral production of parathyroid hormone–related protein. Distant metastases may be seen in up to 20% to 30% of patients at presentation.[11] The most common locations of distant metastases include the lungs, liver, bones, and brain. Synchronous or metachronous head/neck SCCs are present in 3% to 10% of patients, which are thought to be related to smoking.[12]

Pathologic Reatures

ESCC develops through a stepwise progression from histologically normal squamous mucosa to squamous cell dysplasia and finally to invasive squamous cell carcinoma. Squamous cell dysplasia (or intraepithelial neoplasia) is considered a histologic precursor and is characterized by cellular atypia, abnormal differentiation, and disorganized architecture.[12,13] Not only is intraepithelial neoplasia found adjacent to invasive ESCC in a majority of cases, but also its presence at biopsy places a patient at significantly increased risk for the future development of ESCC.

With neoplastic cell invasion through the basement membrane, a lesion is considered invasive ESCC. Defining tumor depth of invasion on a microscopic level is an important prognostic factor. As tumor invasion becomes progressively deeper, there is a concomitant increase in frequency of lymph node metastases.[12] The frequency of lymph node metastases nearly doubles as a tumor invades into each progressively deeper one-third of the submucosal layer.[14]

Imaging Features

Multimodality imaging plays a crucial role in the clinical staging of ESCC and treatment planning (**Figs. 1** and **2**). Clinical staging is defined by the American Joint Committee on Cancer staging system using TNM subclassifications, which is currently in its eighth edition, for esophageal and esophagogastric carcinomas.[15] Tumor location no longer is considered a factor in determining clinical staging but should be noted, because it determines the expected location of potential regional lymph node metastases and can have an impact on surgical approach. With imaging and endoscopy, the tumor's epicenter rather than its upper edge is described. A majority of ESCCs are located within the mid-esophagus followed by the lower esophagus.

Traditionally, barium esophagography was considered part of the work-up for esophageal carcinomas. This examination no longer is considered routine but occasionally may be performed prior to endoscopies, acting as a road map. On double-contrast esophagrams, superficial tumors appear as small protruded lesions, manifesting as plaque-like lesions with or without central ulcers, sessile polyps, and/or focal irregularities of the esophageal wall.[16] The more commonly seen advanced tumors are infiltrating, manifesting as irregular luminal narrowing with nodularity and/or ulceration and abrupt shouldering margins.[16,17] Morphology on barium studies typically matches those on endoscopy and pathology,[14] and, although not specific, occasionally can suggest depth of invasion and risk of lymph node metastases.[18]

Computed tomography (CT) is an important modality in the work-up of esophageal cancers. Primary tumors are detected on CT through evaluation of esophageal wall thickness, which is considered abnormal if greater than 5 mm.[15] ESCC typically manifests as esophageal wall thickening or a mass that causes luminal obstruction if advanced. Earlier tumors may be difficult to detect but occasionally can present as asymmetric thickening of the esophageal wall. Beyond displaying

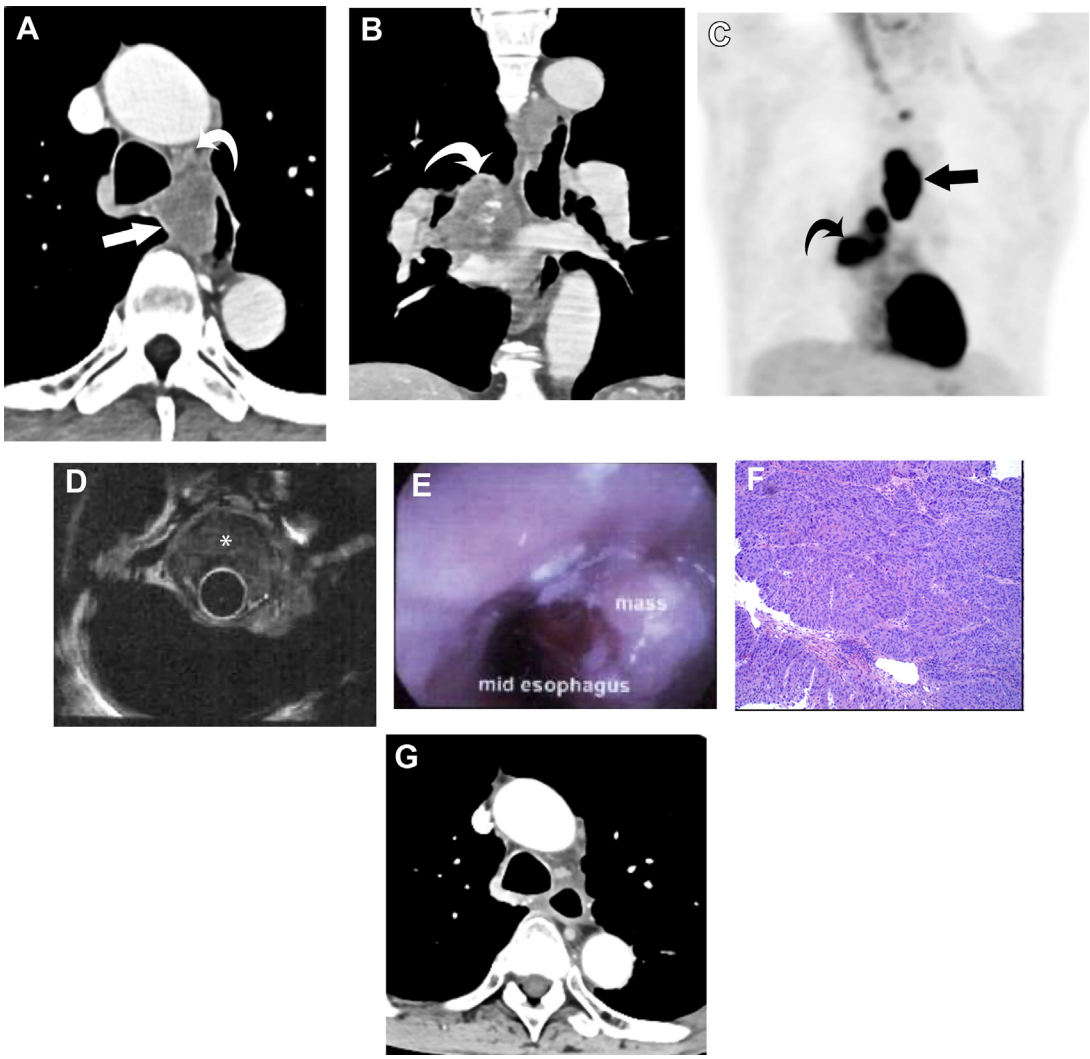

Fig. 1. A 67-year-old man with ESCC. (*A*) Contrast-enhanced axial CT of the chest at initial staging shows a large hypoattenuating mass in the right anterolateral esophageal wall in the mid-esophagus (*straight arrow*) and small left paratracheal lymph node adjacent to the mass (*curved arrow*). (*B*) Coronal reformatted contrast-enhanced CT of the chest shows subcarinal lymphadenopathy (*curved arrow*) compatible with locoregional spread. (*C*) Maximum intensity projection (MIP) image from the initial FDG PET/CT shows a hypermetabolic mid-esophageal mass with a SUVmax of 9.0 (*straight arrow*) and moderately hypermetabolic locoregional disease (*curved arrow*). (*D*) EUS shows a large hypoechoic mass in the mid-esophagus that extends through the muscularis propria without invasion into adjacent organs (*asterisk*), compatible with T3 staging. (*E*) EGD demonstrates a near-circumferential mid-esophageal mass, which appears polypoid and contains ulcerated, friable surfaces. (*F*) Photomicrograph (original magnification, ×4; hematoxylin-eosin stain) of a section from the patient's biopsy shows an infiltrative tumor composed of cohesive nests of tumor cells consistent with invasive keratinizing squamous cell carcinoma arising in a background of high-grade squamous dysplasia. (*G*) Contrast-enhanced axial CT of the chest following neoadjuvant chemoradiation illustrates a good response to treatment with near resolution of the mid-esophageal mass and interval decrease in lymphadenopathy.

the primary tumor, CT is unable to illuminate the depth of tumor invasion, particularly in early cancers, significantly limiting its usefulness in T staging.[11] Despite this limitation, CT does have some utility in the assessment of T4 lesions. In general, local invasion is suggested with a loss of fat planes between the tumor and adjacent structures in the mediastinum. For example, if the tumor and aorta have an interface greater than 90°, this can suggest aortic invasion.[19] CT has a moderate sensitivity for detection of locoregional disease and even higher for distant metastases.[10,11] CT evaluation of nodal

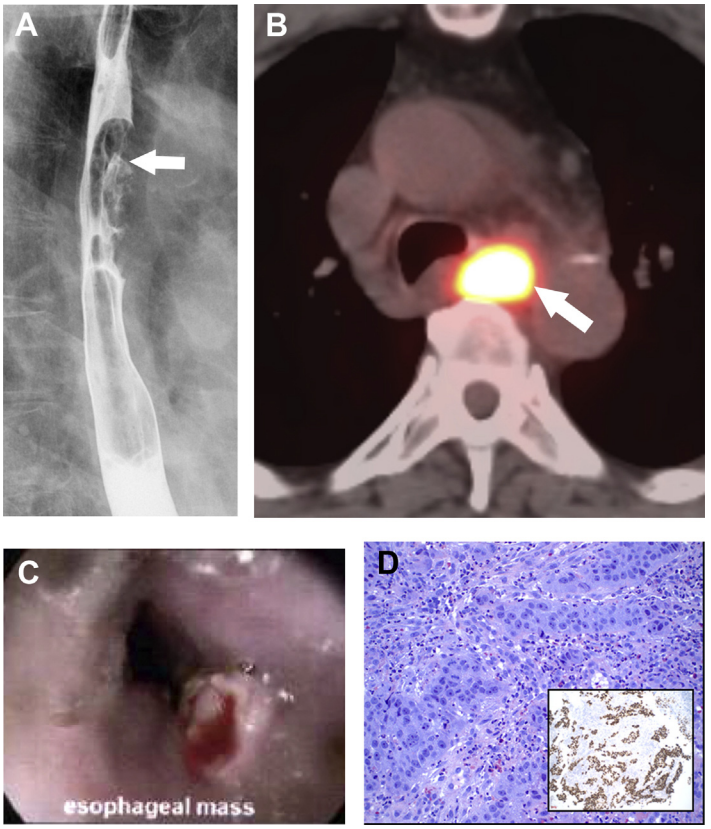

Fig. 2. A 57-year-old man with a history of HIV (undetectable viral load) and ESCC. (*A*) Right anterior oblique image from an esophagram shows an infiltrating mass (*arrow*) that has acute, shouldering margins with ulceration and moderate lumen compression. (*B*) Fused axial FDG PET/CT image shows hypermetabolic (SUVmax of 24.9) circumferential thickening of the mid-esophagus (*arrow*). (*C*) Esophagogastroduodenoscopy (EGD) shows a 2.5-cm friable, oozing polypoid mass with ulceration in the mid-esophagus. (*D*) Photomicrograph (original magnification, ×10; hematoxylin-eosin stain) shows markedly atypical cells with amphophilic cytoplasm and no apparent keratin formation, high nuclear-to-cytoplasmic ratio, prominent nucleoli, and anisonucleosis. Inset image shows a positive immunohistochemical stain for p40 supporting squamous differentiation.

disease is based primarily on size criteria, which characterizes a lymph node as pathologic if it measures greater than 1 cm in short-axis in the presence of known esophageal malignancy.[20] This is, however, neither specific or sensitive and is less accurate than endoscopic ultrasound (EUS).

Fluorodeoxyglucose (FDG) PET/CT can delineate the location of the primary tumor, because most ESCCs are FDG-avid. Similar to CT, however, this imaging modality has little role in T staging, because it is unable to differentiate among the different layers of the esophageal wall. It can less commonly delineate locoregional disease[21] but is inferior to EUS. FDG PET/CT's true importance lies in its ability to detect distant metastases and recurrence. It has been shown to be far superior in the detection of distant metastases compared with CT and EUS.[22] Additionally, FDG PET/CT is critical in evaluating treatment response following neoadjuvant chemoradiation, which has been shown to provide prognostic information regarding survival.[23,24] For example, patients whose FDG PET/CT scans demonstrated maximum standardized uptake values that decreased by greater than 50% compared with their pretreatment scans had a longer overall survival and decreased risk of death following surgery.[25]

EUS currently is the most accurate tool available in determining depth of tumor invasion (T stage) because it can directly visualize all of the layers of the esophageal wall. ESCC presents as a hypoechoic mass that, depending on its depth of invasion, obscures 1 or more of the 5 alternating echogenic and hypoechoic lines that represent the normal layers of the esophageal wall. EUS also is the most accurate means for determining N staging, with a reported accuracy rate of 72% to 80% (compared with the 46%–58% accuracy of CT).[10,11] In contrast, EUS has limited value in the assessment of distant metastases and generally is not recommended for treatment response assessment.[25]

Management

Because a majority of patients present in advanced stages of the disease, the prognosis for ESCC is poor, with a 5-year survival rate of less than 17%.[26] A multidisciplinary approach is considered the mainstay of treatment of ESCC. Esophagectomy alone is recommended for T1-

T2 cancers or patients who cannot tolerate combined modality therapy. Endoscopic techniques, including endoscopic mucosal resection and ablation therapy, can be considered as esophageal-preserving alternatives to surgery in patients with T1a lesions.[24] Patients with T1b lesions generally are not considered candidates for these approaches due to their increased risk of lymph node involvement. That said, more recently, an increasing number of select patients with superficial submucosal invasion T1b lesions with additional favorable features are being treated with endoscopic mucosal resection with good outcomes.[24]

For locally advanced cancers, multimodality therapy is the standard of care and consists of neoadjuvant chemoradiation followed by restaging and consideration for esophagectomy.[25] Adjuvant chemoradiation may benefit some patients with ESCC, particularly if a patient has not previously received neoadjuvant chemoradiation.[10]

Definitive chemoradiation or, less preferably, radiation therapy without subsequent surgery is a possibility for patients who either opt out of or are not candidates for surgery; this approach has been used increasingly to treat ESCC in the cervical and proximal esophagus.[10] Compared with patients who subsequently underwent esophagectomy, those who solely receive definitive chemoradiation have worse locoregional control, but overall survival does not appear to be significantly different between these groups.[25] A palliative approach generally is pursued for patients with T4b tumors or distant metastases, which can include palliative chemoradiation, surgical debulking, and/or esophageal stenting for better symptom control.[24]

ESOPHAGEAL ADENOCARCINOMA

Clinical Features

EAC is a malignant epithelial tumor of the esophagus with glandular or mucinous differentiation that typically arises within Barrett esophagus in the lower one-third of the esophagus. It is strongly tied to chronic GERD and the development of Barrett esophagus. The latter is diagnosed in 1% to 2% of the general population and its annual incidence of malignant transformation to EAC is reported to be 0.2% to 0.5% per year.[10,26] Other risk factors in the development of EAC include tobacco smoking and obesity.

Similar to ESCC, EAC demonstrates a slight male predominance and a mean age at presentation within the seventh decade of life. Unlike ESCC, EAC is more common among whites.[6] The clinical presentation generally mirrors that for ESCC. It is, however, more common for patients with EAC to endorse chronic reflux symptoms.[6] Furthermore, patients with EAC are more likely to demonstrate intra-abdominal metastases in contrast to ESCC, which more commonly metastasizes to intrathoracic or cervical sites.[26] The prognosis for EAC is variable but generally tends to be poor, albeit slightly more favorable than that for ESCC.[27]

Pathologic Features

Grossly, EACs can be described using the same classification system as ESCCs. EAC lesions tend to be infiltrating, polypoid, or ulcerative. If EAC has developed in a background of Barrett esophagus, this can be visualized on endoscopy as reddish mucosa in the distal esophagus compared with the pale grayish color of the normal esophageal squamous cell epithelium.[28,29]

EAC develops through a multistep pathway characterized by a Barrett metaplasia–dysplasia–adenocarcinoma sequence.[29] Less commonly, it develops within heterotopic gastric mucosa or from esophageal mucosal/submucosal glands.[12] Although other types of columnar metaplasia are possible, it is the presence of intestinal metaplasia with goblet cells that specifically defines Barrett esophagus in the United States.[28]

Similar to ESCC, invasive EAC is defined as the invasion of neoplastic cells through the basement membrane. Microscopically, invasive EAC is characterized by mucinous or gland differentiation.[29] The percentage of glandular formation in the carcinoma forms the basis for EAC grading.[28]

Imaging Features

Similar to ESCC, multimodality imaging is central to the work-up for EAC (**Fig. 3**). EACs appear similar to ESCCs on each of the imaging modalities. One feature that can suggest one lesion over the other is its location. EAC tends to occur mainly in the distal esophagus and, unlike ESCC, it has a strong predilection for invading the gastric cardia and/or fundus.[7] Another difference is that EACs are more likely to have adjacent strictures secondary to the presence of chronic GERD, a finding that commonly is appreciated on barium esophagrams.[17] Patients with EACs also are more likely to have intra-abdominal metastases, as discussed previously, which tend to be well visualized on FDG PET/CT and CT. Finally, EACs generally are less FDG-avid than ESCCs and tend to be more heterogeneous in their avidity. As was reported for gastric adenocarcinomas, EACs that are poorly differentiated or demonstrate diffuse-type, signet cell, or mucinous patterns

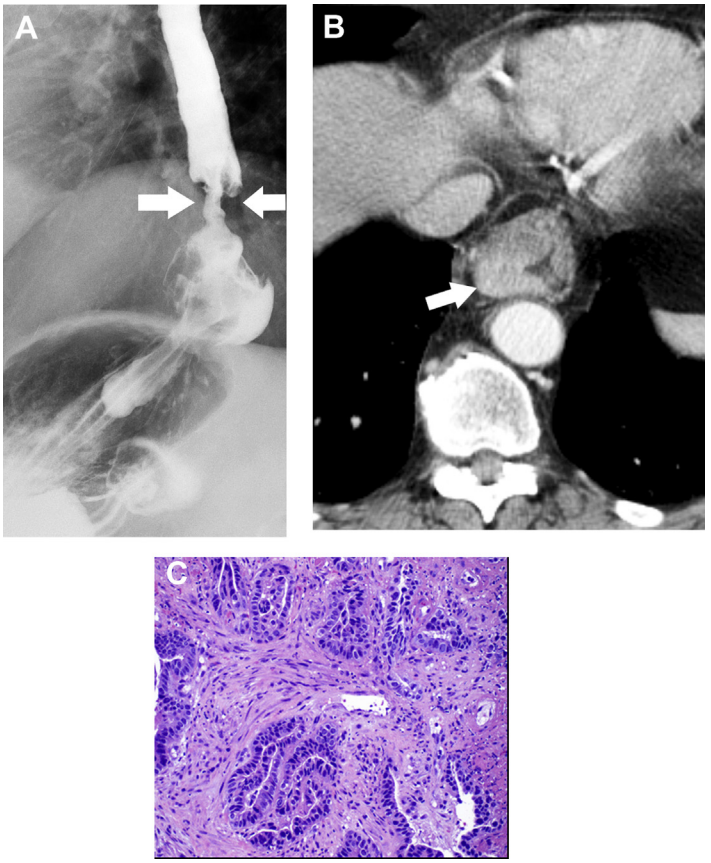

Fig. 3. A 78-year-old man with a history of GERD and EAC. (*A*) Oblique image from an esophagram shows an irregular infiltrating mass in the distal esophagus with circumferential stenosis of the lumen and shouldering margins (*arrows*). (*B*) Contrast-enhanced axial CT of the chest at initial staging demonstrates a hypoattenuating mass in the right anterolateral wall of the distal esophagus (*arrow*). (*C*) Photomicrograph (original magnification, ×10; hematoxylin-eosin stain) shows atypical cells lining irregular and incomplete glandular structures, single infiltrating cells, and desmoplastic stromal reaction.

tend to be on average less FDG-avid (Stahl and colleagues, 2008).[30] In general, however, imaging cannot reliably differentiate EAC from ESCC.

Management

EAC is managed similarly to ESCC with minor differences. For example, because EAC tends to be less radiosensitive compared with ESCC, there is less of a benefit of combined neoadjuvant chemoradiation compared with neoadjuvant chemotherapy alone.[24] Thus, the latter may be considered in patients with EAC. Furthermore, trastuzumab, a humanized monoclonal antibody that targets the human epidermal growth factor receptor family member ERBB2 (HER2), is approved for the treatment of patients with advanced or metastatic HER2-positive EAC. Trastuzumab plus chemotherapy has been shown to improve overall survival in these patients.[31]

LEIOMYOMAS

Leiomyomas are the most common benign tumors of the esophagus, although they occur approximately 50 times less frequently than esophageal carcinoma.[32] In contrast to carcinoma, most patients with leiomyomas are asymptomatic, especially patients with tumors less than 5 cm in size.[32] Tumors commonly are detected incidentally on imaging or endoscopy performed for other reasons.

Pathologic Features

Leiomyomas are neoplasms of mature smooth muscle cells and appear grossly as firm white smoothly marginated masses, with a whorled appearance on cut surface. Tumors almost always are intramural in location, arising from the muscularis mucosae or muscularis propria layers.[32] Microscopically, tumors are composed of bundles of well-differentiated spindled smooth muscle cells.[33] Cells contain abundant eosinophilic neoplasm and are arranged in an interlacing or palisading pattern.[32] Cells have a bland appearance, without mitotic activity or nuclear pleomorphism.[8] On immunohistochemical analysis, lesional cells typically are positive for SMA and desmin, but, unlike gastrointestinal stromal tumors (GISTs), lack affinity for KIT (CD117), CD34, and DOG1.

Imaging Features

On imaging, esophageal leiomyomas most commonly are an intramural, submucosal mass in the mid to lower esophagus, the portions of the esophagus lined by smooth muscle, and which may distort the azygoesophageal edge (Fig. 4). Barium swallow characteristically demonstrates a smooth-surfaced, crescent-shaped filling defect, which forms obtuse angles with the esophageal wall.[16] These masses may be isoattenuating or hypoattenuating to muscle on unenhanced CT, and they commonly are slightly hyperintense on T2-weighted MR imaging.[8] Homogeneous enhancement, without necrosis, is seen most frequently.[34] Leiomyomas may demonstrate coarse calcification, a feature that helps differentiate them from other benign and malignant esophageal tumors, in which calcification is uncommon.[35,36] FDG PET/CT usually is negative in patients with leiomyomas, attributable to the lack of mitotic activity.[8]

Management

No cases of sarcomatous degeneration of leiomyoma to leiomyosarcoma have been reported to date.[7] Therefore, surgical removal of small leiomyomas in asymptomatic patients is not performed commonly. Symptomatic lesions, however, may necessitate surgical resection. Although complete resection of a small leiomyosarcoma often can be accomplished through local excision, larger tumors may require a total or partial esophagectomy.[37]

SARCOMAS

Although sarcomas are uncommon in the esophagus, leiomyosarcoma is the most common type.[8] These tumors are characterized by slow growth and late metastases and are thought to arise de novo rather than from preexisting leiomyomas.[38] Liposarcomas rarely are reported in the esophagus.[39]

Pathologic Features

On gross examination, leiomyosarcomas arise in the wall of the esophagus and appear as polypoid or lobulated intraluminal tumors, which may feature ulceration.[40] Microscopically, these are cellular tumors, with long intersecting fascicles of spindle cells. Tumor cells contain ample eosinophilic cytoplasm and pleomorphic enlarged nuclei. Increased mitotic activity is common in gastrointestinal leiomyosarcomas, and necrosis may be present.[40]

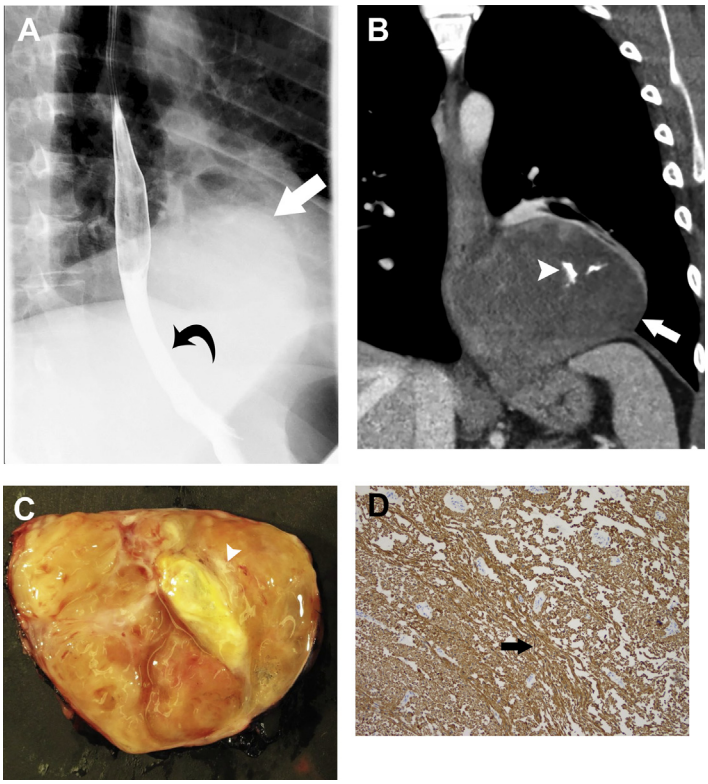

Fig. 4. A 42-year-old man with leiomyoma of the esophagus. (*A*) Coned-down image from an esophagram shows the contour of a mass (*straight arrow*) at the left aspect of the esophagus with smooth mass effect on the left aspect of the esophagus (*curved arrow*). (*B*) Coronal reformatted contrast-enhanced CT of the chest shows a homogeneous hypoattenuating mass (*arrow*) contiguous with the esophagus containing internal focus of calcification (*arrowhead*). (*C*) Sectioned gross specimen shows a gelatinous tan-red cut surface with partially calcified (*arrowhead*) necrosis. (*D*) Photomicrograph (original magnification, ×40; desmin immunohistochemical stain) shows spindle cells (*arrow*) uniformly positive.

Adipose tissue and fibrous septa are characteristic of liposarcoma of the esophagus, and some liposarcomas may be grossly pedunculated and mimic a fibrovascular polyp histologically, differentiated by irregular spindle cells and MDM2 amplification in liposarcomas.[39]

Imaging Features

On CT, these malignant tumors are heterogeneous and feature central areas of necrosis. Large exophytic components may cause mass effect on the trachea or other nearby structures. Ulceration may allow extraluminal gas or contrast material to track into the tumor.[41] On MR imaging, these lesions typically are isointense with skeletal muscle on T1-weighted images and hyperintense on T2-weighted images. A central signal void may be present, as the result of extraluminal gas within the tumor.[41] Fat attenuation on CT or chemical fat signal on MR imaging may suggest liposarcoma (Fig. 5).

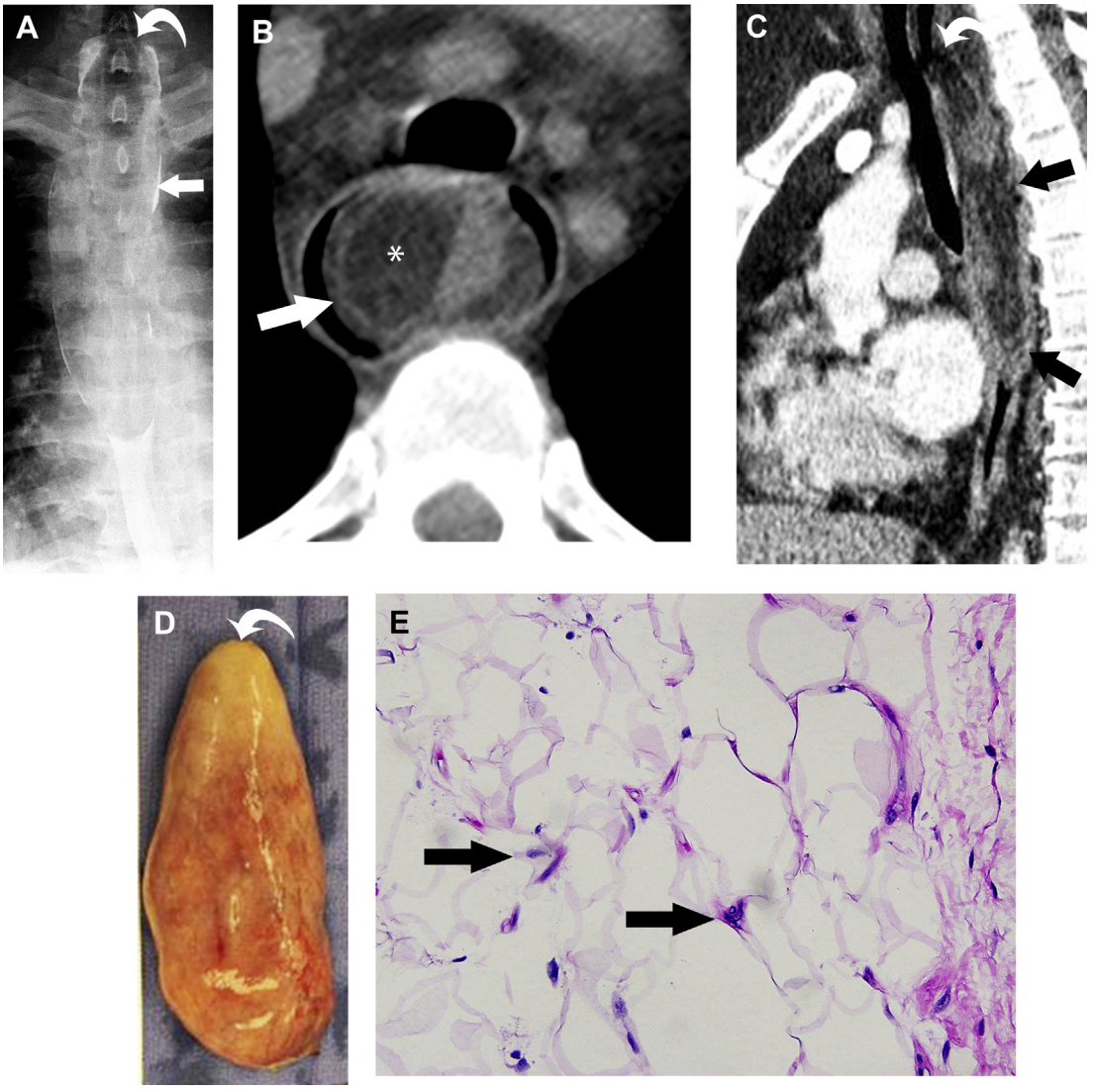

Fig. 5. A 39-year-old man with liposarcoma of the esophagus. (*A*) Coned-down image from an esophagram shows a mass (*straight arrow*) filling the esophagus with stalk-like attachment at the cervical esophagus (*curved arrow*). (*B*) Contrast-enhanced axial CT of the chest shows the esophageal mass (*arrow*) with extensive fat attenuation component (*asterisk*) and soft tissue nodularity. (*C*) Sagittal reformatted contrast-enhanced CT of the chest image shows a polypoid esophageal mass (*arrows*) with stalk-like attachment at the upper esophagus (*curved arrow*). (*D*) Gross specimen shows an elongated, encapsulated esophageal mass (*curved arrow*) with macroscopic fat most notably at the superior aspect near its attachment to the cervical esophagus. (*E*) Photomicrograph (hematoxylin-eosin, original magnification ×40) shows mild atypia of adipocytes with enlarged, hyperchromic nuclei (*arrows*).

Management

Five-year survival rates of approximately 30% to 40% are reported in patients with surgically resected leiomyosarcoma, and survival is influenced strongly by tumor differentiation and size.[42] Tumors eventually may spread by direct extension to the pleura, pericardium, diaphragms, and stomach or metastasize hematogenously to the liver, lungs, and bones.[7]

GASTROINTESTINAL STROMAL TUMORS

Until recently, almost all mesenchymal neoplasms arising from the esophagus were thought to be benign esophageal leiomyomas.[7] It is now known that another stromal tumor, GISTs, also may occur in the esophagus. Differentiation is important, because of the known malignant potential of GISTs, which warrants consideration for surgical resection or treatment with tyrosine kinase inhibitors, such as imatinib.[43] Although GISTs can occur anywhere in the gastrointestinal tract, most commonly in the stomach, approximately 1% arise in the esophagus.[44] When occurring in the esophagus, these tumors most commonly involve the lower third.

Pathologic Features

Similar to GISTs elsewhere, small tumors often are intramural and arise in the muscularis propria, whereas larger tumors may exhibit exophytic growth and areas of necrosis.[36] Immunohistochemical analysis often is required to differentiate esophageal leiomyomas from GISTs, because both may demonstrate spindle cells and calcification on histology. Positivity for KIT (CD117), DOG1, and CD34 are more consistent with GIST.[8]

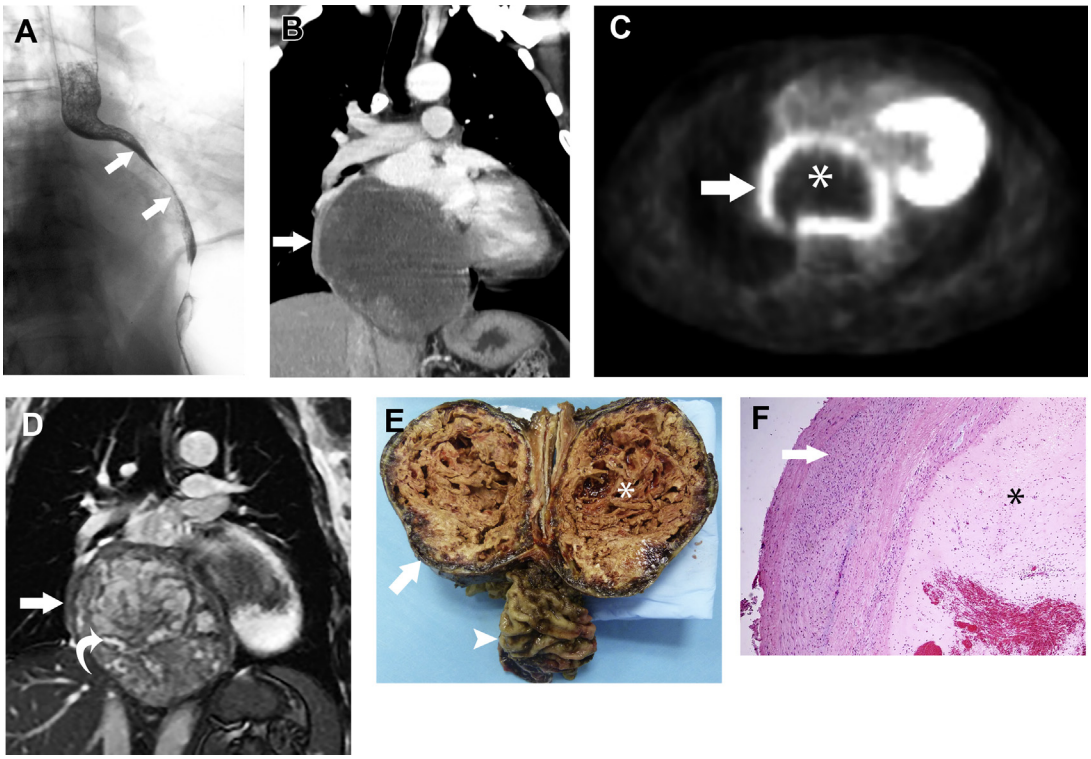

Fig. 6. A 72-year-old man with GIST of the esophagus. (*A*) Coned-down image from an esophagram shows leftward deviation (*arrows*) of the esophagus without mucosal ulceration, characteristic of mesenchymal tumors of the esophagus. (*B*) Coronal reformatted contrast-enhanced CT of the chest shows the mass (*arrow*) with peripheral enhancement and central hypoattenuation indicating necrosis. (*C*) Axial FDG PET shows the mass (*arrow*) with peripheral uptake and no central uptake (*asterisk*), indicating central necrosis. (*D*) Coronal T2-weighted MR image shows the mass (*straight arrow*) with central heterogeneous and hyperintense regions (*curved arrow*) indicating necrosis or hemorrhage products. (*E*) Esophagogastrectomy specimen transected in the coronal plane show the mass (*arrow*) arising from the esophagus and extending to the origin of the stomach (*arrowhead*). Note the extensive intratumoral hemorrhage and necrosis (*asterisk*). (*F*) Photomicrograph (original magnification, ×4; hematoxylin-eosin stain) shows peripheral epithelioid and spindle cells (*arrow*) characteristic of GIST with extensive necrosis and hemorrhage (*asterisk*) centrally.

Imaging Features

Esophageal GISTs have a similar clinical, endoscopic, and imaging appearance to leiomyomas. Features that favor GIST over leiomyoma include more distal location, larger size, more heterogeneous appearance, greater enhancement on contrast-enhanced CT, and marked avidity on FDG PET/CT (**Fig. 6**).[43] In some cases, EUS-guided fine-needle aspiration for immunohistochemical staining may be required for differentiation. Imaging appearance also may be similar to sarcomas, with areas of necrosis and calcification resulting in heterogeneous attenuation.[35]

FIBROVASCULAR POLYPS

Fibrovascular polyps include a variety of lesions, which are differentiated by the predominant

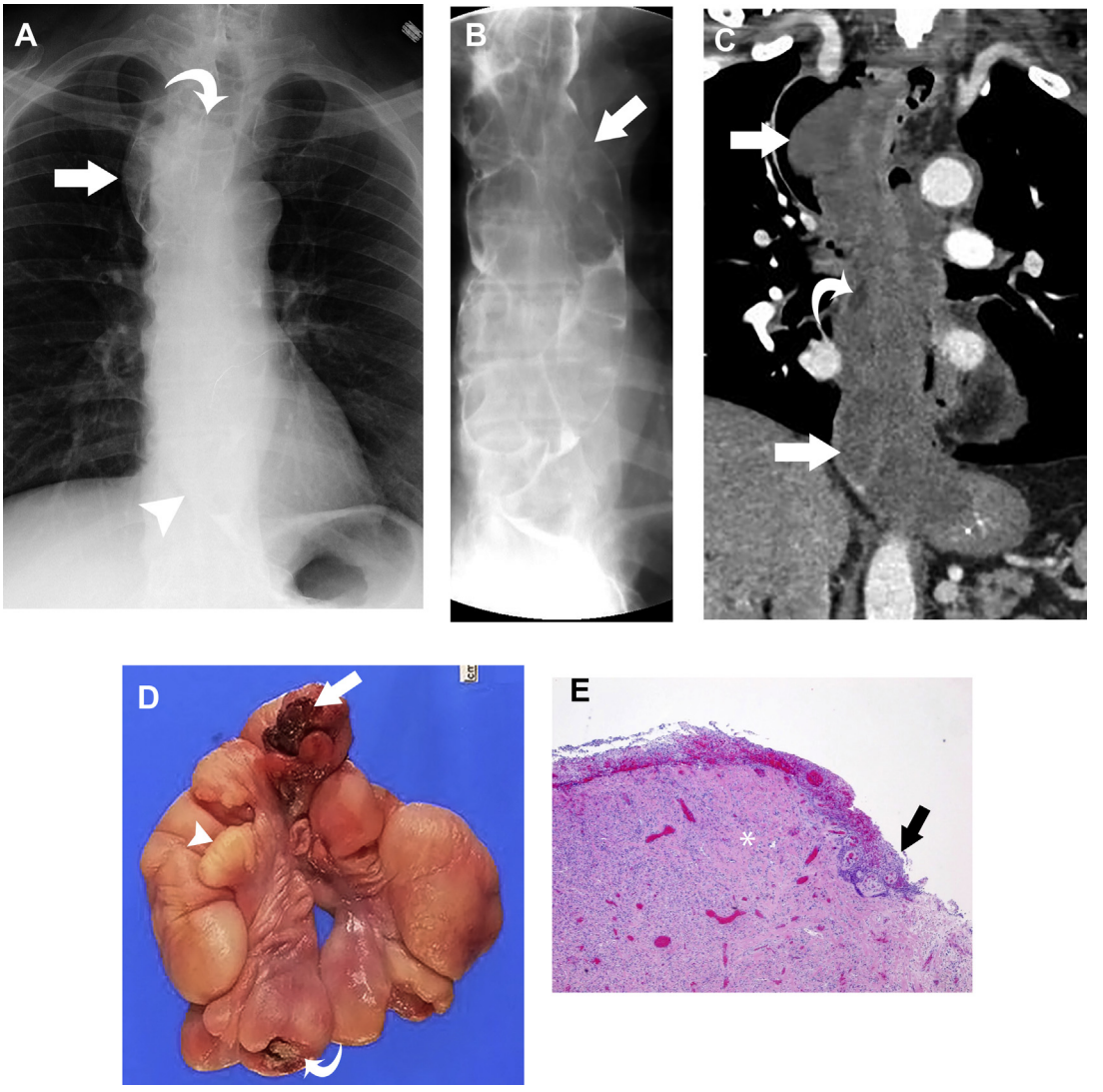

Fig. 7. A 59-year-old man with a fibrovascular polyp of the esophagus. (*A*) Frontal chest radiograph shows opacity at the right paratracheal stripe (*straight arrow*), an abnormal air-soft tissue interface at the upper mediastinum (*curved arrow*), and an abnormal edge near the gastroesophageal junction (*arrowhead*), indicating esophageal pathology. (*B*) Coned-down image from an esophagram shows a polypoid mass (*arrow*) filling the esophagus and outlined in barium without mucosal abnormality. (*C*) Coronal reformatted contrast-enhanced CT of the chest shows the polypoid mass (*straight arrows*) filling and distending the esophagus with a small focus of fat attenuation (*curved arrow*). (*D*) Gross specimen shows the cut stalk (*straight arrow*), regions of ulceration (*curved arrow*), and yellow tissue (*arrowhead*) consistent with fat. (*E*) Photomicrograph (original magnification, ×40; hematoxylin-eosin stain) shows loose fibromyxoid stroma (*asterisk*) with spindle cells, and small thin-walled blood vessels. Focal mucosal ulceration (*arrow*) with granulation tissue also is present.

mesenchymal component histologically. These include fibromas, lipomas, fibrolipomas, fibromyxomas, and fibroepithelial polyps. These tumors contain a mixture of fibrous, vascular, and adipose tissue, which are covered by squamous epithelium. They are found most commonly as pedunculated intraluminal masses in the cervical esophagus, attached by a pedicle near the level of the cricopharyngeus.[8] Most of these polyps are 7 cm or longer at the time of presentation, and they can extend as far as 20 cm into the distal esophagus, occasionally traversing the gastroesophageal junction to enter the gastric fundus.[45] It has been theorized that these tumors gradually elongate over a period of years as the result of esophageal peristalsis pulling on them.[46] Gross pathology demonstrates a white myxoid appearance mixed with yellow adipose tissue, with microscopic examination revealing varying amounts of adipose tissue and loose or dense fibrovascular tissue, covered by normal squamous epithelium. Their cross-sectional appearance largely depends on the proportions of fat and fibrous tissue. A heterogeneous appearance from areas of fat attenuation, hyperechogenicity, or high T1 signal from adipose tissue, mixed with areas of soft tissue attenuation, hypoechogenicity, or low T1 signal from fibrovascular tissue, on CT, ultrasound, and MR imaging, respectively, is most common (**Fig. 7**).[47] Although malignant degeneration is thought to be extremely rare, removal is recommended due to their progressive and eventually debilitating nature.[7]

SUMMARY

The imaging appearance of esophageal neoplasms and tumor-like conditions, such as fibrovascular polyps, is driven by the pathology of these tumors. The locations of tumors within the esophageal wall, along the length of the esophagus, and relative to the lumen of the esophagus are key reporting elements at initial imaging diagnosis. Although pathologic diagnosis is necessary in almost all cases, a modern clinical imager must be aware of what pathologic and imaging features drive clinical management and prognosis.

DISCLOSURE

J.P. Lichtenberger—Author, Elsevier. A.R. Dulberger—The opinions and assertions contained herein are the private views of the authors and are not to be construed as official nor as representing the views of the Departments of the Army, Navy, Air Force, or Defense.

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