Endoscopy and Endoscopic Ultrasound for Esophageal Cancer

Stephen Gowing

Abstract

Esophageal cancer is a serious malignancy and cause of cancer death worldwide with an ever-increasing incidence. The initial management and staging of esophageal cancer are crucial to determine optimal treatment and potential for cure. Optical endoscopy and endoscopic ultrasound (EUS) are key components of this, enabling tissue diagnosis, tumor localization, tumor characterization, and locoregional staging. Endoscopy is also employed in the treatment of dysplastic Barrett's esophagus and the resection of early-stage esophageal malignancies. Additionally, endoscopy can be utilized as a bridge for enteral nutrition as well as for palliation for unresectable disease.

Keywords

Esophageal cancer · Endoscopy · Endoscopic ultrasound · Barrett's esophagus · Endoscopic submucosal resection

Introduction

Esophageal cancer remains one of the major causes of cancer death worldwide and its incidence is continuing to increase. While squamous cell carcinoma (SCC) remains a common diagnosis in Asia, in North America and Europe esophageal adenocarcinoma (EAC) remains the most common presentation [1]. Treatment of this aggressive cancer in its most common presentation requires multimodality treatments including chemotherapy, potential radiation therapy and esophagectomy. Tissue diagnosis, tumor location and clinical stage are paramount in determining potential therapies and often rely on an initial upper gastrointestinal endoscopy.

Initial Endoscopic Assessment for Esophageal Malignancy

The initial diagnosis and treatment of esophageal cancer most often begins with a traditional optical endoscopic assessment of the esophagus in response to patient clinical concerns such as dysphagia, odynophagia, esophageal stasis, regurgitation, weight loss, anemia, gastrointestinal bleeding, esophageal food bolus impaction or symptoms as benign as gastroesophageal reflux disease. Alternatively, upper gastrointestinal contrast swallow examination under fluoroscopy or computed tomography (CT) scanning

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may point towards this diagnosis and direct the clinician to perform endoscopy.

Initial assessment with an optical gastroscope begins with examining the entirety of the upper gastrointestinal tract from the upper esophageal sphincter/cricopharyngeal muscle passing into the esophagus, stomach and ending in the second or third stage of the duodenum. Retroflexion is performed in the stomach to assess the gastrointestinal junction for proximal gastric or distal esophageal tumors as well as hiatus hernia. Care must be taken on withdrawing the endoscope to examine for the presence of Zenker's diverticulum and the rare potential for esophageal cancer within.

Specific endoscopic findings alert the clinician to the potential for esophageal malignancy and will direct further sampling or investigations. These include but are not necessarily limited to: Barrett's esophagus, endoluminal nodules or masses, and mucosal or submucosal strictures that may prevent or hinder passage of the endoscope requiring esophageal dilation or stenting. Submucosal bulging or masses can alert to metastatic adenopathy or submucosal tumors to be further assessed with endoscopic ultrasound (EUS) or cross-sectional imaging.

When esophageal endoluminal masses suggestive of cancer are identified, the proximal and distal aspect of the lesion from the incisors are noted as well as the circumferential extent of the lesion. For lesions present close to the gastroesophageal junction (GEJ) the Siewert-Stein classification of GEJ adenocarcinomas (commonly denoted as Siewert) is additionally noted as follows:

- Siewert 1: epicentre 1–5 cm above the GEJ
- Siewert 2: epicenter up to 1 cm above and 2 cm below the GEJ
- Siewert 3: epicenter 2–5 cm below the GEJ

Siewert 3 lesions are most often referred to as gastric cancers and may treated with esophagectomy or total gastrectomy depending on patient and tumor characteristics [2].

High definition white light endoscopy (WLE) with targeted forceps biopsy remains the standard approach for diagnosis of mucosal based esophageal malignancies. Care must be taken to sample tissue from multiple areas of tumor to avoid non-diagnostic results in the event of necrotic tumor specimen. EUS linear fine needle aspiration (FNA) or core needle biopsy can be helpful as adjunct in assisting with tissue diagnosis sampling nodal metastases or through biopsying submucosal lesions. Tunneling deep endoscopic forceps biopsy in a bite-on-bite fashion with endoscopic clip mucosal closure remains an additional biopsy technique for submucosal lesions when linear EUS is not available or possible [3].

Barrett's Esophagus, Dysplasia and Early-Stage Esophageal Cancers

Barrett's esophagus (BE) (also known as intestinal metaplasia of the esophagus) is the conversion of the pale salmon pink squamous esophagus mucosa to a reddish columnar epithelium in response to chronic acid exposure to the esophagus. Unfortunately, these changes are known to predispose patients to the development of dysplastic Barrett's epithelium and eventually EAC. Thankfully, the incidence of progression of Barrett's epithelium to cancer remains low at a rate of 0.33% per year. However, the diagnosis of BE necessitates lifelong surveillance or the eradication of BE in event of the development of dysplasia or cancer [4]. Newer research has demonstrated that patients with higher aneuploidy (genomic copy number) in their Barrett's epithelium are at higher risk for progression to dysplasia and malignancy [5].

Barrett's esophagus is classified endoscopically according to the Prague Classification [6] denoting the circumferential extent (C) and the maximal extent (M) of BE. This measurement commences from the top of the gastric mucosal folds to denote the gastroesophageal junction. For example: Barrett's epithelium that is 4 cm in circumferential extent and 6 cm in maximal length (from non-circumferential tongues of Barrett's) would be classified as C4M6.

BE greater than or equal to 3 cm in length is referred to as long segment BE and has an increased risk of harboring dysplasia and malignancy. Short segment BE is consequently classified as less than 3 cm in maximal length [7].

High Definition WLE with 4 quadrant biopsies every one to two cm, according to the Seattle protocol [8], remains the standard method of assessment for dysplasia in Barrett's epithelium and for biopsy of endoluminal masses. Current recommendations for non-dysplastic BE surveillance are once every 3 years [9].

Optical Chromoendoscopy: Optical Chromoendoscopy works based on the principles of longer wavelengths of light such as red having deeper tissue penetration than shorter wavelengths of light such as green (540–560 nm) and blue (440–460 nm). Narrow-band imaging (NBI) utilizes green and blue wavelengths of light to improve visualization of mucosal patterns of capillaries and veins. NBI is easy to switch to and from WLE examination modes and does not require special staining for visualization. Mucosal bleeding, however, can quickly overwhelm NBI viewing models due to hemoglobin preferentially absorbing blue light [10]. Due to it's ease of use optical chromoendoscopy is strongly recommended to be combined with WLE and Seattle protocol biopsies for BE surveillance. Additionally, it is strongly beneficial for planning resection margins for endoscopic resection [9]. NBI classification schema exist for Barrett's esophagus indicating the presence of dysplasia and malignancy based on the presence or absence of mucosal pits and regularity/irregularity of vasculature. Similarly, classification schema exist for early esophageal squamous cell carcinoma regarding degree of vascular irregularity and presence/absence of vascular loop-like formations [11].

Chemical Chromoendoscopy: The addition of chemical washes to the esophagus and Barrett's epithelium can optically enhance malignant and dysplastic lesions to aid in their detection. Various washes and stains have been utilized in the detection of foregut malignancy including methylene blue (now replaced primarily by NBI techniques), crystal violet [12], Lugol's iodine and acetic acid [13].

The diagnosis of Barrett's esophagus as well as the detection of dysplasia and intramucosal adenocarcinoma in Barrett's epithelium are enhanced using a dilute acetic acid solution. A 2–3% acetic acid solution is sprayed onto the esophageal epithelium leading to acetowhitening of Barrett's epithelium. Combined with magnification endoscopy villous patterns of intestinal metaplasia characteristic of BE can be visualized versus the reticular mucosa characteristic of cardiac epithelium. With time the acetowhitening effect is lost and Barrett's epithelium returns to its characteristic reddish colour [14]. Importantly, dysplastic BE and intramucosal carcinoma demonstrates early loss of acetowhitening (LAW) compared to non-dysplastic BE resulting in the transient appearance of reddish lesions within whitened Barrett's epithelium allowing for the detection of dysplasia and malignancy [13].

For the detection of esophageal SCC, a dilute 2–3% Lugol's iodine solution can be used. Lugol's iodine stains normal mucosa brown or green–brown. Conditions that deplete cellular glycogen such as dysplasia and early squamous malignancy limit's Lugol's staining resulting in whitish appearing lesions. Lugol's staining is contraindicated in patients with iodine allergy [15].

Confocal Laser Endomicroscopy: Confocal laser endomicroscopy utilizes a catheter-based laser fluorescent probe allowing for microscopic real time imaging of live patient mucosal tissues. This technique allows for imaging for mucosal tissue only, unable to visualize the submucosa and deeper layers. Cellular architecture of squamous and Barrett's epithelium can be visualized allowing for the detection of mucosal dysplasia and malignancy. Currently, the widespread application of this emerging technology is limited by cost and expertise is only present in ultra-specialized centers [16].

Endoscopic Treatment of Dysplastic Barrett's Esophagus

In the event of the development of dysplastic BE, (either low-grade dysplasia (LGD) or high-grade dysplasia (HGD)) eradication of BE is recommended. An alternative to eradication of Barrett's remains intensive surveillance, elected for most commonly in cases of LGD where access to endoscopic eradication therapies such as radiofrequency ablation (RFA) are constrained by cost and reserved for HGD BE. For LGD BE, surveillance endoscopy once a year is commonly employed when endoscopic Barrett's eradication is not available [9].

Currently, BE with LGD or HGD is routinely treated with RFA. Any nodular Barrett's mucosa is excised by cap-based endoscopic mucosal resection (EMR) prior to RFA treatment to rule out invasive malignancy. Alternatively, short segment BE can also be treated with multiple EMRs, albeit with a higher rate of esophageal stricture post-resection [17]. Photodynamic therapy (PDT) and cryotherapy are alternative treatments for dysplastic BE. PDT has largely been abandoned due to high rates of esophageal stricture formation and the need for patients to take photosensitizing agents [18]. Cryotherapy appears to be less efficacious than RFA for eradicating dysplastic BE, however remains an alternative therapy when RFA fails or is contraindicated [19].

Extensive nodular HGD BE where suspicion of intramucosal carcinoma (IMC) is high is becoming more commonly treated with endoscopic submucosal dissection (ESD). With ESD the BE of concern is resected en bloc as a single specimen with dissection down to the level of the muscularis propria. This is accomplished through injection of a methylene blue saline solution into the submucosal space, thereby separating the mucosa from submucosa, allowing for resection via needle knife. Most commonly the high-risk areas of BE are excised with ESD followed by RFA treatment of the residual Barrett's epithelium [20, 21]. Circumferential ESD is performed in selected cases, often for salvage therapy, however significant stricture formation is to be expected and managed afterwards [22, 23]. Although less commonly performed today, esophagectomy can still be performed for operable patients with dysplastic BE where endoscopic management has failed or is not available.

Endoscopic Resection of Early-Stage Esophageal Cancer

Early-stage esophageal cancers localized to the mucosa and upper submucosal space can be treated with endoscopic resection. Once esophageal cancers invade the submucosal space, they access the esophageal lymphatics, and are at significantly higher risk for lymph node metastases [24]. EMR remains an option for small mucosal only lesions less than 2 cm, however for larger lesions ESD is favored due its ability to obtain en bloc resection as well as deeper resection margins than EMR. ESD is also superior for resection of lesions that span across the GEJ (Fig. 1). Piecemeal EMR for lesions larger than 2 cm can be performed, however caution must be taken due to the inability to determine accurate lateral margins, as well as higher rates of R1 resection [20].

Determination of the suitability of solid esophageal lesions for endoscopic resection is dependent on tumor factors, clinical staging, as well as patient factors including operability. Various tumor characteristics including tumor size, differentiation, presence of lymphovascular invasion and depth of invasion are key for determination of the risks of lymph node metastasis [24]. Submucosal invasion is divided into three levels (SM1, SM2 and SM3) with increasing risk of lymph node metastases the deeper the tumor invades [25]. For comparable lesions based on size, depth of invasion, and degree of differentiation, SCC is noted to have much higher rates of lymph node metastasis compared to adenocarcinoma [24, 26]. Expert analysis of ESD specimens by specialized pathologists is critical as for patients with elevated risk of lymph node metastasis based on final pathology endoscopic resection alone is likely not sufficient for cure of disease.

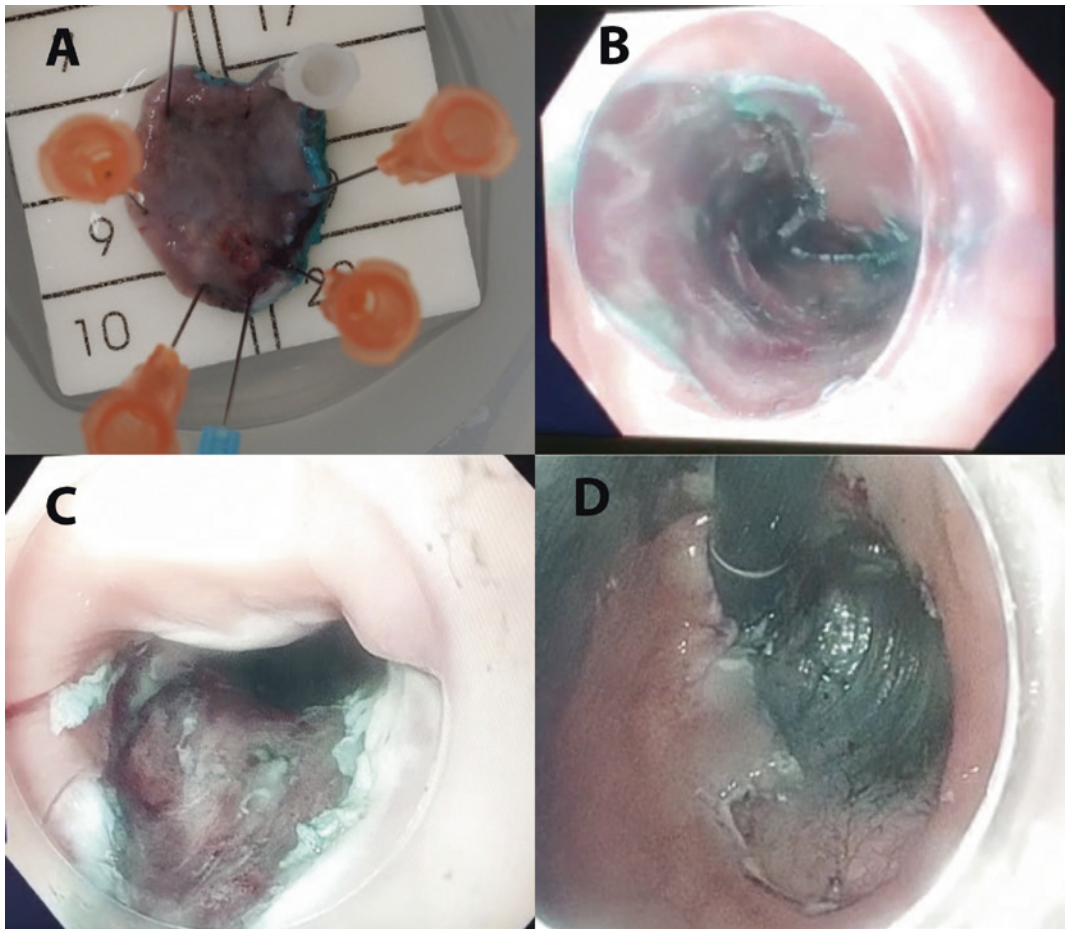

Fig. 1 Endoscopic Submucosal Dissection for Esophageal Cancer. (A, B) Pinned resection specimen (A) and resection bed (B) for T1a esophageal adenocarcinoma. (C, D) Resection bed for T1b Adenocarcinoma of the distal esophagus and gastroesophageal junction

Clinical staging investigations including CT scan, PET scan and EUS are preferred for solid luminal tumors prior to ESD whenever possible, however ultimately the determination of the risk of systemic metastasis is determined by the final pathology of the resection specimen. ESD functions as an excellent excisional biopsy and prognosticator in this regard. We often reserve staging investigations for patients with larger solid lesions whereas smaller superficial lesions are often selected for upfront ESD.

Operable patients who are found to be at elevated risk for lymph node metastasis following ESD routinely proceed with esophagectomy to excise the esophagus and surrounding lymphatic tissues. In our centre, despite elevated risks of lymph node metastases on final pathology, patients who are not otherwise operative candidates are often considered for adjuvant therapies including chemotherapy, radiation therapy and immunotherapy following ESD. Furthermore, patients with residual local mucosal disease only following induction chemotherapy or chemoradiation, and who are not found to be candidates for esophagectomy, may be considered for ESD to resect their residual mucosal disease.

Endoscopic Ultrasound for Esophageal Cancer

Initial Clinical Staging

The assessment of esophageal cancers often includes EUS for clinical staging. Initial assessment with radial endoscopic ultrasound often accompanies standard esophagogastroduodenoscopy and can provide important information for tumor staging including depth of invasion and presence of locoregional nodal metastases (Table 1).

Tumor (T) Stage: Using a 360° radial EUS scope depth of tumor invasion can be directly assessed. The layers of the esophagus on radial EUS are visualized as follows: mucosa (first hyperechoic layer), muscularis mucosa (first hypoechoic layer), submucosa (second hyperechoic layer), muscularis propria (second hypoechoic layer), adventitia (esophageal) or serosa (gastric) (third hyperechoic layer) (Fig. 2).

Solid tumors are visualized and their interface with the various mucosal layers allows for sonographic determination of depth of invasion [27]. Depth of esophageal tumor invasion is referred to as T-stage and is described as followsaccording to the TNM 8th edition: Tis—carcinoma in situ, T1a—mucosal lesion only invading lamina propria or muscularis mucosa, T1b—invading submucosa, T2—invading into but not through muscularis (Fig. 3), T3—invading through muscularis propria into adventitia (Fig. 4), T4—invasion of adjacent structures (T4a—invasion of azygous vein, pericardium, peritoneum or diaphragm (Fig. 5), T4b—invasion of other adjacent structures such as aorta, vertebral body or airway) [28].

Regarding T1 tumors, high frequency radial EUS can be used to differentiate T1a from T1b lesions, however, the most reliable assessment for depth of invasion remains complete mucosal resection with EMR or ESD. In this regard, ESD remains the superior determinant of T stage due to its en bloc dissection down to the level of the muscularis propria. EUS remains more reliable

Table 1 Esophageal TNM staging 8th edition [28]

Tumor invasion (T)	
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1a	Tumor invades the mucosa (lamina propria or muscularis mucosa)
T1b	Tumor invades the submucosa
T2	Tumor invades into but not through muscularis propria
T3	Tumor invades adventitia
T4a	Tumor invades pericardium, diaphragm, pleura, peritoneum, azygous vein
T4b	Tumor invades aorta, vertebral body, trachea
Lymph Nodes (N)	
N0	No lymph nodes metastases
N1	Metastases in ≤ 2 lymph nodes
N2	Metastases in 3–6 lymph nodes
N3	Metastases in ≥ 7 lymph nodes
Distant Metastases (M)	
M0	No evidence of distant metastases
M1	Distant metastases

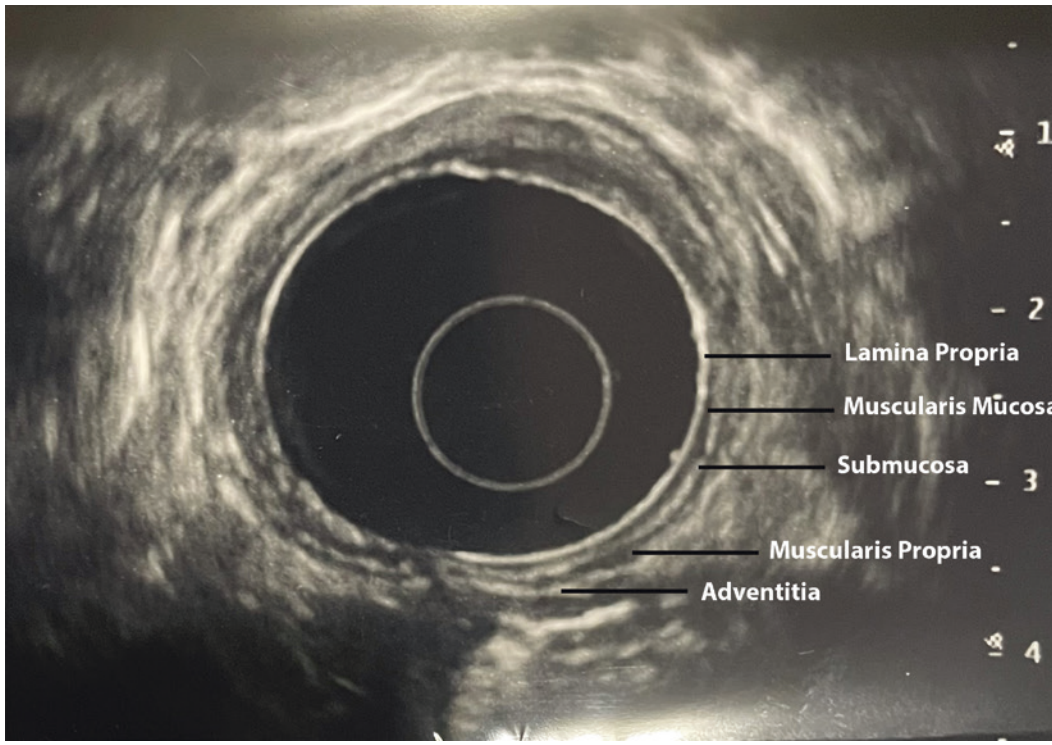

Fig. 2 Sonographic layers of the esophageal wall

for differentiation between numerical T stages (T1 from T2, T2 from T3, T3 from T4) [27].

Nodal (N) Staging: EUS allows for the assessment of locoregional lymph nodes for nodal staging. For esophageal cancer lymph nodal basins in the periesophageal, subcarinal, perigastric, celiac, splenic artery and hilum, left gastric artery and common hepatic artery are visualized and assessed for potential metastatic involvement. Sonographic features of nodal metastases include: size greater than 1 cm, round shape, hypoechoic, discrete borders, and absence of lymphatic hilar structures or intranodal vessels [27]. TNM 8th edition nodal staging is described as: NX—lymph node status cannot be assessed, N0—no nodal metastases, N1—metastases in ≤ 2 regional nodes, N2—metastases in 3–6 regional nodes, N3—metastases in ≥ 7 regional nodes [28] (Fig. 6).

Metastasis (M) Staging: EUS can occasionally be utilized to provide information regarding local metastatic disease to solid organs, predominantly the liver. However, this information is most commonly obtained from the complementary staging imaging investigations of CT and PET imaging [29].

Accuracy of EUS

The overall accuracy of radial EUS for T and N staging of esophageal cancer is approximately 90%, however there is significant variation according to stage [30]. EUS accuracy for T staging increases for more advanced compared to early disease, particularly for differentiating T1a from T1b tumors [31]. EUS restaging following chemoradiation is reported to be decreased potentially from radiation

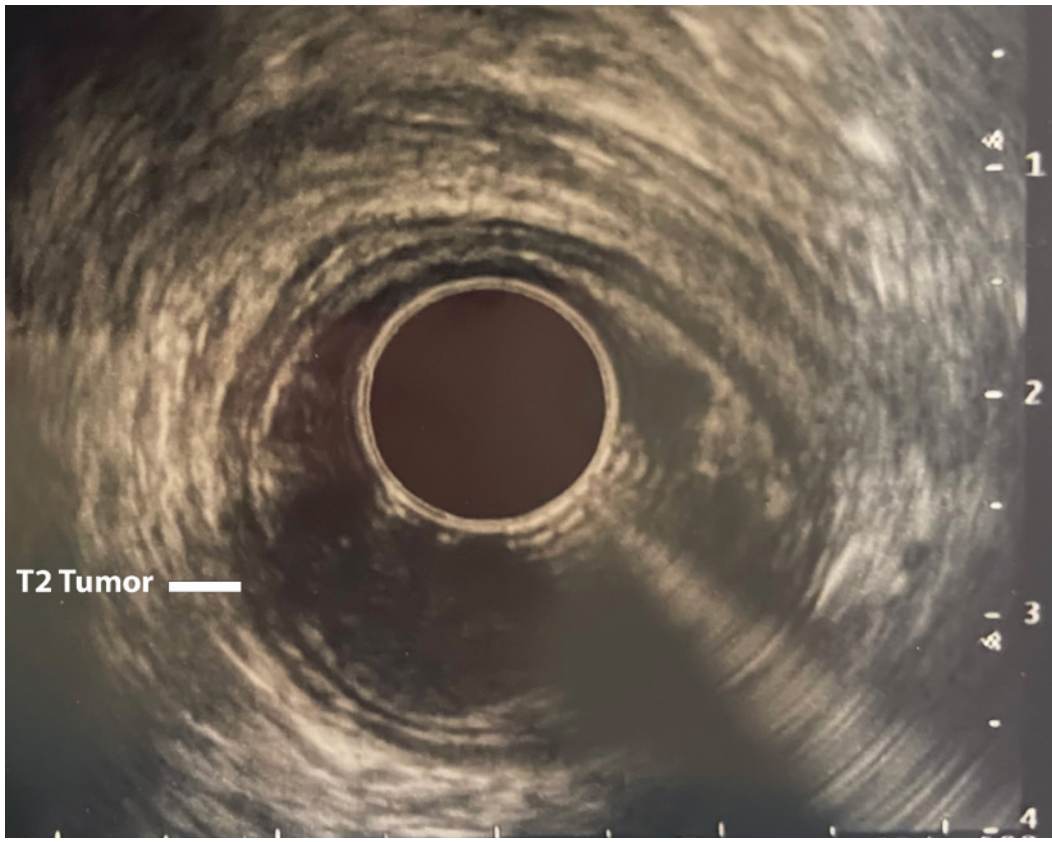

Fig. 3 Radial EUS example of T2 esophageal adenocarcinoma

induced changes in periesophageal tissues [32]. However, in our centre we perform routine EUS surveillance in the addition to cross-sectional CT imaging in higher risk patients following ESD resection or definitive chemoradiation therapy.

Limitations of EUS

EUS has limitations in staging for esophageal cancer in the presence of malignant strictures that prevent the passage of the echoendoscope beyond the lesion. This can necessitate endoscopic dilation to assist in endoscope passage or the use of EUS miniprobe as staging adjuncts. While EUS miniprobe can assist in the sonographic assessment of small mucosal lesions, their high frequencies diminish depth of tissue penetration. Unfortunately, this often prevents accurate EUS nodal assessment of the stomach and retroperitoneal lymph node basins beyond the malignant stricture [27]. It is often argued however, that given the high risk of nodal metastases from T3 lesions, and the supplemental staging information for nodal metastases given by CT/PET imaging, that once a T3 lesion is visualized, passage of the EUS scope beyond the cancer provides minimal treatment-plan impacting information. Other patient factors that can prevent EUS staging include benign esophageal strictures or patient factors that prevent the passage of the large EUS scope beyond the upper esophageal sphincter. Thankfully next-generation EUS scopes are slimmer in diameter reducing the incidence of these problems [33].

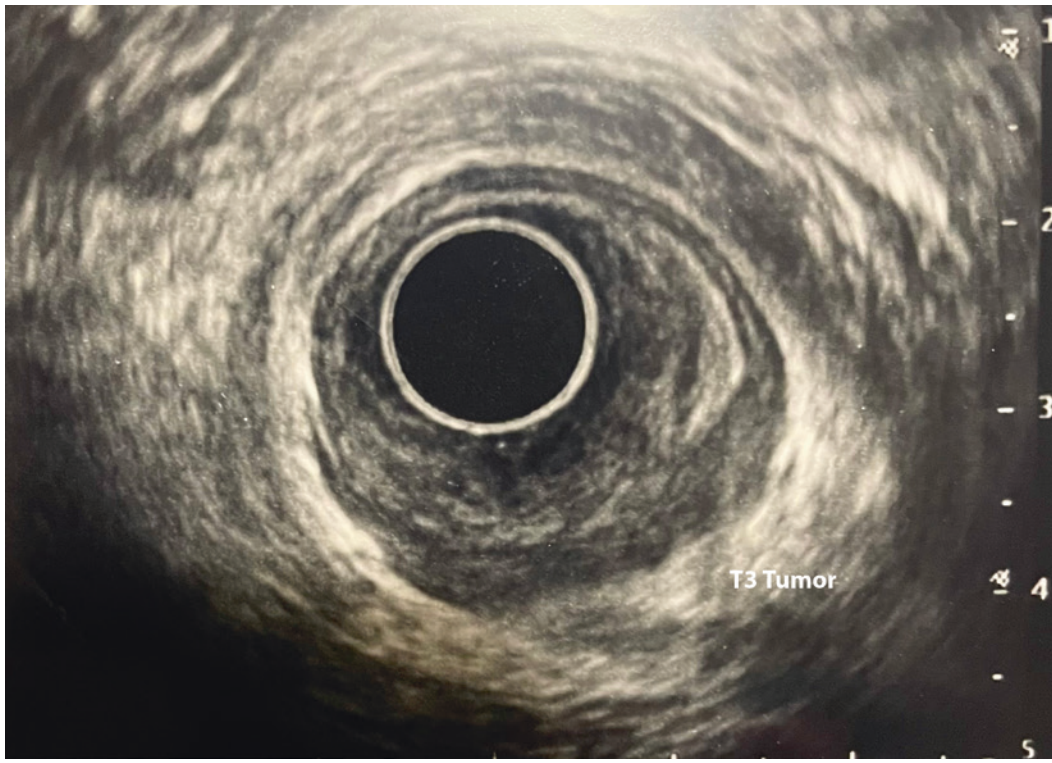

Fig. 4 Radial EUS example of T3 esophageal adenocarcinoma

EUS for Other Benign and Malignant Esophageal Tumors

Radial EUS is routinely utilized for the characterization and identification of benign and malignant lesions of the submucosal and muscularis propria. These most commonly include esophageal leiomyoma and gastrointestinal stromal tumors (GIST) but may also include other rarer benign and malignant tumor varieties. Additionally, congenital lesions such as esophageal duplication and bronchogenic cysts can be identified and characterized. EUS image characterization and esophageal layer localization are key components for lesion identification. Linear EUS FNA or Core Needle biopsy becomes a useful adjunct for tissue sampling when the diagnosis is not clear based on EUS and CT imaging characteristics alone and clinical management will be impacted [34] (Fig. 7).

Linear EUS and Linear Endobronchial Ultrasound (EBUS)

Mediastinal, periesophageal, hilar, pulmonary, perigastric and retroperitoneal adenopathy may be detected in patients during clinical staging for esophageal cancer. Occasionally lymph node enlargement may be secondary to infection and inflammation, autoimmune conditions such as sarcoidosis or low-grade malignancies unrelated to the patient's esophageal cancer. In this setting enlarged lymph nodes on CT or EUS or metabolically active lymph nodes on PET scan may require nodal sampling with linear EUS or linear EBUS guided FNA to determine the presence of esophageal cancer nodal metastases. While EBUS can biopsy pulmonary, hilar and mediastinal lymph nodes surrounding the airway, EUS can biopsy mediastinal, periesophageal, perigastric and retroperitoneal lymph nodes. Linear EUS and EBUS needles are offered in sizes

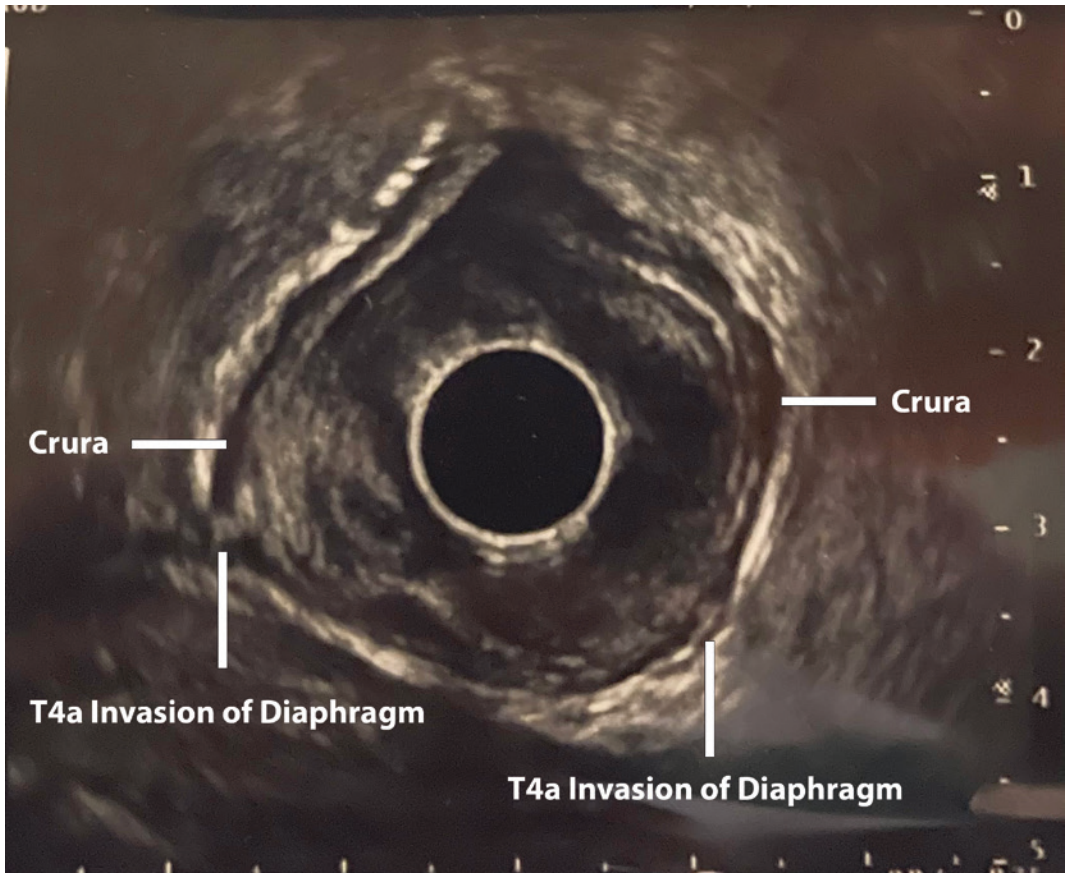

Fig. 5 Radial EUS example of T4a esophageal adenocarcinoma invading diaphragmatic crura

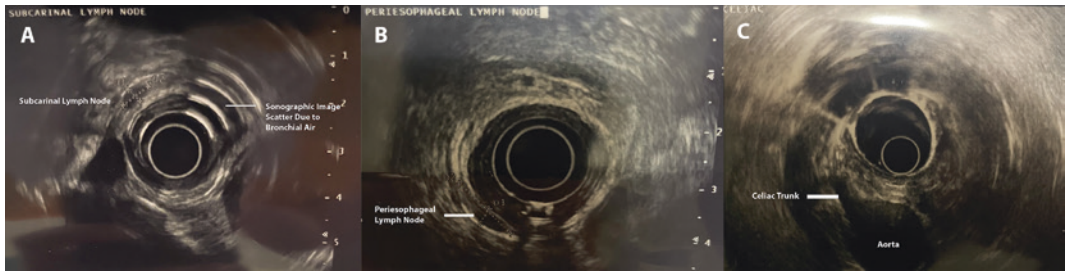

Fig. 6 Examples of Nodal Stations Evaluated for Radial EUS Nodal Staging. (A) Subcarinal lymph node (B) Periesophageal Lymph Node (C) Celiac Trunk location of Celiac lymph nodes

between 19 and 25 gauge depending on the clinical application suspected [35, 36].

EUS for Surveillance

Following endoscopic or surgical resection of esophageal cancer, surveillance EUS has been advocated to look for early signs of locoregional nodal metastases. Surveillance EUS performed at intervals of every six months following resection can result in earlier detection of disease recurrence. It remains unclear however if earlier detection of disease recurrence results in any improvement in long-term survival [37, 38]. Regarding the need for EUS surveillance

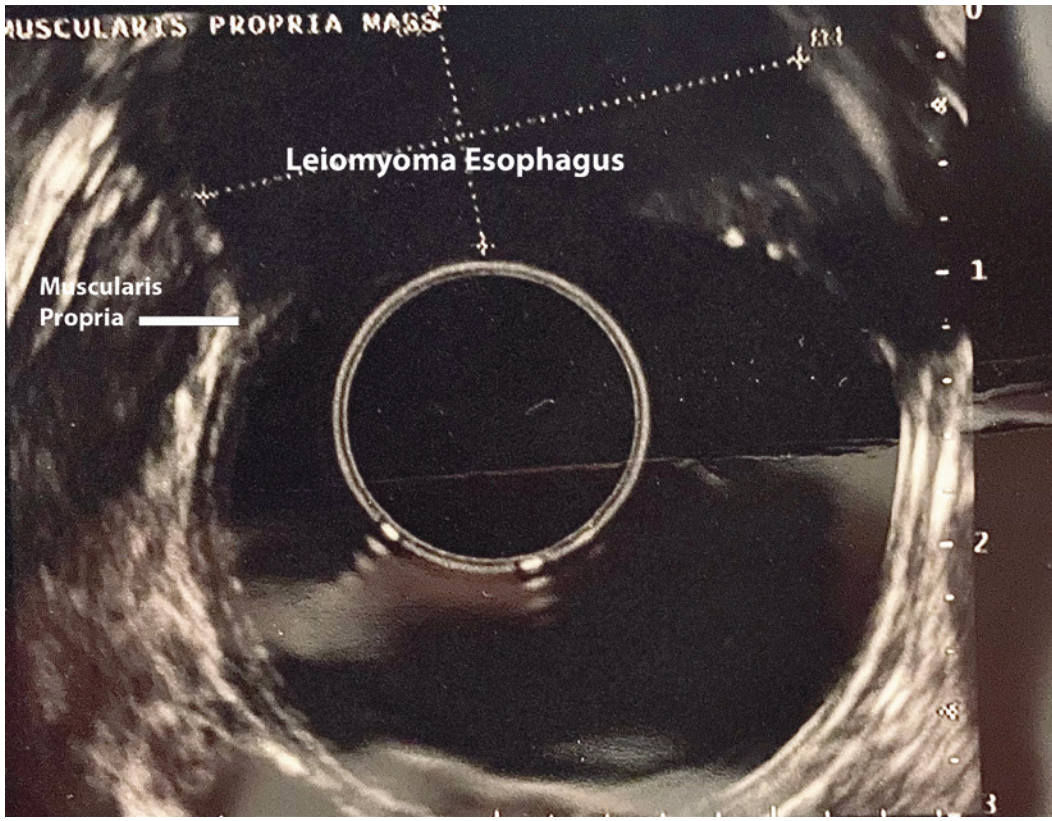

Fig. 7 Esophageal Leiomyoma arising from muscularis propria

following ESD tumor resection, when tumors resected are T1a or greater surveillance EUS in addition to cross-sectional imaging CT scan is suggested to monitor for locoregional nodal recurrence.[39] If suspicious enlarged lymph nodes are identified, linear EUS FNA biopsy can be performed for tissue diagnosis (Fig. 8).

Endoluminal Stenting

Esophageal Cancer and Endoluminal Stenting

A variety of partially and fully covered self-expanding metal (SEM) esophageal stents are available for the treatment of luminal stenosis induced by esophageal cancers. Stents are routinely inserted under direct endoscopic vision through proximal or distal release mechanisms. Fluoroscopy-guided stent insertion is also possible after endoscopic guidewire placement and marking with radio-opaque materials on the patient's skin [40].

Fully covered stents have a silicone membrane covering and take longer for tissue ingrowth and are removable up to 4–6 weeks following insertion. Partially covered stents have coverage of most of the stent with proximal and distal ends uncovered and exposed to allow for tissue ingrowth. Once inserted for greater than 2 weeks times tissue ingrowth occurs and following this removal may not be possible or may cause significant mucosal injury with removal. Therefore, the use of partially covered esophageal stents are often reserved for patients with unresectable cancers [41].

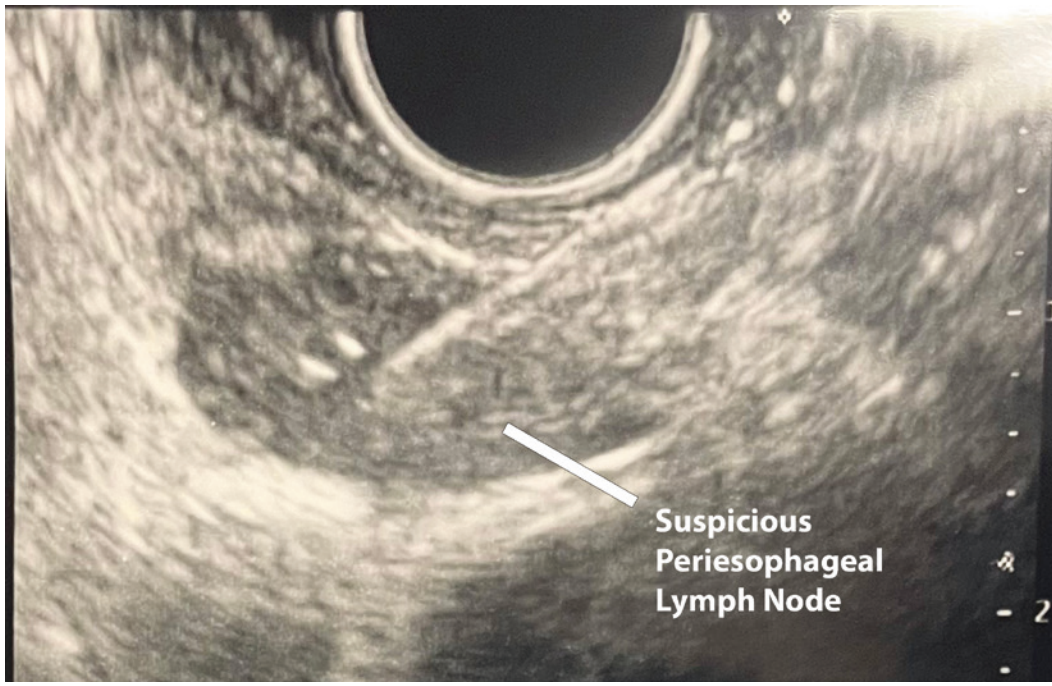

Fig. 8 An enlarging periesophageal lymph node detected by surveillance radial EUS. Sampled with 22-gauge linear EUS FNA. Note the size greater than 1 cm, sharp borders and lack of central hilar/vascular structures suggestive of malignant involvement

Treatment of Esophageal Perforations Related to Cancer

Perforated esophageal cancers induced by retching or iatrogenically through endoscopy by scope passage or dilation can be similarly treated with endoluminal esophageal stenting. Often, once perforation occurs related to a malignancy, esophageal stents may require permanent placement [42]. ESD of esophageal superficial mucosal cancers results in exposure of esophageal muscularis propria. Occasionally full thickness perforation occurs secondary todissection of submucosal fibrosis or injury. If these injuries are present in the esophagus and do not involve the gastroesophageal junction, fully covered esophageal stenting can be performed to allow for healing and stentremoval in approximately 4 weeks. In similar fashion, following fully circumferential ESD stricture formation can occur, leading to prophylactic fully covered esophageal stent placement following resection to reduce severity of stricturing [43].

Complications of endoluminal stenting for esophageal cancer include retrosternal chest pain, tissue ingrowth, tumor overgrowth, stent migration, stent food bolus obstruction and aspiration pneumonia (particularly when stents are placed across the GEJ). Rare complications include tracheoesophageal fistula formation, bleeding, and esophageal perforation. Incidence of complications from stenting is estimated to be between 40 and 50% [44].

Endoscopy and Enteral Nutrition

Endoscopy is important for the support of enteral nutrition in patients with resectable and non-resectable esophageal cancers. While esophageal stenting routinely allows for passage of orally ingested food into the stomach

and small bowel, stenting is not always possible. This can commonly occur for proximal esophageal cancers involving the UES, esophageal collapse secondary to external compression from nodal metastases, or lengthy tumors that prevent esophageal stenting [41].

Upper gastrointestinal nutritional support can be re-established using endoscopy. Nasojejunal feeding tubes can be directed endoscopically with the potential addition of fluoroscopy to enter the proximal jejunum. Nasojejunal feeding tubes are problematic however as they can routinely be removed accidentally through traction. Percutaneous endoscopic gastrostomy (PEG) tubes can be placed with the aid for a gastro-scope or fluoroscopy as a palliative support for patients with unresectable cancers or for patients requiring neoadjuvant therapy with goal of curative intent esophagectomy. Care must be maintained to not injure the right gastroepiploic artery for any potential esophagectomy candidates. PEG tubes can also be modified to direct feeding tubes into thejejunum beyond the stomach. While PEG tubes have advantages, foreign body site infections can necessitate their removal. Insertion of PEG tubes can be complicated by gastric perforation necessitating endoscopic or surgical closure [45].

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