

Minimally Invasive Resection of Benign Esophageal Lesions[☆]

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Benign esophageal lesions include a wide variety of rare neoplasms, polyps, and cysts. In general, these lesions are asymptomatic and have little clinical importance. However, on occasion these lesions become symptomatic due to esophageal obstruction, airway obstruction, or compression of mediastinal structures. In these cases, as well as cases when it is unclear if the lesion is malignant or benign, surgical resection is recommended. Resection is most often performed by extramucosal enucleation, a procedure that is oftentimes well-suited for a minimally invasive approach. Here we discuss the general approach and operative techniques used for minimally invasive resection of benign esophageal lesions.

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Introduction

Benign esophageal lesions are a heterogeneous group of rare neoplasms, polyps, and cysts. It is estimated that benign tumors represent less than 1% of all esophageal neoplasms and less than 10% of all resected esophageal neoplasms.¹ The majority are located in the middle or distal esophagus and can arise from any layer of the esophageal wall. Therefore, lesions are most often described as intraluminal, submucosal, intramural, or extraesophageal in origin.² Intraluminal lesions arising from the mucosa include various polyps, papillomas, and inflammatory pseudotumors. Submucosal and intramural neoplasms include leiomyomas, granular cell tumors, hemangiomas, neurofibromas, lipomas, schwannomas, and rhabdomyomas. Leiomyomas are by far the most common histologic cell type, accounting for more than 80% of all benign esophageal tumors.³ Cysts, including intramural inclusion cysts and extraesophageal congenital duplication cysts, represent the

second most common benign esophageal lesion.⁴ Gastrointestinal stromal tumors may be found anywhere in the gastrointestinal tract, including the esophagus. These neoplasms have malignant potential, and management of these lesions is beyond the scope of this discussion.

Most benign esophageal neoplasms are asymptomatic, in part because of their indolent rate of growth and the ability of the esophagus to dilate. Because of this, the true incidence remains unknown. Most symptoms are caused by obstruction of the esophageal lumen or compression of surrounding mediastinal structures. Intraluminal lesions and large submucosal or intramural lesions, typically >5 cm, are most likely to be symptomatic.⁴ As a result, dysphagia is the most common complaint on initial presentation. Other reported symptoms include chest pain, dyspnea, chronic cough, wheezing, and upper gastrointestinal bleeding.⁴ Circumferential growth of benign esophageal tumors can have the typical appearance and presentation of an esophageal stricture or malignant tumor.

Because dysphagia is the most common initial complaint, many patients undergo a contrast esophagram as part of the initial workup. This study identifies the most symptomatic lesions, most often revealing a smooth-surfaced filling defect. Computed tomography scans are not typically necessary but may be helpful in determining the degree of mass effect caused by the lesion on the esophagus or other mediastinal structures, as well as identifying other extraesophageal causes of symptoms. Upper endoscopy is important for confirmation of the diagnosis and for operative planning. Intraluminal lesions can be differentiated from lesions

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originating from deeper layers of the esophagus, which will have an intact layer of mucosa overlying the lesion. Endoscopic biopsy of intraluminal or submucosal lesions is recommended if there is any suspicion of malignancy. Biopsy is not recommended for intramural lesions, as adequate tissue is rarely obtained to rule out malignant disease (ie, leiomyoma vs leiomyosarcoma),² and the biopsy can create mucosal scarring, which may increase the risk of perforation during enucleation.³ Establishing the location of the lesion relative to the cricopharyngeus and gastroesophageal junction is necessary to determine the optimal surgical approach (cervical, transthoracic, or transabdominal). Endoscopic ultrasound can be useful for assessing which layers of the esophageal wall are involved and evaluate for evidence of invasion into surrounding layers, which would raise suspicion for a malignant lesion.

Incidentally encountered lesions with minimal or no symptoms are typically observed. Symptomatic lesions (typically >5 cm) and lesions that are clearly growing warrant consideration of removal, particularly if there is concern for malignant transformation or to facilitate other esophageal procedures. Smaller intraluminal lesions can typically be resected with endoscopic techniques, such as endoscopic mucosal resection or polypectomy snaring for

pedunculated lesions. Larger lesions may require resection via an esophagotomy. Submucosal, intramural, and extraesophageal lesions are typically amenable to extramucosal enucleation. Resection can be performed with low morbidity using traditional open techniques (thoracotomy and laparotomy)⁵; however, the safety, feasibility, and improved outcomes associated with minimally invasive approaches (video-assisted thoracoscopic surgery and laparoscopy) are well documented, and they are preferred over open approaches in the hands of surgeons with expertise in minimally invasive esophageal surgery.³ There are no strict contraindications to a minimally invasive approach for the enucleation of benign lesions. However, lesion size, previous chest or abdominal surgery, prior biopsy, and surgeon experience all contribute to the technical difficulty of the procedure and should be considered when deciding between open or minimally invasive approach. Symptoms suggesting other esophageal pathology, such as reflux disease, hiatal hernia, and motility disorders should be appropriately worked up and addressed at the time of operation.

Given that most benign esophageal lesions are submucosal or intramural in origin, the remaining discussion and illustrations focuses on the operative techniques used for minimally invasive extramucosal enucleation of these lesions (Figs. 1-13).

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