# Benign lesions of the esophagus

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## INTRODUCTION

A variety of benign esophageal lesions are encountered during endoscopic or radiologic evaluation of the esophagus. Many are uncommon, cause no symptoms, and have no malignant potential. Nevertheless, they can pose a challenge in establishing an accurate diagnosis and thereby formulating a management plan. Benign esophageal lesions can be classified as raised, flat, or cystic.

This topic will review many of the benign lesions that may be found in the esophagus. Esophageal strictures, Barrett's esophagus, and esophageal cancer are discussed elsewhere. (See "Endoscopic interventions for nonmalignant esophageal strictures in adults" and "Barrett's esophagus: Epidemiology, clinical manifestations, and diagnosis" and "Epidemiology and pathobiology of esophageal cancer" and "Clinical manifestations, diagnosis, and staging of esophageal cancer".)

## RAISED LESIONS

**Gastrointestinal stromal tumors, leiomyomas, and leiomyosarcomas** — Gastrointestinal stromal tumors, leiomyomas, and leiomyosarcomas are described separately. (See "Clinical presentation, diagnosis, and prognosis of gastrointestinal stromal tumors".)

**Schwannomas** — Schwannomas are rare, benign tumors that arise from perineural elements of the Schwann cell in the peripheral nerves [1]. They are characterized by peripheral lymphoid

cuffing, benign nuclear atypia, and spindle-shaped cells. Symptomatic esophageal schwannomas most often present with dysphagia, but dyspnea has been documented in tumors compressing the trachea. While the majority of the tumors are benign, malignant schwannomas have been described [2]. Small benign tumors may be removed by surgical enucleation [1]. Large symptomatic tumors typically require surgical resection, though newer, submucosal resection techniques have been used to remove these lesions endoscopically [3,4].

**Lymphangiomas** — Lymphangiomas are benign lesions that are believed to result from malformations of sequestered lymphatic tissue. They are most commonly found on the skin but have been described everywhere in the body except the brain. There are few reported cases of esophageal lymphangiomas, most of which were diagnosed in children younger than two [5].

On endoscopy, they appear as translucent, yellowish, easily compressible nodules that are usually less than 5 mm. Standard endoscopic biopsies are often normal or show nonspecific esophagitis since the lesions are submucosal [5,6]. Histologic examination of resection specimens reveals dilated endothelial spaces with cavities lined by flat endothelial cells containing eosinophilic material [5].

Diagnosis can be suggested on endoscopic ultrasound (EUS) in which the lesion is solid and is located in the submucosal layer of the esophagus [5] and confirmed with tunneled biopsies.

Conservative management is generally appropriate. However, on occasion, these lesions can grow and reach sizes up to 4 to 5 cm, in which case resection is warranted [5,7]. Although data are scarce, band and saline-assisted endoscopic mucosal resection and endoscopic submucosal dissection can be employed to completely remove these lesions [8]. Excision using a $CO_2$ laser has also been reported [9].

**Hemangiomas** — Esophageal hemangiomas are rare; their prevalence in the general population is 0.04 percent based on an autopsy series [10]. The majority are cavernous (ie, have cavernous vascular spaces seen histologically), although capillary lesions have been described.

Though usually solitary, multiple lesions can be seen in Osler-Weber-Rendu disease, Klippel-Trénaunay syndrome, or congenital blue rubber bleb nevus syndrome [11]. (See "Clinical manifestations and diagnosis of hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)" and "Klippel-Trenaunay syndrome: Clinical manifestations, diagnosis, and management".)

On endoscopy, they appear nodular, soft, bluish-red, and typically blanch when pressed with biopsy forceps. They must be differentiated from Kaposi sarcoma, which may have a similar

endoscopic appearance. (See "Classic Kaposi sarcoma: Clinical features, staging, diagnosis, and treatment", section on 'Extracutaneous involvement'.)

Esophageal hemangiomas are usually found incidentally. When symptomatic, they have most often been associated with bleeding or dysphagia. In such cases surgical resection has traditionally been performed, but endoscopic resection has also been accomplished safely [12].

**Fibrovascular polyps** — Fibrovascular polyps refer to a variety of lesions, including fibromas, fibrolipomas, myomas, and lipomas, that are differentiated by their histologic appearance. They contain a mixture of fibrous, vascular, and adipose tissue covered by squamous epithelium. They are rare, accounting for 0.5 to 1 percent of all benign esophageal lesions [13]. They are most commonly encountered in the upper third of the esophagus and typically attach directly to the inferior aspect of the cricopharyngeus. Approximately 75 percent of cases have been reported in men, for reasons that are unclear [14]. Most have been in their 50s or 60s.

Their pathogenesis is incompletely understood, but they likely arise from nodular thickening of a redundant mucosal fold. Such areas can elongate as the result of propulsive forces during repeated swallowing [14,15].

Fibrovascular polyps do not generally cause symptoms. However, case reports have described prolapse of large lesions into the larynx causing asphyxiation. Lesions as large as 20 cm have been reported ( picture 1) [14-16]. Other symptoms associated with large lesions include dysphagia, chronic cough, nausea, and vomiting. Polyps that extend into the proximal stomach are exposed to an acidic environment, which can lead to ulceration and bleeding [15].

Symptomatic polyps often have a stalk, permitting them to be removed endoscopically using a snare. We agree with other authors that EUS should be performed before excision to rule out the presence of a large vessel feeding the stalk of the lesion [14]. Endoscopic resection can be technically difficult when the stalk is attached to the proximal esophagus. Surgical resection may be required if a large feeding vessel is present or if the base of the stalk is endoscopically inaccessible. In the case of a large feeding vessel, endoscopic resection may still be possible with use of an endoloop or clips placed across the stalk of the polyp before polypectomy. (See "Management and prevention of bleeding after colonoscopy with polypectomy".)

**Granular cell tumors** — Granular cell tumors occur in the skin, tongue, breast, and gastrointestinal tract. Approximately 10 percent arise in the gastrointestinal tract, most commonly in the esophagus, which is involved in up to 65 percent of cases [17]. Nevertheless, granular cell tumors of the esophagus are rare; their incidence in endoscopy series has been estimated to be approximately 0.033 percent, representing approximately 1 percent of benign

esophageal tumors [17-19]. (See "Endoscopic ultrasound for the characterization of subepithelial lesions of the upper gastrointestinal tract".)

The mean age at diagnosis is 45 years, with lesions found more commonly in males [17,20]. Up to one-third of patients with granular cell tumors of the esophagus report dysphagia; the others are mainly asymptomatic [17].

On endoscopy, these lesions are typically sessile, yellowish-white, and are covered by normal-appearing mucosa. They feel firm or rubbery when prodded with a biopsy forceps [21]. Most patients have solitary lesions but up to 10 percent have two or more lesions at diagnosis [17].

Histologically, granular cell tumors are composed of large polygonal cells containing numerous eosinophilic granules. In one patient, the diagnosis of granular cell tumors preceded the diagnosis of eosinophilic esophagitis [21,22]. They resemble Schwann cells under electron microscopy and stain positive for S100 protein, suggesting that they originate from cells of neural origin [23].

Superficial biopsies may be normal or can miss the diagnosis, showing only fragments of normal squamous mucosa or revealing hyperplastic changes that can be confused with squamous cell carcinoma, potentially leading to misdiagnosis [20,21]. We advocate tunneled biopsies with adequate tissue capture to facilitate an accurate diagnosis.

Unlike most of the other esophageal lesions discussed in this topic review, granular cell tumors have malignant potential. Malignancy is more likely with large lesions and those that exhibit growth [24]. In a review of 183 cases, eight lesions (4 percent) were malignant; all of these lesions were greater than 4 cm and had histological features of infiltrative growth [17]. They have also been synchronously diagnosed with esophageal adenocarcinoma [25].

Although rare, because there is malignant potential, we suggest endoscopic resection of all granular cell tumors. Small lesions can often be removed with a biopsy forceps, but lesions larger than 1 cm typically require endoscopic mucosal resection or submucosal tunnel endoscopic resection [26]. Recurrence after resection has not been described [20].

**Adenomas** — Esophageal adenomas arise almost exclusively in segments of Barrett's esophagus and likely represent a polypoid or nodular form of dysplasia rather than an isolated polypoid adenoma [27]. Adenomas occurring in the absence of Barrett's esophagus have been described only in older case reports [28]. The association with Barrett's esophagus is further supported by the concomitant finding of esophageal adenocarcinoma in many patients [29,30].

Lesions as large as 1.5 cm have been reported [31]. Multiple lesions giving the appearance of esophageal polyposis have also been described [29,30].

There is limited experience to guide management of esophageal adenomas. If an adenoma-like lesion is apparent, we suggest carefully inspecting the adjacent mucosa to evaluate for areas of Barrett's mucosa. Enhanced endoscopic techniques such as narrow band imaging or chromoendoscopy can be considered in these cases. Even if not apparent endoscopically, we would suggest biopsying surrounding mucosa to evaluate for intestinal metaplasia. If Barrett's is confirmed, then we would advocate management similar to that used for nodular and/or dysplastic Barrett's. (See "Barrett's esophagus: Treatment of high-grade dysplasia or early cancer with endoscopic resection" and "Barrett's esophagus: Surveillance and management", section on 'High-grade dysplasia or intramucosal carcinoma'.)

If the lesion is an isolated adenoma without associated Barrett's, we advocate using a strategy similar to that used for colonic adenomas; lesions smaller than 1 cm should be resected endoscopically using a biopsy forceps or snare. Lesions larger than 1 cm and those containing high-grade dysplasia can be removed using advanced endoscopic resection techniques, whereas more advanced lesions may require surgery.

An esophageal gland duct adenoma, an extremely rare variant, has been reported as an indolent submucosal lesion that can be successfully managed with endoscopic submucosal dissection [32].

**Inflammatory fibroid polyps** — Like fibrovascular polyps, inflammatory fibroid polyps describe a variety of lesions that are composed of reparative tissue with reactive blood vessels, fibroblasts, and inflammatory cells. They include hamartomas, inflammatory pseudopolyps, and eosinophilic granulomas [33].

They are rare, with only one case found in 330,000 surgical specimens submitted in one hospital over 60 years [33]. They are more commonly found in the stomach, small bowel, and colon than the esophagus [34].

Their pathogenesis is unclear, but they are thought to occur primarily from acid reflux-induced inflammation, a theory supported by their occurrence mainly in the distal esophagus, including the gastroesophageal junction.

Inflammatory fibroid polyps are considered to be benign, reactive inflammatory lesions whose defining histologic features include a connective tissue stroma and a diffuse eosinophilic infiltrate [35]. They generally are an incidental finding but can occasionally cause hemorrhage or dysphagia [35]. Rapid growth (to as large as 9 cm) has also been described [35].

A standard biopsy generally provides enough tissue to make a diagnosis. Resection is not necessary except for lesions that cause symptoms, which may warrant snare polypectomy or surgical resection.

**Papillomas** — Esophageal papillomas are benign epithelial lesions characterized histologically by fingerlike projections of tissue lined by an increased number of squamous cells and a core of connective tissue that contains small blood vessels [36]. They are rare but studies suggest their incidence is increasing [37]. Studies have reported an incidence in patients undergoing upper endoscopy that ranged from 0.01 to 0.57 percent [37-40], and a prevalence in the general population based on autopsy series ranging from 0.006 to 0.04 percent [39,41,42].

The pathogenesis of esophageal papillomas is uncertain but two theories have been proposed.

- Several lines of evidence support an underlying inflammatory condition. Approximately 70 percent of papillomas occur in the distal one-third of the esophagus [43], and have been associated with documented reflux, esophagitis, or mucosal irritants such as nasogastric tubes [44,45]. In addition, several reports have linked bougie-assisted mechanical esophageal dilation with papillomas [45,46]. Furthermore, animal studies have shown that papillomas can be induced by caustic mucosal irritation with benzopyrene and nitrosamines [46,47].

- There is also evidence to support a role of human papilloma virus (HPV) in the formation of esophageal papillomas, although data are inconclusive. Studies from Italy and Hungary have documented HPV in 21 to 46 percent of esophageal papillomas [39,48], while studies from the United States, Finland, and Poland identified HPV in fewer than 5 percent of cases [49-51]. It is possible that the differences may be related to geographic variability in the frequency of oral HPV infection, but further studies are needed. A role for HPV is also supported by the observation that esophageal cases have been associated with serotypes 6 and 11 (strains commonly found in the human oropharynx and genital tract), implicating sexual transmission [52].

  HPV has been unequivocally linked to cancer in the larynx and cervix, but its role in the pathogenesis of esophageal cancer remains unclear. HPV has been detected in esophageal squamous cell carcinoma, and there are descriptions of the simultaneous detection of squamous cell carcinoma and papillomas [39,48]. However, there is limited direct evidence of malignant transformation related to HPV infection in the esophagus. A similar virus, bovine papilloma virus, has been linked to the malignant transformation of esophageal papillomas in cattle [35,53]. (See "Epidemiology and pathobiology of esophageal cancer".)

Esophageal papillomas have been most commonly diagnosed in patients who are in their 50s [43,45,54]. Some studies suggest they are more common in men (male to female ratio of 1.8 to 3.4 in various reports) [43,45,54], while others have reported an equal sex distribution [38,39].

The majority of papillomas are solitary, although patients with over 10 lesions have been described [45]. Lesions have a classic endoscopic appearance, and the classic triad of exophytic growth, wart-like projections, and crossing surface vessels evident with narrow-band imaging have a positive predictive value for esophageal papilloma that approaches 90 percent ( picture 2) [55]. Lesions must be differentiated from other similar-appearing lesions, such as verrucous squamous cell carcinoma, granulation tissue, and papillary leukoplakia. They can be seen more frequently in patients with tylosis and acanthosis nigricans, and there is an association with Goltz syndrome, a congenital disorder of focal dermal hypoplasia that features hyperpigmentation, sclerodactyly, dysplastic changes of the teeth and bones, and perianal, oroesophageal, and genital papillomas [56]. Interestingly, papillomas have been detected with positron emission tomography [57].

Most lesions do not cause symptoms, but large lesions can cause dysphagia [45,58]. Papillomas are generally amenable to endoscopic resection. Lesions smaller than 1 cm can usually be removed with a cold forceps biopsy, but larger lesions require advanced endoscopic resection techniques. Recurrence after resection is infrequent [45]. (See "Overview of endoscopic resection of gastrointestinal tumors".)

## FLAT LESIONS

**Heterotopic sebaceous glands** — Heterotopic sebaceous glands are generally found in tissues of ectodermal origin such as the external genitalia, parotid gland, palms and soles, eyelashes and lips, and mouth and tongue (where they are known as Fordyce spots). They are rare in tissues of endodermal origin such as the esophagus.

The pathogenesis of esophageal heterotopic sebaceous glands is unclear. They were not found in a large autopsy series of infants and children, suggesting that they are unlikely to be congenital [59]. One theory is that they result from reactive metaplasia of ectopic, pluripotent salivary type mucous glands in the esophagus that were displaced during embryonic development [60]. However, the observation that the number and size of these lesions do not change with time suggest that they result from heterotopic (aberrantly placed) mucosa and not metaplastic change [61].

On endoscopy, the glands appear as yellowish-gray, plaque-like lesions that can resemble xanthelasmas, Candida, eosinophilic crypt abscesses, or pill residue ( picture 3) [62-64]. They frequently occur in clusters, with several cases of up to 100 lesions reported in the literature [60,63]. Microscopically, the lesions show lobules of cells with sebaceous differentiation within the lamina propria [62]. Microscopic foci of nonspecific inflammation have occasionally been described adjacent to the sebaceous lobules [60].

Histologic review of standard cold biopsies is generally sufficient to make the diagnosis. Surveillance or resection is not generally required since there is no known malignant potential [61].

**Glycogen acanthosis** — Glycogen acanthosis has been described in the esophagus on 3.5 to 15 percent of all upper endoscopies [65,66] and in up to 30 percent of patients getting double-contrast esophageal imaging [67]. It typically appears as multiple, uniformly sized round elevations in otherwise normal-appearing mucosa, usually in the midportion of the esophagus ( picture 4) [65]. They are typically 2 to 10 mm in diameter, but lesions up to 15 mm have been described [68].

The pathogenesis is unclear; they have no relation to glucose metabolism, diabetes, or the presence of acanthosis nigricans. Esophageal glycogen acanthosis has been described in association with Cowden disease, a rare autosomal dominant disease manifested by multiple hamartomas frequently involving the gastrointestinal tract [69,70]. There is also some evidence to suggest an association with celiac disease, especially in children [71]. (See "PTEN hamartoma tumor syndromes, including Cowden syndrome", section on 'Esophageal glycogen acanthosis'.)

Most patients have been diagnosed in their 40s and 50s, and for unclear reasons, most are men [66]. The lesions are believed to grow more numerous and larger with age but do not cause symptoms [68].

Diagnosis can be established by mucosal biopsies with standard cold forceps, which show multifocal plaques of hyperplastic squamous epithelium with abundant intracellular glycogen ( picture 5) [68]. However, biopsies are rarely needed since the visual findings on endoscopy are highly predictive when the lesions have been confirmed histologically [66]. Further confirmation can be achieved by application of Lugol's solution, which turns the glycogen-containing plaques dark brown, thereby distinguishing glycogenic acanthosis from leukoplakia, candidiasis, and bullous pemphigoid, all of which can have a similar endoscopic appearance.

**Inlet patch** — An esophageal inlet patch (also referred to as heterotopic gastric mucosa of the upper esophagus [HGMUE]) consists of a discrete area resembling gastric mucosa that is typically found in the proximal esophagus. The prevalence of the inlet patch in the general

population approached 5 percent in an autopsy series [59], while endoscopic studies have reported it to be present in 0.4 to 11 percent of patients undergoing diagnostic upper endoscopy [72-79]. The lower prevalence in endoscopic studies may be due to the proximal location of inlet patches. They are most commonly found in the proximal 3 cm of the esophagus, usually just distal to the upper esophageal sphincter, an area that is not always seen well during routine upper endoscopy [72,74,76]. Inlet patches have been described in people of all ages, but the most common age of detection is in the mid-50s [77].

**Pathogenesis** — The pathogenesis of the esophageal inlet patch is not known precisely. The term "heterotopic" gastric mucosa, sometimes used to describe it, may be a misnomer since some evidence suggests that the patch is composed of embryonic (rather than heterotopic) gastric mucosa. Immunohistochemical analysis has shown that the patch contains glucagon reactive cells that are not found in the mature human stomach, but are seen in its embryonic form [80]. In addition, the patch is frequently discovered in children and its prevalence does not increase with age [72,73].

One hypothesis is that the columnar lining of the esophagus in the embryo is not completely replaced by pseudostratified squamous epithelium during fetal development [81]. However, conflicting data have also been published. Some studies based on immunohistochemical analysis found that inlet patches more closely resemble Barrett's mucosa, an acquired lesion, rather than normal gastric mucosa, making a congenital origin seem less likely [82].

Several authors have advanced a "mixed theory," suggesting that the patch occurs as the result of a loss of normal squamous epithelium (from trauma, regurgitation, or infection) and subsequent healing by surfacing ectopic gastric mucosa present in the underlying lamina propria as the result of a congenital anomaly [82-84].

**Clinical features** — The patches range in size from 2 mm to 4.5 cm and can be solitary or multiple [73,75]. They are usually red, velvety, and flat ( picture 6), although raised polypoid patches have been described [85].

Biopsies reveal corpus or fundic-type gastric mucosa [72,74,75,77], sometimes with parietal cells capable of secreting acid [78,86,87]. Microscopically, inlet patches also commonly have an associated inflammatory infiltrate [73].

Most inlet patches are found incidentally and do not cause symptoms. In a prospective study, an inlet patch was found in 11 percent of the 300 studied patients, but there was no association between the presence of an inlet patch and the reported severity of pyrosis, hoarseness, or dysphagia [79]. However, a wide range of clinical sequelae have been attributed to inlet patches, including:

- Perforation and a tracheoesophageal fistula occurring at the site of an inlet patch [88,89]. The presumed mechanism is related to acid production by the patch.

- Dysphagia, commonly from strictures, rings, and webs associated with the patch [90-92]. The association with webs and the potential for acid production and ulceration has led some authors to propose that the inlet patch is a possible underlying etiology for Plummer-Vinson syndrome [93]. (See "Esophageal rings and webs".)

- Interestingly, *Helicobacter pylori* can be localized in the patch in 19 to 73 percent of patients who are colonized in the stomach, raising the possibility that it acts as a reservoir for oral-oral transmission [75,94,95]. In a study with 68 patients with inlet patches, *H. pylori* was detected in 23 percent, all of whom reported globus sensation, suggesting a possible correlation [96]. (See "Globus sensation".)

- Some studies have found that up to 20 percent of patients with an inlet patch have concomitant Barrett's esophagus in the distal esophagus [77], but others have not confirmed such a correlation [73].

- There are numerous reports of esophageal adenocarcinoma arising from an inlet patch in the upper esophagus [97-99]. Additionally, there is one report of laryngeal adenocarcinoma diagnosed in a 22-year-old with an inlet patch [100].

- An association with globus sensation, chronic cough, and laryngopharyngeal reflux has been suggested in various reports [101-104]. A small randomized, sham-controlled trial found improvement in globus symptoms after ablation of the patch with argon plasma coagulation [105]. Thus, it may be reasonable to look for inlet patches in these patients and, if found, attempt ablation with argon plasma coagulation, multipolar electrocoagulation, or radiofrequency ablation. (See "Globus sensation".)

**Diagnosis** — Diagnosis can usually be made based upon visual inspection alone. Biopsies can be obtained if there is doubt about the diagnosis.

**Management** — As noted above, most inlet patches are found incidentally and do not cause symptoms. Thus, treatment is generally required only if complications develop.

Specific risk factors for malignancy are unclear. As a result, consensus has not been reached on the need for surveillance, though the European Society of Gastrointestinal Endoscopy (ESGE) recommended against surveillance for this [106]. There is limited evidence that endoscopic ablation followed by chronic proton pump inhibitor therapy can result in replacement of the inlet patch with normal squamous mucosa [92,105]. The role of ablation remains unclear but

may be an option if a prominent inlet patch is detected in a patient with pronounced globus symptoms.

**Parakeratosis** — Esophageal parakeratosis appears endoscopically as whitish, membranous, linear plaques ( picture 7) that do not stain (turn brown) when sprayed with Lugol's solution [107,108]. Biopsies reveal epithelial acanthosis, basal hyperplasia, and a dense, compact layer of parakeratosis, often featuring cytoplasmic eosinophilia and pyknotic nuclei, covered by an outer layer of nonnucleated squamous cells [107].

The clinical significance of esophageal parakeratosis is unclear; associations with esophageal and head and neck carcinoma have been reported, but the areas of parakeratosis themselves have not been shown to give rise to neoplasia [109,110]. As an example, a prospective study of 400 patients with newly diagnosed squamous cell cancers of the head and neck found that close to 40 percent of patients had esophageal parakeratosis but none of the cancers clearly arose from these areas [109]. Another report described an association with submucosal fibrosis of the oral cavity, particularly in smokers and those who chewed betel nuts [110]. Because of the association with squamous cell cancer, we suggest careful evaluation of the esophagus and head and neck.

**Esophagitis dissecans superficialis** — Esophagitis dissecans superficialis is a rare occurrence characterized by sloughing of the entire length of esophageal mucosal epithelium ( picture 8). The sloughed mucosal lining usually appears as a tubular cast within or tethered to the esophageal lumen ( picture 9 and picture 10). It is most commonly seen in association with desquamating dermatologic disorders, particularly pemphigus vulgaris, where it has been described in up to 5 percent of patients [111,112]. It has also been described as a complication of rigid endoscopy with esophageal dilation, ingestion of oral bisphosphonates or ferrous sulfate tablets, celiac disease, and the use of certain immunotherapies [113-118]. We have seen it following the accidental ingestion of a fish bone.

## CYSTIC LESIONS

**Bronchiogenic and enterogenous cysts** — Cystic lesions of the esophagus were present in 1 in 8200 patients based upon review of four autopsy studies that included a total of close to 50,000 patients [119]. Most esophageal cysts arise from mediastinal structures (ie, bronchiogenic or enterogenic), although there have been a few case reports of esophageal teratomas [120,121].

Bronchiogenic and enterogenic cysts are distinguished mainly by content and lining of the cysts. Bronchogenic cysts contain milky white material and are lined by columnar epithelium

containing smooth muscle, hyaline cartilage, or a focus of seromucous glands, while enterogenous cysts are filled with greenish mucous and are lined with intestinal or gastric epithelium [122].

Both types of cysts form as the result of abnormal budding of the primitive tracheobronchial tree. They can be periesophageal, but are more commonly intrapulmonary or found in the mediastinum [119]. Their average size is 4 cm [119].

On endoscopy or barium studies, the cysts appear as a protruding, submucosal mass covered by normal mucosa. If suspected based upon these studies, endoscopic ultrasound can confirm the diagnosis. Because there is no clinical relevance to differentiating these two types of cysts, we do not advocate obtaining a fine-needle aspiration biopsy, especially since it can cause infection of the cysts or bleeding.

Large cysts can cause dysphagia, which generally requires surgical resection [122]. Neither of these cysts has been reported to harbor neoplastic change and thus no treatment or surveillance is necessary in patients who are asymptomatic.

**Esophageal duplication/duplication cysts** — Duplication cysts are congenital anomalies that arise during early embryonic development. They are most frequently found in the proximal small intestine, although they can also be found in the esophagus, stomach, and colon. (See "Endoscopic ultrasound for the characterization of subepithelial lesions of the upper gastrointestinal tract".)

Duplication cysts of the esophagus have been estimated to occur in 1 out of 8000 live births [119]. They are defined by three criteria [119]:

- They lay within the esophageal wall
- They are covered by two muscle layers
- They contain squamous epithelium or a lining compatible with that found in the embryonic esophagus

Up to one-third of these cysts contain heterotopic gastric mucosa and they can also contain heterotopic pancreatic mucosa or mucosa consistent with Peyer's patches [123,124].

Approximately 80 percent of cysts do not communicate with the esophageal lumen; the others generally run parallel to and communicate with the esophageal lumen [119]. They most commonly occur on the right lateral aspect of the esophagus due to the dextrorotation of the stomach during embryogenesis [119].

Esophageal duplication cysts frequently cause symptoms, unlike duplication cysts in other parts of the gastrointestinal tract. As a result, approximately 80 percent are diagnosed before the age of 2 [123]. Because of compression of adjacent structures, they most commonly cause dysphagia (70 percent), epigastric pain (20 percent), retrosternal pain (10 percent) [119], and respiratory symptoms including cough, stridor, and wheezing. Hematemesis has also been reported in a patient with a communicating, tubular duplication [125]. Surgical resection is generally required for cysts that cause symptoms. Malignancy within these cysts is rare but has been reported [126-128]. Because of the low risk, we do not perform surveillance endoscopy.

## SUMMARY AND RECOMMENDATIONS

- Benign esophageal lesions can be classified as raised, flat, or cystic. Most can be diagnosed based upon their endoscopic appearance, findings on routine biopsy and, in the case of submucosal lesions, by endoscopic ultrasound.
- Management is generally based upon the confidence in the diagnosis and whether the lesion is causing symptoms. However, a few of the lesions have the potential to progress to malignancy or may be a marker for an increased risk of malignancy, while not directly having malignant potential. Our approach to such lesions is summarized below.
  - Esophageal schwannomas have a low risk of malignancy. Symptomatic lesions require surgical enucleation or partial esophagectomy.
  - Esophageal parakeratosis has been associated with esophageal and head-and-neck carcinoma. As a result, we suggest careful examination of the esophagus and refer patients to an otolaryngologist for examination of the mouth and pharyngeal structures. (See 'Parakeratosis' above.)
  - Esophageal papillomas have been associated with esophageal squamous cell carcinoma in some reports. As a result, we suggest removing these lesions endoscopically. (See 'Papillomas' above.)
  - Esophageal adenomas are almost always found in areas of Barrett's esophagus. Thus, their management is usually based upon the management Barrett's esophagus with dysplasia. (See 'Adenomas' above and "Barrett's esophagus: Surveillance and management".)
  - We perform endoscopically resection of all granular cell tumors because, while rare, these lesions have malignant potential. Small lesions can often be removed with a

biopsy forceps, but lesions larger than 1 cm typically require endoscopic mucosal resection. (See 'Granular cell tumors' above.)

- The risk of malignancy is probably too low to warrant surveillance or prophylactic measures for inlet patches and duplication cysts and thus we suggest **not** performing surveillance in such patients. (See 'Inlet patch' above and 'Esophageal duplication/duplication cysts' above.)

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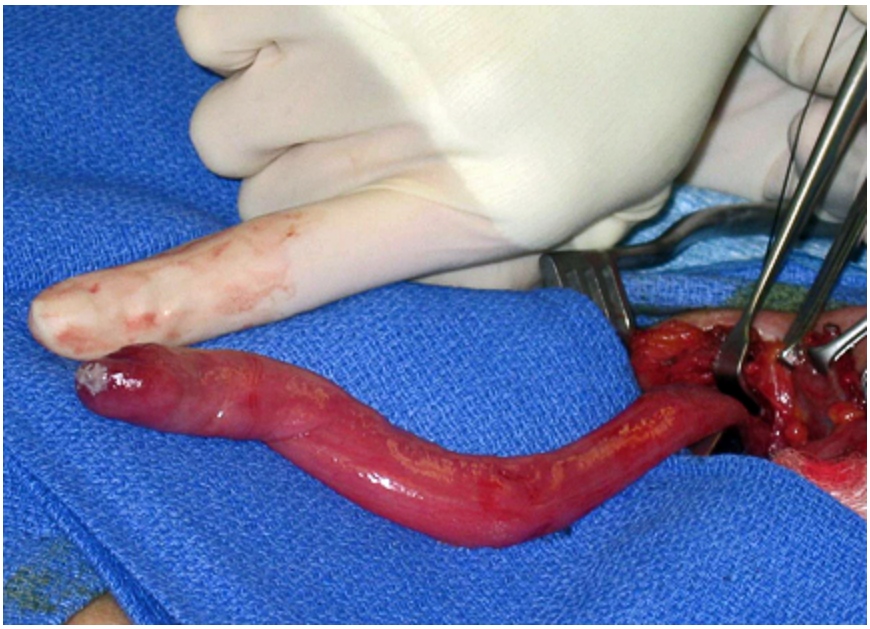
Topic 2253 Version 24.0

## GRAPHICS

### Esophageal fibrovascular polyp



Intraoperative view of an esophageal fibrovascular polyp before resection.

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*Courtesy of Amy Lo, MD, Daniel Wild, MD, and Moises Guelrud, MD.*

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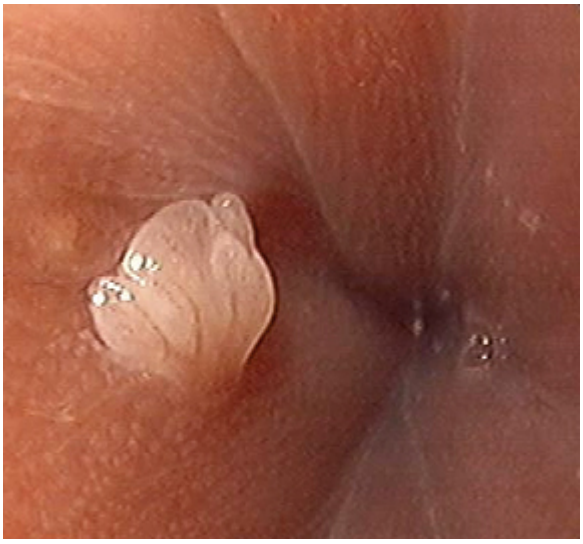
Graphic 70481 Version 2.0

## Esophageal papilloma



Endoscopic view of an esophageal papilloma appearing as an isolated, wart-like exophytic projection.

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*Courtesy of Daniel Wild, MD, and Moises Guelrud, MD.*

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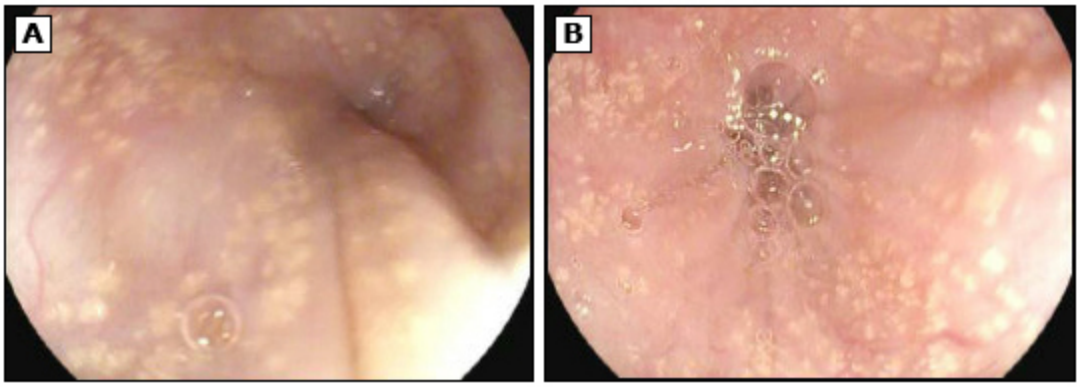
Graphic 63177 Version 2.0

## Heterotopic sebaceous glands




Endoscopic view of dense clusters of heterotopic sebaceous glands in the esophagus.

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*Courtesy of Daniel Wild, MD.*

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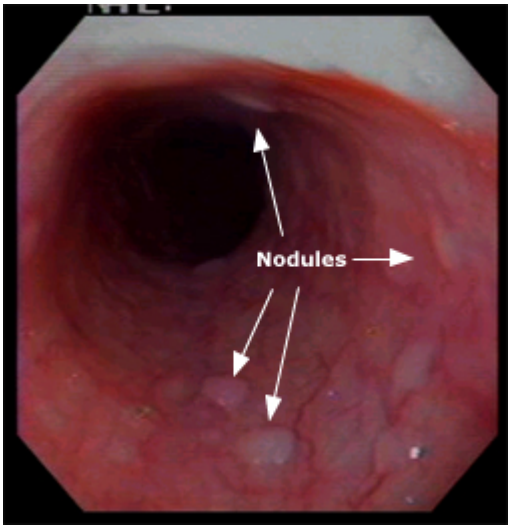
Graphic 140855 Version 3.0

## Esophageal glycogen acanthosis




Endoscopy revealed multiple elevated gray-white nodules (white arrows) in the upper and middle third of the esophagus. A long segment of salmon colored mucosa was seen in the distal esophagus.

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*Courtesy of Andres Gelrud, MD and Kenneth Falchuk, MD.*

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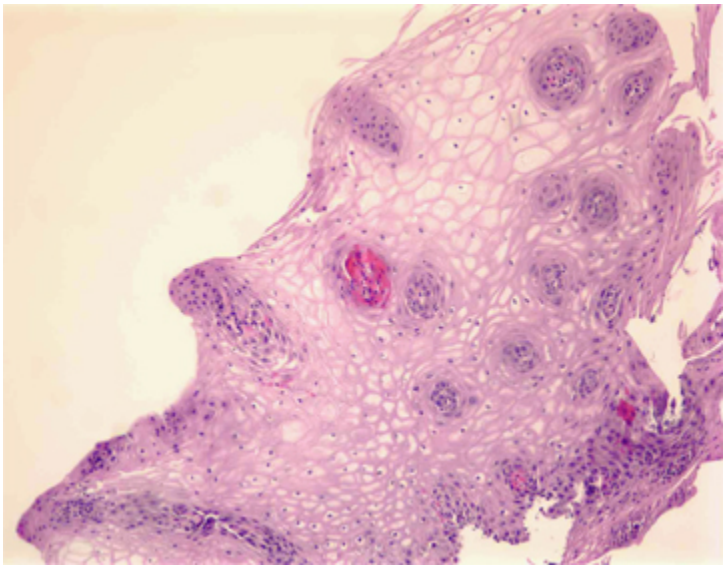
Graphic 56274 Version 2.0

## Glycogen acanthosis



Medium power view of a biopsy specimen from the upper and mid esophagus revealed glycogen filled squamous cells, known as glycogenic acanthosis, a benign condition with no clinical significance.

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*Courtesy of Donald Antonioli, MD.*

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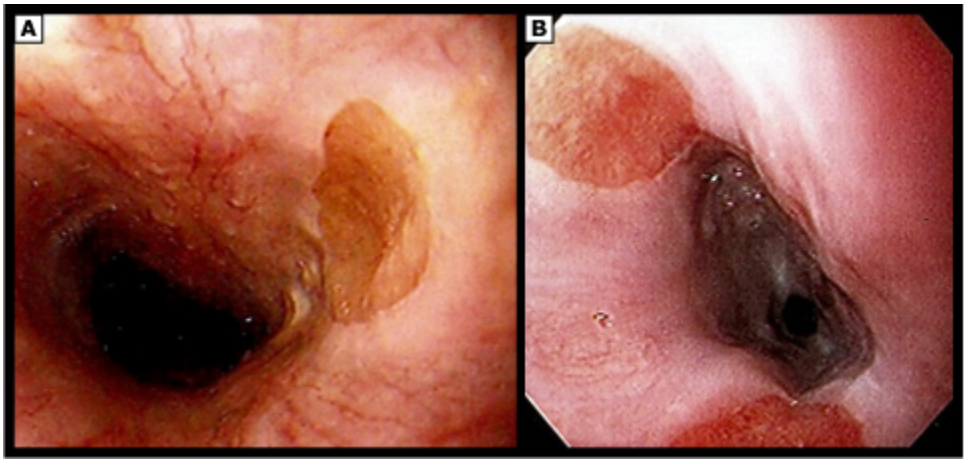
Graphic 67987 Version 1.0

## Esophageal inlet patch




Endoscopic views showing inlet patches in two different patients. Both show well-circumscribed areas of salmon-colored mucosa in the proximal esophagus. The patient on the right had two areas (11 and 6 o'clock).

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*Courtesy of Daniel Wild, MD, and Moises Guelrud, MD.*

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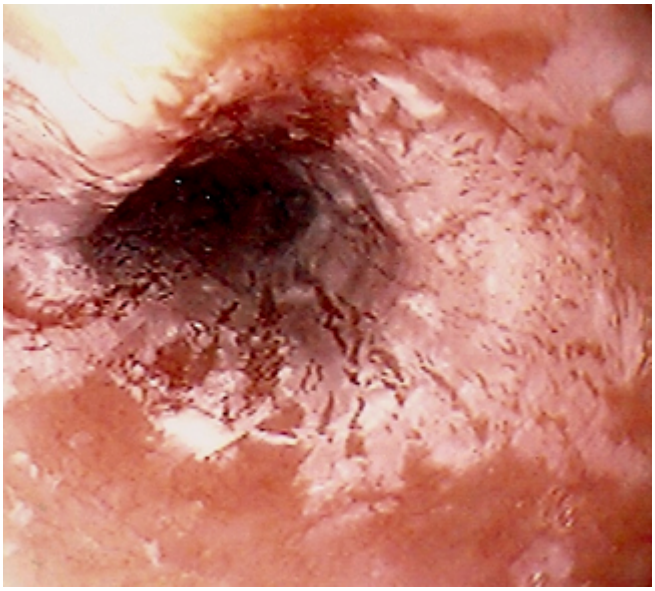
Graphic 57315 Version 1.0

## Esophageal parakeratosis



Endoscopic view of esophageal parakeratosis showing a characteristic whitish, membranous plaque.

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*Courtesy of Daniel Wild, MD, and Moises Guelrud, MD.*

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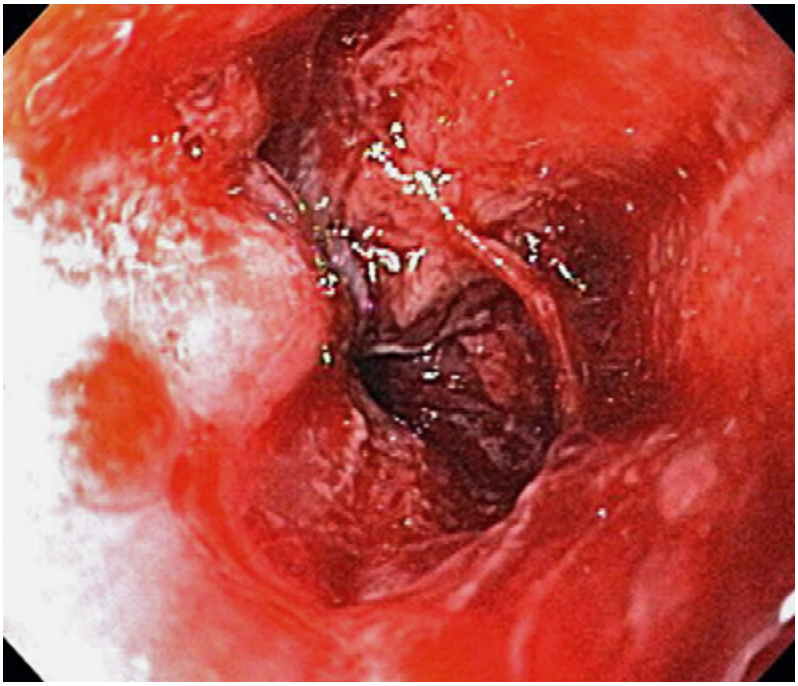
Graphic 70116 Version 1.0

## Esophagitis dissecans superficialis



Upper endoscopy showing a denuded esophagus in esophagitis dissecans superficialis.

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*Courtesy of S. Ian Gan, MD, and Daniel Wild, MD.*

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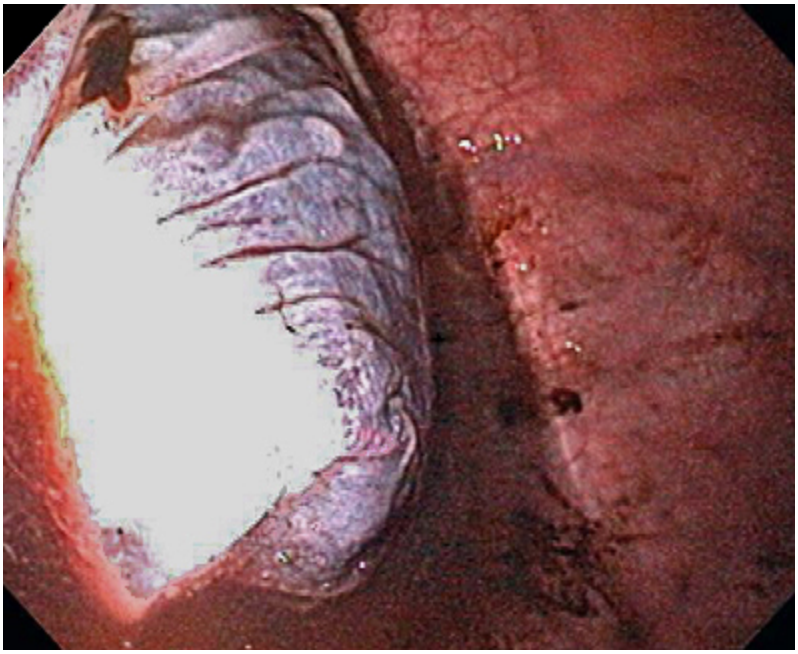
Graphic 68821 Version 2.0

## Esophagitis dissecans superficialis



Upper endoscopy showing an esophageal cast still tethered to gastroesophageal junction in a patient wth esophagitis dissecans superficialis.

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*Courtesy of S. Ian Gan, MD, and Daniel Wild, MD.*

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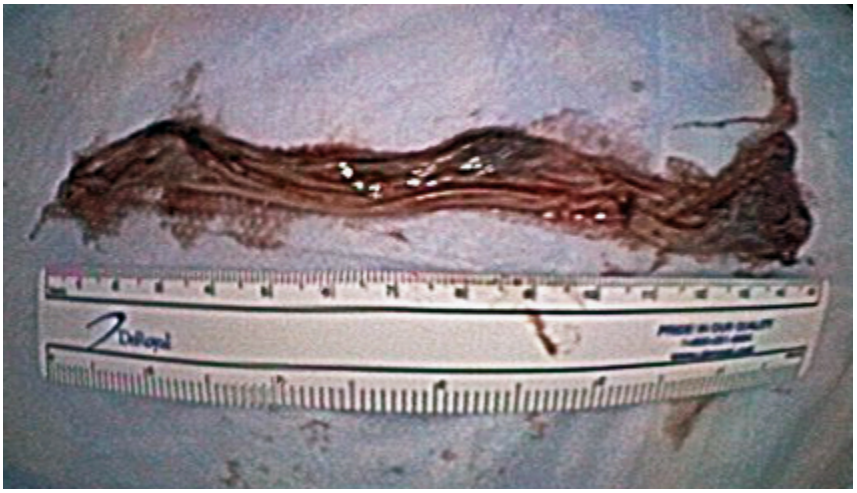
Graphic 78182 Version 2.0

## Esophagitis dissecans superficialis



Gross specimen of an esophageal cast in a patient with esophagitis dissecans superficialis after endoscopic removal.

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*Courtesy of S. Ian Gan, MD, and Daniel Wild, MD.*

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Graphic 82733 Version 2.0

## Contributor Disclosures

**Daniel Wild, MD** No relevant financial relationship(s) with ineligible companies to disclose. **John R Saltzman, MD, FACP, FACG, FASGE, AGAF** No relevant financial relationship(s) with ineligible companies to disclose. **Shilpa Grover, MD, MPH, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy

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