DYSPHAGIA

Dysphagia is a subjective sensation of difficulty or abnormality of swallowing.

Other terms to differentiate:

- Odynophagia: pain with swallowing.
- Globus sensation: nonpainful sensation of a lump, tightness, foreign body or retained food bolus in the pharynx.

CLINICAL FEATURES

There are a number of key questions to ask:

1. Was the difficulty swallowing solids and liquids from the start? Yes: motility disorder or pharyngeal causes

No: solids then liquid: suspect a stricture

2. Is it difficult to make the swallowing movement?

Yes: suspect bulbar palsy (esp if he coughs on swallowing)

3. Is swallowing painfull (odynophagia)?

Yes: suspect cancer, esophagitis, achalasia, esophagial spasm

4. Is the dysphagia intermittent or is it constant and getting worse?

Intermittent: suspect esophageal spasm

Constant and worsening: suspect malignant stricture

5. Dose the neck bulge or gurgle on drinking?

Yes: suspect a pharyngeal pouch

Questions to ask patients with dysphagia

Do you have problems initiating a swallow or do you feel food getting stuck a few seconds after swallowing? (Helps distinguish oropharyngeal from esophageal dysphagia.)

Do you cough or choke or is food coming back through your nose after swallowing? (Coughing, choking, or nasal regurgitation suggests aspiration and oropharyngeal dysphagia.)

Do you have problem swallowing solids, liquids, or both? (Liquids, not solids, suggests a motility disorder; solids progressing to liquids suggests a benign or malignant obstruction.)

How long have you had problems swallowing and have your symptoms progressed, remained stable, or are they intermittent? (Rapidly progressive dysphagia is concerning for malignancy.)

Could you point to where you feel food is getting stuck? (Ability to localize source of dysphagia is unreliable; best with oropharyngeal dysphagia.)

Do you have other symptoms such as loss of appetite, weight loss, nausea, vomiting, regurgitation of food particles, heartburn, vomiting fresh or old blood, pain during swallowing, or chest pain?

Do you have medical problems such as diabetes mellitus, scleroderma, Sjögren's syndrome, overlap syndrome, AIDS, neuromuscular disorders (stroke, Parkinson's, myasthenia gravis, muscular dystrophy, multiple sclerosis), cancer, Chagas' disease or others?

Have you had surgery on your larynx, esophagus, stomach, or spine?

Have you received radiation therapy in the past?

What medications are you using now (ask specifically about potassium chloride, alendronate, ferrous sulfate, quinidine, ascorbic acid, tetracycline, aspirin and NSAIDs)? (Pill esophagitis can cause dysphagia.) Dysphagia can be classified as oropharyngeal dysphagia or esophageal dysphagia.

- Oropharyngeal or transfer dysphagia is characterized by difficulty initiating a swallow. Swallowing may be accompanied by coughing, choking, nasopharyngeal regurgitation, aspiration, and a sensation of residual food remaining in the pharynx.
 - Liquid more than solids.

- Causes can be neurologic or muscular and include stroke, Parkinson's disease, myasthenia gravis, prolonged intubation, and Zenker's diverticula.

Esophageal dysphagia is characterized by difficulty swallowing several seconds after initiating a swallow and a sensation of food getting stuck in the esophagus. Should be characterized according to types of food that produce symptoms (ie, solids, liquids or both), the time course (ie, progressive or intermittent), severity, and associated symptoms (ie, weight loss, heartburn, or regurgitation)

- If clue to obstruction, usually involves solids more than liquids (strictures, Schatzki rings, webs, carcinoma) and is progressive.

- If clue to a motility disorder (achalasia, scleroderma, esophageal spasm), usually presents with both liquid and solid dysphagia.

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Diagnosis of dysphagia



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A detailed and accurate history is the mainstay of clinical assessment in patients who complain of dysphagia.

The esophagus is a deep-seated structure that does not lend itself to direct physical examination.

- A general look at the patient should be done looking for pallor, cachexia and characteristic features for example in a patient scleroderma features. (CREST)
- Head and neck exam is performed.
- We examine the thyroid gland, lymph nodes or any mass that can cause an extrinsic compression on the esophagus and thus leading to dysphagia.

- A neurological assessment should be performed it may reveal signs of cerebrovascular accident, myasthenia gravis, or Parkinson's disease.
- A careful examination of the cranial nerves may demonstrate deficits contributing to oropharyngeal dysphagia.
- A complete physical exam should be done, of the chest and abdomen.

Diagnostics

Diagnostics

- Many diagnostic tests can be used in assessing dysphagia. It could be endoscopic, radiologic or manometric diagnostics.
- When to use these investigations depends on the history and physical exam.
- In cases of suspected or confirmed caustic ingestion the first test is emergency upper flexible endoscopy to assess the extent of the injury.

Barium Swallow

- In most cases of dysphagia the barium swallow is the ideal first test as its readily available, cost effective and quick.
- It's performed after an overnight fast. The patient swallows solution containing barium sulfate, followed by a single static image.
- It shows the size and shape of the esophagus as well as contractions and peristaltic waves.
- What can be gained: anatomic relations, esophageal transit patterns and if there is presence of mass lesions or diverticula.



Modified Barium Swallow (Videofluoroscopy)

- ► A real time contrast-enhanced imaging.
- Patient swallow the barium solution while a continuous Xray beam is focused on the esophagus.
- Ideal for continuous evaluation of swallowing mechanism.
- This is the test of choice for oropharyngeal dysphagia.



Upper Endoscopy

- Upper endoscopy allows for visual assessment of the mucosa.
- It can be diagnostic for example biopsies and brushings and therapeutic such as dilataions.





Endoscopic Ultrasound (EUS)

- EUS is an excellent tool for assessing the depth of tumor invasion, as well as locating and sampling potentially affected lymph nodes.
- It provides a detailed view of the layers of the esophageal wall.



Interface
 Mucosa
 Submucosa
 Muscularis – inner layer
 Muscularis – outer layer



24-hour pH monitoring

- It's used when reflux disease is suspected.
- A gold standard in diagnosing GERD.
- Measurement of esophageal pH over 24-48 hours using a transnasal catheter.
- Documentation of relevant events by the patient.



Esophageal Manometry

- It measures the propagation, speed, and vigor of peristaltic waves via an esophageal catheter fitted with pressure sensors.
- Used in dysmotility disorders.





CT-scan and MRI

- In cases where extrinsic compression is suspected or demonstrated CT or MRI may be useful in identification of malignant masses or vascular anomalies.
- ▶ They are also useful in staging if esophageal cancer is the cause of dysphagia.

Dysmotility Syndromes

- Motility disorders can be considered within a spectrum that includes:
 - o Diffuse esophageal spasm (DES),
 - Nutcracker esophagus, and
 - Hypertensive LES
- These disorders are traditionally considered separate entities; however, the manometric findings and the mainstays of medical treatment are similar.

Diffuse Esophageal Spasm

DES has unknown etiology.

Nonperistaltic spontaneous contractions of the esophageal body – several segments of the esophagus contract simulatenously and prevent appropriate advancement of the food bolus It is characterized in 50% of patients by intermittent
dysphagia to solids and liquids.

Up to 5% of patients with unexplained chest pain are found to have DES on manometric testing.

Clinical features:

chest pain that mimics angina and may radiate to the jaw, arms, back. Dysphagia is common; however, regurgitation of food is rare. **Diagnosis:** esophageal manometry is diagnostic --> simultaneous multiphasic repetitive contractions after swallowing, the sphincter response may be normal. Upper GI barium swallow (corckscew esophagus) in 50%, which represents multiple simulatenous contractions.

Evidence for DES on manometry includes:

- periodic prolonged,
- o multipeaked,
- high-amplitude contractions in more than one in five wet swallows, with observation of normal peristalsis in intervening periods.

Incomplete LES relaxation or hypertensive LES may also be observed.



<u>DES</u>

The classic corkscrew appearance of the esophagus is evident in this barium study in a middle-aged patient presenting with dysphagia and intermittent chest pain. Treatment:

In general, there is no completely effective therapy- treatment failure rates are high Medical treatment involves nitrates and calcium channel blcokers (decrease amplitude of contractions). Tricyclic antidepressants may provide symptomatic relief. Esophagomyotomy is usually not perfromed and its efficacy is controversial.

Nutcracker Esophagus

- Nutcracker esophagus presents more commonly with chest pain rather than dysphagia.
- Manometry also forms the mainstay of diagnosis: a <u>normal peristaltic pattern</u> is noted, with extremely increased pressure amplitudes of more than 180 mm Hg.
- □ In contrast to DES, normal peristalsis is not observed within trains of high-pressure waves.
- □ Barium swallow is of normal appearance

Hypertensive LES

□ Hypertensive LES may be found in isolation but often coexists with other dysmotility syndromes.

Resting pressures at the LES by manometry are found to be 45 mm Hg or greater.

Treatment

- Treatment for nutcracker esophagus, and hypertensive LES is based on smooth muscle relaxation using nitrates such as isosorbide dinitrate or calcium channel blockers such as diltiazem.
- Balloon dilatation may be effective for isolated hypertensive LES.

Esophagitis

- Eosinophilic esophagitis is an immune-mediated chronic inflammatory disorder of the esophagus.
- Rare, most commonly in men 20-40 years old.
- Strongly associated with allergies (environmental, food, asthma)
- Most common symptom: intermittent dysphagia to solids with food impaction.

Barium study: often normal

Manometry: hyperperistalsis in around half of the patients

Endoscopy: classically, scalloped appearance with ridges.

Definitive diagnosis: esophageal biopsy showing dense eosinophilic infiltrates in the esophageal epithelium.



- Treatment:
- Avoidance of potential allergens.
- Glucocorticoids (Fluticasone or Budnesone)
- Some patients benefit from PPI therapy



Pill- Induced Esophagitis

- Caused by direct mucosal injury to the esophagus by the drug.
- Presentation: sudden onset, intermittent, and self-limiting odynophagia, dysphagia, or retrosternal pain. The onset of the symptoms may occur within a few hours to one month after ingestion of the drug.

Pill- Induced Esophagitis

- Risk factors:
- Taking the pill before lying down
- > When pills are taken with little or no water
- Large size of the pill
- Altered esophageal anatomy
- Increasing age.
Pill- Induced Esophagitis

Seen especially in patients taking:

- Doxycycline
- ASA
- o NSAIDS
- Iron
- Bisphosphonates

Pill- Induced Esophagitis

Diagnosis can be made by history alone.

- Sometimes we need to perform EGD in patients with hematemesis, abdominal pain, and weight loss or patients with persistent symptoms after one week of discontinuation of the offending drug.
- Treatment is by stopping the offending agent, reassurance and patient education on how to take the pills appropriately.

Infections Esophagitis

□ Infectious causes of dysphagia associated with odynophagia include intrinsic infections such as esophageal candidiasis, herpetic infections, and cytomegaloviral illness, HIV, TB. □ Rarely, extrinsic infection of the esophagus (originating in neighboring necrotizing mediastinal lymph nodes) can occur, causing dysphagia, odynophagia, and other potentially disastrous complications.

- Infection with Candida species is the most common type of infectious esophagitis.
- Candidal esophagitis is typically seen in immunocompromised patients, such as patients with HIV who have advanced immunosuppression, those who have hematologic malignancies or are hematopoietic cell transplant recipients, and those who have solid organ cancer and are receiving cytotoxic chemotherapy.

Other conditions associated with an increased incidence of Candida esophagitis include esophageal stasis, alcoholism, malnutrition, and advanced age. Occasionally, Candida esophagitis can occur in otherwise healthy individuals with no underlying esophageal or systemic disease.

- *The hallmark of esophageal candidiasis is odynophagia or pain on swallowing.
- Patients with esophagitis often have evidence of oropharyngeal disease (thrush) on exam; however, the absence of thrush does not preclude the diagnosis.

- **The diagnosis of Candida esophagitis is usually made through endoscopy with a biopsy of white mucosal plaque-like lesions. The plaques may be localized or diffuse and usually are located in the upper or mid esophagus. Histology demonstrates the presence of yeast and hyphae invading mucosal cells with a culture confirming Candida.
- **Treatment: Candida esophagitis is usually self-limiting, and most patients have a marked response to treatment with antifungal agents.



Extensive esophagitis due to Candida albicans in a patient with AIDS.

- Herpes simplex virus type I is the second most common cause of infectious esophagitis.
- Herpes esophagitis usually manifests as multiple, small, superficial ulcers in the upper esophagus or midesophagus on an otherwise normal background mucosa

Herpes esophagitis



Barium swallow in a patient with Herpes simplex esophagitis shows a single aphthous ulcer in the distal esophagus (arrow) on a background of normal esophageal mucosa. Other viruses, such as cytomegalovirus and HIV, and medications may also produce this appearance.

Herpes esophagitis

Upper endoscopy showing shallow ulcers in the esophagus



Upper endoscopy showing multiple discrete shallow ulcers in the distal third of the esophagus.

- Asymptomatic cytomegalovirus (CMV) infection is common worldwide.
- Unlike herpes esophagitis, CMV esophagitis almost never occurs in immunocompetent patients, and the vast majority of affected individuals are found to have AIDS.
- CMV esophagitis is typically manifested by 1 or more giant and relatively flat ulcers, sometimes associated with small satellite ulcers

CMV esophagitis



► HIV Esophagitis

- Giant esophageal ulcers have been described in the esophagus in patients with AIDS in whom no other infectious etiology for the ulcers can be found.
- I or more giant, flat ulcers (>1 cm in diameter) of the esophagus. This finding is sometimes associated with a cluster of small satellite ulcers. The ulcers are often surrounded by a radiolucent rim of edema.



Tuberculosis esophagitis occurs primarily in patients with advanced pulmonary or mediastinal tuberculosis or in immunocompromised patients who have disseminated tuberculosis or another mycobacterial disease. The esophagus is usually involved by erosion of the involved mediastinal lymph nodes abutting the esophagus. Barium studies or computed tomography (CT) scans may reveal extrinsic compression or displacement of the esophagus due to enlarged collections of nodes in the adjacent mediastinum. In some patients, traction diverticula may develop in the upper esophagus or midesophagus.



Conclusion

- Evaluation of the patient presenting with dysphagia represents a challenge for the surgeon.
- □ A careful history is key in determining likely etiologies.
- The barium swallow should be the first diagnostic test to be considered, endoscopy to follow.
- Esophageal manometry represents the gold standard for diagnosing benign, functional (motor) disorders.
- Treatment is varied and depends on the etiology of the dysphagia.

Achalasia

- Ninety-eight percent of all cases of achalasia are idiopathic.
- The disease is thought to result from a loss of inhibitory neurons in the Auerbach plexus, altering neural input to the LES and preventing normal relaxation.
- Achalasia affects females and males equally at a rate of 1 per 100,000 individuals per year.
- The usual presentation is between 20 and 50 years, but it has been described in all age groups.
- The disease is slowly progressive, and presentation is typically at advanced stages.

Symptoms - Achalasia

- Symptoms include progressive dysphagia to both solids and liquids, accompanied by regurgitation of food particles, chest pain, and weight loss.
- GERD-like symptoms were present in up to 48% of patients in a study of 32 patients; these symptoms are a consequence of stasis esophagitis (secondarily to fermentation of retained food) rather than reflux of gastric acid.

- Plain x-rays may reveal an air-fluid level in the distal esophagus, and a barium swallow will demonstrate a dilated and atonic esophagus with the pathognomonic "bird's beak" narrowing of the gastroesophageal junction (GEJ).
- Long-standing achalasia may manifest with an extremely dilated and tortuous esophagus (often described as a sigmoid esophagus).



Barium swallow demonstrates the proximal dilatation and classic "bird's beak" narrowing at the esophagogastric junction, consistent with achalasia, in a 22- year-old woman being evaluated for dysphagia.



Barium study demonstrates dilated esophagus with rightsided deviation and tortuous course of the distal esophagus. <u>Treatment</u> most often involves resection of diseased esophagus with conduit interposition.

Achalasia

 Manometric findings of aperistalsis and failure of LES relaxation are key in establishing the diagnosis.

 Resting LES pressures may be normal or elevated.
Endoscopic assessment is required to visually assess mucosal appearance to rule out cancer. (it can increase the risk for esophageal cancer).

Treatment Modalities

- AIM: Treatment modalities for achalasia must achieve enhanced LES compliance and lower resting LES pressures.
- *Medical management* with calcium channel blockers or nitrates has no meaningful benefit.
- Endoscopic management includes endoscopically injected botulinum toxin, or balloon dilatation, to mechanically disrupt the lower esophageal muscle fibers.

Recurrent dysphagia (up to 50%) has been noted in some studies at 5 years after balloon dilatation, with a 5% periprocedural risk of esophageal rupture.

 In comparison, a laparoscopically performed "<u>Heller</u> <u>esophagomyotomy with partial anterior (Dor)</u> <u>fundoplication</u>" is considered to be the standard of care in terms of both durable outcomes (90 to 95% resolution of dysphagia) and low complication rates. Long-standing achalasia is a risk factor for esophageal squamous cell carcinoma, and tumors of the GEJ may present with symptoms similar to those of achalasia

Secondary Motor Disorders

- In secondary dysmotility syndromes, the esophageal symptoms are a manifestation of a generalized systemic process.
- The etiology is thought to be progressive neuropathy and fibrosis.
- Common diseases associated with secondary dysmotility include:
- 1. •Rheumatologic syndromes, such as scleroderma
- 2. •Diabetes mellitus

Webs

- Esophageal webs are one or more thin horizontal membranes of squamous epithelium within the upper and mid esophagus. In contrast to rings, they rarely encircle the lumen but protrude from the anterior wall extending laterally but not posteriorly.
- Congenital webs are rare and usually restricted to the pediatric population.
 - These are located in the middle and lower thirds of the esophagus.
 - Acquired webs are normally located in the postcricoid cervical esophagus and are mostly asymptomatic.

Etiologies for acquired webs include iron deficiency anemias (Plummer-Vinson)and dermatologic diseases, This condition has also been associated with celiac sprue.

Webs are twice as common in female patients.





Symptoms

- This syndrome (iron deficiency anemia and postcricoid esophageal webs or strictures) has been associated with an increased incidence of squamous cell carcinoma.
- Biopsy of webs in patients with this syndrome may show reactive epithelial changes with basal cell proliferation, parakeratosis, chronic inflammation, and fibrosis.
- This syndrome is most common in Scandinavian women with poor nutrition.
- Dysphagia occurs intermittently with solids, the patients becomes symptomatic when the diameter of the orofice is less than 1.3 cm

Treatment

Diagnosis is by barium swallow, and treatment involves mechanical dilatation using <u>Savary bougies</u> or endoscopic balloons.

Underlying anemias and dermatologic conditions should also undergo assessment and appropriate treatment "there is no difference between bougie and balloon dilation of BESs regarding symptomatic relief, recurrence rate at 12 months, bleeding, and perforation. Patients undergoing balloon dilation present less severe post-procedure pain."



Rings

Esophageal rings are typically located in the lower third of the esophagus.

□ Two types are typically described:

- Muscular rings and
- Mucosal or Schatzki rings.

Muscular rings are rarely associated with dysphagia and are often found incidentally in children undergoing barium swallow for other reasons.

Schatzki Rings

- Schatzki rings are located at the Z-line (squamocolumnar junction) and are almost always seen in patients with GERD; consequently, the upper surface of a Schatzki ring is covered by squamous epithelium, whereas the lower surface is covered by columnar epithelium.
- Associations with eosinophilic esophagitis and GERD have been proposed.
- Diagnosis and treatment are as for esophageal webs.



Schatzki ring

Barium swallow demonstrates a ring in a middle-aged man with severe gastroesophageal reflux disease symptoms and recent-onset dysphagia
Peptic Stricture

- Peptic stricture was previously found in up to 10% of patients with GERD(chronic) and represents the end stage of reflux associated ulcerative esophagitis.
- The incidence of peptic strictures has been drastically reduced with the increased use of effective antireflux medications, chiefly the proton-pump inhibitors (PPIs).
- Symptoms are described as progressive in nature and involve initial solid food dysphagia, progressing to liquid dysphagia.

Peptic Stricture

Initial assessment is by barium swallow followed by Upper endoscopy.

Peptic strictures are short segment, circumferential, and located at the squamo-columnar junction.

A high index of suspicion for concomitant Barrett esophagus or frank cancer at the site of stricture must be kept.

Peptic Strictures

- Treatment of isolated peptic strictures includes acid suppression and endoscopic dilatation.
- In the past, peptic strictures were usually an indication for surgical correction; however, recent clinical experience with careful repeated dilatations combined with effective acid suppression therapy (using PPIs) has rendered surgical treatment an uncommon occurrence.





Diverticulae

- Diverticulae are classified according to the degree to which the esophageal wall is involved in the outpouching:
 - <u>True diverticulae</u> involve all layers of the esophageal wall,

• <u>False diverticulae</u> involve only the mucosal layer.

Both types of diverticulae can also be classified by the mechanism underlying their formation:

- <u>True diverticulae</u> usually form in the mid-esophagus near the tracheal bifurcation and are most often related to extrinsic traction from extramural inflammation in adjacent mediastinal lymph nodes. These are also referred to as "**Traction**" type diverticulae.
- <u>False diverticulae</u> relate to dysmotility and consist of the mucosa being extruded through external muscular layers above a high-pressure zone and are thus "**Pulsion**" type diverticulae.

Zenker diverticulum

- Pharyngoesophageal diverticulae (Zenker diverticulum) are the most common diverticulae observed.
- □ These pulsion type false diverticulae arise in the Killian triangle and are located in the upper third of the esophagus just superior to the cricopharyngeus muscle .



► The basis for the formation of a Zenker diverticulum is pharyngo- cricopharyngeal dyscoordination.

When the cricopharyngeal sphincter fails to immediately and fully relax during swallowing, the pharyngeal pump mechanism produces extremely high pressures.

► As a result of these high pressures, progressive bulging of the mucosa occurs in the posterior midline through a potential gap between the pharyngeal constrictors and the cricopharyngeus muscles (the Killian space).

It is typically seen in patients >50 years old .

There has been a long-standing appreciation of an association between the pathologic severity of gastroesophageal reflux and the development of Zenker diverticulae:

• It is thought that repeated exposure of the proximal esophagus and pharynx to low pH refluxate results in cricopharyngeal spasm and loss of normal coordination.

Symptoms

- Symptoms of a cricopharyngeal diverticulum include:
 - o Dysphagia,
 - o Halitosis,
 - Throat discomfort, and chronic cough
 - o a palpable mass, and weight loss
 - Regurgitation of undigested food.

Some patients may suffer from recurrent aspiration pneumonia and in severe cases may develop lung abscesses.

Diagnostic Evaluation

The first diagnostic test should be a barium swallow, which will delineate the size and position of the diverticulum.

Initial assessment by endoscopic means is not recommended as there is an increased risk of perforating the pouch with the endoscope.



<u>Pharyngoesophageal</u> <u>diverticulum</u>

A large Zenker diverticulum is shown in an elderly patient who presented with dysphagia, recurrent pneumonia, and regurgitation

Treatment

Treatment is surgical and must include the division of the cricopharyngeus muscle.

Smaller pouches may be treated by myotomy alone, whereas those larger than 2 cm should be excised.

Endoscopic (transoral) approaches to diverticulostomy have also been described.

Midesophageal diverticulae

Midesophageal diverticulae are not typically associated with dysphagia.

These true diverticulae are formed by traction from extraesophageal inflammation, most often granulomatous disease in subcarinal lymph nodes.

Midesophageal diverticulae are usually asymptomatic, and treatment is focused on the underlying inflammatory process.

Epiphrenic diverticulae

Epiphrenic diverticulae arise in the distal esophagus.

These pulsion-type diverticulae are associated with underlying esophageal dysmotility and are also occasionally an isolated finding.

In the absence of symptoms, expectant management is appropriate.

Surgical Treatment

- When dysphagia is present, surgical management is necessary.
- The surgical approach, should incorporate "triple therapy," which must include:
 - Excision of the diverticulum,
 - An esophageal myotomy, and
 - An antireflux procedure

Cancer

Esophageal cancer typically presents with progressive dysphagia (first solids and then liquids), associated with weight loss, hematemesis. they generally have poor prognosis

Anorexia, and chest pain may also be present.

Locally advanced cancers causing airway fistulization may present with aspiration (swallow-cough sequence) \rightarrow Aspiration pneumonia due to presence of tracheoesophageal or bronchoesophageal fistula

There are two pathologic types: SCC & adenocarcinoma

in the past, SCC accounted for up 90% of cases. However, the incidence of adenocarcinoma has increased dramatically especially in the US, and it now accounts for up to 50% of new cases.

SCC:

Incidence is higher in african american men than in other groups. most common locations are the upper and midthoracic esophagus. About 1/3 may be in the distal 10cm of the esophagus



<u>Mid-esophageal squamous cell</u> <u>carcinoma</u>

- Shown is the classic appearance of a midesophageal squamous cell carcinoma.
- Mucosal irregularity is apparent within the lesion, along with proximal dilatation and shouldering at the upper and lower borders.
- Bronchoscopy confirmed anterior penetration of tumor into the airway mucosa

Risk factors of eSCC:

- Smoking and alcohol use
- **Diet** (nitrosamines, toxin-producing fungi (eg, aflatoxin), red meat, zinc deficiency, low folate intake, low selenium levels, Areca nuts or betel quid)... Higher intake of fruits and vegetables reduces the risk of esophageal SCC
- Certain infections (HPV)
- History of achalasia
- Tylosis (hyperkeratosis of the palms and soles)



Tylosis is a **rare autosomal dominant** disease caused by a mutation in **TEC** (tylosis with esophageal cancer), a **tumor suppressor gene** located on chromosome 17q25. Tylosis is associated with **hyperkeratosis of the palms and soles** and a high rate of esophageal SCC (40% to 90% by the age of 70 years).



Other eSCC risk factors:

- strictures
- Achalasia cardia
- Prior gastrectomy
- Use of oral bisphosphonates
- Drinking scalding-hot liquids (hotter than 65° C [149° F])
- Poor oral hygiene
- Plummer-Vinson syndrome
- Nasopharyngeal carcionma

Adenocarcinoma typically affects the lower 1/3 of the esophageus (at gastroesohageal junction in 80% of cases) more common in men (5:1 men:women)

Risk factors of esophageal adenocarcinoma:

- Chronic GERD (Barrett esophagus)
- Obesity and metabolic syndrome
- Alcohol and Smoking (less common than SCC)
- Achalasia

Staging:

Stage I- tumor invades lamina propria or

submucosa, nodes negative

Stage IIa- tumor invades muscularis propria or

adventitia, nodes negative

Stage IIb- tumor invades up to muscularis propria, positive regional nodes

Stage III- tumor invades adventitia (positive regional nodes) or tumor invades adjacent structures

(positive or negative nodes)

Stage IV- distant metastasis.

Diagnosis: barium swallow

upper endoscopy with biospy and brush cytology for definitive diagnosis (it confirms the diagnosis in 95% of cases) Transesopagheal ultrasound helps determine the depth of penetration of the tumor and is the most reliable test for staging local cancer.

Full metastatic workup (chest/abdomen CT, CXR, bone scan)

Treatment:

Pallaition is the goal in most pateints because the disease is usually advanced at presentation

Surgery (esophagectomy) may be curative for patients with disease in stage 0, 1 or 2A.

Chemotheraphy plus radiation before surgery has been shown to prolong survival more than surgery alone.