Barrett's Oesophagitis

Overview

Acquired premalignant condition 2° to chronic GORD in which normal squamous lining is replaced by a metaplastic columnar epithelium which is visible endoscopically and confirmed histologically. The metaplastic columnar epithelium is at risk of ↑grades of dysplasia → invasive adenoCa.

Epidemiology: Incidence: 1% of the older adult population and in 3- 5% of persons with GORD.

Risk Factors

- M>F, Caucasians, ↑age.
- Chronic GORD
- Hiatus hernia. Size of hernia correlated with length of Barrett's oesophagus.
- Some studies indicate obesity, smoking and alcohol intake as RF.
- Risk factors for progression to adenoCa include male gender, age>45yrs, extended segment (>8cm) disease, duration of reflux history, early age of GORD onset, duodeno-gastrooesophageal reflux, mucosal damage (ulceration and stricture) and family history.
- Interestingly NSAIDs and H. pylori appear to be protective and assoc with a lower risk of developing oesophageal adenocarcinoma.

Presentation

- Maybe asymptomatic. Symptoms of GORD & strictures are ↓ in affected segment.
- Typical case: middle-aged white man with a long hx of GORD &, occasionally, dysphagia.

Investigations

- Endoscopy + biopsy ± endoscopic USS

Management

- The recommended diet is the same as that recommended for patients with GORD.
- Low-grade dysplasia: PPI for 2-3mo & then rebiopsy & surveillance
- High-grade dysplasia: PPI. Ablative therapy (photodynamic therapy, argon plasma coagulation, multipolar electrocoagulation and various forms of lasers) may ↓dysplasia. Unclear if it ↓progression to invasive Ca.
- If high-grade dysplasia persists oesophagectomy or endoscopic ablation / mucosal resection if unfit for surgery, should be considered.
- Anti-reflux surgeries, e.g. Nissen fundoplication, have not been shown to prevent the progression of Barrett's oesophagus to oesophageal cancer.

Complications

- Adenocarcinoma in 2-5% (but 40-50% of patients with severe dysplasia within 5yrs.)

Prognosis

- Majority do not develop oesophageal cancer and will die of other causes.

Screening

- Surveillance endoscopy controversial and not proven to increase survival.
- However, ↑incidence of oesophageal adenoCa, and the poor results of Rx for established adenoCa screening may be considered worthwhile in the future.

Achalasia

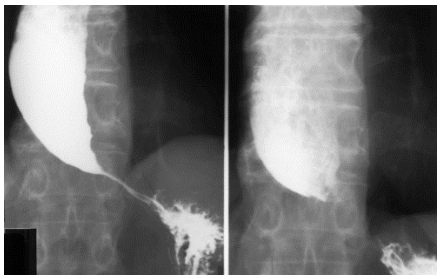
Definition: Absent/incomplete relaxation of the LOS and impairment of oesophageal peristalsis.

Pathophysiology: Acquired aganglionic segment/s of distal oesophagus of unknown aetiology.

Causes: Idiopathic. Similar condition occurs (without aganglionic segment) in Chagas' disease, intestinal pseudo-obstructive syndrome, as a manifestation of paraneoplastic neural dysfunction, and secondary to oesophageal amyloidosis, DM.

Epidemiology: Annual incidence of ~1/100000. All ages, most common early to mid-adulthood.

Clinical Features: Dysphagia with solids, regurgitation (± risk of aspiration), cramping chest pain in early, hypercontracting phase, heartburn, wt loss.



Investigations: CXR (?aspiration, may show dilated oesophagus). Barium swallow may be normal or show dilated oesophagus tapering smoothly to closed LOS. Oesophageal manometry is sensitive for motor dysfunction. Endoscopy/CT to exclude malignancy.

Differential Diagnosis: Ca oesophagus, Ca stomach/cardia, oesophageal stricture, Plummer-Vinson syndrome, GORD, pseudochalasia.

Management: CCB, nitrates & β-blockers not often effective. Balloon dilatation often needs repeating, SE: 25% failure, 5% perforation risk. Endoscopic injection of botulinum toxin. Oesophagomyotomy best but 10% risk of GORD.

Prognosis: Excellent if treated before major dilatation. 2-7% risk of oesophageal Ca. (ave. latency 20-30yrs)

Diffuse oesophageal spasm

Description: Episodic chest pain ± dysphagia from spastic contractions of distal half of oesophagus in the absence of any precipitating structural stenosis. There may be an underlying disorder of neural control but evidence for this is lacking and the aetiology is unknown.

Clinical Features: Episodic, crushing, central retrosternal pain. Intermittent dysphagia in ~⅔

Investigations: Ba swallow may be normal, or show trapping of beads of contrast in distal oesophagus (the corkscrew oesophagus) or sustained obliteration of distal oesophageal lumen. Manometry may show intermittent, simultaneous, prolonged, and vigorous oesophageal contractions interspersed with normal swallow-induced peristalsis. Relaxation of LOS is normal.

DDx: Achalasia, GORD, oesophageal strictures, webs or rings.

Treatment: No specific therapy. Nitrites, nitrates, CCB and TCAs may reduce symptoms; but reassurance is the most important measure. If disabling, botulinum toxin or long oesophagomyotomy can be used.

Oesophageal Strictures, Webs and Rings

Causes

Oesophageal strictures may be benign or malignant.

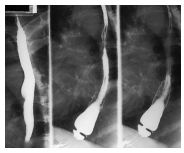
- Benign: usually from severe and persistent GORD (M>F, 25% w/o dyspepsia). Also post-ingestion of corrosives, post-operative, 2° to drugs (alendronate, iron, NSAIDs, KCl)
- Malignant: usually from oesophagus Ca but may ascend from stomach Ca.

Oesophageal webs (smooth extension of normal mucosa/submucosa often about 2-3 mm wide)

- Paterson-Brown-Kelly aka Plummer-Vinson syndrome. Anterior postcricoid web assoc with Fe deficiency. F>M. May be koilonychia, cheilosis and glossitis.
- Most upper oesophageal webs are not associated with Paterson-Brown-Kelly syndrome.

Oesophageal rings Concentric, smooth, extensions of normal tissue (mucosa, submucosa and muscle) that are ~3-5mm thick. Most are asymptomatic. Multiple rings more common in young men. Classified as A, B and C rings.

- **A ring:** uncommon muscular ring several cm proximal to the squamocolumnar junction.
- **B ring** or **Schatzki ring:** really a web as only has mucosa and submucosa. Tends to mark the proximal part of a hiatus hernia in patient >50 whose main complaint is chronic intermittent dysphagia to solids. Non progressive.
- **C ring:** Rare. Indentation on CXR caused by the diaphragmatic crura. Rarely symptomatic.



Extrinsic lesions: thoracic aortic aneurysm, lung Ca, ↑LN (sarcoidosis, TB or lymphoma), grossly enlarged heart, aberrant right subclavian artery.

Clinical Features

Heartburn, progressive dysphagia, food impaction, weight loss and chest pain. Occasionally persistent cough and wheeze due to aspiration of food or acid. May be history of GORD.

Differential Diagnosis: Achalasia, GORD

Investigations

Bloods: FBC - Fe deficiency anaemia, non-anaemic Fe deficiency. Abnormal LFTs –mets.
Chest x-ray: may show a mass, gross dilatation of achalasia, or aspiration.
Barium swallow: usually required to clarify nature and length of stricture before endoscopy.
CT or endoscopy: delineate & stage tumours.

Management

- Benign strictures or rings: oesophageal dilation at endoscopy. PPIs may ↓frequency of repeated dilatations. Poor results is an indication for surgery.
- Malignant strictures will require oesophagectomy or palliative stent (Atkinson tube).
- In Paterson-Brown-Kelly syndrome, correcting Fe deficiency may reverse symptoms.

Complications

- Aspiration pneumonitis, food impaction, malnutrition.
- Dilatation can cause bleeding or perforation
- Paterson-Brown-Kelly syndrome is assoc with risk of malignant

Prognosis

Benign causes: Generally good. Surgery rarely needed. Potential progression from normal oesophagus→lower oesophageal ring→oesophageal stricture that occurs in assoc with reflux oesophagitis. Anti-reflux therapy may slow or prevent progression.
For malignant causes, see the article on carcinoma of oesophagus.

Oesophageal muscle diseases

Systemic sclerosis commonly involves the smooth muscle of the oesophagus.

Primary striated muscle disorders (e.g. dermatomyositis, polymyositis, muscular dystrophy, & myasthenia gravis) may present with high dysphagia in assoc with oropharyngeal dysfunction.

Hiatus hernia

Sliding - About 90 per cent of hiatus hernias. The gastro-oesophageal junction (GOJ) is displaced upwards into the thorax→simple pouch of intrathoracic stomach→symptoms of GORD.
Rolling – Remaining 10% where part of the stomach herniates alongside a normally situated GOJ. Some rolling hernias are assoc with displacement of the GOJ above the hiatus (mixed hernias).
Cx: Obstruction/distension of the pouch→upper abdominal discomfort and can→strangulation. Gastric volvulus may obstruct the GOJ. Both Cx have a high mortality and req urgent surgery. Elective surgery is normally recommended to reduce and anchor rolling hiatus hernias to ↓risks.

Oesophageal diverticula and pseudodiverticula. In scleroderma wide-mouthed, multiple diverticula are characteristic. Diverticula can otherwise occur in the mid & distal parts, 2° to hypercontraction motor disorders. Rarely symptomatic. Conservative Mx best.
Pseudodiverticulosis - Characteristic multiple intramural out-pouchings on barium swallow due to dilatation of submucosal gland ducts by an unknown process.

Mechanical, chemical, and radiation trauma

Mallory-Weiss tear

These mucosal tears extend across the GOJ usually induced by vigorous straining/vomiting. Bleeding may cause hypovolaemia in 10%. Continued bleeding requires endoscopic injection or electrocoagulation, vascular embolization, or vasopressin infusion. Very rarely, surgery is req.

Boerhaave's syndrome

Straining and vomiting→oesophageal rupture, most often in the left lower third. Spillage of the gastric contents into the pleural space → shock and chest and upper abdo pain with radiation to the back, left chest, or shoulder. CXR becomes abnormal only some hours after rupture. Surgical repair and drainage usually necessary, and if delayed beyond 24h, mortality is high.

Traumatic perforation

May complicate oesophageal instrumentation. *Symptoms:* chest or epigastric pain directly after instrumentation, sometimes with dyspnoea. *Signs*: Pneumothorax and surgical emphysema are diagnostic. *Mx:* Often treated non-surgically with NG, antimicrobials, and IV nutrition.

Caustic ingestion

See Toxicology.

Medication-induced oesophagitis

High risk for severe injury: Slow-release KCl, NSAIDs, tetracycline, quinidine
High risk for moderate injury: Many antibiotics, Fe supplements
Occ. injury: Ascorbic acid, mexiletine, slow-rel. theophylline, captopril, phenytoin, zidovudine
Chemotherapy may impair mucosal defences, ↓resistance to damage from other agents, and ↑susceptibility to infective oesophagitis from immune suppression. Oesophageal transit and acid clearance may be impaired through neurotoxic effects of some agents. Fistulation or perforation may occur through cytotoxic effects on malignancy in the oesophageal wall.

Infective oesophagitis

Causes:

Immunocompetent patients

Pathogen	Management	Remarks
Candida albicans	Topical/oral antifungals	By far most common
Herpes simplex	Acyclovir if severe	Unusual; may denude mucosa
Varicella zoster	Acyclovir if severe	In association with chickenpox/herpes zoster
Bacteria		Rare in well individuals

Immunocompromised patients

Pathogen	Management	Remarks
Candida albicans	Systemic antifungals	Most common; oral disease almost diagnostic
Cytomegalovirus	Prophylaxis and treatment with ganciclovir or foscarnet	Part of systemic infection. Sepiginous → giant ulcers distal half
Herpes simplex	Prophylaxis and treatment with acyclovir or foscarnet	Circumscribed ulcers, raised edges → coalescence. Oral lesions
Tuberculosis	Conventional	From military and local spread
Gram-positive cocci, Gram-negative bacilli	IV antibiotics	Often with systemic infection
Syphilis	Conventional	Associated with tertiary syphilis elsewhere → inflammatory stricture

Notes:

- Viral oesophagitis can cause major haemorrhage.
- Infective oesophagitis can damage the full thickness of the oesophageal wall and → stricturing.
- Dx is often aided by the setting. Cutaneous or oral disease can suggest cause.
- Endoscopy is the diagnostic method of choice. Appearance and biopsies/brushings allow diagnosis ± identification of infectious agents.