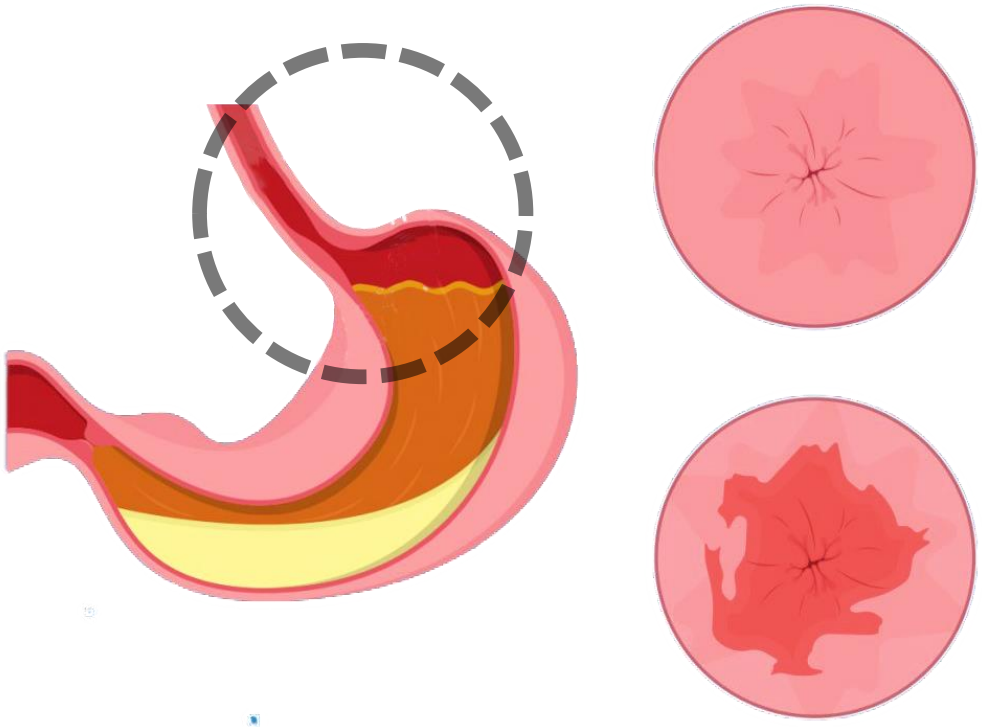
Barrett's Oesophagus



Anatomy

- Muscular tube
- Approximately 25 cm long
- Extending from the upper Oesophageal sphincter to the junction with the cardia of the stomach
- Upper sphincter – Striated muscle
- Transitional zone - Striated and smooth muscle
- Lower half - Only smooth muscle

Histology

- 4 concentric layers Mucosal layer
- Submucosal layer
- Muscular layer
- Adventitial layer

- Mucosa - Nonkeratinizing stratified squamous epithelium
- Changes from squamous cell epithelium to columnar cell epithelium at the gastroesophageal junction
- This junction has been termed the "Z line" or squamocolumnar junction

Barrett's oesophagus

- Metaplastic change in the lining mucosa of the oesophagus in response to chronic gastrooesophageal reflux

- Adaptive response involves a mosaic of cell types, probably beginning as a simple columnar epithelium that becomes 'specialised' with time

Epidemiology

- Average age -55-65 years
- 2:1 male-to-female ratio
- Prevalence -0.9-10% of the general adult population

Pathophysiology

- Prolonged exposure of the esophagus to the refluxate
- Erode the esophageal mucosa
- Inflammatory cell infiltrate
- Epithelial necrosis
- Chronic damage
- Replacement of healthy esophageal epithelium with the metaplastic columnar cells

- An adaptive response of the esophagus
- Gastroesophageal reflux disease - amount of gastric juice that refluxes into the esophagus exceeds the normal limit, causing symptoms with or without associated esophageal mucosal injury

Aetiology

- GERD
- Abdominal obesity
- Use of oral bisphosphonates

Types

- Classic Barrett's (3 cm or more columnar epithelium)
- Short-segment Barrett's (less than 3 cm of columnar epithelium)
- Cardia metaplasia (intestinal metaplasia at the oesophagogastric junction without any macroscopic change at endoscopy).

Clinical features

- Chronic history of gastroesophageal reflux
- Pyrosis(heart burn)
- Occasionally dysphagia
- Bleeding
- Physical examination- normal

Complications

- Oesophageal ulceration and stricture-
- Pain
- Bleeding
- Obstruction
- Penetration
- Perforation
- Dysplasia
- Malignant transformation - adenocarcinoma

Diagnosis

- Esophagogastroduodenoscopy (EGD)
- Biopsy confirm the diagnosis
- Ultrasonography
- When high-grade dysplasia or cancer is found on surveillance endoscopy
- Endoscopic ultrasonography -evaluate for surgical resectability

- Endoscopy - Difficult to distinguish a Barrett's oesophagus from a tubular, sliding hiatus hernia
- Because two often coexist or where the visible Barrett's segment is very short
- Mucosa in the body of the stomach has longitudinal folds
- Columnar lining of Barrett's oesophagus is smooth

Treatment

- Screening and Surveillance- Endoscopy and biopsy
- Treatment to underline gastro-oesophageal reflux disease (GORD)
- Management of dysplasia

Screening and Surveillance

- Endoscopy and biopsy
- No dysplasia - 2 consecutive yearly endoscopies
- Low-grade dysplasia - every 6 months for 2 cycles
- If no progression of disease is noted, surveillance may be extended to yearly follow-up
- Management of high-grade dysplasia is more controversial

Treatment to underlying GORD

1) Proton pump inhibitors (PPIs) – Omeprazole
   Esomeprazole
2) Histamine 2 (H2)-receptor antagonists - Ranitidine
   Famotidine
3) Antireflux surgery

- Drugs and foods that should avoided
- Fried or fatty foods
- Peppermint
- Coffee
- Chocolate
- Alcohol
- Carbonated beverages
- Citrus fruits or juices
- Mustard
- Tomato sauce
- Vinegar

- Aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs)

High grade dysplasia

- Surveillance endoscopy with intensive biopsy at 3-month intervals until cancer is detected
- Endoscopic ablation
- Surgical resection.

Endoscopic methods of ablating Barrett's Oesophagus

- Laser
- Photodynamic therapy
- Argon-beam plasma
- Coagulation and endoscopic mucosal resection
- Eliminating the risk of cancer development