## Candidiasis

The yeast *Candida albicans* is a normal mouth commensal but it may proliferate to cause thrush. This occurs in babies, debilitated patients, patients receiving corticosteroid or antibiotic therapy, patients with diabetes and immunosuppressed patients, especially those receiving cytotoxic therapy and those with HIV infection. White patches are seen on the tongue and buccal mucosa. Odynophagia or dysphagia suggests pharyngeal and oesophageal candidiasis.
A clinical diagnosis is sufficient to start therapy, although brushings or biopsies can be obtained for mycological examination. Oral thrush is treated using nystatin or amphotericin suspensions or lozenges. Resistant cases or immunosuppressed patients may require oral fluconazole.

## Parotitis

Parotitis is due to viral or bacterial infection. Mumps causes a self-limiting acute parotitis. Bacterial parotitis usually occurs as a complication of major surgery. It is a consequence of dehydration and poor oral hygiene, and can be avoided by good post-operative care. Patients present with painful parotid swelling and this can be complicated by abscess formation. Broad spectrum antibiotics are required, whilst surgical drainage is necessary for abscesses. Other causes of salivary Calculi ,Sjogren's syndrome Sarcoidosis Tumours Benign: pleomorphic adenoma (95% of cases) Intermediate: mucoepidermoid tumour Malignant: carcinoma.

## Aphthous ulceration

Aphthous ulcers are superficial and painful;. Recurrent ulcers occur in 30% of the population and are particularly common in women prior to menstruation. The cause is unknown. Management is with topical corticosteroids (such as 0.1% triamcinolone),Symptomatic relief is achieved using local anaesthetic mouthwashes. Rarely, patients with very severe, recurrent aphthous ulcers may need oral corticosteroids . other causes of oral ulceration:

**Infection**

• Fungal (candidiasis) • Viral (herpes simplex, HIV) • Bacterial, including syphilis, tuberculosis

**Gastrointestinal diseases**

• Crohn's disease • Coeliac disease

**Dermatological conditions**

• Lichen planus• • Dermatitis herpetiformis • Erythema multiforme

**Drugs**

• Nicorandil, NSAIDs, methotrexate, penicillamine, losartan, ACE inhibitors • Cytotoxic drugs

**Systemic diseases**

• Systemic lupus erythematosus • Behçet's syndrome

**Neoplasia**

• Carcinoma • Leukaemia • Kaposi's sarcoma

## Gastro-oesophageal reflux disease

GERD resulting in heartburn affects 30% of the general population.

***Pathophysiology***

GERD develops when the oesophageal mucosa is exposed to gastroduodenal contents for prolonged periods of time, resulting in symptoms and oesophagitis. Several factors are known to be involved in the development of GERD

1- *Abnormalities of the lower oesophageal sphincter:*

The lower oesophageal sphincter is tonically contracted under normal circumstances, relaxing only during swallowing .Some patients with GERD have reduced LES tone, permitting reflux when intra-abdominal pressure rises

*2-Hiatus hernia:*

Hiatus hernia causes reflux because the pressure gradient between the abdominal and thoracic cavities, , is lost. In addition, the oblique angle between the cardia and oesophagus disappears. Many patients who have large hiatus hernias develop reflux symptoms, but the relationship between the presence of a hernia and symptoms is poor. Hiatus hernia is very common in individuals who have no symptoms, and some symptomatic patients have only a very small or no hernia. Nevertheless, almost all patients who develop oesophagitis, Barrett's oesophagus or peptic strictures have a hiatus hernia.

*3-Delayed oesophageal clearance*

Defective oesophageal peristaltic activity is commonly found in patients who have oesophagitis .Poor oesophageal clearance leads to increased acid exposure time.

*4-Gastric contents*

Gastric acid is the most important oesophageal irritant. Pepsin and bile also contribute to mucosal injury.

*5-Defective gastric emptying*

Gastric emptying is delayed in patients with GERD. The reason is unknown.

*6-Increased intra-abdominal pressure*

Pregnancy and obesity are established predisposing causes. Weight loss may improve symptoms.

*7-Dietary and environmental factors*

Dietary fat, chocolate, alcohol and coffee relax the lower oesophageal sphincter and may provoke symptoms.

***Clinical features***

The major symptoms are heartburn and regurgitation, often provoked by bending, straining or lying down.

'Waterbrash', which is salivation due to reflex salivary gland stimulation . The patient is often overweight. Some patients are woken at night by choking as refluxed fluid irritates the larynx. Others develop odynophagia or dysphagia. other features such as atypical chest pain which may be severe and can mimic angina, and may be due to reflux-induced oesophageal spasm. Others include hoarseness ('acid laryngitis'), recurrent chest infections, chronic cough and asthma.

***Investigations***

▲Young patients who present with typical symptoms of GERD, without worrying features such as dysphagia, weight loss or anaemia, can be treated empirically without investigation.

▲Investigation is advisable if patients present over the age of 50–55 years, if symptoms are atypical or if a complication is suspected.

1- Endoscopy is the investigation of choice. This is performed to exclude other upper gastrointestinal diseases that can mimic gastro-oesophageal reflux and to identify complications. A normal endoscopy in a patient with compatible symptoms should not preclude treatment for GERD.

2- 24 hr pH monitoring is indicated if the diagnosis is unclear or surgical intervention is under consideration. This involves tethering a slim catheter with a terminal radiotelemetry pH-sensitive probe above the gastro-oesophageal junction. The intraluminal pH is recorded whilst the patient undergoes normal activities, and episodes of symptoms are noted and related to pH. A pH of less than 4 for more than 6–7% of the study time is diagnostic of reflux

***Complications***

*1-Oesophagitis*

A range of endoscopic findings, from mild redness to severe, bleeding ulceration with stricture formation, are recognised, although appearances may be completely normal There is a poor correlation between symptoms and histological and endoscopic findings.

*2-Barrett's oesophagus*

Barrett's oesophagus is a pre-malignant condition, in which the normal squamous lining of the lower oesophagus is replaced by columnar mucosa (columnar lined oesophagus; CLO) that may contain areas of intestinal metaplasia .is found in 10% of patients undergoing gastroscopy for reflux symptoms. the true prevalence may be up to 1.5–5% of the population, as the condition is often asymptomatic until discovered when the patient presents with oesophageal cancer. The relative risk of oesophageal

cancer is 40–120-fold increased. The epidemiology and etiology of CLO are poorly understood. The prevalence is increasing, and it is more common in men (especially white), the obese and those over 50 years of age. It is weakly associated with smoking but not alcohol intake. The risk of cancer seems to relate to the severity and duration of reflux rather than the presence of CLO per se and it has been suggested that duodenogastro oesophageal reflux of bile, pancreatic enzymes and pepsin, as well as gastric acid, may be important in pathogenesis.

The molecular events underlying progression of CLO to dysplasia and cancer are incompletely understood but inactivation of the tumour suppression protein p16 followed by somatic inactivation of p53, which promotes tumour progression.

***Diagnosis.*** This requires multiple systematic biopsies to maximise the chance of detecting intestinal metaplasia and/or dysplasia.

***Management.*** Neither potent acid suppression nor antireflux surgery stops progression or induces regression of CLO, and treatment is only indicated for symptoms of reflux or complications, such as stricture. Endoscopic therapies, such as radiofrequency ablation or photodynamic therapy, can induce regression but, at present, are used only for those with dysplasia or intramucosal cancer. Regular

endoscopic surveillance can detect dysplasia at an early stage and may improve survival but, because most CLO is undetected until cancer develops, surveillance strategies are unlikely to influence the overall mortality rate of oesophageal cancer.

**Surveillance**

♠-patients with CLO without dysplasia --endoscopy at 3–5-yearly intervals

♠- those with lowgrade dysplasia at 6–12-monthly intervals.

♠- those with high-grade dysplasia (HGD) or intramucosal carcinoma, the treatment options are either oesophagectomy or endoscopic therapy with a combination of endoscopic resection (ER) of any visibly abnormal areas and radiofrequency ablation (RFA) of the remaining Barrett's mucosa as an 'organ-preserving' alternative to Sx.

*3-Anaemia:*

Iron deficiency anaemia can occur as a consequence of occult blood loss from long-standing oesophagitis. Most patients have a large hiatus hernia and bleeding can stem from subtle erosions in the neck of the sac ('Cameron lesions'). Nevertheless, hiatus hernia is very common and other causes of blood loss, particularly colorectal cancer, must be considered in anaemic patients, even when endoscopy reveals oesophagitis.

*4-Benign oesophageal stricture*

Fibrous strictures especially in the elderly and those with poor oesophageal peristaltic activity. The typical presentation is with dysphagia that is worse for solids than for liquids. many elderly patients presenting with strictures have no preceding heartburn. Diagnosis is by endoscopy, when biopsies of the stricture can be taken to exclude malignancy. Endoscopic balloon dilatation helpful., long-term therapy with a PPI drug should be started to reduce the risk of recurrent oesophagitis and stricture formation.

*5-Gastric volvulus*

Occasionally, a massive intrathoracic hiatus hernia may twist upon itself, leading to a gastric volvulus. This gives rise to complete oesophageal or gastric obstruction and the patient presents with severe chest pain, vomiting and dysphagia. The diagnosis is made by chest X-ray (air bubble in the chest) and barium swallow. Most cases spontaneously resolve but recurrence is common, and surgery is usually advised after the acute episode has been treated by NG decompression

***Management of GERD:***

♠Lifestyle advice: weight loss, avoidance of diet worsen symptoms, elevation of the bed head , avoidance of late meals and giving up smoking.

♠ Patients who fail to respond to these measures should be offered PPIs, which are usually effective in resolving symptoms and healing oesophagitis.

♠Recurrence of symptoms is common when therapy is stopped and some patients require life-long treatment at the lowest acceptable dose.

♠ When dysmotility features are prominent, domperidone can be helpful.

♠There is no evidence that *H. pylori* eradication has any therapeutic value.

♠ Proprietary antacids and alginates can also provide symptomatic benefit. H2-receptor antagonist drugs also help symptoms without healing oesophagitis..

♠Patients who fail to respond to medical therapy, those who are unwilling to take long-term PPIs and those whose major symptom is severe regurgitation should be considered for laparoscopic anti-reflux surgery. Although heartburn and

regurgitation are alleviated in most patients, a small minority develop complications, such as inability to vomit and abdominal bloating ('gas-bloat' syndrome').

## Other causes of oesophagitis

***Infection***

Oesophageal candidiasis occurs in debilitated patients and those taking broad-spectrum antibiotics or cytotoxic drugs. It is a particular problem in patients with HIV.

***Corrosives***

Suicide attempt by strong household bleach or battery acid is followed by painful burns of the mouth and pharynx and by extensive erosive oesophagitis. This may be complicated by oesophageal perforation with mediastinitis and by stricture formation. At the time of presentation, treatment is conservative, based upon analgesia and nutritional support; vomiting and endoscopy should be avoided because of the high risk of oesophageal perforation. After the acute phase, a barium swallow should be performed to demonstrate the extent of stricture formation. Endoscopic dilatation is usually necessary but it is difficult and hazardous because strictures are often long, tortuous and easily perforated.

## *Achalasia*

***Pathophysiology***

Achalasia is characterised by:

• a hypertonic lower oesophageal sphincter, whichfails to relax

• failure of propagated oesophageal contraction, leading to progressive dilatation of the gullet.

The cause is unknown. Defective release of nitric oxide by inhibitory neurons in the lower oesophageal sphincter has been reported, and there is degeneration of ganglion cells within the sphincter and the body of the oesophagus. Loss of the dorsal vagal nuclei within the brainstem can be demonstrated in later stages.

***Clinical features***

The presentation is with *dysphagia*. This develops slowly, is initially intermittent, and is worse for solids and eased by drinking liquids, and by standing and moving around after eating. Heartburn does not occur because the closed oesophageal sphincter prevents GERD. Some patients had *chest pain* due to oesophageal spasm. As the disease progresses, dysphagia worsens, the oesophagus empties poorly and *nocturnal pulmonary aspiration* develops. *Achalasia predisposes to squamous carcinoma of the oesophagus.*

***Investigations***

1-Endoscopy should always be carried out because carcinoma of the cardia can mimic the presentation and radiological and manometric features of achalasia ('pseudo-achalasia').

2-A barium swallow shows tapered narrowing of the lower oesophagus and, in late disease, the oesophageal body is dilated, aperistaltic and foodfilled

3- Manometry confirms the highpressure, non-relaxing lower oesophageal sphincter with poor contractility of the oesophageal body

***Management***

*Endoscopic*

Forceful pneumatic dilatation using a 30–35-mm diameter fluoroscopically positioned balloon disrupts the oesophageal sphincter and improves symptoms in 80% of patients. Some patients require more than one dilatation but those needing frequent dilatation are best treated surgically.

Endoscopically directed injection of botulinum toxin into the lower oesophageal sphincter induces clinical remission but relapse is common.

*Surgical*

Surgical myotomy (Heller's operation), performed either laparoscopically or as an open operation, is effective but is more invasive than endoscopic dilatation. Both pneumatic dilatation and myotomy may be complicated by GERD, and this can lead to severe oesophagitis because oesophageal clearance is so poor.

For this reason, Heller's myotomy is accompanied by a partial fundoplication anti-reflux procedure. PPI therapy is often necessary after surgery

Other oesophageal motility disorders

**Diffuse oesophageal spasm**

presents in late middle age with episodic chest pain that may mimic angina, but is sometimes accompanied by transient dysphagia. Some cases occur in response to GERD.

Treatment is based upon the use of PPI drugs when gastro-oesophageal reflux is present. Oral or sublingual nitrates or nifedipine may relieve attacks of pain. The results of drug therapy are often disappointing, as are the alternatives: pneumatic dilatation and surgical myotomy.

**Nutcracker' oesophagus**

is a condition in which extremely forceful peristaltic activity leads to episodic chest pain and dysphagia. Treatment is with nitrates or nifedipine. The patients are usually elderly and present with dysphagia and chest pain.Manometric abnormalities, ranging from poor peristalsis to spasm, occur. Treatment is with dilatation and/or vasodilators for chest pain.