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Chapter 2

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad Chapter 2

Pulmonology

Smoking cessation Action of smoking • Nicotine is a stimulant and releases dopamine in the brain that leads to addictive effects of smoking. • Its effects can be replaced in other ways using nicotine replacement therapy and this reduces the addiction to cigarette smoking. General points of treatment • Advise all people who smoke to stop • Offer referral to a local smoking cessation service for behavioural support and drugs (a combination of drug treatment and behavioural support may be the best option) • Advise to stop abruptly. • Patients should be offered nicotine replacement therapy (NRT), varenicline or bupropion. • NRT, varenicline or bupropion should normally be prescribed as part of a commitment to stop smoking on or before a particular date (target stop date) • If unsuccessful using NRT, varenicline or bupropion, do not offer a repeat prescription within 6 months unless special circumstances have intervened • Do not offer NRT, varenicline or bupropion in any combination • Starting date of the treatment Start NRT on the quit date. Start varenicline or bupropion 7-14 days before the quit date. • Duration of treatment Prescribe NRT for 2 weeks after stop date Prescribe varenicline or bupropion FOR 3- 4 weeks after stop date. • Varenicline or combination NRT (a patch plus a short-acting preparation) have been shown to be the most effective treatments. • Varenicline or bupropion should not be prescribed to pregnant or breastfeeding women or young people aged under 18. • No one form of NRT is more effective than another. • Reviewed 2 weeks after stopping smoking, and the CO level measured at 4 weeks.

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Nicotine replacement therapy (NRT) • Available in a choice of formats, including Gum, Inhalator, Sublingual tablet, Nasal and Oral spray, and Transdermal patch • Nice recommend offering a combination of nicotine patches and another form of NRT (such as gum, inhalator, lozenge or nasal spray) to people who show a high level of dependence on nicotine or who have found single forms of NRT inadequate in the past. • Duration The duration of treatment with NRT is 8–12 weeks (depending on which form of NRT is used and which dose is initiated), followed by a gradual reduction in dose. For children over the age of 12 years, treatment should be limited to 12 weeks. Treatment with NRT can be stopped abruptly or tapered gradually • No absolute

contraindications • Adverse effects: Headache, dizziness, Nausea, vomiting, Rash, urticaria. Varenicline • Mode of action: a partial nicotinic receptor agonist → reduces the rewarding and reinforcing effects of smoking by preventing nicotine binding to the receptors. • Duration Advise the person to stop smoking 7–14 days after starting varenicline. The recommended course of treatment is 12 weeks. Varenicline may be stopped without tapering the dose. However, immediately after stopping treatment with varenicline, up to 3% of people experience an increase in irritability, urge to smoke, depression, or insomnia. Consider tapering the dose in these people. • Contraindications Aged under 18 years. Pregnancy. End-stage renal disease. • Common adverse effects Nervous system: headache; somnolence, dizziness, dysgeusia. Psychiatric: abnormal dreams, insomnia GIT upset and dry mouth Bupropion • Mode of action: selective dopamine and noradrenaline re-uptake inhibitor • Duration Advise the person to stop smoking 7–14 days after starting bupropion. If no effect is seen after 7 weeks, discontinue treatment with bupropion. • Contraindications Age under 18 years Pregnancy History of seizures. CNS tumour. History of bulimia or anorexia nervosa. History of bipolar disorder. Severe hepatic cirrhosis. • Common adverse effects Psychiatric : insomnia, depression, agitation, anxiety Nervous system: tremor, concentration disturbance, headache, dizziness, taste disorders. GIT upset and dry mouth

Chapter 2

Pulmonology Medications used for Smoking cessation NRT Varenicline Bupropion Action Nicotine replacement therapy Effectiveness Effective with combination of two forms of NRT Date of starting on the quit date 7–14 days before quit date. Duration 8–12 weeks 12 weeks 7 weeks Absolute contraindications NO absolute contraindications Age ˂ 18 years Pregnancy History of seizures CNS tumour bulimia or anorexia nervosa bipolar disorder Severe hepatic cirrhosis Prescribe with Cation Conditions lower the seizure threshold (e.g. Alcohol abuse, head trauma, diabetes, antipsychotics). Hepatic & renal impairment. Common adverse effects Chronic diseases (DM, HTN , RF, MI , CVA) Epilepsy. Headache, dizziness, Nausea Pregnant women • Assessment NICE recommended in 2010 that all pregnant women should be tested for smoking using carbon monoxide detectors, partly because 'some women find it difficult to say that they smoke because the pressure not to smoke during pregnancy is so intense.' All women who smoke, or have stopped smoking within the last 2 weeks, or those with a CO reading of 7 ppm or above should be referred to NHS Stop Smoking Services. • Adverse effects of smoking in pregnancy Reduces birth weight increases risk of miscarriage and still birth. The infant has a greater risk of sudden infant death syndrome. affect ovarian function in female children. Notes & Notes for MRCP
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partial nicotinic receptor agonist Norepinephrine and dopamine reuptake inhibitor Effective Less effective than NRT and Varenicline 7–14 days before quit date Aged ˂ 18 years Pregnancy End-stage renal disease Cardiovascular, renal & psychiatric illness. Epilepsy Headache, abnormal dreams, insomnia & GI upset Headache, insomnia, depression, tremor.

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 increases lung maturity, possibly by enhancing the production or secretion of cortisol. This makes neonates less likely to develop respiratory distress syndrome, but as lung maturation is often abnormal babies may have reduced lung function and increased rates of other respiratory illnesses.
• Interventions in pregnant smoker first-line: cognitive behaviour therapy and support from stop Smoking Services second line: NRT NRT should only be used if smoking cessation without NRT fails. Pregnant women should remove the patches before going to bed varenicline and bupropion are contraindicated

Third edition Notes & Notes For MRCP part 1 & 11 By Dr. Yousif Abdallah Hamad Gastroenterology Updated

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad Chapter 3

Gastroenterology

Oesophageal diseases Achalasia

Definition • Failure of oesophageal peristalsis and of relaxation of lower oesophageal sphincter (LOS) due to degenerative loss of ganglia from Auerbach's plexus i.e. LOS contracted, oesophagus above dilated.

Pathophysiology • Atrophy of inhibitory neurons in the Auerbach plexus → lack of inhibitory neurotransmitters (e.g., NO, VIP) → inability to relax and increased resting pressure of the LES, as well as dysfunctional peristalsis → esophageal dilation proximal to LES Epidemiology • prevalence of around 10 /100,000 persons.
• typically presents in middle-age • equally common in men and women.

Causes
• Primary achalasia (most common): cause is unknown • Secondary achalasia (pseudoachalasia):
 mechanical obstruction (e.g., a malignancy)
 Chagas disease Features • Symptoms usually develop years before the patient presents • Dysphagia of BOTH liquids and solids. • Regurgitation of food → heartburn, cough, aspiration pneumonia etc • Oesophageal spasm → vague chest discomfort (common)
• Weight loss Complications • Increased risk of esophageal cancer.

Investigations

• Barium swallow initial investigation
 will show: dilated oesophagus, fluid level, 'bird's beak' appearance • Manometry:
 The confirmatory test of choice Will show: excessive LOS tone which doesn't relax on swallowing
 Lack of peristalsis in the lower two-thirds of the esophagus • Upper endoscopy to rule out pseudoachalsia Usually normal, May show retained food in esophagus or increased resistance of LES during passage with endoscope • CXR: Will show:
 wide mediastinum, (˃6 cm on an upright PA chest X-ray or ˃ 8 cm on supine AP chest film). fluid level

Treatment • Heller cardiomyotomy (Laparoscopic myotomy)
 The best initial treatment for most patients with achalasia.
• Balloon dilation (Pneumatic dilatation)
 the preferred option for older unfit patients or patients who choose a nonsurgical treatment. Current guidelines recommend obtaining gastrograffin study followed by barium esophagram in all patients after pneumatic dilation to exclude esophageal perforation long-term efficacy is less than that of surgical myotomy. 25% of patients treated with pneumatic dilation required re-dilation.
• Intra-sphincteric injection of botulinum toxin
 Reserved for the elderly and who cannot tolerate dilatation or surgery. Reduces the LOS pressure and provides symptomatic relief. However, the effects are temporary, and patients need to undergo repeat injections every six to twelve months. • Drug therapy has a role but is limited by side-effects Short-term improvement in clinical symptoms may occur with isosorbide mononitrate, a long-acting nitrate or with nifedipine, a calcium-channel blocker.
• Contraindications Promotility agents like metoclopramide increase the lower oesophageal sphincter pressure and so are contraindicated in achalasia.

Images

This film demonstrates the classical 'bird's beak' appearance of the lower oesophagus that is seen in achalasia. An air-fluid level is also seen due to a lack of peristalsis

Gastroenterology

Mediastinal widening secondary to achalasia. An air-fluid level can sometimes be seen on CXR but it is not visible on this film

Barium swallow - grossly dilated filled oesophagus with a tight stricture at the gastroesophageal junction resulting in a 'bird's beak' appearance. Tertiary contractions give rise to a corkscrew appearance of the oesophagus TOP TIPS

The most appropriate initial investigation of a high dysphagia is a barium swallow, which identifies the site of pathology and forewarns of pitfalls such as a pharyngeal pouch, which if unidentified can increase the risk of perforation at endoscopy.

Dysphagia

The table below gives characteristic exam question features for conditions causing dysphagia:

Diagnosis
Characteristic features Oesophageal cancer • Dysphagia may be associated with weight loss, anorexia or vomiting during eating • Past history may include Barrett's oesophagus, GORD, excessive smoking or alcohol use Oesophagitis • May be history of heartburn • Odynophagia (Painful swallowing) but no weight loss and systemically well Oesophageal candidiasis • There may be a history of HIV or other risk factors such as steroid inhaler use • Treatment oral or IV therapy (usually with fluconazole or itraconazole for at least 14-21 days). Achalasia • Dysphagia of both liquids and solids from the start • Heartburn • Regurgitation of food - may lead to cough, aspiration pneumonia etc Pharyngeal pouch • More common in older men • Represents a posteromedial herniation between thyropharyngeus and cricopharyngeus muscles • Usually not seen but if large then a midline lump in the neck that gurgles on palpation • Typical symptoms are dysphagia, regurgitation, aspiration and chronic cough. Halitosis may occasionally be seen Systemic sclerosis • Other features of CREST syndrome may be present, namely Calcinosis, Raynaud's phenomenon, oesophageal dysmotility, Sclerodactyly, Telangiectasia • As well as oesophageal dysmotility the lower oesophageal sphincter (LES) pressure is decreased. This contrasts to achalasia where the LES pressure is increased Myasthenia gravis • Other symptoms may include extraocular muscle weakness or ptosis • Dysphagia with liquids as well as solids Globus hystericus • May be history of anxiety • Symptoms are often intermittent and relieved by swallowing • Usually painless - the presence of pain should warrant further investigation for organic causes

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Causes of dysphagia - by classification Classification Examples Extrinsic • Mediastinal masses • Cervical spondylosis Oesophageal wall • Achalasia • Diffuse oesophageal spasm • Hypertensive lower oesophageal sphincter Intrinsic • Tumours • Strictures • Oesophageal web • Schatzki rings Neurological • CVA • Parkinson s disease • Multiple Sclerosis • Brainstem pathology • Myasthenia Gravis

Dysphagia
• Dysphagia to both solids and liquids → Achalasia (motility disorder) • Dysphagia to solids only → Oesophageal obstruction (structural disorder, e.g. malignancy) Investigations • Barium contrast oesophagram is the initial test (prior to upper endoscopy)

Oesophageal disorders The table below lists a small group of oesophageal disorders that are not covered elsewhere in the notes. Disorder Notes PlummerVinson syndrome Triad of: • dysphagia (secondary to oesophageal webs) • glossitis • iron-deficiency anaemia Treatment includes iron supplementation and dilation of the webs Mallory-Weiss syndrome Severe vomiting → painful mucousal lacerations at the gastroesophageal junction resulting in haematemesis. Common in alcoholics Boerhaave syndrome Severe vomiting → oesophageal rupture

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Oesophageal web

Diffuse oesophageal spasm

• Features Dysphagia Chest pain • Diagnosis barium swallow demonstrates a 'corkscrew appearance' Manometry reveals: prolonged, repetitive and high amplitude contractions.
 The lower oesophageal sphincter pressure is increased and there is incomplete relaxation of the sphincter.

Differential diagnosis manometry findings: • Absence of peristalsis in the body of the oesophagus + high lower oesophageal sphincter → Achalasia • Normal contractions in the body of the oesophagus + high lower oesophageal sphincter pressure → Hypertensive lower oesophageal sphincter • High amplitude contractions in the body of the oesophagus + high lower oesophageal sphincter pressure → Diffuse oesophageal spasm

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Gastro-oesophageal reflux disease (GORD)

Definition: regurgitation of stomach contents into the esophagus

Pathophysiology • Decreased tone of the lower esophageal sphincter. • The most important physiological mechanism that prevents reflux → Parasympathetic stimulation of the lower circular smooth-muscle fibres of the oesophagus

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Cause • Transient lower esophageal sphincter relaxation is the most common cause • Pregnancy → decreased motility secondary to progesterone • gastric acidity • gastric outlet obstruction • decreased esophageal motility • hiatal hernia: ≥ 90% of patients with severe GORD • Obesity →Transient relaxations of the lower esophageal sphincter (TRLES). The main stimulus for TRLES is gastric distension, particularly in the fundus. • Lifestyle habits such as smoking, caffeine and alcohol consumption
• Scleroderma • Angle of His enlargement (> 60°)

Features • Heartburn and regurgitation when lying down.
• GORD is the most common non-cardiac cause of chest pain.
• Extraesophageal symptoms (eg, chronic cough, hoarseness, wheezing) The three most common causes of a persistent cough are postnasal drip, asthma, and GORD.
• Acid reflux in chronic GORD can lead to damage of the enamel layer of teeth. • May present with over-the-counter antacids side effects which may include magnesium hydroxide.
 Magnesium hydroxide can act as an osmotic laxative, resulting in the adverse effect of diarrhea.
Investigations • Endoscopy: Indications for upper GI endoscopy: No symptomatic improvement after PPI trial Alarm features
 New onset dyspepsia in patient ≥60 years Dysphagia Odynophagia Early satiety

Persisting vomiting Unintentional weight loss Aspiration pneumonia
 Evidence of gastrointestinal bleeding (hematemesis, melena, hematochezia, occult blood in stool) Iron deficiency anemia Anorexia Gastrointestinal cancer in a first-degree relative The most common endoscopic finding is reflux esophagitis.
 Symptoms do not correlate with mucosal status at endoscopy appearance • 24-hr oesophageal pH monitoring: If endoscopy is negative (the gold standard test for diagnosis)

 To confirm the diagnosis of GORD in patients with persistent symptoms of GORD despite a trial of PPI therapy. Evaluation before surgical or endoscopic antireflux procedure • Oesophageal manometry In patients with suspected GORD and a normal upper endoscopy to exclude an esophageal motility disorder.
 To evaluate esophageal peristaltic function prior to antireflux surgery.

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Treatment • Lifestyle changes
 Small portions;
 avoid eating (< 3 hours) before bedtime. Avoid foods with high fat content Avoid: nicotine, alcohol, coffee, and certain drugs (e.g., calcium channel blockers, diazepam) • Pharmacological: if lifestyle changes are ineffective Proton pump inhibition (PPI):
 full-dose PPI (e.g. 20 mg omeprazole OD) for 4 or 8 weeks. In those failing to respond to two months of full dose therapy doubling the dose of proton pump inhibitor for one month increases response rate. If no response, increase the dose to (twice daily therapy) If symptoms recur after initial treatment, offer a PPI at the lowest dose possible to control symptoms. H2 receptor antagonist
 The 2nd line if there is an inadequate response to a PPI In those failing to respond to a double dose of proton pump inhibition an H2 receptor antagonist may be added or substituted in treatment or a prokinetic agent added to treatment.
• Laparoscopic Nissen fundoplication the treatment of choice for patients with GORD refractory to or intolerant of proton pump inhibitor therapy. The patient should have had an endoscopy within the six months prior to surgery to exclude any unsuspected pathology such as Barrett's oesophagus or adenocarcinoma. the most useful in assessing the role of surgery → Oesophageal motility and pH study • Severe oesophagitis 1st line: full-dose PPI (e.g. omeprazole 20 mg OD) for 8 weeks 2nd line: high dose PPI (double standard dose e.g. 40 mg omeprazole OD) of the initial PPI, switching to another full-dose PPI or switching to another high-dose PPI Maintenance treatment → long-term full-dose PPI. If fail to respond to maintenance treatment, → switch to another PPI at full dose or high dose. • Management of GORD in pregnancy includes 1st line: lifestyle and dietary modification
 2nd line: antacids and sucralfate. Antacids containing sodium bicarbonate and magnesium trisilicate should be avoided in pregnancy.
 3rd line: similar to nonpregnant patients, H2RAs and then PPIs.

Complications • Barrett esophagus: Metaplasia of the lower esophagus • Esophageal strictures occur in 10%
 Two types of rings (Schatzki rings):muscular ring, or A ring: located approximately 2 cm above

the gastroesophageal junction. Rare mucosal ring, or B ring: most common. located at the squamo-columnar junction. mechanical cause of dysphagia most patients respond well to dilatation therapy.
 People who have had dilatation of an oesophageal stricture should remain on longterm full-dose PPI therapy • Adenocarcinoma of the lower esophagus.

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GORD management • 1st line → lifestyle changes don't lie down after eating avoid spicy foods eat small servings • 2nd line → proton pump inhibitors (omeprazole, lansoprazole) for at least 8 weeks (once daily therapy) No response: → further diagnostic evaluation Partial response: → increase the dose to (twice daily therapy)
 Good response: → discontinue PPI after 8 weeks If symptoms recur after discontinuation of PPIs → Maintenance therapy
 After 8 weeks of initial treatment, reduce PPI to lowest effective dose • 3rd line → H2 receptor antagonists(cimetidine, ranitidine) • 4th line → Surgical Nissen fundoplication or hiatal hernia repair

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Barrett's oesophagus

Overview
• Metaplasia of the lower oesophageal mucosa 1 cm or more proximal to the gastroesophageal junction. (the normal squamous epithelium of the oesophagus replaced by a columnar epithelium) (ie, intestinal metaplasia)
• The physiological transformation zone ("Z-line") between squamous and columnar epithelium is shifted upwards • Metaplasia is defined as the replacement of one type of cells with another type whereas dysplasia is the disordered growth of the cells. • Barrett esophagus is a premalignant condition of the lower esophagus caused by chronic esophageal reflux • the columnar epithelium may resemble that of either the cardiac region of the stomach or that of the small intestine (e.g. with goblet cells, brush border)

Risk factors
• Age ˃ 50 years • Male gender • Ethnicity: more common in white populations than Hispanic, Black, or Asian. • Long duration or frequency of Gastro-oesophageal reflux disease (GORD) symptoms
• Previous oesophagitis • Hiatus hernia • Central obesity

Diagnosis • Endoscopic evaluation, with: Visualization of columnar epithelium 1 cm or more above the gastroesophageal junction
 Biopsy sampling of esophageal epithelium with histologic confirmation of intestinal metaplasia (American guidelines) or histologic confirmation of columnar epithelium (British guidelines).
Management (based on presence or absence of dysplasia) • Nondysplastic: proton pump inhibitors + surveillance endoscopy with repeated biopsy every 3 to 5 years

 NO dysplasia and <3 cm segment of Barrett's → endoscopy every three to five years with biopsies
 NO dysplasia and segment Barrett's >3 cm → Endoscopy every two to three years • Low-grade dysplasia (LGD) Repeat endoscopic biopsy in 6 months. If LGD is found → Radiofrequency ablation If ablation is not undertaken, 6-monthly surveillance is recommended • Moderate to high grade dysplasia or recurrent disease 1st line: Endoscopic ablation therapy (endoscopic resection of any visible mucosal irregularities, followed by Radiofrequency Ablation (RFA) to ablate the remaining metaplastic epithelium.) 2nd line: Oesophagectomy • High-dose proton pump inhibitor: The best next line of management whilst this is commonly used in patients with Barrett's the evidence base that this reduces the change of progression to dysplasia or induces regression of the lesion is limited

Histology at biopsy Endoscopy frequency Actions No dysplasia Every 2 - 5 years

Low-grade dysplasia Every 6 months Repeat endoscopy with quadrantic biopsies every 1cm. High-grade dysplasia Every 3 months If a visible lesion is present, consider endoscopic ablation with mucosal resection (EMR) or radiofrequency ablation.

Prognosis • ↑↑ risk of oesophageal adenocarcinoma (50-100 fold), although the absolute risk is low (< 1%).

Barrett's oesophagus

Gastroenterology

Oesophagitis in immunosuppressive patients

Causes • Candidal esophagitis is the most common cause of symptomatic disease.
• Ulcerative esophagitis resulting from cytomegalovirus is the next most important etiologies.
 Cytomegalovirus (CMV) most commonly causes multiple ulcers at the lower esophageal sphincter • Herpes simplex virus esophagitis appears to be relatively uncommon.
Features • The hallmark of oesophagitis is odynophagia or pain on swallowing. Diagnosis
• Endoscopy with a biopsy Treatment
• Candida spp.
 Nonpregnant patients, we suggest initial therapy with fluconazole Pregnant patients → amphotericin B is the treatment of choice during the first trimester since oral azoles are teratogenic. • Herpes simplex virus (HSV) → oral or iv acyclovir • Cytomegalovirus → ganciclovir or valganciclovir

• Oesophagitis in the immunocompromised that presents with punched-out ulcers → Herpes simplex virus-1
• Oesophagitis in the immunocompromised that presents with a white pseudomembrane → Candida spp.
• Oesophagitis in the immunocompromised that presents with linear ulcers → Cytomegalovirus

Candida oesophagitis
Although oropharyngeal candidiasis may be treated with topical antifungal agents (such as nystatin, clotrimazole, and amphotericin B oral suspension/lozenges) Candida oesophagitis requires oral or IV therapy (usually with fluconazole or itraconazole for at least 14-21 days).

HIV patient presented with painfull swallowing difficulty, an upper GI endoscopy shows ulcerative oesophagitis, what is the most likely diagnosis?

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Eosinophilic oesophagitis

Overview

• Chronic immune-mediated eosinophil-predominant inflammation of the esophageal mucosa Should be considered in adults wit: History of food impaction, with persistent dysphagia, or Gastroesophageal reflux disease that fails to respond to medical therapy. • Associated with Allergies : (e.g., asthma, rhinitis, atopic dermatitis, alimentary allergies) Features • Episodic oesophageal spasm and intermittent dysphagia Diagnosis
• Upper endoscopy with esophageal biopsy → presence of epithelial infiltrate of ≥ 15 eosinophils per high-power microscopy field

Treatment • Diet modification. Refer for allergy testing (Eosinophilic esophagitis commonly associated with allergies). Once an antigen is identified, avoidance can improve symptoms • Proton pump inhibitors. If the patient does not respond to a low-dose proton pump inhibitor, it is appropriate to increase the dose to a maximal dose before trying other treatment strategies. • Topical “swallowed” corticosteroids for 8 weeks

Patients who present with food impaction, dysphagia, and history of atopy should undergo an upper endoscopy evaluation with esophageal biopsy to diagnose eosinophilic esophagitis.

Oesophageal cancer

Epidemiology • Sex: ♂ > ♀ (3:1)
• Squamous cell carcinoma (SCC) is the most common type of esophageal cancer worldwide.
• Adenocarcinoma: most common type of esophageal cancer in the UK and US

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Types
• Adenocarcinoma
 The most common form of esophageal cancer in the UK and US. Affects primarily white men.
 begins in the cells of mucus-secreting glands (glandular cells of the submucosa) in the esophagus.
 Occurs most often in the lower portion of the esophagus.
• Squamous cell carcinoma (SCC) The most prevalent esophageal cancer worldwide. Develops in the thin, flat cells of the mucosa , which line the oesophagus. Occurs most often in the upper two-thirds of the esophagus.
Risk factors
• Risk factors for SCC Smoking Alcohol (Unlike adenocarcinoma). Diet Red meat consumption Low selenium levels. selenium supplementation reduces the risk Zinc deficiency
 Low dietary folate intake low intake of fruits and vegetables
 Hot liquids
 Tylosis (rare, autosomal dominant disorder characterized by hyperkeratosis of the palms and soles, with thickening and fissuring of the skin.) Achalasia cardia Plummer-Vinson syndrome
 Oral bisphosphonates
 Poor oral hygiene Infection with the human papillomavirus (HPV)

• Risk factors for adenocarcinoma Gastroesophageal reflux (GORD) → the most common predisposing factor Barrett esophagus Smoking (twofold risk) Obesity Male sex Older age (50–60 years)

Oesophageal cancer risk factors • Alcohol is NOT a risk factor for Adenocarcinoma
• H. pylori infection associated with DECREASE incidence of oesophageal cancer. Helicobacter pylori may actually be protective against oesophageal cancer

The most important risk factors for esophageal adenocarcinoma are gastroesophageal reflux and associated Barrett esophagus.

The primary risk factors for squamous cell esophageal cancer are alcohol consumption, smoking, and dietary factors (e.g., diet low in fruits and vegetables).

Dermatological conditions associated with oesophageal carcinoma →Tylosis (95% will get squamous oesophageal cancer)

Localization
• Squamous cell esophageal cancer: mostly in the upper two-thirds of the esophagus • Adenocarcinoma: mostly in the lower third of the esophagus

Features • Early stages: Often asymptomatic • Late stage: progressive dysphagia, initially worse on solids and then later to include liquids

• Sudden onset of hiccups is common when tumor spreads to diaphragm • General signs: Weight loss, dyspepsia, anaemia

Diagnosis • Upper GI endoscopy is the first line test • Staging: For local tumor extent (mural invasion or tumour depth): Endoscopic ultrasound. For distant metastases: CT of the Chest, abdomen and pelvis. PET/CT scan is more sensitive than CT for detecting metastatic disease and are now widely used for detecting occult metastases if metastases are not seen on the initial staging CT scans.

Treatment • Superficial intramucosal oesophageal cancer is best managed by endoscopic resection and surveillance. • Early-stage cancers in surgical candidates are best treated by oesophagectomy. • For locally advanced disease, combined modality therapy is considered the current standard. This involves chemotherapy or chemoradiotherapy followed by surgery. • High-risk patients should be treated with a combination of chemotherapy and radiotherapy for best results, but local recurrence rates remain high. • Palliative Opioid for pain relief
 Nifedipine helps relieve painful oesophageal spasm and tenesmus associated with gastrointestinal tumours and could be used to relieve his odynophagia. Oesophageal cancer • Most oesophageal cancers are not resectable at presentation
• Chemo-radiotherapy then surgery is preferred to surgery alone.

What is the most common type of Oesophageal cancer? • Squamous cell carcinoma is the most prevalent esophageal cancer worldwide. • Adenocarcinoma is the most common form of esophageal cancer in the UK and United States

Squamous cell carcinoma (SCC) Prevalence More common worldwide More common in UK/US Major risk factors Smoking, alcohol Achalasia, Plummer Vinson Notes & Notes for MRCP
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Adenocarcinoma Barrett's oesophagus, GORD, smoking, and obesity.

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Part of oesophagus affected
Upper 2/3 Lower 1/3 Prognosis
Poor long -term prognosis after resection Better long-term prognosis after resection than that of SCC Treatment
More sensitive to chemo-radio therapy than adenocarcinoma

Prognosis • Poor prognosis due to advanced disease at presentation. • Most patients present with stage 3 disease (late stage) and survival at 5 years is only 9%.

Pharyngeal pouch

Definition • A pharyngeal pouch is a posteromedial diverticulum through Killian's dehiscence.
 Killian's dehiscence is a triangular area in the wall of the pharynx between the thyropharyngeus and cricopharyngeus muscles.
• Upper esophageal diverticulum (Common site) • Zenker’s diverticulum is the most common type of esophageal diverticula defined as a posterior "false" diverticulum that has a neck proximal to the cricopharyngeal muscle.

Epidemiology • more common in older patients
• 5 times more common in men

Associations • Achalasia → Inadequate relaxation of the esophageal sphincter ↑intraluminal pressure → outpouching of the esophageal wall → pulsion diverticulum (e.g., Zenker diverticulum) • Inflammation of the mediastinum with scarring and retraction (e.g., secondary to tuberculosis or fungal infection) → traction diverticulum (Common site: the middle esophagus) • Gastro-oesophageal reflux disease

Features • Dysphagia (most common) • Regurgitation of undigested food • Aspiration • Coughing after food intake • Retrosternal pressure sensation and pain • Halitosis (a bad breath) • Neck swelling which gurgles on palpation (Boyce’s sign) • Weight loss

Diagnosis • Barium studies (best confirmatory test) detected best by using lateral X-ray shows a contrast-filled pouch protruding dorsally from the hypopharynx at the level of C5/C6
 Upper gastrointestinal endoscopy is risky, since the pouches are thin-walled and easy to perforate; this is the reason why a barium swallow may be the preferable first-line investigation in elderly patients with dysphagia.

• Upper endoscopy under direct vision should be performed to exclude malignancy.

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Treatment
• Diverticula of the upper esophagus :Surgical, with either an open or endoscopic approach. • Diverticula of the middle and distal esophagus (traction diverticula and epiphrenic diverticula) usually do not require treatment (most of them are asymptomatic).

Acute upper gastrointestinal bleeding (UGIB) (NICE 2012) Definition • bleeding derived from a source proximal to the ligament of Treitz.

Causes: Most commonly due to either:
• peptic ulcer disease or
• oesophageal varices. Risk assessment • use the Blatchford score at first assessment, and • the full Rockall score after endoscopy Blatchford score • The Blatchford score is based on clinical parameters alone: Elevated blood urea nitrogen Reduced haemoglobin A drop in systolic blood pressure Raised pulse rate The presence of melaena or syncope, and Evidence of hepatic or cardiac disease.

Admission risk marker Score Urea (mmol/l) 6.5 - 8 = 2 8 - 10 = 3 10 - 25 = 4

> 25 = 6 Haemoglobin (g/l) Men • 12 - 13 = 1 • 10 - 12 = 3 • < 10 = 6 Women

Chapter 3

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Admission risk marker Score Systolic blood pressure (mmHg) 100 - 109 = 1 90 - 99 = 2 < 90 = 3 Other markers Pulse >=100/min = 1 Presentation with melaena = 1 Presentation with syncope = 2 Hepatic disease = 2 Cardiac failure = 2 Patients with a Blatchford score of 0 may be considered for early discharge

Rockall score
• Used to: determine the prognosis of upper GIT bleeds.
 assess severity of GIT bleeds and / or to triage patients for emergency endoscopy. • Consists of 5 categories:

1. age
2. shock
3. co-morbidity e.g. ischaemic heart disease (IHD)
4. diagnosis and
5. evidence of bleeding (the latter two can only be categorised after endoscopy). Each category is scored between 0 and 2 points, with the exception of co-morbidities which has a maximum score of 3.
   Renal failure, liver failure and metastatic cancer carry the highest points, and thus confer the highest risk of death, of any of the other parameters included in the scoring system. • The full Rockall scoring system is shown in the table below:

Score 0 Score 1 Score 2 Score 3 Age <60 60-79

> 80

-

Shock No shock Pulse >100 Systolic blood pressure <100 mmHg Co-morbidity Nil major

CCF, IHD, major morbidity Diagnosis MalloryWeiss tear All

other diagnoses Evidence of bleeding None

Blood, adherent clot, spurting vessel • Interpretation: Increasing scores are strongly correlated with increasing risk of mortality,
 The total score predicts mortality as follows:
Notes & Notes for MRCP
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• 10 - 12 = 1 • < 10 = 6

Renal or liver failure, metastatic cancer GI malignancy

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 Score 0, 0.2%;
 score 2, 5%;
 score 4, 24%;
 score 6, 49%.
 correlation with risk of re-bleeding is also present but not as strong. Grades of hypovolaemic shock
The table below outlines the signs and symptoms of the different grades of hypovolaemic shock:
Grade

• Up to about 15% loss of effective blood volume (~750ml in an average adult who is assumed to have a blood volume of 5 litres).
• This leads to a mild resting tachycardia and can be well tolerated in otherwise healthy individuals.
• In the elderly or those with underlying conditions such as ischaemic heart disease the additional myocardial oxygen demands may not be tolerated so well. Grade

• Between 15-30% loss of blood volume (750-1500ml)
• will provoke a moderate tachycardia and begin to narrow the pulse pressure.

• The capillary refill time will be extended. Grade

• At 30 - 40% loss of effective blood volume (1500 - 2000 ml)
• the compensatory mechanisms begin to fail and hypotension, tachycardia and low urine output (<0.5ml/kg/hr in adults) are seen. Grade

• At 40-50% loss of blood volume (2000-2500 ml)
• profound hypotension will develop and if prolonged will cause end-organ damage and death.

Blood test evidence of upper gastrointestinal haemorrhage • Reactive thrombocytosis
• Urea elevated in excess of creatinine

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Gastroenterology

Treatment • Resuscitation ABC, wide-bore intravenous access
 platelet transfusion if actively bleeding platelet count of less than 50 x 10*9/litre fresh frozen plasma to patients who have either a fibrinogen level of less than 1 g/litre, or a prothrombin time (international normalised ratio) or activated partial thromboplastin time greater than 1.5 times normal prothrombin complex concentrate to patients who are taking warfarin and actively bleeding • Endoscopy should be offered immediately after resuscitation in patients with a severe bleed all patients should have endoscopy within 24 hours Recent NICE guidelines do not recommend proton pump inhibition (PPIs) before endoscopy.
 He may have alcohol dependency and therefore should be prescribed Pabrinex whilst waiting for endoscopy.
• Management of non-variceal bleeding NICE do not recommend the use of proton pump inhibitors (PPIs) before endoscopy to patients with suspected non-variceal upper gastrointestinal bleeding although PPIs should be given to patients with non-variceal upper gastrointestinal bleeding and stigmata of recent haemorrhage shown at endoscopy The best evidence for pharmacological intervention post-stabilisation of bleeding peptic ulcer disease is for proton pump inhibitors.
 the most appropriate intervention to prevent further bleeding IV omeprazole reduction in risk of recurrent bleeding of over 50%,
 reduction in need for surgical intervention of approximately 40%.
 if further bleeding then options include repeat endoscopy, interventional radiology and surgery

• Management of variceal bleeding terlipressin and prophylactic antibiotics should be given to patients at presentation (i.e. before endoscopy) band ligation should be used for oesophageal varices and injections of N-butyl-2cyanoacrylate for patients with gastric varices transjugular intrahepatic portosystemic shunts (TIPS) should be offered if bleeding from varices is not controlled with the above measures

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Oesophageal varices

Overview • Oesophageal varices are tributaries of the left gastric vein , found in lower 1/3 of esophagus lower 1/3 of oesophagus is drained into the superficial veins lining the esophageal mucosa, left gastric vein portal vein. upper 2/3 of oesophagus are drained via esophageal veins azygos vein superior vena cava. (These veins have no part in the development of esophageal varices) • Esophageal varices are the most common cause of death in cirrhosis.