MODULE: CLINICAL PATHOLOGY

UNIT: PATHOLOGY OF GASTROINTESTINAL SYSTEM

Carey Francis Okinda
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Outline

	Topic	Duration (Hrs)
1.	Introduction to Pathology of Digestive System	2
2.	Disorders of the Oral Cavity **(TAKE AWAY ASSIGNMENT)**	1
3.	Disorders of the Oesophagus	1
4.	Disorders of the stomach	1
5.	PUD	1
6.	GIT Bleeding	1
7.	Disorders of the Small Intestines	3
8.	Disorders of the Large Intestines	1
9.	Disorders of the Rectum and Anus	1
10.	Disorders of the peritoneum	2
	TOTAL	**14**

Lesson 1: INTRODUCTION TO GIT PATHOLOGY

Learning Objectives

At the end of the lesson the learner will be able to: -

1) Describe the structure and function(s) of all the organs of the digestive tract
2) Explain the pathophysiology of GIT disorders
3) Investigate GIT disorders

1.0 INTRODUCTION

- GIT is the portal through which nutritive substances and fluid enter the body
- Digestion and absorption are the two major functions of the digestive system
- Primary function of the digestive system is to bring essential nutrients into the internal environment so that they are available to each cell of the body

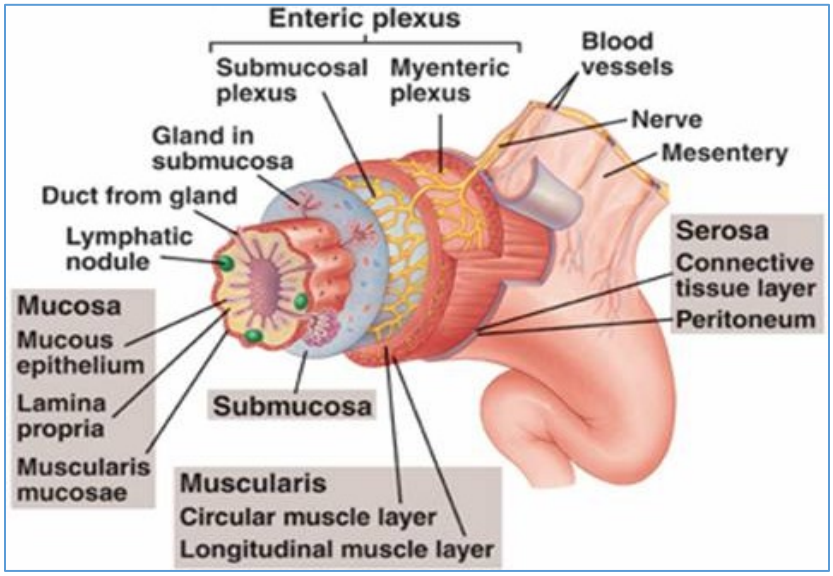
2.0 ORGANIZATION

- Comprises of the digestive tract and digestive glands (salivary glands, pancreases) and biliary system (liver, gall bladder and bile ducts)
- Organs include – oral/buccal cavity, oesophagus, stomach, small intestine (duodenum, jejunum and ileum), large intestine (ascending colon, transverse colon, descending colon and sigmoid), rectum and anal canal
- Accessory organs include teeth, tongue, salivary glands, liver, gall bladder, pancreas & vermiform appendix

3.0 STRUCTURE

- Alimentary canal (digestive tract) is a tubular approximately 9 m (30 ft) long
- Extends from the mouth to the anus
- Walls have 4 layers (mucosa, sub mucosa, muscularis and serosa) with different modifications at various levels

Diagram 1.1: Layers of Alimentary Tract



4.0 GASTROINTESTINAL BLOOD FLOW

- Extensive splanchnic circulation which includes blood flow to the gut, spleen, pancreas and liver
- All the blood that passes through the gut, spleen, and pancreas then flows into the liver through the portal vein and passes through the liver sinusoids and leaves via the hepatic veins to empty into the vena cava of the general circulation

5.0 NERVE SUPPLY

- By the enteric nervous system

i) Intrinsic Innervation

- Enteric nervous system composes of two plexuses –
  a) Myenteric (Auerbach's) plexus
     - Between longitudinal and circular muscle layers)
     - Innervates longitudinal and circular smooth muscles layers (motor control of the GIT movements)
  b) Sub-mucosal plexus (Meissner's plexus)
     - Lies in the sub mucosa
     - Innervates glandular epithelium, intestinal endocrine cells and sub mucous blood vessels (control GIT secretions and local blood flow)
- Plexuses are connected to the sympathetic and parasympathetic divisions of the autonomic nervous system as well as to the prevertebral ganglia of the sympathetic nervous system, spinal cord and brain stem (via the vagus nerve)
- Neurotransmitters of enteric neurones include acetylcholine and norepinephrine (most important) and adenosine triphosphate, serotonin, dopamine, cholecystokinin, substance P, vasoactive intestinal polypeptide and somatostatin.

ii) Extrinsic Innervation

- Intestines receive dual extrinsic innervation from autonomic nervous system
  a) Parasympathetic cholinergic activity
     - Increases activity of intestinal smooth muscle
     - Has cranial and sacral divisions
     - Cranial division
       - Via the vagus nerve except those for the mouth and pharynx
       - Innervation to the oesophagus; stomach, pancreas and a less extend to the small intestine and first half of the large intestine
     - Sacral division
       - Originate in the S2, S3 and S4 segments of the spinal cord and pass through the pelvic nerve to the distal half of the large intestine
     - Sigmoid, rectal and anal regions of the large intestine are considerably better supplied with parasympathetic fibres than other intestinal areas.
  b) Sympathetic adrenergic activity
     - Reduces intestinal activity and contraction of sphincters
     - Fibres to the GIT originate in the spinal cord between T5 and L2
     - Innervate essentially all portions of the GIT rather than being more extensively supplied to the portions nearest the oral cavity and anus as the parasympathetic fibres.

Sensory Nerves

- Many afferent nerve fibres arise in the gut
- Can be stimulated by irritation of the gut mucosa, excessive distension of the gut and presence specific chemical substances.

Gastrointestinal Reflexes

- 3 gastrointestinal reflexes essential to gastrointestinal function control
  i) Reflexes integrated entirely within the enteric nervous system control gastrointestinal secretion, peristalsis, mixing contractions and local inhibitory effects.
  ii) Reflexes from the gut to the pre-vertebral sympathetic ganglia and back to the GIT. They transmit signals for long distances in the GIT e.g. gastro-colic reflex, entero-gastric reflexes and colono-ileal reflex
  iii) Reflexes from the gut to the spinal cord or brain and back to the GIT
     a. From stomach and duodenum to the brain stem and back through the vagus nerve controls gastric motor and sensory activity
     b. Pain reflexes – cause general inhibition of the entire GIT
     c. Defecation reflexes – from the colon and rectum to the spinal cord and back to produce powerful colonic, rectal and abdominal contractions

6.0 FUNCTIONS

1) Break up food into smaller pieces
2) Transporting food through the GI tract (gastrointestinal)
3) Secreting digestive enzymes
4) Absorbing nutrients into the blood
5) Excreting solid waste products (waste)

6) Immune system

7.0 DEFENCE MECHANISMS

Diagram 1.2: Defence Mechanisms

Non-Specific Resistance		Specific Resistance
First line	Second line	Third line
• Intact skin • Mucous membranes • Secretions • Normal microbiota	• Phagocytic WBCs • Inflammation • Fever • Antimicrobial substances	• Specialized lymphocytes B and T cells • Antibodies

Physical and Chemical

- Mucus membranes – layers of mucosal cells that line body cavities that open to the outside (digestive, genitourinary and respiratory tracts). Mucus is produced by the mucosal cells contains antimicrobial substance such as lysozymes, lactoferrin (sequester iron). Mucosal cells are rapidly dividing → flush out of body along with attached bacteria
- Mouth and lower digestive tract – lots of bacteria (mostly anaerobes e.g. *Bacteroides,* anaerobic streptococci [*Streptococcus mutans* in mouth] and *Clostridium* in colon)
- Saliva (contains lysozyme), bile (alkaline) in small intestine and stomach acids
- Defecation (faeces contains up to 50% bacteria)
- Mucus contain antibacterial agents, antibodies and immune cells called phagocytes

8.0 PATHOPHYSIOLOGY OF GIT DISORDERS

- Include trauma and physical damage; Genetic disorders; Congenital malformations; Disorders of blood supply (ischaemia); Tumours; Poor diet and nutrition; Infections; Insufficient or excessive digestive acids; Malfunction of the liver, pancreas or gallbladder; Immune system dysfunction

9.0 FEATURES OF GIT DISORDERS

Discussion:

- What are the features of GIT disorders?
- How do these features come about (pathophysiology)?

1) NAUSEA AND VOMITING

Discuss the causes and pathophysiology of nausea and vomiting

2) DIARRHOEA

1.1 Introduction

- Diarrhoeal disease is the second leading cause of death in children under five years old killing around 760 000 children every year.
- Globally, there are nearly 1.7 billion cases of diarrhoeal disease every year.
- Diarrhoea is a leading cause of malnutrition in children under five years old.

1.2 Definition

- Passage of three or more loose or liquid stools per day (or more frequent passage than is normal for the individual)

- Diarrhoea is an increase in the volume of stool or frequency of defecation
- Is the too rapid evacuation of too fluid stools more than 200 grams of stool in 24 hours
- Pseudo diarrhoea – more frequent bowel movement but less than 200 grams in 24 hours
- Incontinence – involuntary loss of stool due to anal sphincter dysfunction or neurologic impairment
- **NOTE**: Frequent passing of formed stools is not diarrhoea, nor is the passing of loose, "pasty" stools by breastfed babies

1.3 Classification

- Acute diarrhoea – diarrhoea lasts less than 2 weeks
- Chronic diarrhoea – diarrhoea lasts more than 2 weeks
- Persistent diarrhoea lasting longer than two weeks but resolving within a month
- There are three clinical types of diarrhoea:
  - Acute watery diarrhoea – lasts several hours or days, and includes cholera;
  - Acute bloody diarrhoea – also called dysentery; and
  - Persistent diarrhoea – lasts 14 days or longer.

1.4 Causes of Diarrhoea

	Description	Examples
1.	Secretory diarrhoea	Infectious (damage mucosal epithelium) e.g. Rota virus, Enteric adenoviruses; Infectious (enterotoxin mediated injury) e.g. Vibrio cholera; E. coli, Clostridium Neoplastic; Excess laxative use
2.	Osmotic diarrhoea	Antacids; lactose deficiencies; gastric lavage
3.	Exudate diseases	Infectious – damage to mucosal epithelium e.g. Shigella; Salmonella; Campylobacter; E. histolytica Idiopathic inflammatory bowel disease - Ulcerative colitis and Chron's disease
4.	Malabsorption	Defective intraluminal digestion; Primary mucosal cell abnormalities Reduced small intestinal surface area; Lymphatic obstruction; Infectious – impaired mucosal cell absorption
5.	Deranged Motility	Decreased intestinal transit time e.g. surgical reduction of the gut length; Neural dysfunction; Hyperthyroidism; Diabetic neuropathy; Carcinoid syndrome Decreased motility (increased intestinal transit time) e.g. small intestinal diverticula; surgical creation of a blind loop and bacterial overgrowth in the small intestine

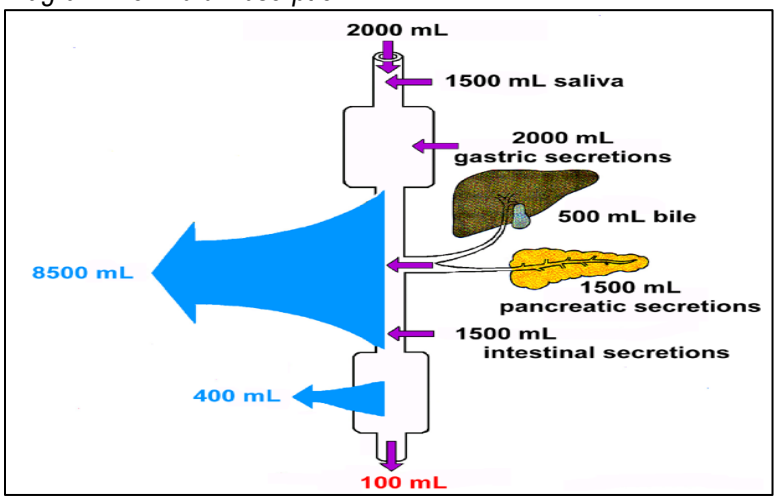
1.5 Physiology

Intestinal absorption of water and electrolytes

- Absorption of water from the small intestine is caused by osmotic gradients created when solutes (particularly sodium) are actively absorbed from the bowel lumen by the villous epithelial cells through several mechanisms
- To enter the epithelial cells, sodium is linked to the absorption of chloride, or absorbed directly as sodium ion, or exchanged for hydrogen ion, or linked to the absorption of organic materials such as glucose or certain amino acids
- Addition of glucose to an electrolyte solution can increase sodium absorption in the intestine as much as threefold.
- After being absorbed, sodium is transported out of the epithelial cells by an ion pump (Na+K+ ATPase) which transfers sodium into the extracellular fluid (ECF) elevating the osmolality that causes water and other electrolytes to flow passively from the bowel lumen through intercellular channels and into the ECF

- This process maintains an osmotic balance between fluid in the bowel and ECF in the intestinal tissue.

Diagram 1.3: Fluid Absorption



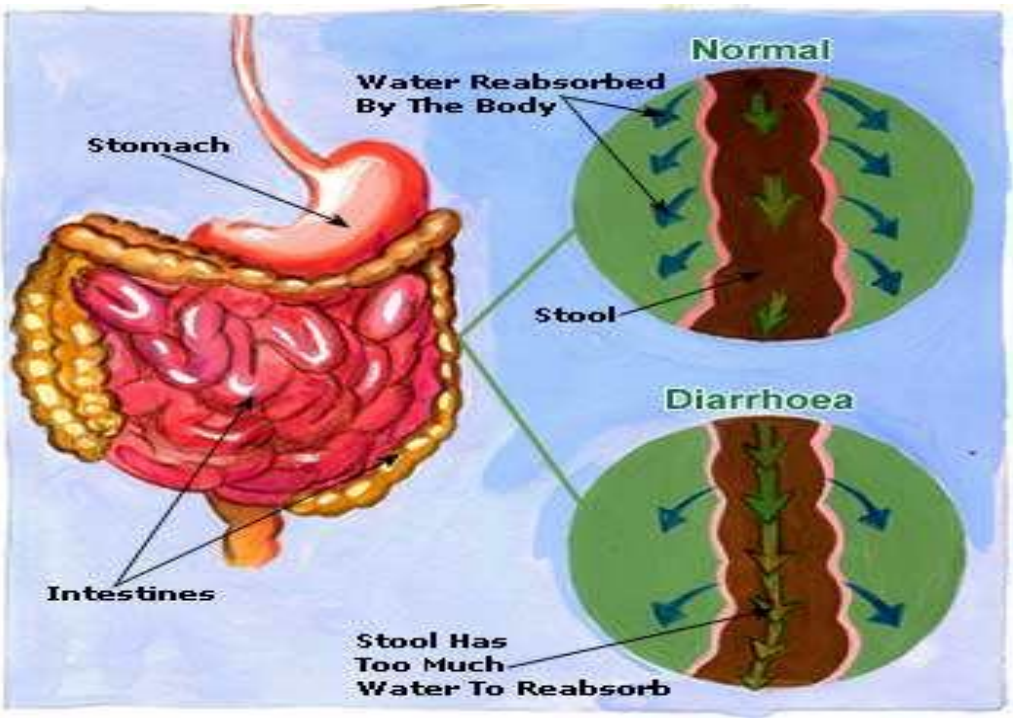
Intestinal secretion of water and electrolytes

- Normally occurs in the crypts of the small bowel epithelium where NaCl is transported from ECF into the epithelial cell across its basolateral membrane
- Sodium is then pumped back into the ECF by Na+K+ ATPase
- At the same time, secretory stimuli increase the ability of chloride to pass through the luminal membrane of the crypt cells, allowing that ion to enter the bowel lumen
- Movement of chloride ion creates an osmotic gradient that causes water and other electrolytes to flow passively from the ECF into the bowel lumen through the intercellular channels.

1.6 Pathophysiology (Mechanisms of Diarrhoea)

- Two principal mechanisms include secretion and osmotic imbalance

Diagram 1.4: Mechanisms of Diarrhoea



Secretory diarrhoea

- Caused by the abnormal secretion of fluid (water and salts) into the small bowel
- Secretion of water into the intestinal lumen exceeds absorption and the absorption of sodium by the villi is impaired while the secretion of chloride in the crypts continues or is increased
- Net fluid secretion causes loss of water and salts from the body as watery stools causing **dehydration**
- Causes
    - In infectious diarrhoea, changes result from the action of bacterial toxins or viruses on the bowel mucosa
        a) *Vibrio cholerae*, produces a toxin, which activates adenylyl cyclase causing prolonged increase in intracellular concentration of cAMP within crypt enterocytes resulting in prolonged opening of the chloride channels that are instrumental in secretion of water from the crypts, allowing uncontrolled secretion of water. Additionally, the toxin affects the enteric nervous system resulting in an independent stimulus of secretion
        b) Exposure to toxins from types of bacteria (e.g. *E. coli* heat-labile toxin) induce the same series of steps and massive secretory diarrhoea that is often lethal unless the person or animal is aggressively treated to maintain hydration
        c) Other bacterial toxins and agents induce secretory diarrhoea by turning on the intestinal secretory machinery including:
            i. Some laxatives
            ii. Hormones secreted by certain types of tumours
            iii. Drugs (e.g. some types of asthma medications, antidepressants, cardiac drugs)
            iv. Certain metals, organic toxins, and plant products (e.g. Arsenic, insecticides, mushroom toxins, caffeine)

Osmotic diarrhoea

- Small bowel mucosa is a porous that allows water and salts to move across rapidly to maintain osmotic balance between the bowel contents and the blood
- Diarrhoea can occur when a poorly absorbed, osmotically active substance is ingested

- If the substance is taken as an isotonic solution, the water and solute will simply pass through the gut unabsorbed, causing diarrhoea.
- Causes
  a) Purgatives, such as magnesium sulphate, work by this principle
  b) Lactase deficiency or glucose malabsorption (occasional complications of enteric infections)
  c) Ingestion of a poorly absorbed substrate - offending molecule is usually a carbohydrate or divalent ion. Common examples include mannitol or sorbitol, epson salt ($MgSO_4$) and some antacids ($MgOH_2$).
  d) Malabsorption: Inability to absorb certain carbohydrates is the most common deficit in this category of diarrhoea, but it can result virtually any type of malabsorption

Inflammatory and Infectious Diarrhoea

- Epithelium of the digestive tube is protected by a number of mechanisms constituting the gastrointestinal barrier
- Destruction of the epithelium results in exudation of serum and blood into the lumen and widespread destruction of absorptive epithelium
- Water absorption occurs very inefficiently and diarrhoea results
- Pathogens frequently associated with infectious diarrhoea include - Bacteria: *Salmonella, E. coli, Campylobacter*; Viruses: rotaviruses, coronaviruses, parvoviruses (canine and feline), Noro virus; Protozoa: coccidia species, *Cryptosporium*, *Giardia*

Altered Intestinal Motility

- ENS contains many neurotransmitters including 5HT, substance P, VIP and CGRP
- ENS controls motility and secretory functions of the intestine
- ENS functions autonomously but may be modified by the sympathetic and parasympathetic nervous systems
- Causes - autonomic diabetic neuropathy, after vagotomy (peptic ulcer surgery) and irritable bowel syndrome

1.7 Types of Dehydration

1) Isotonic dehydration
2) Hypertonic (hypernatraemic) dehydration
3) Hypotonic (hyponatraemic) dehydration

Outline the causes, pathophysiology and clinical features of the different types of dehydration (REF: CIRCULATORY DISORDERS LEVEL 1)

1.8 Effects of Diarrhoea

1) Dehydration
   - Most dangerous because it can cause hypovolaemia, cardiovascular collapse, electrolyte imbalance and death if not treated promptly.
2) Metabolic acidosis
   - Due to loss of a large amount of bicarbonate may be lost in the stool
   - If the kidneys continue to function normally, much of the lost bicarbonate is replaced by the kidneys and a serious base deficit does not develop. However, this compensating mechanism fails when renal function deteriorates, as happens when there is poor renal blood flow due to hypovolaemia. Then, base deficit and acidosis develop rapidly
   - Acidosis also results from excessive production of lactic acid when patients have hypovolaemic shock
   - Features of base-deficit acidosis include

i) Reduced serum bicarbonate concentration (<10 mmol/l)
ii) Arterial pH is reduced (< 7.10)
iii) Breathing becomes deep and rapid, which helps to raise arterial pH by causing a compensating respiratory alkalosis
iv) Increased vomiting

3) Electrolyte imbalance - Potassium depletion
- Occur due to large faecal losses of this ion (losses are greatest in infants and malnourished children)
- Loss of potassium and bicarbonate together does not cause hypokalaemia because the metabolic acidosis that results from the loss of bicarbonate causes potassium to move from ICF to ECF in exchange for hydrogen ion, thus keeping the serum potassium level in a normal or even elevated range.
- Signs of hypokalaemia may include general muscular weakness, cardiac arrhythmias and paralytic ileus

3) Other features
- Dyspepsia; pain and discomfort; abdominal swelling; constipation; obstipation; dysphagia; odynophagia; haematemesis; belching; malena stools; haematochazia

10.0 INVESTIGATIONS IN GIT DISORDERS

1) Stool examination (analysis)
   a. Macroscopy
      - Pus + blood - dysentery (rbc clumps – amoebic, discrete – bacillary
      - Bulky - steatorrhoea
      - Red streaks - fissure-in-ano, fresh – haemorrhoids/dysentery
      - Black - malignancy/polyp, Fe, constipation
      - Green - children – bacterial infection (pseudomonas)
      - Rice water - cholera / viral diarrhoea
   b. Microscopy
      - Ova - tapeworm, roundworm, threadworm etc.
      - Cysts – E. histolytica; E. coli, Giardiasis, Enterobius etc.
      - Benzidine test (occult blood), benedicts (reducing sugars) and
      - Stercobilinogen (haemolytic anaemia)
   c. Bacteriological – stool culture

2) Endoscopy - oesophagostomy; gastroscopy; sigmoidoscopy; colonoscopy
3) Blood counts - Red and white blood cell counts
4) Blood cultures
5) H. pylori tests
   a. Biopsy – culture and detection of urease enzyme activity by placing the biopsy specimen onto a substrate containing urea and monitoring change in pH.
   b. ^{13}C-breath test - *Helicobacter pylori* is a bacterium that is capable of living in the low pH environment of the stomach; adaptation to this hostile environment involves production of high levels of urease (which can help raise the local pH).
6) Immunoassay for IgG antibody
7) Radiological and imaging - plain abdominal x-rays; Barium studies – meals, swallow, follow through, enema; MRI; Ultrasound; CT scan
8) Biopsy
9) LFTs
10) Blood sugars
11) Tests of nonspecific biochemical abnormalities seen in malabsorption
    - Serum calcium; serum albumin; serum alkaline phosphatase; serum proteins; serum urea nitrogen; hypocholesterolaemia; The prothrombin time; The glucose tolerance curve; Vitamin A
12) Tests used in the evaluation of malabsorption/maldigestion
    i) Carbohydrate malabsorption - D-xylose absorption test (decreased); Disaccharide test (decreased); Breath hydrogen test (increased)
    ii) Fat malabsorption - Faecal fat determination (elevated); ^{14}C-triolein breath test (decreased)
    iii) Bacterial overgrowth - ^{14}C-Xylose breath test (increased)
    iv) Specific disorders - Celiac disease (Endomysial antibody present); Pernicious anaemia (Schilling test); Cystic fibrosis (sweat test)

Lesson 2: DISORDERS OF THE ORAL CAVITY

Learning Objectives

At the end of the lesson the learner will be able to: -

1) Describe the structure and function of all the organs in the oral cavity
2) Describe pathological conditions affecting the oral cavity

DISORDERS OF THE MOUTH

- Mouth cavity is the anterior opening of the digestive system which ingests food. It has a muscular tongue on which are arranged the taste buds. There are two rows of teeth - upper and lower.

1.0 THE TEETH

Outline the disease conditions affecting the teeth

2.0 ORAL MUCOSA

Outline the disease conditions affecting the oral mucosa

3.0 SALIVARY GLANDS

Outline the disease conditions affecting the salivary glands

4.0 OROPHARYNX

Outline the disease conditions affecting the oropharynx

Lesson 3: DISORDERS OF THE OESOPHAGUS

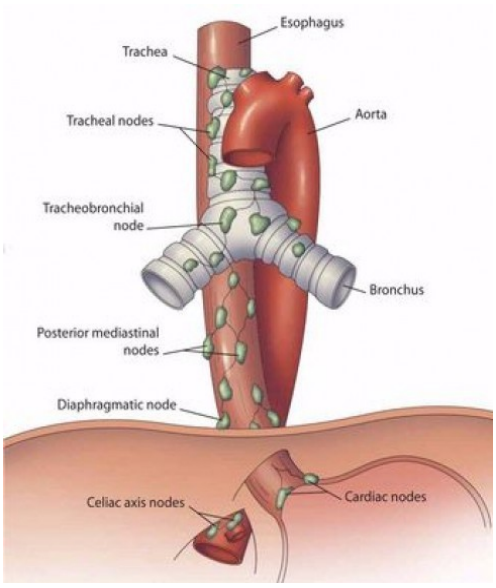
Learning Objectives

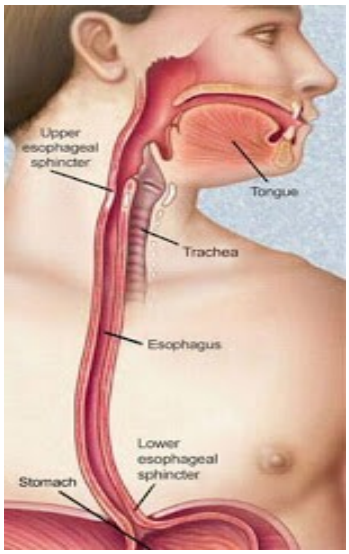
At the end of the lesson the learner will be able to: -
1) Describe the structure and function oesophagus
2) Discuss the pathology of disorders of the oesophagus
3) Investigate disorders of the oesophagus

1.0 INTRODUCTION – ANATOMY AND PHYSIOLOGY

- The oesophagus produces limited symptoms of disease because of its simplicity in function. The common complains include heart burn, dysphagia, odynophagia and hematemesis.

Diagram 3.1: The Oesophagus



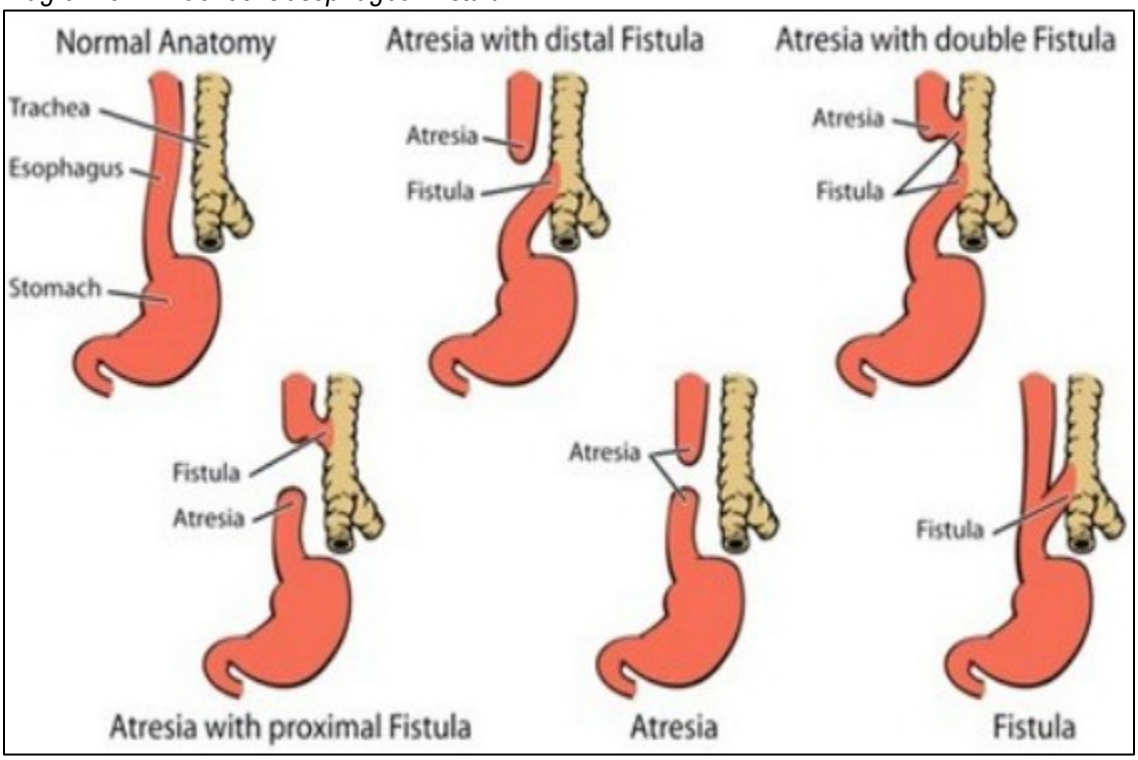


2.0 CONGENITAL ABNORMALITIES

1. **Agenesis**
   - Congenital absence of the oesophagus
   - Rare and incompatible with life

2. **Oesophageal atresia and tracheo-oesophageal fistula**
   - Most cases associated with trachea-oesophageal fistula
   - Tracheo-oesophageal fistula is congenital disorder suggested in a new-born by copious salivation associated with choking, coughing, and cyanosis on attempts at food intake.
   - It occurs in three distinct variants
     i)Communication between lower oesophagus-trachea (near bifurcation)- 90%
     ii)A fistulous connection between the upper oesophagus and the trachea
     iii)A fistulous connection between trachea and a completely patent oesophagus

Diagram 3.2: Tracheo-Oesophageal Fistula



3. Oesophageal Stenosis and webs

- May occur as a developmental anomaly or following oesophagitis
- Involves fibrous thickening of the oesophageal wall and atrophy of the muscularis propria
- Frequently results from severe oesophageal injury with inflammatory scarring
- Usually develops in adulthood
- Presents with progressive dysphagia (solids – liquids)
- These anomalies have been classified histologically as follows:
  Group I - Tracheobronchial rests (cartilage, respiratory mucus glands, ciliated epithelium)
  Group II - Membranous diaphragm
  Group III - Fibromuscular stenosis

4. Duplication of Oesophagus

- The is a double oesophagus

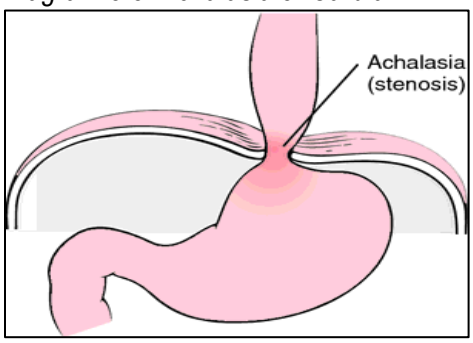
3.0 MUSCULAR DISORDERS

- There is motor dysfunction of the oesophagus and presents with dysphagia
- Include achalasia, hiatus hernia, oesophageal diverticula, webs and rings

3.1. Achalasia of Cardia (Cardiospasm)

- Achalasia means failure to relax
- A neuromuscular dysfunction in which the cardiac sphincter fails to relax during swallowing causing progressive dysphagia and dilatation of the oesophagus (mega-oesophagus).
- The normal peristaltic wave initiated by swallowing dies before it reaches the cardiac sphincter.

Diagram 3.3: Achalasia of Cardia



Clinical Features

- Progressive dysphagia, nocturnal regurgitation and aspiration of undigested food

Differential Diagnosis

- Poliomyelitis, diabetic autonomic neuropathy and infiltrative disorders e.g. malignancy, amyloidosis

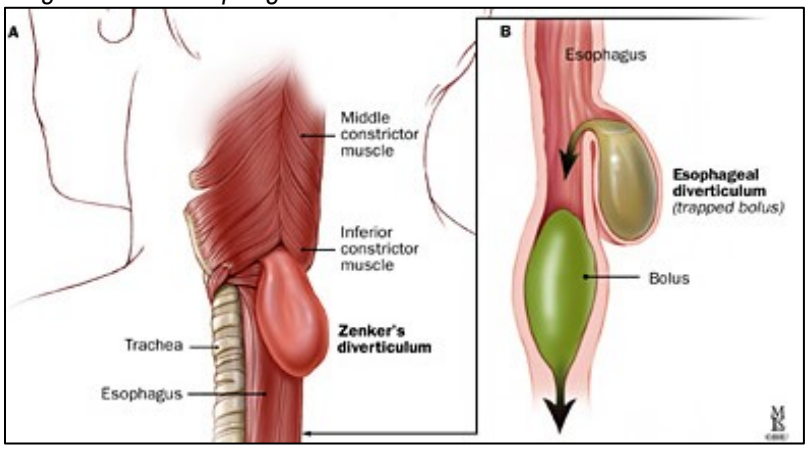
Complications

1. Oesophageal squamous cell carcinoma
2. Oesophagitis
3. Diverticula
4. Aspiration pneumonia
5. Airway obstruction

3.2. Oesophageal Diverticula

- A diverticulum is a pouch extending out from the normal wall of the swallowing channel.
- Diverticula (the plural of diverticulum) can develop in either the pharynx or oesophagus
- Small diverticula may not cause symptoms but larger diverticula can cause dysphagia for liquids and solids.
- Regurgitation of undigested food, often hours after ingestion is a characteristic symptom of patients with diverticula

Diagram 3.4: Oesophageal Diverticula



Aetiology

- Congenital diverticula
- Acquired diverticula (Pulsion/Zenker's and traction type)

Features

Task: What are the features of oesophageal diverticula?

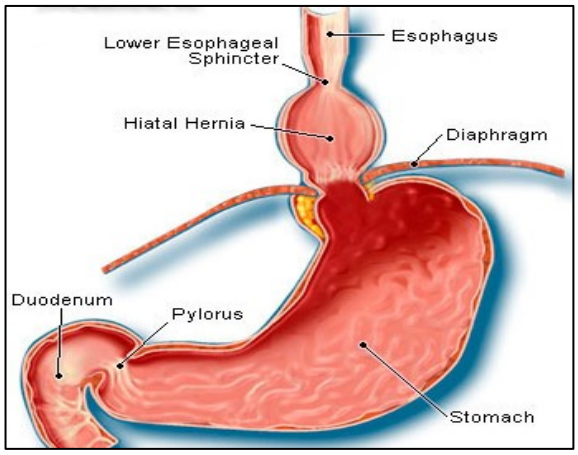
Complications

1) Obstruction
2) Infection
3) Perforation
4) Haemorrhage
5) Carcinoma

3.3. Hiatus Hernia

- Herniation or protrusion of the stomach though the oesophageal hiatus of the diaphragm due to defects in muscle fibres that form the margin of the oesophageal hiatus

Diagram 3.4: Hiatus Hernia



Risk Factors

- Increased pressure within the abdomen caused by - heavy lifting or bending over, frequent or hard coughing, hard sneezing, pregnancy and delivery, violent vomiting, straining with constipation, obesity and se of the sitting position for defecation; heredity, smoking, drug use, stress and diaphragm weakness

Aetiology

1) Congenital
2) Acquired –
   a) Fibrous scarring of the oesophagus due to degeneration (in aging)
   b) Increased intra-abdominal pressure (pregnancy, tumours)
   c) Oesophageal regurgitation and increased fatty tissue (obesity)
   d) Permanent shortening of the oesophagus (perhaps caused by inflammation and scarring from the reflux or regurgitation of stomach acid) which pulls the stomach up.

Clinical Features

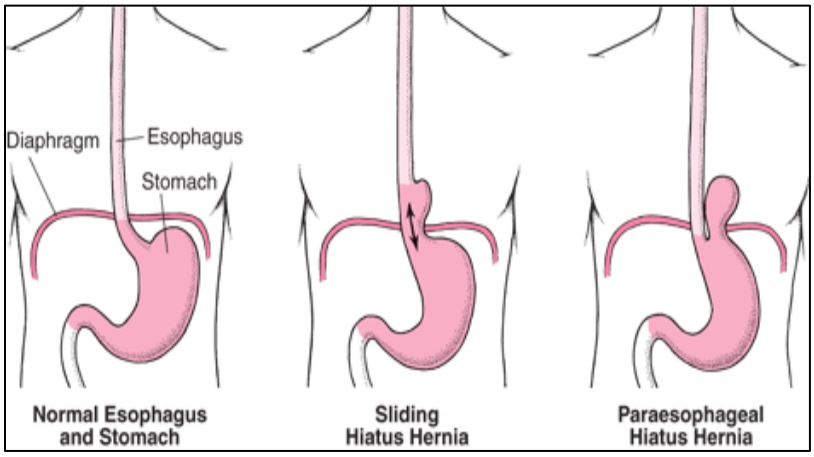
- Heartburn
- Regurgitation of gastric juices

Types

i) Sliding(rolling) hernia - the junction between the oesophagus and the stomach as well as a portion of the stomach itself, all of which are normally below the diaphragm, protrude above it

ii) Paraesophageal hiatus hernia - the junction between the oesophagus and stomach is in its normal place below the diaphragm, but a portion of the stomach is pushed above the diaphragm and lies beside the oesophagus.
iii) Mixed – combination of sliding and paraoesophageal types

Diagram 3.5: Types of Hiatus Hernia



Diagnosis

1) Barium swallow — show outlines the oesophagus, fairly accurate at diagnosing paraesophageal hernias,
2) Endoscopy —check the inner lining of the stomach
3) Oesophageal manometry or motility studies —studies can measure how tightly the LES shuts, and they can also check for abnormalities in oesophageal pressure and movement.
4) Cardiac evaluation — electrocardiogram and an exercise stress test to rule out heart disease.
5) Oesophageal pH monitoring —test uses electrodes to measure the pH (acid level) in the oesophagus, usually over a 24-hour period.
6) Abdominal ultrasound — look for other abnormalities that account for your symptoms including gallbladder problems.

Complications

1) Ulceration
2) Bleeding
3) Perforation
4) Strangulation
5) Obstruction
6) Reflux esophagitis

4.0 VASCULAR ABNORMALITIES

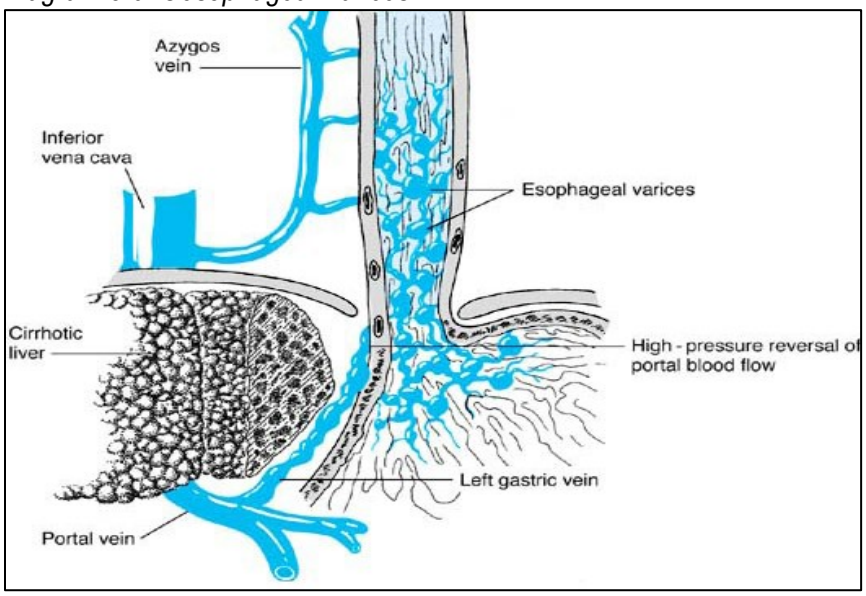
Out all the vascular abnormalities of the oesophagus

5.0 OESOPHAGEAL VARICES

Introduction

- Variceal haemorrhage occurs from dilated veins (varices) at the junction between the portal and systemic venous systems
- Tend to be in the distal oesophagus and/or the proximal stomach, isolated varices may be found in the distal stomach, large and small intestine
- Majority of patients with variceal bleeding have chronic liver disease (bleeding is characteristically severe and may be life-threatening)

Diagram 3.6: Oesophageal Varices



Causes

1) Pre-hepatic - Portal vein thrombosis, portal vein obstruction, congenital atresia/stenosis; increased portal blood flow – fistula and increased splenic flow
2) Intrahepatic - cirrhosis, idiopathic portal hypertension (hepatoportal sclerosis), acute hepatitis (especially alcoholic), schistosomiasis, congenital hepatic fibrosis
3) Post hepatic e.g. compression (e.g. from tumour); Budd-Chiari syndrome; Constrictive pericarditis (and rarely right-sided heart failure)

1.9 Factors that increase the risk of variceal bleeding

- Same factors that increase the risk of portal hypertension e.g.: -decompensation of liver disease; malnourishment; alcohol intake; physical exercise; increased intra-abdominal pressure; aspirin and Non-steroidal anti-inflammatory drugs (NSAIDs); bacterial infection (cause of initial, and recurrence of, bleeding)

1.10 Presentation

Symptoms	Haematemesis (most commonly), melena; abdominal pain; features of liver disease and specific underlying condition; dysphagia/odynophagia (uncommon); confusion secondary to encephalopathy (even coma)
Signs	Peripherally shut down; pallor; hypotension and tachycardia (i.e. shock); reduced urine output; melena; signs of chronic liver disease; reduced Glasgow Coma Scale; signs of sepsis may also commonly be present; splenomegaly; haemorrhoids

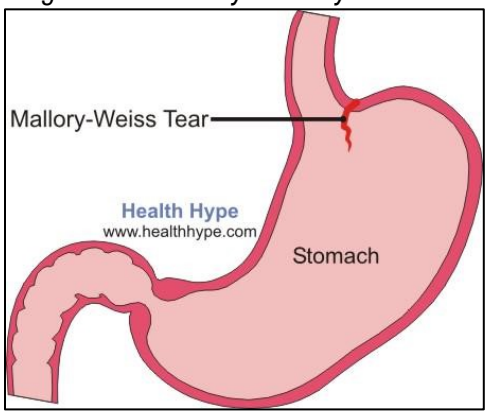
1.11 Investigations

1. Endoscopy
2. TBC - haemoglobin may be low; MCV may be high, normal or low; platelets may also be low
3. Renal function
4. LFTs
5. Group and cross-match
6. CXR - patients may have aspirated or have chest infection
7. Ascitic tap may be needed if bacterial peritonitis is suspected
8. Investigations as indication for the underlying cause of portal hypertension (see separate article Portal Hypertension)

6.0 MALLORY-WEISS SYNDROME

- Longitudinal tears in the oesophagus at the gastroesophageal junction or gastric cardia as a consequence of severe retching and vomiting
- Second most common cause stomach bleeding.
- Tear occurs as a result of excessive pressure or force on the stomach as a result of retching, vomiting, chronic coughing and convulsions (these causes are usually persistent and extremely forceful in order for the tear to occur)

Diagram 3.7: Mallory Weis Syndrome



Clinical Features

- Bleeding (upper GIT); Haematemesis

7.0 INFLAMMATORY LESIONS (OESOPHAGITIS)

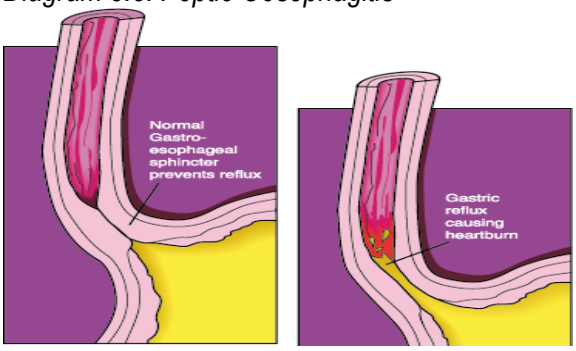
1) Reflux (Peptic) Oesophagitis

- Most common
- Due to persistent regurgitation of gastric juice into the lower oesophagus
- Reflux normally prevented by tonal activity of the cardiac sphincter, constriction effect of the diaphragm and acuteness of the cardio-oesophageal angle
- Predisposing factors are due to increased intra-abdominal pressure e.g. obesity, pregnancy
- Effects - high gastric acid levels damage oesophageal mucosa and reduce the tone of cardiac sphincter

Causes

- Reduced efficacy of oesophageal anti-reflux mechanism
- Presence of sliding hiatus hernia
- Delayed gastric emptying
- Certain foods can relax the muscular ring at the lower end of the oesophagus, responsible for acid reflux. Such foods include citrus fruit, tomatoes, garlic, chocolate, tea, coffee, alcohol, peppermints.
- Dishes high in fat and oil.
- Excess weight can cause extra pressure on the sphincter
- Certain medications, like aspirin.
- Stress, because this strain the nerves controlling the muscular ring.
- Smoking which stimulates stomach acid.

Diagram 3.8: Peptic Oesophagitis



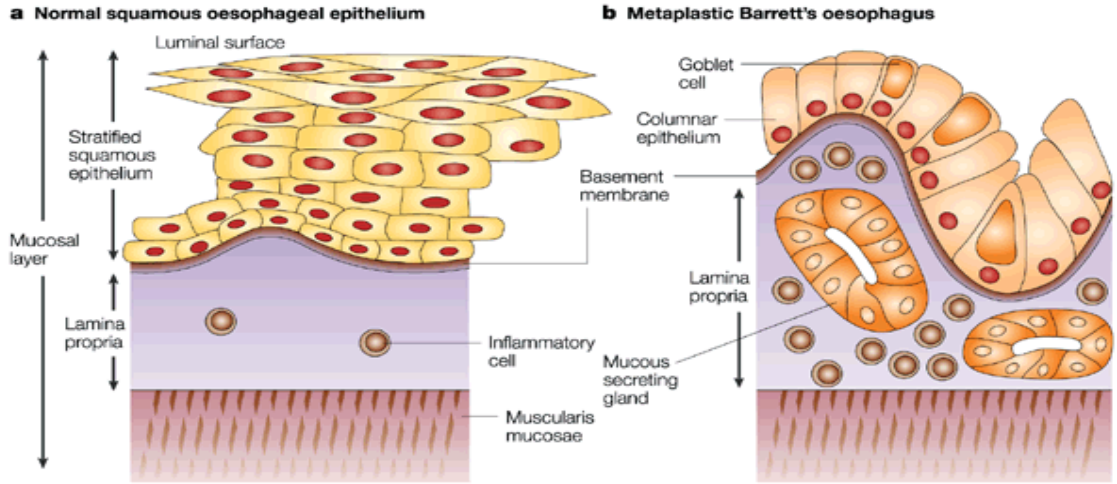
Clinical Features

- Dysphagia; heartburn; hematemesis; melena; a painful burning sensation in the upper chest; sour and burning taste of reflux when it reaches the mouth; hoarse voice from irritated larynx; difficulty breathing; excessive belching; chronic coughing; wheezing and other asthma-like symptoms in adulthood

2) Barret's Oesophagitis

- Consequence of reflux oesophagitis whereby the stratified squamous epithelium of the lower oesophagus is replaced by columnar epithelium
- Metaplasia may lead to chronic peptic ulceration (risk of erosion and perforation), dysphagia and invasive adenocarcinoma

Diagram 3.9: Berret's Oesophagitis



Clinical Features - What are the clinical; features?

3) Infective oesophagitis

- Follows a number of opportunistic infections such as Candidiasis, Herpes, CMV and tuberculosis. This occurs mainly when there is some defect in mucosal resistance

8.0 OESOPHAGEAL OBSTRUCTION

Causes

1. External compression e.g. mediastinal tumours e.g. bronchogenic carcinoma; mediastinal lymphadenopathy e.g. tuberculosis; enlargement of the left atrium; vascular disorders e.g. aneurysms; pharyngeal pouch
2. Intrinsic lesions (strictures) - carcinoma; reflux oesophagitis; corrosive liquids; Chron's disease
3. Oesophageal occlusion - foreign material/bodies and polypoid tumours
4. Functional obstruction – Achalasia; Kelly-Paterson (Plummer-Vinson) syndrome

9.0 NEOPLASMS

9.1. Classification

i) Benign Neoplasms - Are uncommon

ii) Malignant Neoplasms

- Two types originating from the epithelium
- Squamous cell carcinoma is related to diet and smoking
- Adenocarcinoma is increasing in incidence and commonly associated with Barret's oesophagus
- Originate from the epithelium

9.2. Risk Factors

- Age; Gender; Oesophageal disorders (gastroesophageal reflux disease, Barrett's oesophagus, Achalasia, oesophageal webs, injury, diverticula, Plummer-Vinson syndrome); Tobacco and alcohol; Obesity; Diet; Workplace exposures to chemical fumes in certain workplaces; People who have had certain other cancers, (lung cancer, mouth cancer, and throat cancer); Human papilloma virus; Family/genetic

9.3. Clinical Features and Pathology

- What are the clinical features of Ca Oesophagus?
- What are macroscopic and microscopic features of Ca oesophagus
- What are the radiological features of Ca oesophagus?

9.4. Spread

i) Local spread – stomach, hypopharynx, trachea (trachea-oesophageal fistula), larynx, mediastinum, lungs, bronchi, pleura and aorta
ii) Lymphatic spread – cervical, paraoesophageal, trachea-bronchial, sub diaphragmatic nodes
iii) Haematogenous spread – lungs, liver and adrenals

9.5. Complications

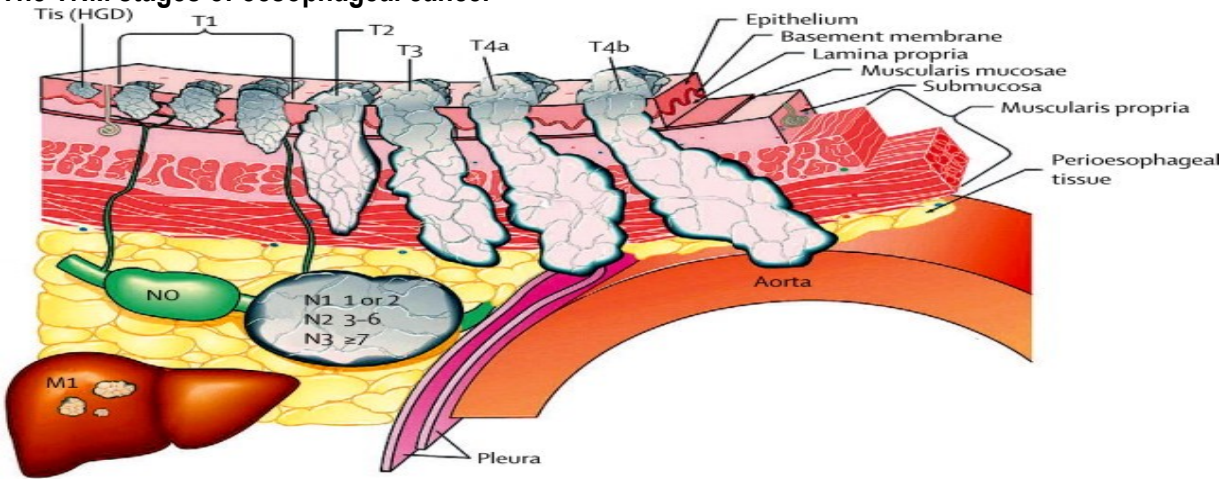
What are the complications of Ca oesophagus?

9.6. Investigations

What are the relevant investigations in Ca oesophagus?

9.7. Staging

The TNM stages of oesophageal cancer



Clinical Staging

Stage	Substage	Characteristics
0	CIN	• High grade dysplasia (HGD) carcinoma in situ (CIS), Severely abnormal cells in the inner lining of the oesophagus.
1	1A	• In the submucosa - T1, N0, M0
	1B	• Grown into the muscle layer - T2, N0, M0.
2	2A	• Membrane covering the outside of the oesophagus, but not spread to nearby lymph nodes (T3, N0, M0)
	2B	• Within the muscle layer and 1 or 2 lymph nodes (T1 or T2, N1, M0) • Has not spread to any other organs
3	3A	• grown into the pleura, pericardium and diaphragm - (T4a, N0, M0), or adventitia and is in 1 or 2 nearby lymph nodes (T3, N1, M0), or spread to 3 to 6 nearby lymph nodes (T1 or 2, N2, M0)
	3B	• adventitia and has spread to 3 to 6 lymph nodes but nowhere else (T3, N2, M0)
	3C	• Pleura, pericardium, diaphragm and is in up to 6 lymph nodes (T4a, N1 or 2, M0), or trachea, vertebra or the aorta and has spread to any number of local lymph nodes (T4b, any N, M0) or is any size and has spread to 7 or more nearby lymph nodes. But has not spread to another part of the body (Any T, N3, M0)
4	4A	• Cancer is advanced and has spread to other parts of the body, such as the liver or lungs (any T, N, M1)
	4B	•

Lesson 4: DISORDERS OF THE STOMACH

Learning Objectives

At the end of the lesson the learner will be able to: -

1) Describe the structure and function of the stomach
2) Discuss the pathology of disorders of the stomach
3) Investigate disorders of the stomach

1.0 NORMAL STRUCTURE AND FUNCTION

- Has four layers - inner two layers (mucosa and the sub-mucosa) which produces mucus, muscle layer that churns the contents of the stomach and outer membrane - the serosa, which holds the stomach together
- Food enters the stomach from the oesophagus through the connection between the stomach and the oesophagus called the cardiac sphincter
- Cardiac sphincter prevents food from passing back to the oesophagus and the other end of the stomach empties into duodenum
- The pyloric sphincter separates the stomach from the duodenum.
- Stomach has five anatomical parts that include the cardia, fundus, body (main part), pyloric antrum and the pylorus
- Posterior relations of the stomach - the diaphragm, spleen, left suprarenal gland, left kidney, anterior surface of the pancreas, left colic flexure, and the upper layer of the transverse mesocolon

Diagram 4.1: Structure of the Stomach

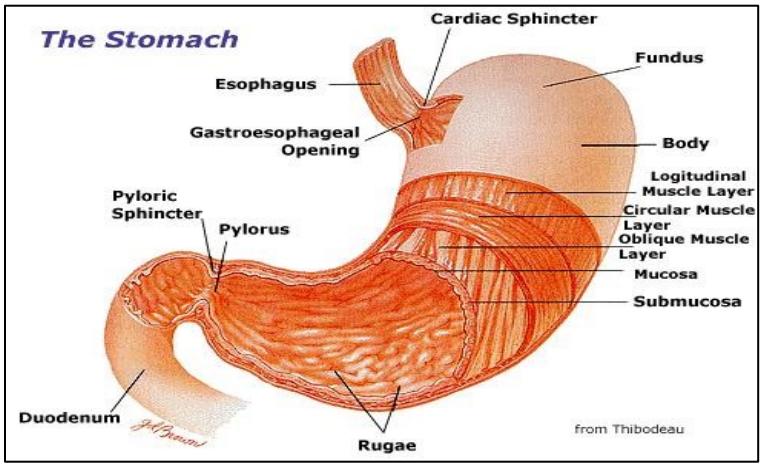
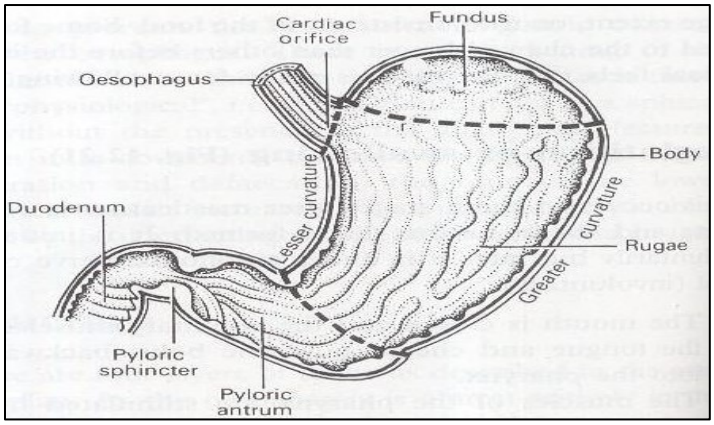


Diagram 4.2: Inner Structures of the stomach



Blood supply
- Coeliac artery and its branches (right and left gastric arteries and splenic artery)
- Spleenic artery gives off left gastroepiploic artery which anastomoses with the right gastroepiploic artery
- Venous - the portal vein

Nerve supply
- Sympathetic supply from coeliac plexus
- Parasympathetic supply from the vagus nerves

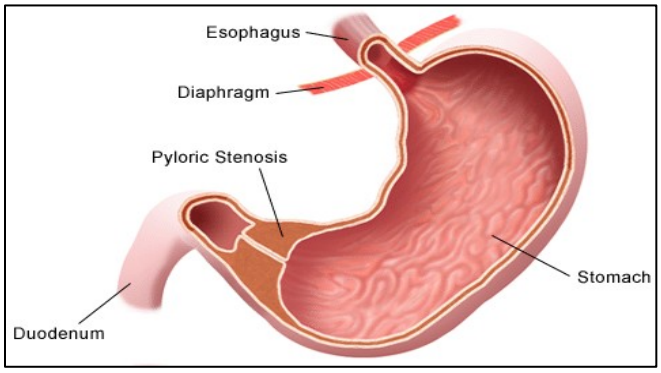
Functions
1) Temporary storage allowing time for the digestive enzymes, pepsins to act
2) Mechanical and chemical digestion
3) Absorption of water, alcohol and some lipid soluble drugs; Fe
4) Non-specific defence against microbes - by hydrochloric acid, vomiting
5) Production of intrinsic factor needed for absorption of vitamin B12
6) Controlled release of the stored contents into the duodenum

2.0 CONGENITAL ABNORMALITIES

2.1. PYLORIC STENOSIS

- Narrowing of the pylorus (opening from the stomach into the small intestine)
- Blockage is also referred to as a gastric outlet obstruction
- Normally, food passes easily from the stomach into the duodenum through a valve called the pylorus but in pyloric stenosis, the muscles of the pylorus are abnormally thickened preventing the stomach from emptying into the small intestine and food backs up into the oesophagus
- Cause of the thickening is unknown, although genetic factors may play a role
- Condition is usually diagnosed by the time a child is 6 months old.

Diagram 4.3: Pyloric Stenosis



Clinical Features

1) Vomiting is the first symptom in most children:
   - May occur after every feeding or only after some feedings
   - Usually starts around 3 weeks of age, but may start any time between 1 week and 5 months of age
   - Is forceful (projectile) and the vomit itself is usually clear or has the appearance of partially digested (curdled) milk.

2) The infant is hungry after vomiting and wants to feed again
3) Abdominal pain
4) Belching
5) Constant hunger
6) Dehydration (gets worse with the severity of the vomiting)
7) Failure to gain weight or weight loss
8) Wave-like motion of the abdomen shortly after feeding and just before vomiting occurs

Diagnosis

- Usually diagnosed before the baby is 6 months' old
- A physical exam may reveal signs of dehydration
- An ultrasound of the abdomen may be the first imaging test performed
- A barium X-ray to show the shape of the stomach and pylorus

Complications

1) Malnutrition
2) Dehydration
3) Failure to thrive
4) Anaemia

3.0 INFLAMMATORY DISORDERS

3.1. GASTRITIS

- Condition in which the stomach lining - known as the mucosa - is inflamed
- May be acute or chronic.
- Sudden, severe inflammation of the stomach lining is called acute gastritis.
- Inflammation that lasts for a long time is called chronic gastritis

Causes

Non-erosive

1) Helicobacter pylori (H. pylori) infection causes most cases of chronic non-erosive gastritis. H. pylori are bacteria that infect the stomach lining. H. pylori are primarily transmitted from person to person. In areas with poor sanitation, H. pylori may be transmitted through contaminated food or water.
2) Autoimmune disorders in which the immune system attacks healthy cells in the stomach lining
3) some digestive diseases and disorders, such as Crohn's disease and pernicious anaemia
4) Viruses, parasites, fungi, and bacteria other than H. pylori

Erosive

1) Prolonged use of non-steroidal anti-inflammatory drugs (NSAIDs) such as aspirin and ibuprofen
2) Alcohol, cocaine,
3) Radiation
4) Traumatic injuries, critical illness, severe burns, and major surgery can also cause acute erosive gastritis. This type of gastritis is called stress gastritis.

Features

- Upper abdominal discomfort or pain, nausea and vomiting (these symptoms are also called **dyspepsia)**

- Erosive gastritis may cause ulcers or erosions in the stomach lining that can bleed. Signs of bleeding in the stomach include blood in vomit, black, tarry stools and red blood in the stool

Complications

1. Peptic ulcer disease
2. Gastric polyps
3. Benign and malignant gastric tumours
4. Atrophic gastritis – can lead to two types of cancer: gastric cancer and gastric mucosa-associated lymphoid tissue (MALT) lymphoma.

Diagnosis

1. Endoscopy with a biopsy of the stomach.
2. Barium studies
3. Blood test - check for anaemia
4. Stool test - check for the presence of blood in the stool, another sign of bleeding in the stomach.
5. Tests for *H. pylori* infection

ACUTE GASTRITIS

- Is an acute mucosal inflammatory process usually of transient nature

Pathogenesis

- Deranged mucosal protection
- Frequently associated with heavy use of NSAIDS, excessive alcohol consumption, heavy smoking, uraemia, severe stress, ischaemia and shock, suicidal attempts, gastric irritation and mechanical trauma

Clinical Features

- Epigastric pain, nausea and vomiting, haemorrhage, massive hematemesis, malena

Pathology

Macroscopy	• Mucosa is oedematous, congested • Superficial erosion (site of blood loss) • Erosive – superficial epithelium is lost
Microscopy	• Capillary congestion • Leakage of RBCs into lamina propria • H. pylori – neutrophilia

CHRONIC GASTRITIS

- Presence of chronic mucosal inflammatory changes leading to mucosal atrophy and intestinal metaplasia usually in the absence of erosions

Causes

- Chronic infection by H. pylori
- Autoimmunity
- Toxic – alcohol and smoking
- Post-surgical
- Radiation

- Granulomatous conditions

Helicobacter associated

- Causes direct epithelial cell injury and excites vigorous immune responses leading to chronic inflammation (duodenal and gastric ulcer)

Autoimmune

- Associated with B12 deficiency, macrocytic anaemia (pernicious anaemia)
- Circulating antibodies against gastric parietal cells
- Involves mainly the body
- Causes extensive intestinal metaplasia

Chemical

- Chemical injury
- Causes hyperplasia and proliferation of gastric pits

Clinical Features

- Nausea and vomiting
- Upper abdominal discomfort

4.0 GASTRIC NEOPLASMS

BENIGN TUMOURS

- The polyp is applied to nay nodule or mass that projects above the level of the surrounding mucosa
- Mucosal polyps are classified as neoplastic and non-neoplastic
- Include non-neoplastic polyps, adenomas and papilloma

Adenomas

- Also, called neoplastic polys
- Found in the antrum but rare
- Commonly associated with chronic gastritis

Stromal tumours

- Include lipomas, schwannomas and leiomyoma
- Appear as circumscribed nodules

MALIGNANT NEOPLASMS - ADENOCARCINOMA

- Comprises more than 90% of all gastric malignancies and leading cause of cancer related deaths
- 2nd commonest tumour

Causes/ Risk Factors

1. Age - most common around the age of 60 and rare under the age of 40.
2. Gender - men are around twice as likely to develop stomach cancer as women.
3. Environmental factors

a. Diet - diet high in salt and foods that are smoked or cured may increase the risk of stomach cancer. Certain food preservative chemicals known as nitrosamines, which are found cured meats such as bacon and ham, may increase your chance of developing stomach cancer.
b. Helicobacter pylori infection

4. Family history - some people inherit an increased risk of developing stomach cancer.
5. Type A blood group - people with type A blood are at higher risk of stomach cancer.
6. Smoking
7. Atrophic gastritis
8. Pernicious anaemia
9. Geographical factors
10. Racial factors

Mechanisms

- Metaplasia
- Dysplasia

Classification

a) Site of origin - antral (25%), fundic (15%) and cardiac (60%)
b) Macroscopic
   i. Exophytic - Common in fundus and spreads through stomach wall to serosa causing ulceration and fungating mass
   ii. Ulcerative or infiltrative - common at antrum, presents as ulcers with rolled edges
   iii. Diffuse pattern - affects entire gastric wall, presents with minimal ulceration with prominent mucosal folds
c) Microscopic patterns
   i. Gland forming neoplasm
      - Resemble tumours of intestines
      - Common in older individuals
   ii. Diffuse adenocarcinomas
      - Non-cohesive
      - Infiltrate widely through the wall of the stomach
      - 50% of gastric carcinoma
      - Common in younger age group

Assessment

1. Patient presents with the same symptoms as gastric ulcer. Later, evaluation shows the lesion to be malignant.
2. Gastric fullness (early satiety), dyspepsia lasting more than 4 weeks, progressive loss of appetite are initial symptoms.
3. Stool samples are positive for occult blood.
4. Vomiting may occur and may have coffee-ground appearance.
5. Later manifestations include pain in black or epigastric area (often induced by eating, relieved by antacids or vomiting); weight loss; haemorrhage; gastric obstruction.

Diagnostic Evaluation

1. Upper GI X-ray with contrast media may initially show suspicious ulceration that requires further evaluation.

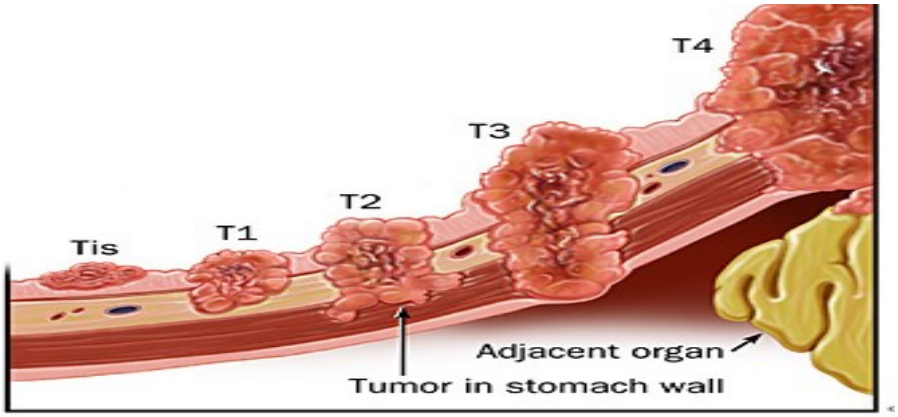
2. Endoscopy with biopsy and cytology confirms malignant disease.
3. Imaging studies (bone scan, liver scan, CT scan) helps determining metastasis.
4. Complete blood count (CBC) may indicate anaemia from blood loss.

CLINICAL FEATURES

- Persistent abdominal pain, gastric distension and vomiting, loss of weight (cachexia) and appetite (anorexia), anaemia, weakness and malaise

TNM STAGING

T1 - to grow into the wall of the stomach
T1a - within the mucosa
T1b - mucosa and into submucosa.
T2 – involve the muscular layer
T3 - outer lining of the stomach.
T4 - through the outer lining of the stomach
T4a - broken through outer lining of stomach wall
T4b - other organs/structures nearby - liver, oesophagus or abdominal wall



SPREAD

1) Direct – local extension to the oesophagus, mucosal and submucosal lymphatic spread to duodenum, draining LN and adjacent viscera e.g. liver
2) Distant
   - Lymphatic – to supraclavicular node - Virchow's node/Troisier's sign
   - Lungs (late)
   - Haematogenous – liver, lungs, brain, bones, kidneys, adrenals
   - Transcoelomic - Krukenberg tumour

Lesson 5: PEPTIC ULCER DISEASE

Objectives

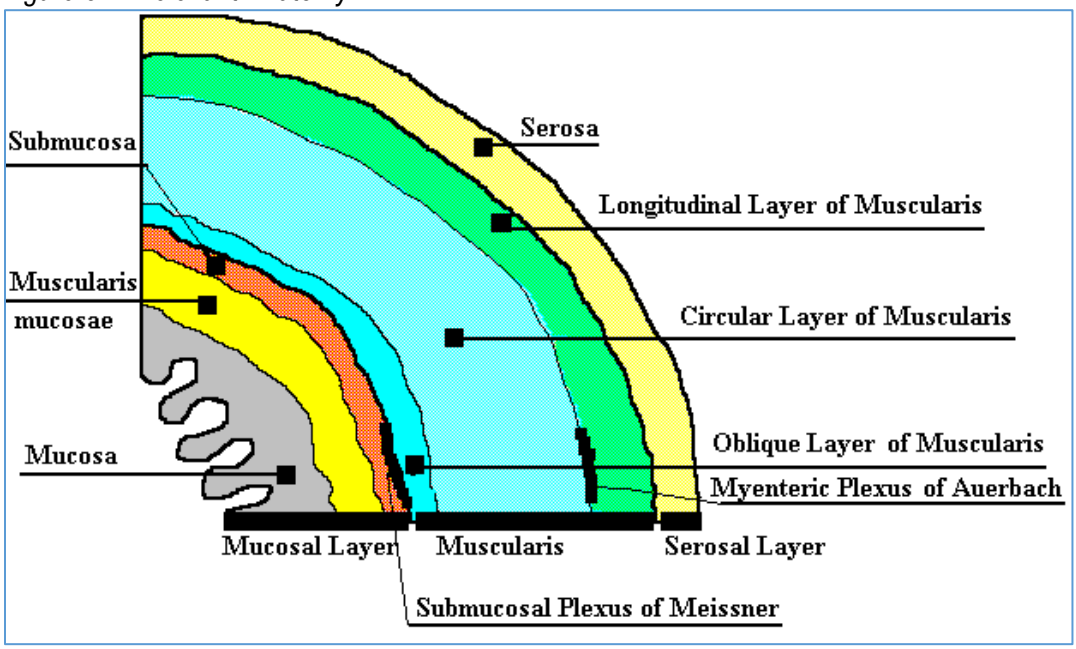
At the end of the lesson the leaner will be able to: -

1) Explain the pathogenesis, pathophysiology and pathology of PUD
2) Investigate PUD
3) Describe the complications of PUD

1.0 Introduction and Definition

- An ulcer is a disruption of mucosal integrity of the stomach and/or duodenum leading to a local defect or excavation due to active inflammation.
- Peptic ulcers are areas of degeneration and necrosis of gastrointestinal mucosa exposed to acid-peptic secretions
- Mucosal erosions or ulcerations occur when damaging effects of aggressive factors such as acid, pepsin, bile, NSAIDS and Helicobacter pylori overwhelm the G.I.T mucosal defence factors namely mucous and bicarbonate secretion, prostaglandins, blood flow and the process of restitution and regeneration after cellular injury.
- The gastric epithelium is under constant assault by several endogenous noxious factors such as HCl, pepsinogen/pepsin and bile acids as well as exogenous substances such as medications, (NSAIDS), alcohol and bacteria (*H. pylori*)
- A biological system which provides defence from mucosal injury and repairs any injury that may occur. Mucosal resistance (protection) is provided by mucous in gastric juice in a soluble phase, insoluble mucous gel which coats the mucosal surface of the stomach and mucosal prostaglandins.

Figure 5.1: Relevant Anatomy



2.0 Risk/Predisposing Factors

Discussion: What are the risk factors in acute PUD?

3.0 ACUTE PEPTIC (STRESS) ULCERS

- Are multiple, small mucosal erosions that involve mainly the stomach and occasionally the duodenum.

Aetiology

i) Psychological stress
ii) Physiological stress e.g. shock, severe trauma, septicaemia, intracranial lesions, drugs and local irritants e.g. alcohol, smoking

Pathogenesis

- Mainly due to ischaemic hypoxic injury to the mucosal cells
- Depletion of the gastric mucosa barrier
- In most cases, gastric acid secretion is normal

Pathology

Macroscopy	oval or circular multiple ulcers less than 1 cm in diameter common in stomach then duodenum
Microscopy	Shallow ulcers that do not invade the muscular layer; Inflammation at margins and base; Heal with complete re-epithelialization

4.0 CHRONIC PEPTIC ULCERS (GU AND DU)

Risk/Predisposing Factors/Aetiology

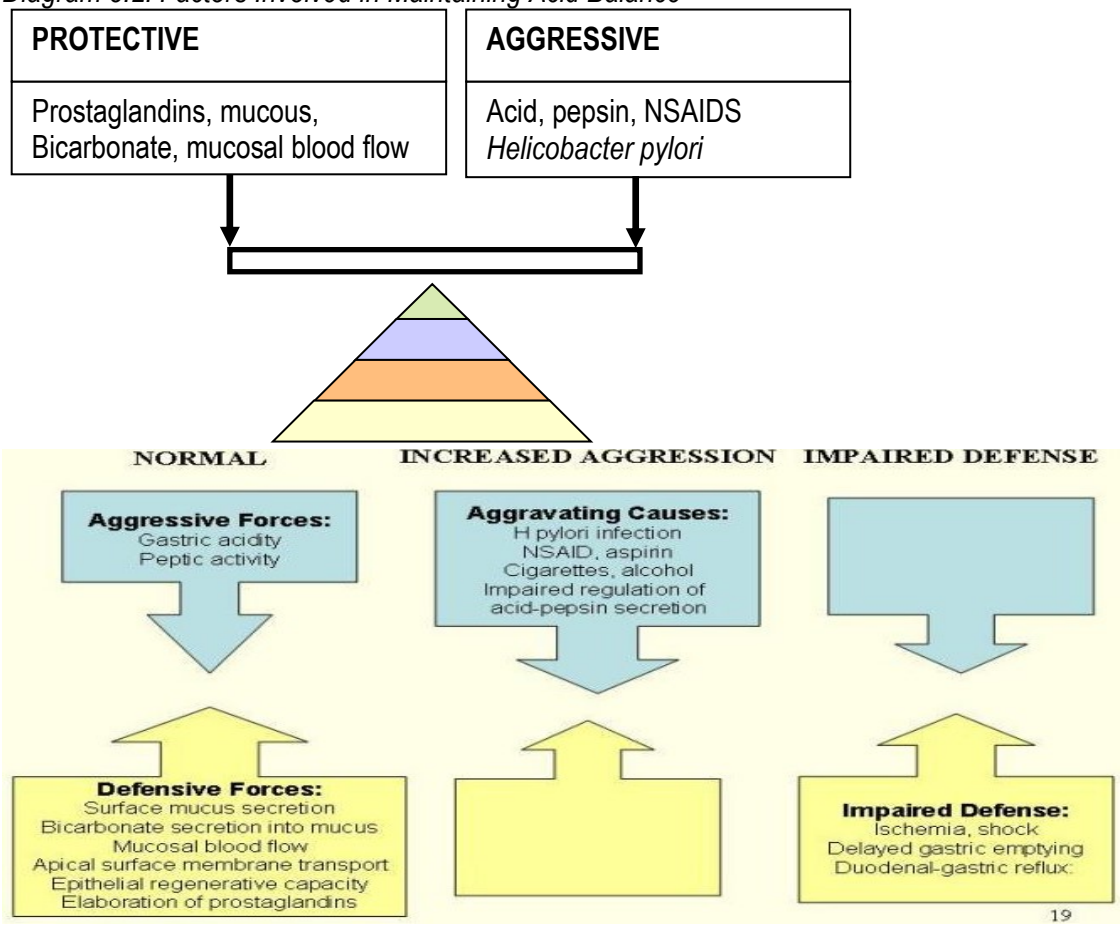
- Helicobacter pylori; Acid-pepsin secretions; Reduced mucosal secretion; Gastritis; Local irritants; Dietary factors; Psychological factors; Genetic factors; Hormonal factors; Miscellaneous (associated with other conditions such as alcoholic cirrhosis, chronic renal failure, hyperparathyroidism, chronic pancreatitis)

Discussion: Explain how these factors predispose to chronic PUD

Pathogenesis

- Aetiopathogenesis is multifactorial
- Peptic ulcers are produced by an imbalance between the gastro-duodenal mucosal defense mechanisms and damaging forces of gastric acid and pepsin, combined with superimposed injury from environmental or immunologic agents
- Immediate cause of PUD is disturbance in normal protective mucosal barrier by acid-pepsin resulting in digestion of the mucosa
- Defense mechanisms normally limit the injury
- Aggressive factors include acid secretion/gastric juice (including hydrochloric acid, pepsin, and bile salts refluxed from the duodenum), H pylori, and NSAIDs. *H. pylori*, Pepsinogen Secretion, Cigarette smoking; Corticosteroid use
- Defensive factors include mucus production, bicarbonate production, mucosal blood flow (more important in the development of stress ulcer) high epithelial cell turnover prostaglandins (PGE2) - stimulate mucus and bicarbonate production, and blood flow
- Key factors in peptic ulcer formation are exposure of the mucosa to gastric acid and pepsin secretions and strong association with H. pylori infection.

Diagram 5.2: Factors Involved in Maintaining Acid Balance



Duodenal Ulcer

- Hyper-secretion of gastric acid into the fasting stomach at night under the influence of vagal stimulation
- Rapid emptying of the stomach (food buffers and neutralizes gastric acid)
- Helicobacter gastritis caused by H. pylori
- Usually associated with gastritis confined to the antrum

Gastric Ulcer

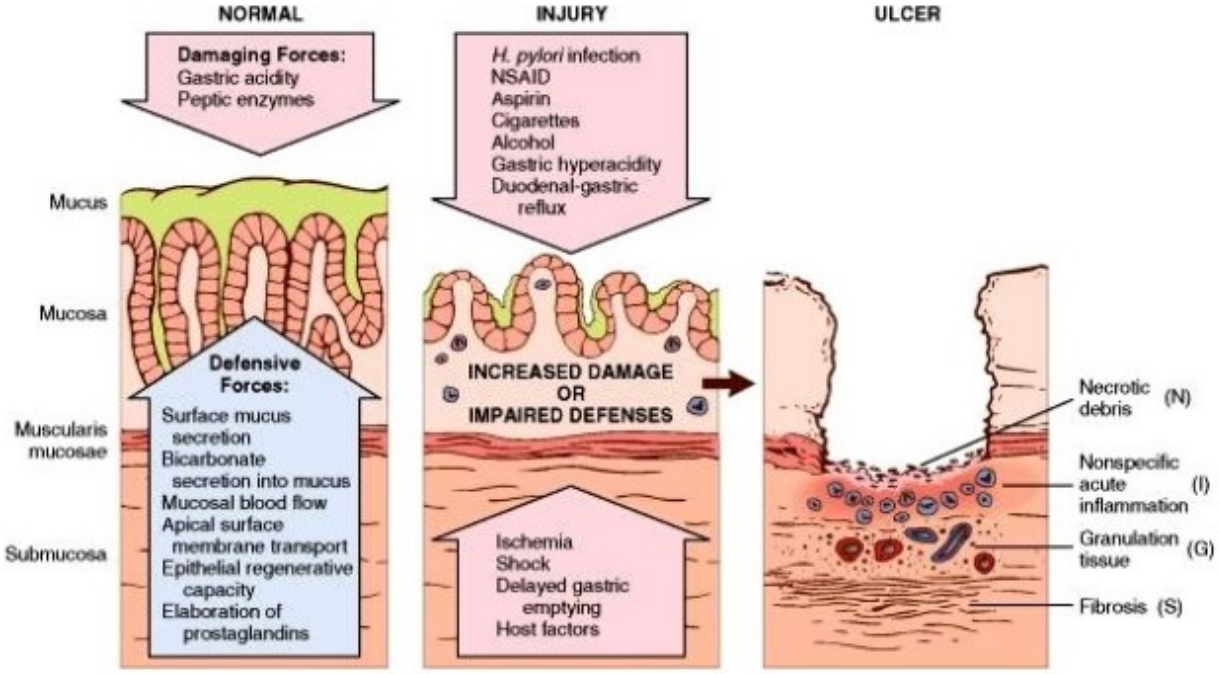
- Impaired gastric mucosal defences against acid-pepsin
- Increased serum gastrin levels
- Usually associated with pangastritis

Pathophysiology

Cause	Mechanism
H. pylori	H. pylori secretes urease (generates ammonia), protease (breaks down glycoprotein in the gastric mucus) or phospholipases. Bacterial lipopolysaccharide attracts inflammatory cells to the mucosa Neutrophils release myeloperoxide. A bacterial platelet-activating factor promotes thrombotic occlusion of surface capillaries. Mucosal damage allows leakage of tissue nutrients in the surface microenvironment, sustaining the bacillus. Damage of the protective mucosal layer The epithelial cells are exposed to the damaging effect of acid-peptic digestion.

Cause	Mechanism
Inflammation	Chronically inflamed mucosa more susceptible to acid- peptic injury and prone to peptic ulceration. Ulcers occur at sites of chronic inflammation e.g. antrum, junction of antral and body- fundic mucosa (division between the inflamed antral mucosa and normal acid secreting mucosa). Pangastritis - When there is extensive gastritis, the ulcers are more proximally situated. In elderly patients, gastric ulcers are more proximally situated as there is proximal migration of the antral-body mucosal junction
Impaired Defence	The gastric acid and pepsin levels are normal and no H. pylori are present. Chronic use of NSAIDs (aspirin) causes suppression of mucosal prostaglandin and direct irritative topical effect Repeated use of corticosteroid in high dose. Cigarette smoking impair healing and favour recurrences. Alcoholic cirrhosis; Personality, psychological stress, ischemia.

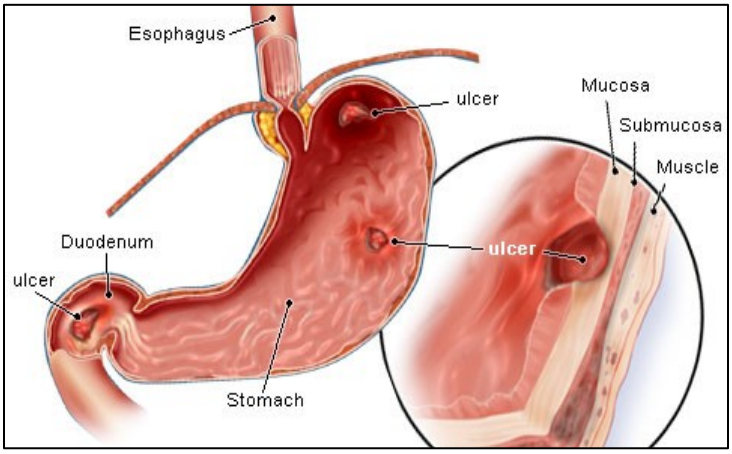
Figure 5.3: Pathophysiology of PUD



5.0 Pathology

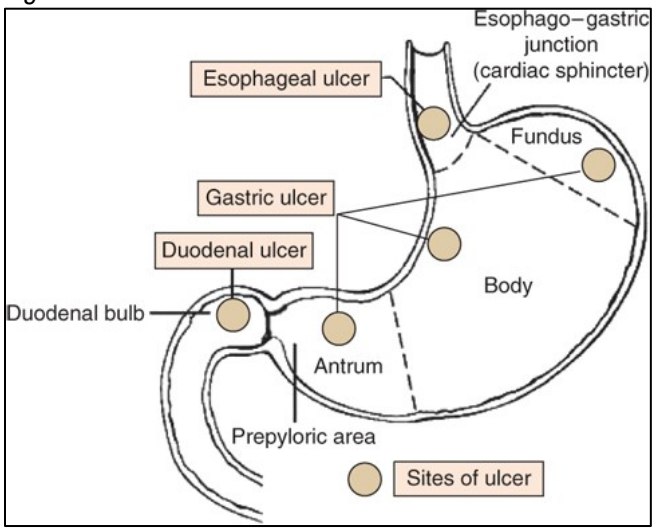
Macroscopy	• Commonly solitary round and oval (punched out) ulcer of 1 – 2.5 cm in diameter • Clean floor, thick firm base; • Vary in depth from superficial (confined to mucosa) to deep ulcers (penetrating into the muscular layer) • Scarring
Microscopy	• 4 histological layers – necrotic zone, superficial exudative zone, granulation tissue zone and zone of cicatrisation

Diagram 5.4: PUD Pathology



6.0 Sites

Figure 5.5: Sites



7.0 Presentation

Discussion

- What are the clinical features of peptic ulcer (GU and DU)?
- What are the similarities and differences between gastric and

8.0 Differential Diagnosis

1. Mesenteric ischemia
2. Angina pectoris
3. Biliary colic
4. Pancreatitis
5. Nonulcer dyspepsia (NUD) also known as *functional dyspepsia* or *essential dyspepsia*
6. Gallstone disease and its complications,
7. Gastroesophageal reflux disease,
8. Chronic pancreatitis,
9. Cancers of the stomach and pancreas,
10. Postgastrectomy gastritis
11. Diseases of the transverse colon

9.0 Investigations

1) Endoscopy -upper endoscopy, EGD (esophagogastroduodenoscopy), colonoscopy, sigmoidoscopy, anoscopy
2) Barium x-rays (barium meal, swallow, enema, follow through)
3) Biopsies
4) Blood counts
5) Blood cultures
6) Stool examination

8.0 Complications

1) Healing and scarring – pyloric stenosis
2) Obstruction
3) Haemorrhage
4) Perforation
5) Penetration
6) Malignant transformation

DIFFERENCES

	Feature	Duodenal ulcer	Gastric ulcer
1.	Incidence	• More common, 25 – 50 years	• Less common, beyond 6th decade
2.	Aetiology	• H. pylori, hypersecretion of acid-pepsin	• H. pylori
3.	Pathogenesis	• Mucosal digestion; Damage of protective mucous barrier	• Damage to mucous barrier
4.	Pathology	• First part of duodenum; Solitary • Composed of 4 layers	• as duodenal
5.	Complications	• Haemorrhage; Perforation; Obstruction	• Perforation; Haemorrhage • Malignancy
6.	Clinical Features	• Pain-food-relief pattern • Night pain • No vomiting • Melena > hematemesis • No loss of weight • Deep tenderness in right hypochondrium • Marked seasonal variation • In people at greater stress	• Pain-food pattern • No night pain • Vomiting • hematemesis > Melena • Significant loss of weight • Deep tenderness in midline in epigastrium • No seasonal variation • In labouring groups

Lesson 6: GIT BLEEDING

Objectives

At the end of the lesson the learner will be able to: -

1) Outline causes of GI bleeding
2) Explain the clinical features of GI bleeding
3) Investigate GI bleeding
4) Describe complications of GI bleeding

1.0 INTRODUCTION

- Massive lower GI bleeding is defined as passage of a large volume of red or maroon blood through the rectum. Other features include hemodynamic instability and shock, initial decrease in haematocrit (Hct) level of 6 g/dL or less, transfusion of at least 2 units of packed red blood cells (RBCs); Bleeding that continues for 3 days or significant re-bleeding in 1 week
- Causes of GI bleeding are classified into upper or lower GIT bleeding

2.0 UPPER GIT BLEEDING

1.12 Causes

- Originates in the first part of the GI tract-the oesophagus, stomach, or duodenum

	Part/organ	Examples
1.	Oesophagus	• Inflammation - oesophagitis; oesophageal varices; tears - Mallory-Weiss syndrome; ulcers; cancer and trauma
2.	In the Stomach	• Peptic ulcers (ulcers may enlarge and erode through a blood vessel, causing bleeding; gastritis; cancer
3.	In the Small Intestine	• Duodenal ulcer; infections; inflammatory; cancer; gall-stones; Cushing's ulcers; Curling's ulcers; diverticulum; duodenitis
4.	Systemic	• Bleeding disorders – Haemophilia, Von Willebrand's disease, scurvy and polycythaemia; Liver disease; anticoagulants; uraemia; DIC; pancreatitis
5.	Miscellaneous	• Aneurysm of the splenic/gastric artery/aorta; Amyloidosis; Polyarteritis nodosa; hereditary haemorrhagic telangiectasia; Neurofibromatosis; Kaposi's sarcoma; Acute infections e.g. (yellow fever, scarlet fever, acute yellow atrophy, malaria, septicaemia)

1.13 Clinical Features

1) Bright red blood, dark clots, or coffee ground-like material in vomit
2) Black, tar like stool
3) Symptoms of Passing only bright red blood, or passing blood mixed in stool
4) Bright red or maroon coloured blood in the stool
5) Acute bleeding - Weakness, shortness of breath, dizziness, rapid pulse, reduced urine flow, crampy abdominal pain; cold, clammy hands and feet; faintness, diarrhoea, confusion, disorientation, sleepiness, bright red blood coating the stool, dark blood mixed with the stool, black or tarry stool, bright red blood in vomit; coffee-grounds appearance of vomit
6) Chronic bleeding - Weakness, fatigue, shortness of breath, pallor, chest pain, dizziness, lethargy, faintness, bright red blood coating the stool, dark blood mixed with the stool, black or tarry stool, bright red blood in vomit, coffee-grounds appearance of vomit

1.14 Differential Diagnosis

i) Peptic Ulcer Disease (PUD) >50% cases
ii) Gastritis/Duodenitis (15-30%), Subset due to NSAID use
iii) Varices from portal hypertension (10-20%)
iv) Mallory-Weiss tears at GE junction (5%)
v) Esophagitis (3-5%)
vi) Malignancy (3%)
vii) Nasopharyngeal bleed – swallowed blood
viii) Other- Aortoenteric fistula, angiodysplasia, Crohn's, hemophilia, hemosuccus pancreaticus

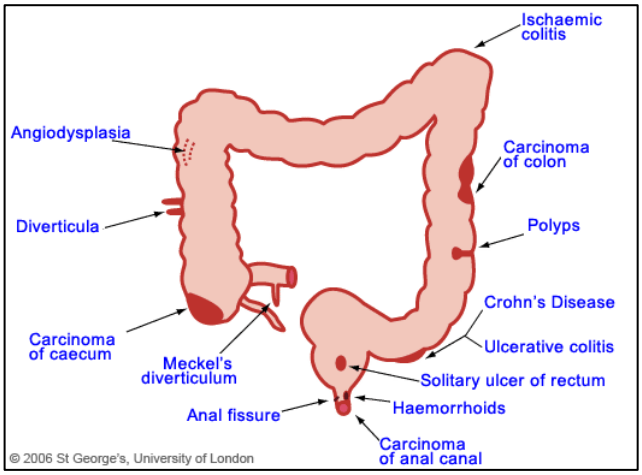
3.0 LOWER GIT BLEEDING

- Originates in the portions of the GI tract further down the digestive system (parts of the small intestine beyond the duodenum, large intestine, rectum, and anus).

1.15 Causes

1) In the Small intestines - tumours (adenocarcinoma, lymphoma); vascular ectasis; NSAIDS; Meckel's diverticulum; intussusception; Chron's disease
2) In the Large Intestines - diverticulosis; inflammatory bowel disease - ulcerative colitis, Chron's disease; infections; polyps; Cancer
3) Rectum and anus – haemorrhoids, fissures; ulcerative colitis; polyps; cancer

Diagram 6.1: Causes of Lower GIT Bleeding

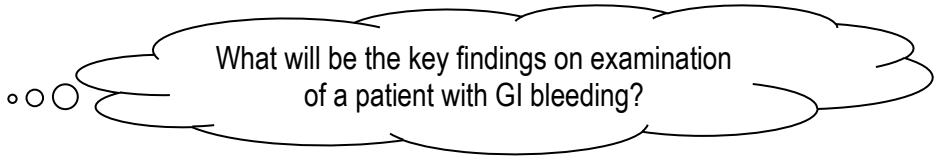


1.16 Clinical Features

History

- Bowel habits (going more or less often than usual)
- Stool colour (black or red) and consistency (looser or more firm)
- Pain or tenderness, and where it's located

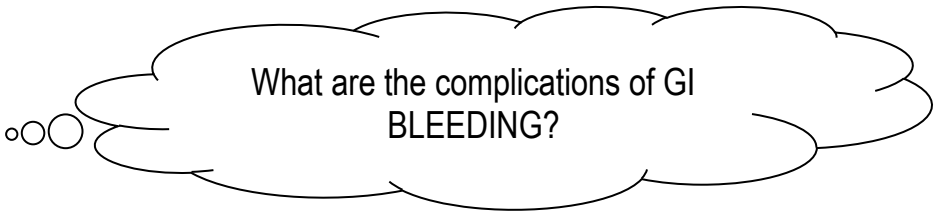
Physical Examination



4.0 Investigations

1) Endoscopy -upper endoscopy, EGD (esophagogastroduodenoscopy), colonoscopy, sigmoidoscopy, anoscopy
2) Barium x-rays (barium meal, swallow, enema, follow through)
3) Biopsies
4) Blood counts
5) Blood cultures
6) Stool examination

5.0 Complications



Lesson 7: DISORDERS OF THE SMALL INTESTINES

Learning Objectives

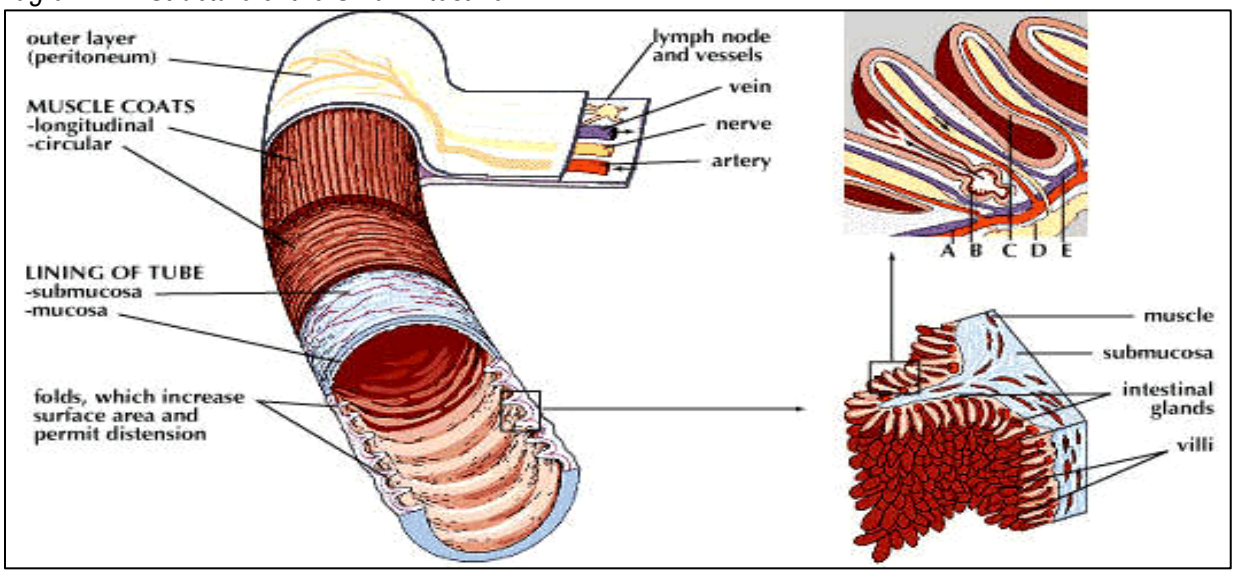
At the end of the lesson the learner will be able to: -

1) Describe the structure and function of the small intestines
2) Discuss the pathology of disorders of the small intestines
3) Investigate disorders of the small intestines

1.0 NORMAL STRUCTURE AND FUNCTION

- Small intestine is 3-7 metres long and approximately 2.5-3 cm in diameter
- Intestinal villi are tiny finger-like outgrowths, in the lining of the small intestine
- Villi increase the surface area of the gut wall allowing for slower movement through the small intestine and greater time for absorption of nutrients
- Each villus has a lacteal and capillary bit that picks up digested nutrients that are then transported by the blood to all the cells of the body
- The wall of the small intestine consists of 4 layers – serosa, muscularis propria, sub mucosa and mucosa.

Diagram 7.1: Structure of the Small Intestine



2.0 CONGENITAL DISORDERS

2.1. INTESTINAL ATRESIA

- Congenital absence (complete occlusion) of the lumen commonly mainly the ileum or duodenum in which the proximal segment has a blind end separated completely from the distal segment or joined by a fibrous cord
- Presentation depends on the level of the obstruction
- Duodenal or jejunal atresia may cause polyhydramnios due to inability of the foetus to absorb swallowed amniotic fluid

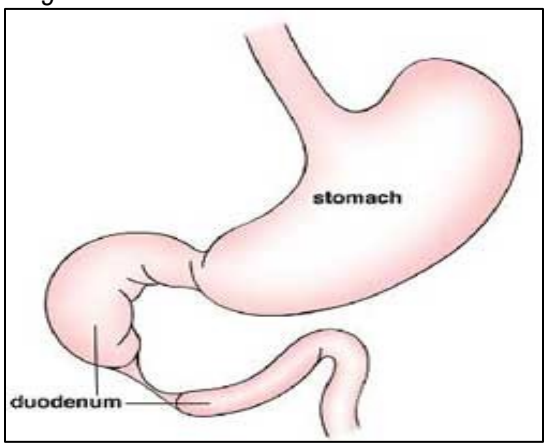
Features

- Vomiting and abdominal distension within 24 hours of birth, or these symptoms may be delayed somewhat if the obstruction is more distal.

Duodenal atresia

- Half of the infants with this condition are born prematurely and approximately two-thirds have associated abnormalities of the heart, genitourinary, or intestinal tract
- Nearly 40% have Down syndrome
- Infants usually vomit within hours after birth, and may develop a distended abdomen
- Abdominal X-rays show a large dilated stomach and duodenum without gas in the remaining intestinal tract.

Diagram 7.2: Duodenal Atresia



Jejunoileal atresia

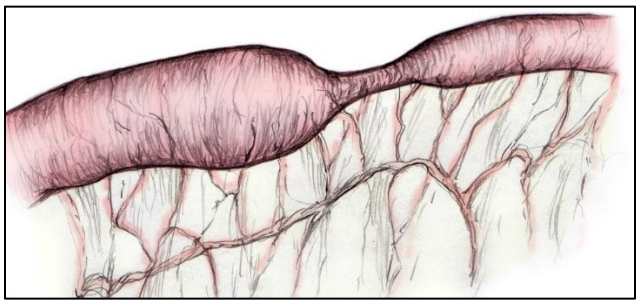
- Involves an obstruction of the jejunum or ileum)
- Segment of intestine just before the obstruction becomes massively enlarged (dilated), thus hindering its ability to absorb nutrients and propel its contents through the digestive tract
- A number of infants also have abnormalities of intestinal rotation and fixation
- Cystic fibrosis is also an associated disorder (screen for cystic fibrosis)

Features

- Vomit green bile within the first 24 hours of life
- Distended abdomen
- Reduced bowel movement

2.2 INTESTINAL STENOSIS

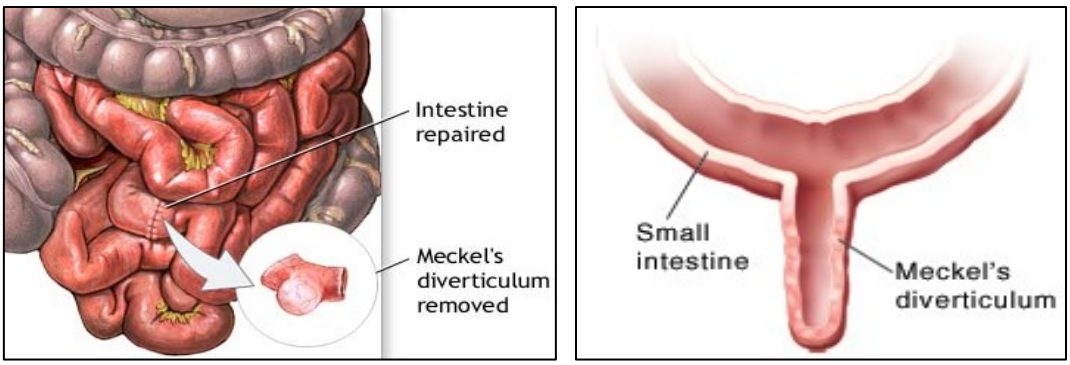
- Congenital narrowing of the lumen affecting a segment of the small intestines
- The intestine above the level of obstruction is dilated while the segment below is collapsed



2.2. MECKEL'S DIVERTICULUM

- A Meckel's diverticulum is a small pouch of tissue on the intestine (bowel)
- One of the most common congenital abnormalities that occurs when the connection between the intestine and the umbilical cord doesn't completely close off during foetal development resulting in a small out pouching of the small intestine

Diagram 7.3: Meckel's Diverticulum



Features

- Many people with a Meckel's diverticulum never have symptoms
- Most common signs of a problem include:
  i) Painless bleeding from the rectum
  ii) Blood in stool (fresh or black/tarry)
  iii) Anaemia (a health problem due to blood loss)
  iv) Signs of infection (fever, chills, or pain or tenderness in the abdomen)

Diagnosis

i) Blood tests - check for signs of bleeding or infection
ii) Stool sample - check for blood
iii) Meckel's scan - a special dye is injected into the child's bloodstream through an IV (intravenous) line. This dye may make the Meckel's tissue show up on a scan.
iv) Ultrasound
v) Other tests - Imaging tests such as an x-ray or CT scan

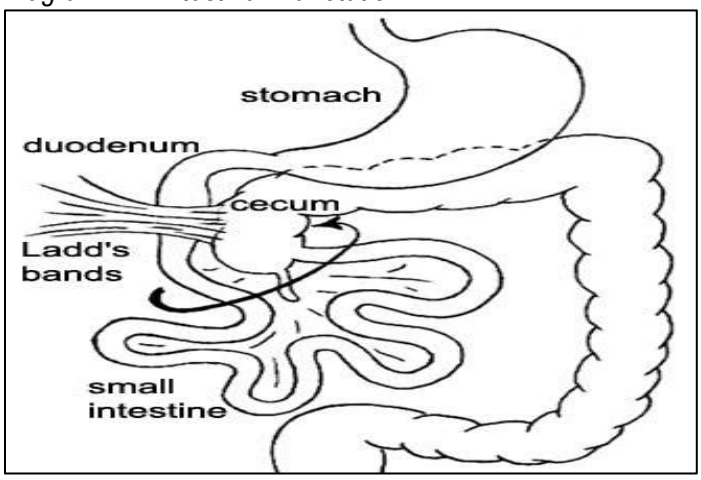
Complications

i) Perforation
ii) Haemorrhage
iii) Diverticulitis (DDx – appendicitis)

2.3. INTESTINAL MALROTATION

- Developmental abnormality of the mid-gut which causes in failure of normal rotation of the mid-gut resulting in exomphalos, misplacement of the caecum, appendix and ascending colon and mobile caecum

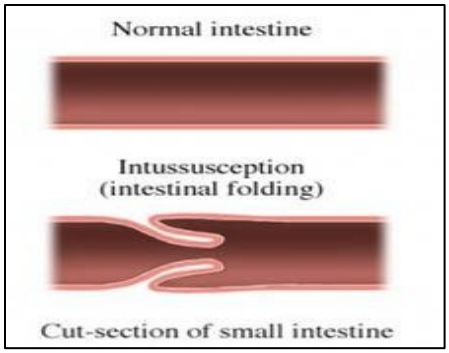
Diagram 7.4: Intestinal Malrotation

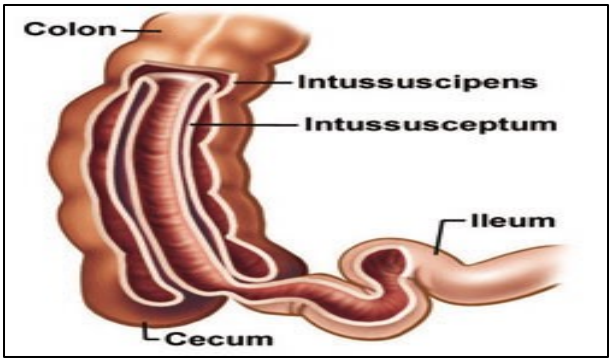


3.0 INTUSSUSCEPTION

- An invagination of a proximal part of the bowel into a distal part of the bowel (one segment of the intestine enfolds within another) or the telescoping of a segment of intestine into the segment below due to peristalsis
- Most common is the ileum passing into the cecum and colon through the ileocecal valve
- Telescoped segment is called the *intussusceptum* and the lower receiving segment is called *intussuscipeins*
- 95% of all intussusceptions occur in children
- Most common cause of intestinal obstruction in infants past the neonatal period

Diagram 7.5: Intussusception





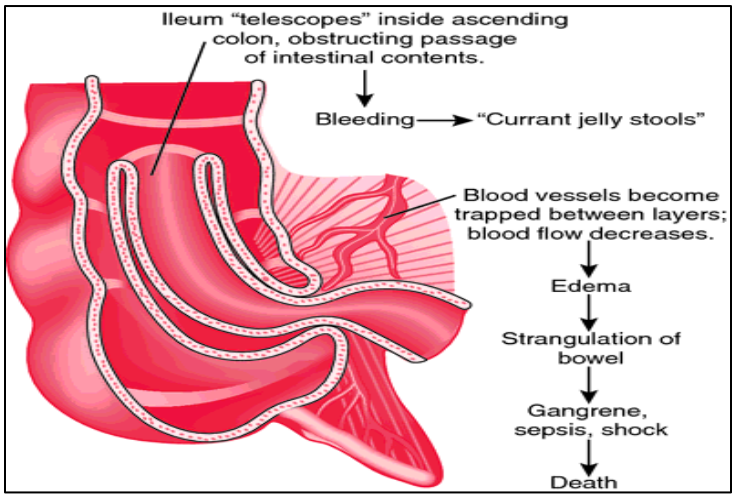
Causes

- Idiopathic
- Complication of medical conditions such as viral infections (especially adenovirus), Meckel's diverticulum, intestinal polyps, tumours, such as lymphosarcoma and neurofibroma, lymphoma, recent abdominal surgery, inflammatory bowel disease and haemophilia

Features

- Abdominal pain (sudden, severe, colicky or cramping), vomiting (sometimes yellow or green tinged), stools mixed with mucus and blood (often described as currant jelly), lethargy, poor feeding, diarrhoea, shock, dehydration, fever

Diagram 7.6: Effects of Intussusception



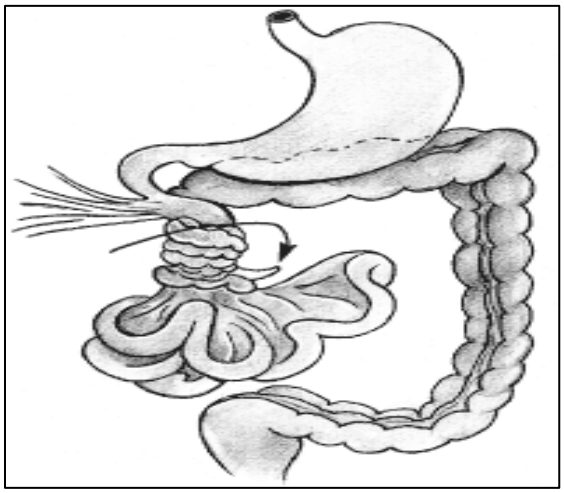
Complications

1) Bowel gangrene
2) Perforation of the intestinal wall
3) Peritonitis
4) Infection
5) Intestinal obstruction

4.0 VOLVULUS

- Condition in which the bowel twists on itself through 180^{o} causing obstruction to the flow of material through the bowel
- Can also lead to obstruction of the blood supply to the intestine itself causing ischaemia and necrosis (gangrene)
- Most commonly due to a birth defect called malrotation (bowel becomes misaligned during foetal development)
- Bowels do not have a normal attachment to the abdominal wall, which makes it possible for the bowels to shift out of their normal position or to rotate.
- Volvulus can also occur in the absence of underlying malrotation

Diagram 7.7: Volvulus



Clinical Features

- Abdominal tenderness, nausea or vomiting (green bile-looking material), bloody or dark red stool, constipation or difficulty expelling stools, distended abdomen, shock

Investigations
- Stool sample test finds blood in the stool
- Upper GI X-ray with small bowel follow-through shows a malrotated bowel or midgut volvulus
- CT scan may show evidence of intestinal obstruction
- Barium enema often shows an abnormal position of the bowel, suggesting malrotation
- Blood tests to check the electrolytes may show abnormalities

5.0 MALABSORPTION

- Malabsorption syndrome (MAS) is characterized by defective or impaired intestinal absorption of nutrients

Aetiopathology
1) Primary MAS – due to primary deficiency of the absorptive mucosal surface and associated enzymes e.g. Coeliac sprue, tropical sprue, Whipple's disease
2) Secondary MAS – mucosal changes result for other factors such as disease, surgery, trauma and drugs
   a) Impaired digestion
   b) Impaired absorption
   c) Impaired transport

Mechanisms of malabsorption

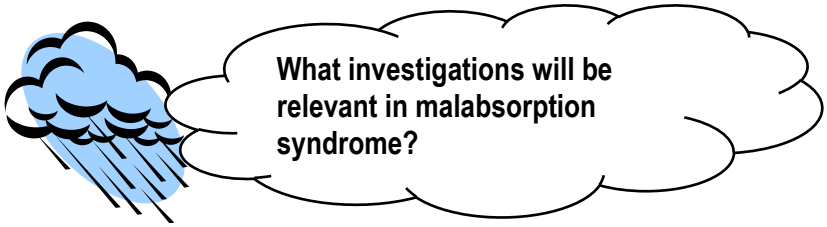
i) Inadequate digestion
ii) Intestinal damage
iii) Altered intestinal flora
iv) Biochemical abnormalities
v) Lymphatic obstruction
vi) Inadequate absorptive surface
vii) Endocrine disturbances
viii) Circulatory disturbances

Using examples explain how these mechanisms bring about malabsorption

Clinical Features
- Passage of abnormally bulky, frothy, greasy, yellow or grey stools (steatorrhoea), chronic diarrhoea, weight loss, anorexia, abdominal distension, borborygmic and flatulence, dehydration, hypotension, muscle wasting
- Specific malnutrition and vitamin deficiencies

Complications
1) Alimentary tract - diarrhoea; flatus; weight loss; mucositis
2) Haemopoietic system - anaemia (iron, pyridoxine, folate, B_{12} deficiency, vitamin K deficiency); pellagra
3) Musculoskeletal - osteopenia; Tetany – low calcium and magnesium; Vitamin D deficiency
4) Endocrine - amenorrhea, impotence and infertility due to generalized malnutrition; Hyperparathyroidism – from calcium and vitamin D deficiency

5) Cardiovascular system
6) Immunodeficiency
7) Skin - purpura and petechie – vitamin K deficiency; Oedema – protein deficiency; Dermatitis and keratosis – Vit A, zinc
8) Nervous system - peripheral neuropathy – Vit A, B6 and B12 deficiency; dementia



6.0 INFECTIVE DISEASES

6.1. Bacterial

Explain the pathology of the following bacteria in terms of causative agent, pathogenesis, pathophysiology, pathology, clinical features, diagnosis, investigations and complications: - Cholera, TB, salmonellosis, shigellosis, clostridial infections, E. coli, Campylobacter, and Staphylococcal

6.2. Viral

Explain the pathology of the following viruses in terms of causative agent, pathogenesis, pathophysiology, pathology, clinical features, diagnosis, investigations and complications: - HIV/AIDS, Rota virus, Enteroviruses, Adenoviruses,

6.3. Parasitic

Explain the pathology of the following parasites in terms of causative agent, life cycle pathogenesis, pathophysiology, pathology, clinical features, diagnosis, investigations and complications: - amoebiasis, schistosomiasis, nematodes (ascariasis, strongyloides, hookworm, trichuris), cestodes (diphyllobothrium, taenia solium and saginata), giardiasis

6.4. Fungal

Explain the pathology of the following fungi in terms of causative agent, pathogenesis, pathophysiology, pathology, clinical features, diagnosis, investigations and complications: - candida

7.0 Neoplasms

Briefly discuss the pathology of neoplasms of the small intestines.

Lesson 8: HERNIAS AND INTESTINAL OBSTRUCTION

Learning Objectives

At the end of the lesson the learner should be to

1) Explain the causes and risk factors for hernias and intestinal obstruction
2) Discuss the pathology of hernias and intestinal obstruction

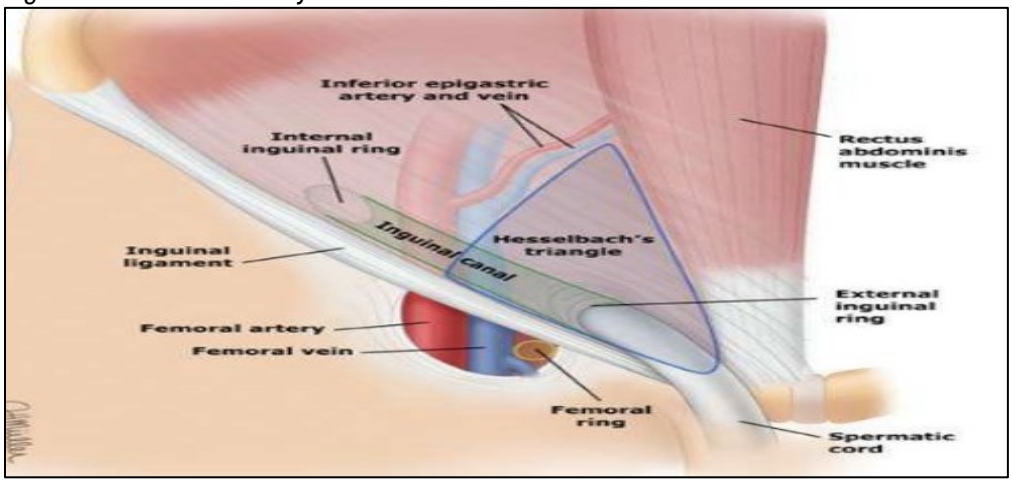
HERNIAS

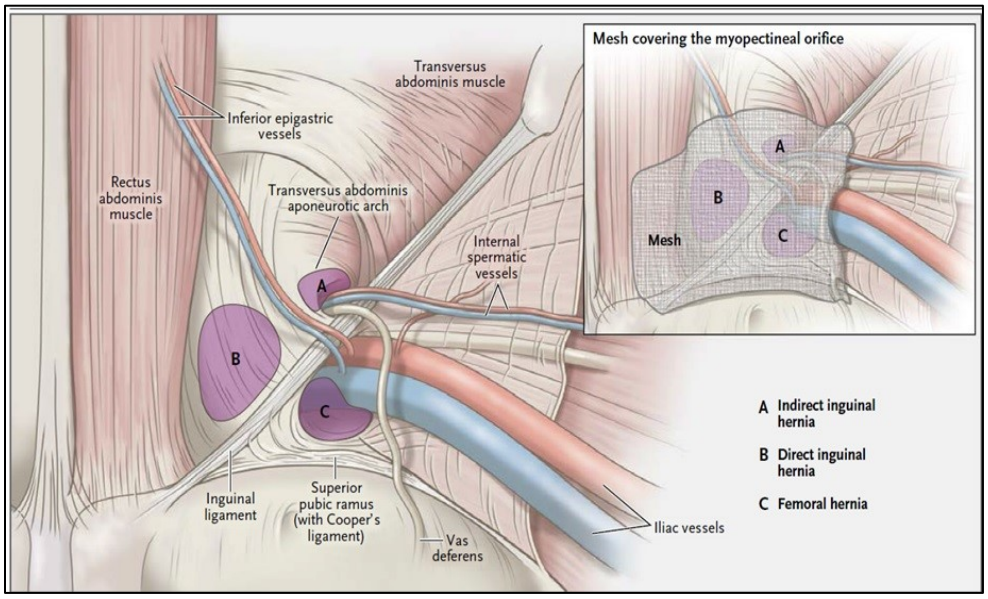
1.0 INTRODUCTION

- A protrusion of a portion of a viscera through an abnormal opening in the wall of the containing cavity.
- A hernia is an abnormal protrusion from one anatomical space to another.
- A weakness or defect in the wall of the peritoneal cavity may permit protrusion of a pouch-like, serosa-lined sac of peritoneum called a hernia sac
- Protrusion of a structure from its normal position to another through an opening that is either congenital or acquired
- Abnormal protrusion of intra-abdominal tissue (a viscus or part of a viscus) through a fascial defect (abnormal opening) in the abdominal wall.
- Most often, a hernial mass consists of covering tissues (skin, subcutaneous tissues, etc.), a peritoneal sac, and any contained viscera
  - i) External hernia: Protrudes to the outside
  - ii) Internal hernia: Protrudes within the body
  - iii) Incisional hernia: Protrudes through a previous incision
  - iv) Reducible hernia: It is one where the contents of the sac may spontaneously or with pressure return to the abdomen.
  - v) Irreducible (incarcerated) hernia: It is one whose contents cannot be returned to the abdomen.
  - vi) A strangulated hernia: It is one where there is compromise to the blood supply of the contents of the sac leading to ischemia and gangrene.
  - vii) Hiatus hernia: Protrusion of the stomach above the diaphragm.

2.0 ANATOMY

Figure 8.1: Groin Anatomy





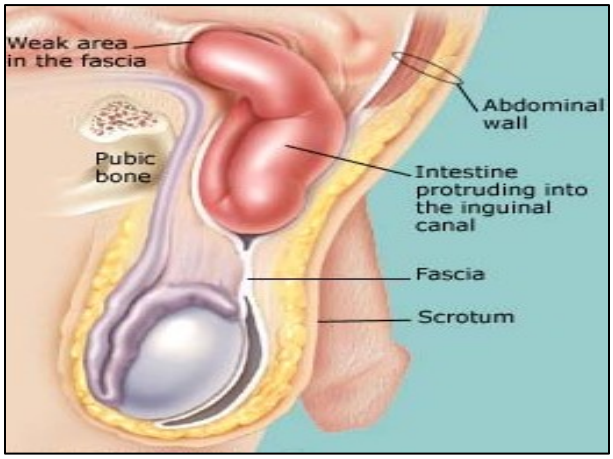
3.0 PREDISPOSITION

- Children - poor nutrition and whooping cough
- Adults - chronic cough; straining – micturition/defecation; intra-abdominal malignancy; obesity – weakness, aponeurosis (hiatus, paraumbilical, direct inguinal hernia); smoking; peritoneal dialysis; overexertion; anything that causes an increase in pressure in the abdomen can then cause a hernia, including obesity, lifting heavy objects, diarrhoea or constipation, or persistent coughing or sneezing.

4.0 PATHOGENESIS

- Caused by a combination of **pressure** and **an opening or weakness** of muscle or fascia; the pressure pushes an organ or tissue through the opening or weak spot
- Pressure results from coughing, straining, obesity and intra-abdominal malignancy

Diagram 8.2: Hernia



5.0 COMPOSITION OF A HERNIA

1) Sac - diverticulum of peritoneum that consists of mouth, neck, body and fundus

2) Coverings - derived from layers of the abdominal wall
3) Contents - omentum (omentocele); intestine (enterocele); portion of the circumference of the intestine (Richter's hernia); portion of the bladder; ovary with or without the Fallopian tubes; a Meckel's diverticulum (Littre's hernia); fluid

5.1. Classification

1) Reducible - contents can be returned to the abdomen
2) Irreducible - contents cannot be returned
3) Obstructed - bowel in the hernia has good blood supply but it is obstructed
4) Strangulated - blood supply of bowel obstructed
5) Inflamed - contents of sac have become inflamed

6.0 CLINICAL FEATURES

Symptoms

i) Swelling or bulge beneath abdominal skin (*may disappear when lying down*)
ii) Pain or discomfort in the area of the bulge or the entire abdomen
- A burning, tearing, sharp, dull or pulling pain
- Due to stretching and tearing of tissues around the hernia with nerve endings
- Can cause localized pain or generalized pain when strangulation or incarceration occurs.

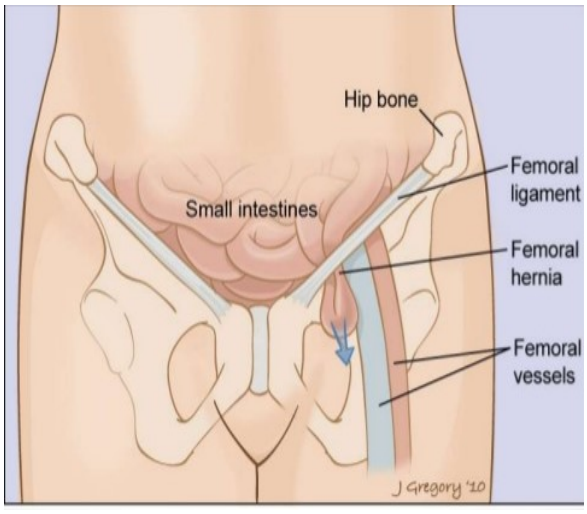
iii) Constipation or bloody stool
iv) Nauseas ness and/or vomiting
v) Urinary Difficulties

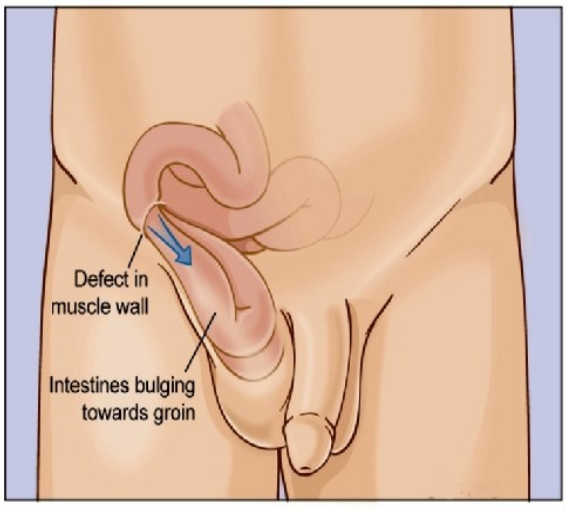
6 TYPES OF HERNIAS

1) Femoral Hernia

- Part of intestine, or other abdominal contents, is forced through a weakness in the femoral canal located near the groin and below the inguinal ligament (crease between the lower abdomen and thigh)
- Common in males but are more likely to occur in females
- Predisposition - straining or coughing, overweight, constipation, pushing or carrying heavy things and smoker's cough

Diagram 8.3: femoral Hernia





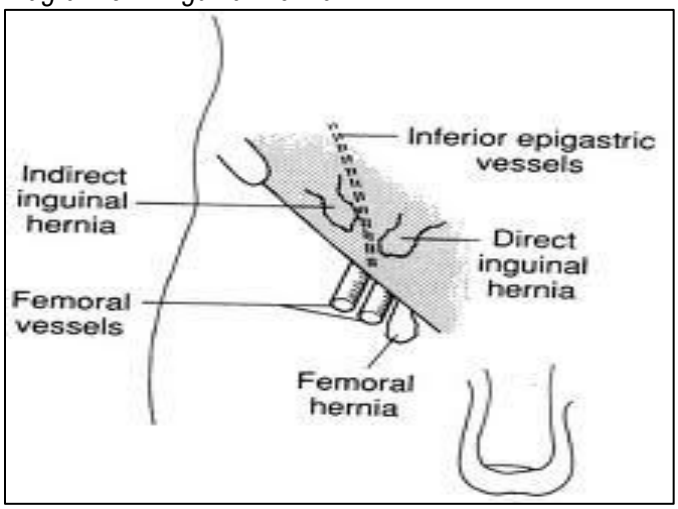
Differential Diagnosis of Groin Swellings

1) Inguinal hernia – direct and indirect
2) Femoral hernia
3) Undescended testis
4) Inguinal lymphadenitis
5) Lipoma of spermatic cord
6) Excysted hydrocele
7) Saphena varix
8) Femoral artery aneurysm
9) Psoas abscess
10) Femoral nerve neuroma

2) Inguinal Hernia

- Occur when intestines (bowel), omentum or other abdominal organs protrude through the abdominal ring within a processus vaginalis in the inguinal canal
- If the processus vaginalis does not remain patent an indirect hernia cannot develop
- Most common type of hernia
- Are typically a result of the testis descending from the abdomen into the scrotum, these types of hernias are found in men more than women at a rate of about 10 to 1.

Diagram 8.4: Inguinal Hernia



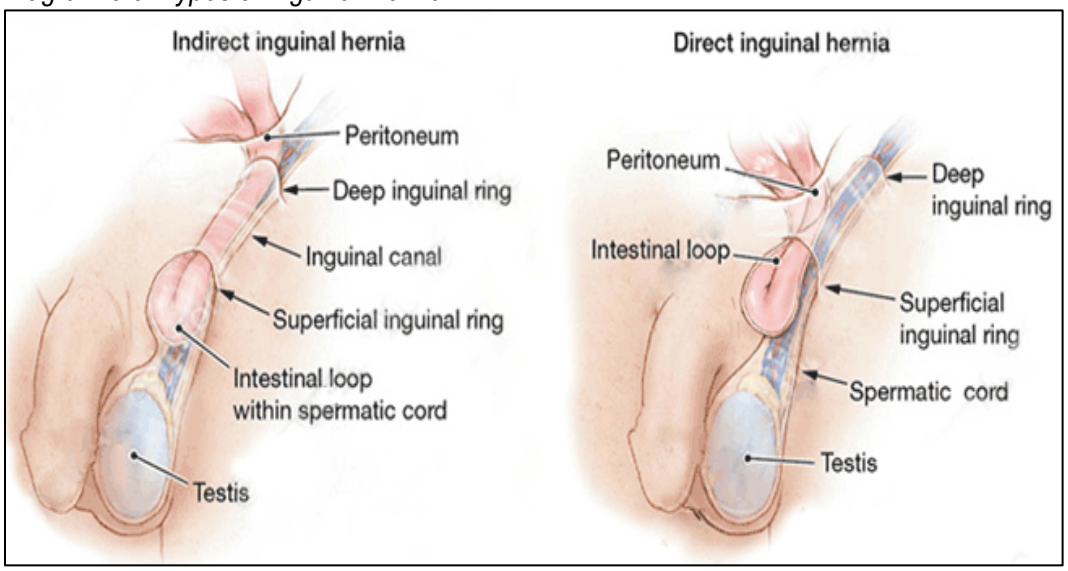
Causes

- Congenital or present at birth
- Acquired as the result of sudden or repetitive strain, pressure or injury which weakens the abdominal wall.

Presentation

- Typically located in the inguinal area and unilateral or bilateral; direct or indirect
- Often present is a painless bulge in the groin area, more visible when straining or coughing and may disappear when lying down
- May become incarcerated or strangulated
- Progressively increase in size and grow more and more uncomfortable with time

Diagram 8.5: Types of Inguinal Hernia



Types of Indirect Inguinal Hernias

1) **Congenital vaginal hernia**
   - The entire processus vaginalis remains patent and the contents of the hernia pass through it into the scrotum
   - Such a hernia is also called a complete hernia
2) **Congenital funicular hernia**
   - A patent processus vaginalis is present in relation to the spermatic cord, but does not communicate with the tunica vaginalis
   - Hernial contents lie above the level of testis
3) **Bubonocele**
   - The hernia is confined to the inguinal canal and does not protrude through the superficial inguinal ring

Diagnosis

1. Medical history
2. Physical examination of the groin.

Table 8.1: Direct and Indirect Hernias

	Direct Inguinal Hernia	**Indirect Inguinal Hernia**
1.	Protrudes directly forwards when the patient stands up	Shows a more oblique route downwards towards the scrotum
2.	Appears as a symmetric, circular swelling at the external ring, i.e. medial to the femoral artery	Indirect hernia is seen as an elliptical swelling
3.	Reduced indirect hernia can be controlled by pressure over the internal ring, classically with a single finger	Direct hernia cannot
4.	On standing, the direct hernias appear immediately	Indirect hernia takes time to reach its full size
5.	On lying down, direct hernias disappear immediately	There is a delay before the reducible indirect retracts fully

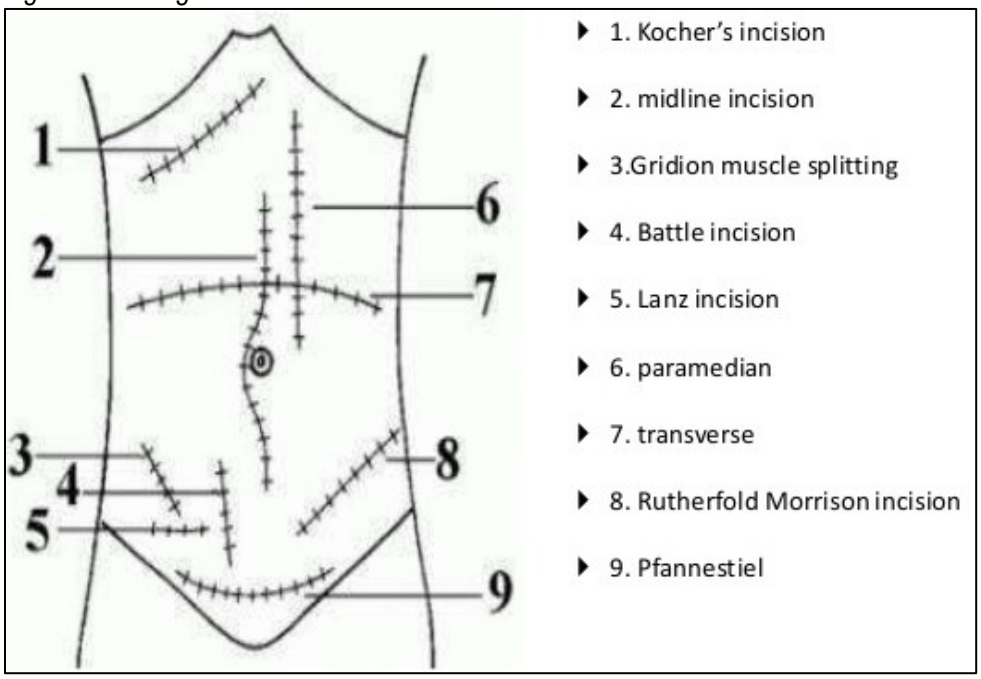
Table 8.2: Differences between Femoral Hernia and Inguinal Hernia

Feature	Inguinal Hernia	Femoral Hernia
Sex	Male	Female
Defect	Pass through inguinal canal	Passes through the femoral canal
Site	Above and medial to the pubic tubercle	Below and lateral to the pubic tubercle
Strangulation	Less common	More common because of rigid neck-Ricter's hernia
Treatment	Can be treated without surgery	Surgery is a must because of risk of strangulation

3) Incisional Hernia

- Occurs at the site of a previous incision in the abdominal wall as a result of the muscles of an old incision breaking down.
- Often identifiable as a bulge at or near the area of prior incision
- Procedures that can result in an incisional hernia are intestinal surgery, vascular surgery, an appendectomy or laparoscopy

Figure 8.6: Surgical Incisions



4) Umbilical Hernia

- Occur in or around the naval, or umbilicus
- Abdominal lining, or any portion of abdominal organs, protrudes through a small hole in the abdominal wall around the navel area
- Can be acquired as the result of a sudden or repetitive strain, pressure or injury which weakens the abdominal wall or can also be congenital, or present since birth
- Caused at birth by a weakness in the navel area where the umbilical cord exited the infant, the umbilical ring never quite heals.
- Signs and symptoms
  - Vary from person to person, however
  - Primary symptom - a small, soft bulge under or around the navel area, bulge may be visible, or it may only be felt when pushed on, sometimes the umbilical hernia is accompanied by pain, or a burning sensation, in the abdomen and may become more severe when lifting, coughing or sneezing. This area may also become swollen and may appear red or a grey-blue on the surface

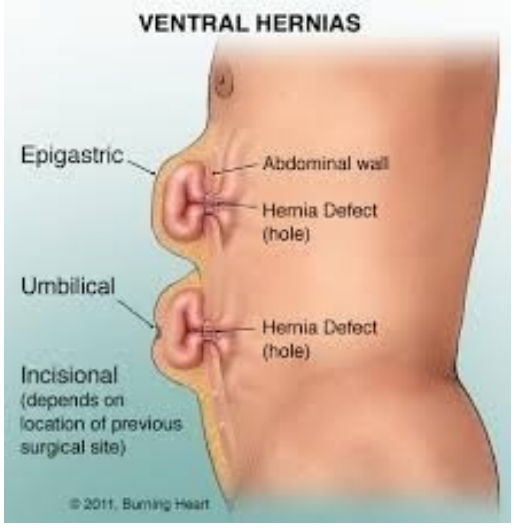
5) Epigastric Hernia

- Occur in the midline in the upper abdomen between the rib cage and the umbilicus
- Usually composed of fat and rarely containing abdominal organs.
- Can be congenital in origin or develop as a result of an increase in intra-abdominal pressure from lifting or straining
- Epigastric hernias are usually asymptomatic but cause pain with straining
- May increase in size and may become strangulated or incarcerated

6) Ventral Hernia (Abdominal)

- Occur anywhere in the front of the abdominal wall usually appear in the midline
- Result from a tear or division in the muscles or fascia
- Composed of fat and rarely containing abdominal organs
- Can be congenital in origin or develop as a result of an increase in intra-abdominal pressure form lifting or straining

Figure 8.7: Ventral Hernias



What are the complications of hernias?

INTESTINAL OBSTRUCTION

1.0 INTRODUCTION

- Is a partial or complete blockage of the bowel that results in the failure of the intestinal contents to pass through
- A mechanical or functional **obstruction** of the **intestines** which prevents the normal movement of the products of digestion
- Interferes with the propulsion of contents in the intestines

2.0 CLASSIFICATION

Table 8.3: Classification of Intestinal Obstruction

Classification	**Features**	
Dynamic or Adynamic	Dynamic: Peristalsis is working against a mechanical obstruction. The obstructing lesion may be: → Intraluminal (impacted faeces, foreign bodies, bezoar, gallstones → Intramural (malignant or strictures) → Extramural (intraperitoneal bands and adhesions, hernias, volvulus or intussusception.	Adynamic: → This may occur in two forms. → Peristalsis may be absent (e.g. paralytic ileus) → Or it may be present in a non-propulsive form (e.g. mesenteric vascular occlusion or pseudo-obstruction). In both types a mechanical element is absent
Mechanical or functional obstruction	Mechanical → Caused by a physical blockage → Complete arrest or serious impairment of the passage of intestinal contents caused by a mechanical blockage → often requires corrective surgery	Functional → Caused by paralysis of intestinal transit → with certain exceptions, relies on conservative management (and can be exacerbated by operative intervention).
Simple or Strangulated (presence or absence of an adequate blood supply)	→ <u>Simple</u> → Blockage without interfering with vascular supply	→ <u>Strangulated</u> → Arterial & venous flow of a bowel segment are cut off → Closed loop obstruction - bowel obstructed at both the proximal & distal end → Requires more urgent operative correction → Most commonly associated with hernia, volvulus, intussusception, vascular occlusion
Complete or incomplete (level)	→ Incomplete (partial) → abdominal pain, emesis	→ complete → abdominal pain, emesis and obstipation (severe constipation).
Small Bowel or Large Bowel	→ Small bowel obstruction → <u>High</u>: Early profuse vomiting, rapid dehydration → <u>Low</u>: Predominant pain, and central distention, vomiting delayed, air-fluid levels seen on AXR	→ Large bowel obstruction → Early pronounced distension, mild pain, vomiting, dehydration late e.g. carcinoma, diverticulitis or volvulus

3.0 CAUSES

Explain these mechanisms

Table 8.4: Causes of Intestinal Obstruction

Class	Description	Site/mechanism	Causes (examples)
Dynamic	peristalsis is working against mechanical obstruction	Luminal	gall stones; foreign bodies; faecoliths; round worms; tumours; bezoars; impaction; imperforate anus
		Intramural	stricture e.g. Chron's disease; Congenital stenosis and malignancy
		Extramural	adhesions and bands; strangulated hernia; intussusception; volvulus and intra-abdominal tumour
Adynamic	peristalsis is absent or may be present in a non-propulsive form (pseudo obstruction)	Neurogenic obstruction	paralytic ileus (paralysis of the muscularis of the intestine)
		Vascular obstruction	thrombosis, embolism and accidental ligation
		Myopathies and neuropathies	Hirchsprung's disease

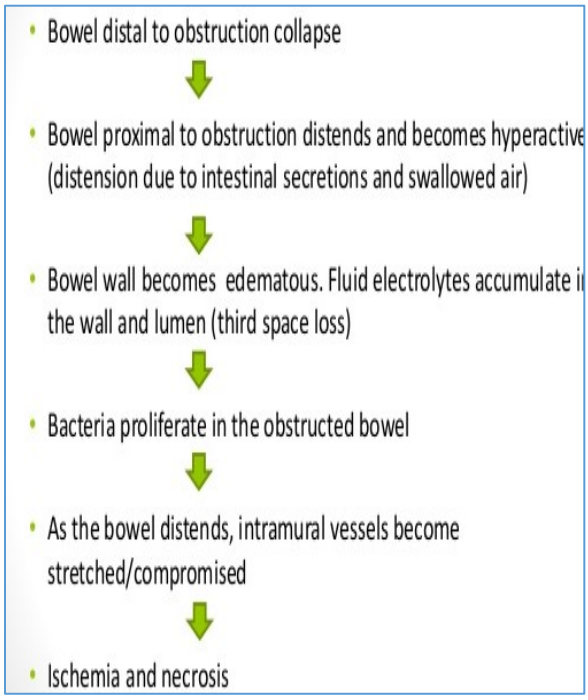
4.0 MECHANISMS OF OBSTRUCTION

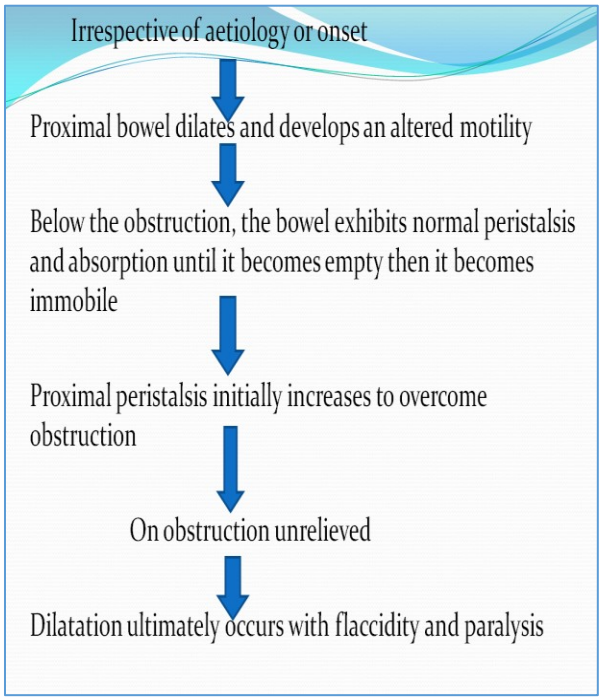
- Volvulus; incarceration; obstruction; Intussusception; Impaired innervation

5.0 PATHOPHYSIOLOGY

- Dynamic obstruction
  - Proximal intestine dilates and develops an altered motility
  - Below the obstruction, the bowel has normal peristalsis and absorption until it becomes empty, contracts and becomes immobile
  - Initially the proximal peristalsis is increased in order to overcome the obstruction
  - If the obstruction is not relieved the bowel distends resulting in reduced peristaltic force and eventually flaccidity and paralysis
    - Causes of proximal distention include gas (overgrowth of aerobic and anaerobic organisms which produce gas) and fluid (accumulation of digestive juices with reduced absorption)
- Dynamics obstruction
  - Peristalsis is affected
  - Bowel distends

Figure 8.8: Pathophysiology of Intestinal Obstruction





6.0 PRESENTATION

- Pain (sudden onset, severe colicky central abdominal); vomiting; constipation (absolute); dehydration; hypokalaemia; pyrexia; abdominal distension and abdominal tenderness
- The four cardinal features of dynamic obstruction include colicky pain, distention, vomiting and absolute constipation.
- The clinical features are also influenced by the site of obstruction whether small bowel or large bowel and the onset of obstruction whether acute, chronic or acute on chronic

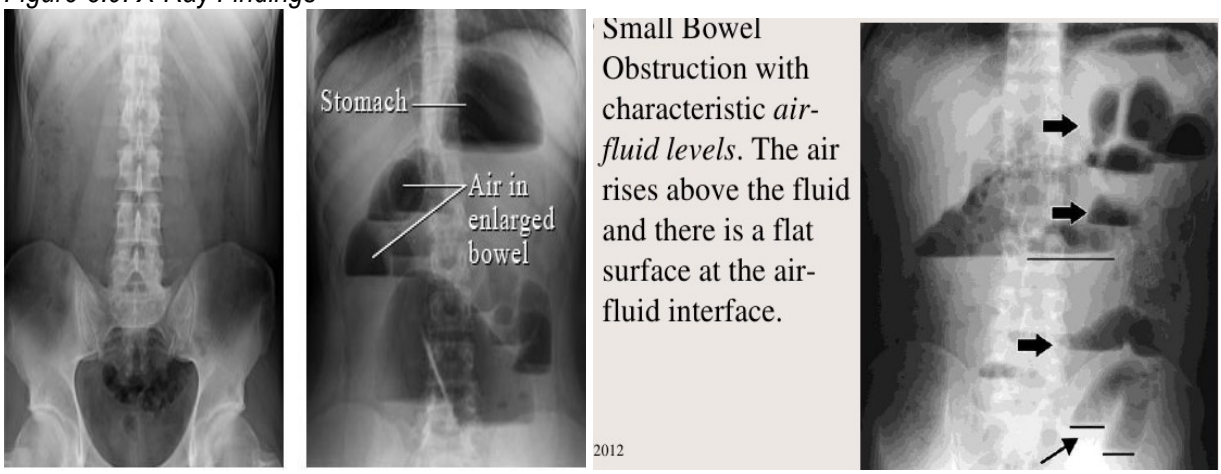
Table 8.5: Differences between Proximal and Distal Small Bowel Obstruction

Symptom	Gastric or proximal small bowel	Distal small bowel or large bowel
Vomiting	Develops early Bilious, watery Large amounts No or little odour	Develops later Small volumes May contain food particles Foul odour May be absent
Pain	Early symptom Peri-umbilical Short intermittent cramps	Late symptom Localised, deep visceral pain Often crampy Long intervals between cramps
Abdominal distension	May be absent	Present
Anorexia	Always	May not be present
Bowel sounds	May be normal	May be hyperactive

7.0 INVESTIGATIONS

- Abdominal ultrasound; plain abdominal x-ray (supine, erect – shows fluid levels; gas shadows; impacted foreign material); Barium enema; CT scan; TBCs

Figure 8.9: X-Ray Findings



8.0 DIAGNOSIS

- Quartet of pain, distension, vomiting and absolute constipation

How would you make a diagnosis of IO?

What are the complications of intestinal obstruction?

Lesson 9: DISORDERS OF THE LARGE INTESTINES

Learning Objectives

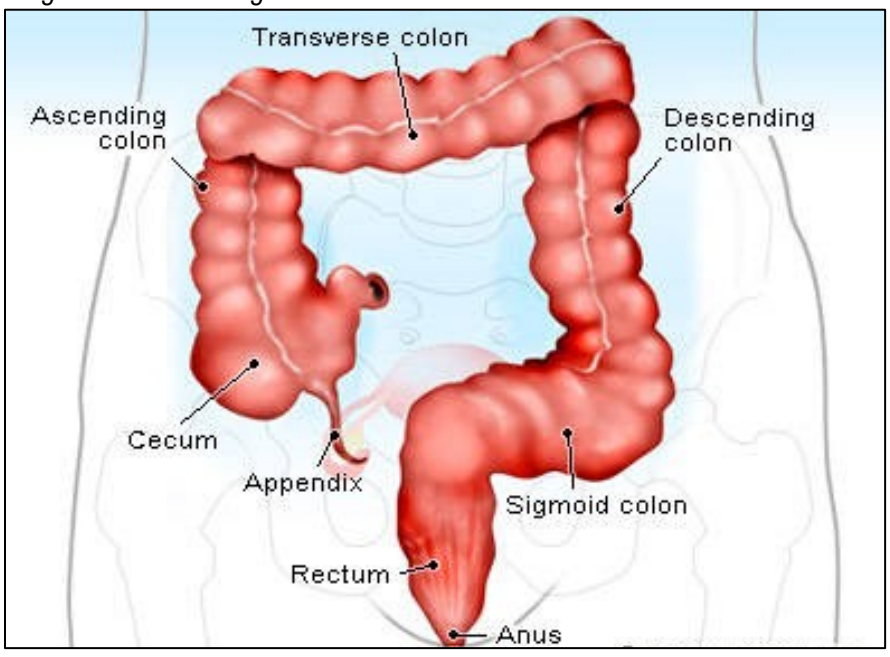
At the end of the lesson the learner will be able to: -

1) Describe the structure and function of the large intestine
2) Discuss the pathology of disorders of the large intestine
3) Investigate disorders of the large intestine

1.0 NORMAL STRUCTURE OF LARGE INTESTINES (COLON)

- Hollow tube - five to six feet (1.5 to 1.8 meters) long and up to five inches (12.7 cm) in diameter
- Divided up into segments - the first segment (the cecum) located in the lower right side of the abdomen; second segment (ascending colon), third (transverse colon), and fourth segment (descending colon)
- Descending part of the colon leads down to the S-shaped portion of the bowel called the sigmoid, which connects to the rectum
- Colon is made up of four different layers of tissue
  i) Innermost mucosal layer (a thin layer, with specialized cells that are constantly dying and being replenished and comes in direct contact with the faecal matter)
  ii) Sub mucosal tissue (supports the mucosal layer)
  iii) Muscular tissue **(**provides strength to the colon and causes contractions which push the faecal matter through the large intestine)
  iv) Outermost serosal layer **(**supports and protects the colon).

Diagram 9.1: The Large Intestine



2.0 ACUTE APPENDICITIS

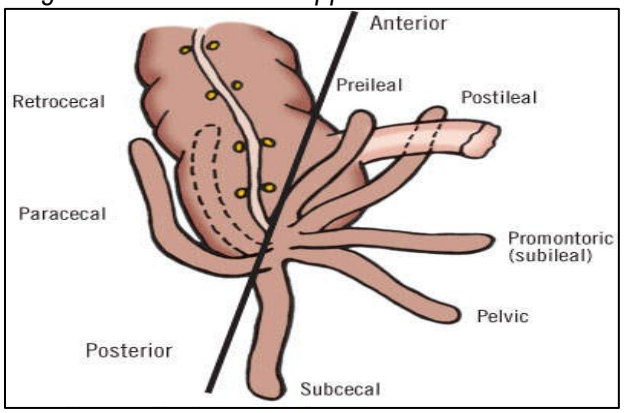
2.1. Definition

- Inflammation of the inner lining of the vermiform appendix that subsequently spreads to its other parts

2.2. Anatomy

- Definition
    - This is a worm-like diverticulum arising from the posteromedial wall, of the caecum, about 2 cm below the ileocaecal orifice
    - It is a blind muscular tube with mucosal, submucosal, muscular and serosal layers.
- Dimensions
    - The length varies from 2 to 20 cm with an average of 9 cm.
    - The average length is between 7.5 and 10 cm
    - It is longer in children than in adults.
    - The diameter is about 5-10 mm.
    - The lumen is quite narrow and may be obliterated after mid-adult life.

Diagram 9.2: Sites of the Appendix



2.3. Causes

1) Obstructive
    a) Foreign bodies
        i. Parasites – threadworms, roundworms, Enterobius vermicularis especially Oxyuris vermicularis(pinworm)
        ii. Vegetable – seeds, date stones
        iii. ''Mineral'' – faecoliths – commonest cause; found in 40% of cases of simple acute appendicitis, 65% of cases of gangrenous appendicitis without rupture, and 90% of cases of gangrenous appendicitis with rupture. A faecolith is composed of inspissated faecal material/barium from previous X-ray studies, calcium phosphates, bacteria and epithelial debris
    b) Strictures and bands of a congenital nature
    c) Tumour – obstruction of the appendiceal orifice by tumour, particularly carcinoma of the caecum, is acute appendicitis in middle age and the elderly
    d) Calculi
    e) Sub mucous lymphoid tissue – diffuse lymphoid hyperplasia, especially in children

2) Non-Obstructive
    a) Infection
    b) Vascular occlusion
    c) Inappropriate diet lacking roughage

2.4. Pathophysiology

- Obstruction appendiceal lumen causes an increase in pressure within the lumen and increased continuous secretion of fluids and mucus from the mucosa and the stagnation of this material
- Mucosal secretions continue to increase intraluminal pressure
- The pressure exceeds capillary perfusion pressure and venous and lymphatic drainage are obstructed
- There is vascular compromise which causes epithelial mucosa breaks down and bacterial invasion by bowel flora occurs
- Increased pressure also leads to arterial stasis and tissue infarction
- Results in perforation and spillage of infected appendiceal contents into the peritoneum
- Intestinal bacteria multiply causing acute inflammation leading to the recruitment of white blood cells and the formation of pus and subsequent higher intraluminal pressure
- If appendiceal obstruction persists, intraluminal pressure rises ultimately above that of the appendiceal veins, leading to venous outflow obstruction which causes appendiceal wall ischemia resulting in a loss of epithelial integrity and allowing bacterial invasion of the appendiceal wall.
- Within a few hours, this localized condition may worsen because of thrombosis of the appendicular artery and veins, leading to perforation and gangrene of the appendix.
- Pelvic appendix may irritate the bladder or rectum causing suprapubic pain, pain with urination, or feeling the need to defecate
- Multiple anatomic variations explain the difficulty in diagnosing appendicitis

2.5. Pathology

Gross (macroscopy)	• Early stages – grossly oedematous with dilatation of serosal vessels • Appendiceal wall is grossly thickened and the lumen is dilated • Serosal exudate (fibrinous or fribropurulent)
Microscopy	• Neutrophil infiltration

2.6. Clinical features

Symptoms

1. Periumbilical Colic
    - Poorly localised colicky abdominal pain, central abdominal pain is usually his first symptom, and it may be severe enough to wake him from sleep.
    - The pain is moderately severe, and is steady, sometimes with intermittent cramping superimposed.
    - Pain shifts to right iliac fossa after 1 – 12 hours average 4 – 6 hours.
2. Anorexia
3. Nausea and vomiting - caused both by neural stimulation and the presence of ileus
5. Vital Signs:
    - Slight pyrexia (37.2-37.7^{o}C) with corresponding increase in the pulse rate to 80 or 90 is usual
    - If his pulse is raised, his appendix has possibly perforated.
    - A steadily rising pulse is always serious
6. Distinction between obstructive & non-obstructive appendicitis:
    - Nature of pain in obstructive type is colicky.
    - In obstructive type - pain starts early in epigastrium.
    - Vomiting more marked in obstructive type.
    - Tenderness and rigidity – less in an early case of obstructive type
7. Bowel Function:

- Most patients have obstipation beginning prior to the onset of abdominal pain, and many feel that defecation would relieve their abdominal pain.
- Diarrhoea occurs in some patients, particularly children

Signs

- General – fever, tachycardia and dehydration
- Inspection – movement with respiration may be absent, distention
- Palpation
  - Maximal tenderness at McBurney's point
  - RLQ tenderness and rebound tenderness
  - Guarding; rigidity
- Auscultation - bowel sounds may be absent in perforated appendix-paralytic ileus or may be increased with obstruction at the caecum
- The cardinal features are those of an unwell patient with low grade pyrexia, localized abdominal tenderness, muscle guarding and rebound tenderness
- **McBurney point:** Gentle superficial palpation of the abdomen, beginning in the left iliac fossa moving anticlockwise to the right iliac fossa, will detect muscle guarding over the point of maximum tenderness, classically McBurney point.
- **Pointing sign**
  - The patient is then asked to point to where the pain began and to where it moved
- **Rovsing's sign**
  - Deep palpation of the left iliac fossa may cause pain in the right iliac fossa, which is helpful in supporting a clinical diagnosis of appendicitis. It is indicative of right-sided local peritoneal irritation
- **Psoas sign**
  - Occasionally an inflamed appendix lies on the psoas muscle and the patient, often a young adult, will lie with the right hip flexed for pain relief'
  - Pain in the right lower quadrant brought on by extension of the right hip (iliopsoas sign), is associated with a retrocecal appendix
  - It suggests that an inflamed appendix is located along the course of the right psoas muscle
- **Obturator test**
  - Spasm of the obturator internus is sometimes demonstrable when the hip is flexed and internally rotated.
  - If an inflamed appendix is in contact with the obturator internus, this manoeuvre will cause pain in the hypogastrium (Zachary Cope).
  - Pain with internal rotation of the hip (obturator sign) is associated with a pelvic appendix
  - These signs are present in a minority of patients with acute appendicitis.
  - Their absence never should be used to rule out appendiceal inflammation
- **Dunphy's sign**
  - Sharp pain in the RLQ elicited by a voluntary cough.
  - It may be helpful in making the clinical diagnosis of localized peritonitis.

2.7 Diagnosis

- The diagnosis of appendicitis rests more on thorough clinical examination of the abdomen than on any aspect of the history or laboratory investigation.
- Similarly, RLQ pain in response to percussion of a remote quadrant of the abdomen, or to firm percussion of the patient's heel, suggests peritoneal inflammation.
- **Markle sign:** Pain elicited in a certain area of the abdomen when the standing patient drops from standing on toes to the heels with a jarring landing, is stated

2.8 Differential Diagnosis

Table 9.1: Differential Diagnosis

	System	Differentials
1.	Pancreatico-Biliary	cholecystitis; Biliary colic; Pancreatitis
2.	GIT	gastroenteritis, enterocolitis; Meckel diverticulitis; Perforated duodenal ulcer; Crohn's disease, ulcerative colitis; Colon carcinoma, peri caecal abscess; Intussusception; Mesenteric adenitis
3.	Urinary system	ureteric colic; Urinary tract infection (UTI); Pyelonephritis
4.	Reproductive (women)	ovarian cyst torsion; Mittel Schmerz; ectopic pregnancy; pelvic inflammatory disease (PID); endometriosis; salpingitis
5.	Respiratory	Lobar pneumonia

Table 8.2: Differential Diagnosis

	Age	Differentials
1.	Children	Acute mesenteric adenitis/lymphadenitis; acute gastroenteritis; meckel's diverticulitis; intussusception; purpura; lobar pneumonia & pleurisy
2.	Adults	Ureteric colic/stone; right-sided acute pyelonephritis; perforated peptic ulcer; acute pancreatitis; rectus sheath haematoma; disease of the male urogenital system (torsion of the testis, acute epididymitis and seminal vasculitis); Crohn's disease; primary peritonitis
3.	Gynaecologic Disorders	PID; Ruptured Graafian follicle/Mittelschmerz; Twisted ovarian cyst or tumour; Endometriosis; Ruptured ectopic pregnancy
4.	Elderly	Sigmoid diverticulitis; Intestinal obstruction; Carcinoma of the caecum
5.	**Other Diseases**	Foreign-body perforations of the bowel; closed-loop intestinal obstruction; mesenteric vascular occlusion; pleuritis of the right lower chest; acute cholecystitis; acute pancreatitis; haematoma of the abdominal wall

2.9 Staging

Table 9.3: Staging of Appendicitis

	Stage	Description
1.0	Oedematous stage	• Appendicitis may have spontaneous regression or may evolve to the 2nd stage • Mesoappendix is commonly involved with inflammation
2.0	Purulent (phlegmonous) stage	• Spontaneous regression occurs rarely • Appendicitis usually evolves beyond perforation and rupture • Peritonitis may be possible
3.0	Gangrenous stage	• Spontaneous regression never occurs; Peritonitis is present

2.10 Scoring - MANTRELS or Alvarado Scoring

- Makes use of clinical signs, symptoms and laboratory findings
- Each of the alphabets represents a sign or symptom, and a score of 1 is award to each, where they exist, except T and S that are scored 2 each

Table 9.4: MANTREALS Staging of Appendicitis

	Description	Score	interpretation	
M	Movement of pain to the RIF	1	**Score**	**Meaning**
A	Anorexia	1	9 - 10	Appendicitis highly likely
N	Nausea and Vomiting	1	7 - 8	Indicative of appendicitis
T	Tenderness in the RIF	2	5 – 6	Appendicitis likely
R	Rebound tenderness	1	< 5	Appendicitis unlikely
E	Elevated temperature/pyrexia	1	A score of greater than 6 in children makes the possibility of appendicitis up to 100% likely	
L	Leucocytosis > 10,000/mm^2	1		
S	Shift in WBC count to the right	2		
	Total	10		

The Paediatric Appendicitis Scoring (PAS) Scoring

- Designed for use in children between the ages of 4 – 15 years.
- It is more or less a modified Alvarado or MANTRELS scoring
- It uses 8 variables (laboratory findings as well as signs and symptoms), to which a score is of 1 or 2 is given to each variable, where they exist.
- Maximum score that can be accumulated is 10.
- Presence of anorexia, pyrexia, nausea or vomiting, leucocytosis and high neutrophils are given a score of 1 each
- Tenderness on coughing, hopping or percussing the abdomen is given a score of 2.
- So too is the presence of tenderness in the right lower abdominal region.
- A Paediatric Appendicitis Score of 6 and above is highly indicative of appendicitis in children

2.11 Investigations

1) Full Haemogram - mild elevation of WBCs (i.e. >12,000/mL) especially neutrophilia (values greater than 17,000 cells indicate complicated appendicitis)
2) Urinalysis
   - Differentiating appendicitis from urinary tract conditions
     i) Mild pyuria may occur in patients with appendicitis because of the relationship of the appendix with the right ureter
     ii) Haematuria in ureteric colic
     iii) Glycosuria in Diabetic ketoacidosis
     iv) Urobilinogen in acute porphyria
3) Urea and Electrolytes - Detect any deranged electrolytes, R/o Renal Pyelonephritis and colic
4) C-reactive protein and ESR – raised
5) Liver and pancreatic function tests (e.g., Transaminases, bilirubin, alkaline phosphatase, serum lipase, amylase) R/o Acute pancreatitis and cholecystitis
6) Pregnancy test-in females of childbearing age
7) CT scan for peri- appendiceal abscess and wall of appendix. May be used in obese patients where ultrasonography may be hampered
8) Diagnostic laparoscopy

2.12 Complications

1. Perforation (Risk factors for perforation - extremes of age, immunosuppression, diabetes mellitus, faecolith obstruction of the appendix lumen, pelvic appendix - free-lying, previous abdominal surgery which limits the ability of the greater omentum to wall off the spread of peritoneal contamination)

2. Peritonitis with paralytic ileus
3. Incisional hernia
4. Abscess formation – intra abdominal, diaphragmatic, liver abscess
5. Toxaemia
6. Wound infection
7. Dehydration
8. Adhesions → intestinal obstruction
9. Faecal fistula
10. Portal pyaemia/pylophlebitis
11. Mucocele

3.0 COLITIS

What are the causes and features of colitis?

4.0 IDIOPATHIC CHRONIC INFLAMMATORY BOWEL DISEASE

4.1. ULCERATIVE COLITIS

- Is an ulcero-inflammatory disease limited to the colon and affecting only the mucosa and submucosa except in severe cases
- It begins in the rectum, and in continuity extends upwards into the sigmoid colon, descending colon, transverse colon and sometimes the entire colon

Aetiology
- Unknown

Pathology
- Involves the rectum and extends proximally in retrograde fashion to involve the entire rectum
- Mucosa - extensive ulceration; slight reddening; granularity with friability; easy bleeding
- Regeneration causes pseudopolys
- Colon may swell and become gangrenous (toxic megacolon)

Clinical Features
- Depends on activity of the disease process
- Relapsing disorder; attacks precipitated by stress
- Bloody mucoid diarrhoea, bleeding, lower abdominal pain (relieved by defecation), abdominal cramps, constipation (paradoxically due to disruption of normal peristalsis)

Complications
1) Local - blood and fluid loss; Cancer; Perfection; Malabsorption; Perianal fistula; Stricture; Toxic megacolon (fulminant colitis)
2) Systemic - erythema nodosum; Pyoderma gangrenosum; Iritis; Arthritis; Chronic liver disease; Ankylosing spondylitis

4.2. CHRON'S DISEASE

- Is a chronic idiopathic bowel disease characterized by transmural non-caseating granulomatous inflammation commonly affecting terminal ileum and/or colon
- May involve the duodenum, stomach, oesophagus

Aetiology

- Unknown but associated with genetic factors, immunological factors and exogenous factors (microbial factors, psychosocial factors, smoking and oral contraceptives)

Pathology

- Multiple well demarcated segmental bowel involvement with intervening uninvolved (skipped areas/lesions)
- Affected segment is thick and hard (hose pipe)
- Lumen markedly narrowed
- Focal mucosal ulcers resembling canker sores (aphthous ulcers), oedema, and loss of normal mucosal texture
- Mucosal inflammation (transmural)

Clinical Features

- Mild diarrhoea, fever, abdominal pain, attacks precipitated by physical and emotional stress, occult or overt faecal blood loss, anaemia

Complications

1) Intestinal - malabsorption; Fistula formation; Stricture formation; Development of malignancy
2) Extra intestinal - migratory polyarthritis; Sarcoilitis; Ankylosing spondylitis; Finger clubbing; Erythema nodosum

Table 8.5: Comparison of Main Features of Chron's Disease and Ulcerative Colitis

	Feature	Chron's Disease	Ulcerative Colitis
1.	Characteristics	• Chronic relapsing inflammatory condition of unknown aetiology	• Chronic relapsing inflammatory condition of unknown Aetiology
2.	Presentation	• Abdominal pain or obstruction	• Bloody diarrhoea
3.	Sites	• Anywhere in the GI tract • Commonest in distal ileum then colon	• Confined to large bowel • May be localized to rectum or in continuity with any length of the colon
4.	Inflammation	• Transmural, patchy often partly granulomatous	• Mucosal, diffuse, non-granulomatous
5.	Complications	• Malabsorption; Fistula formation; Anal lesions; Malignancy (adenocarcinoma); Amyloidosis • Perforation	• Blood loss; Electrolyte disturbances; Toxic dilatation; Malignancy; Extra-colonic complications

5.0 COLORECTAL CANCER

Risk Factors

- Family history and genetics; Age; Diet – red meat; Inflammatory bowel disease - ulcerative colitis and Chron's disease; Obesity; Smoking; Alcohol; Type 2 diabetes mellitus

Features

- Change in bowel habits – diarrhoea, constipation, narrowing of the stool that lasts more than a few days
- Urge to open bowels but not relieved by doing so
- Rectal bleeding (bright red or dark blood), chronic pain, bloating and/or fullness, decreased appetite, anaemia, ulceration or growth, fatigue and weight loss

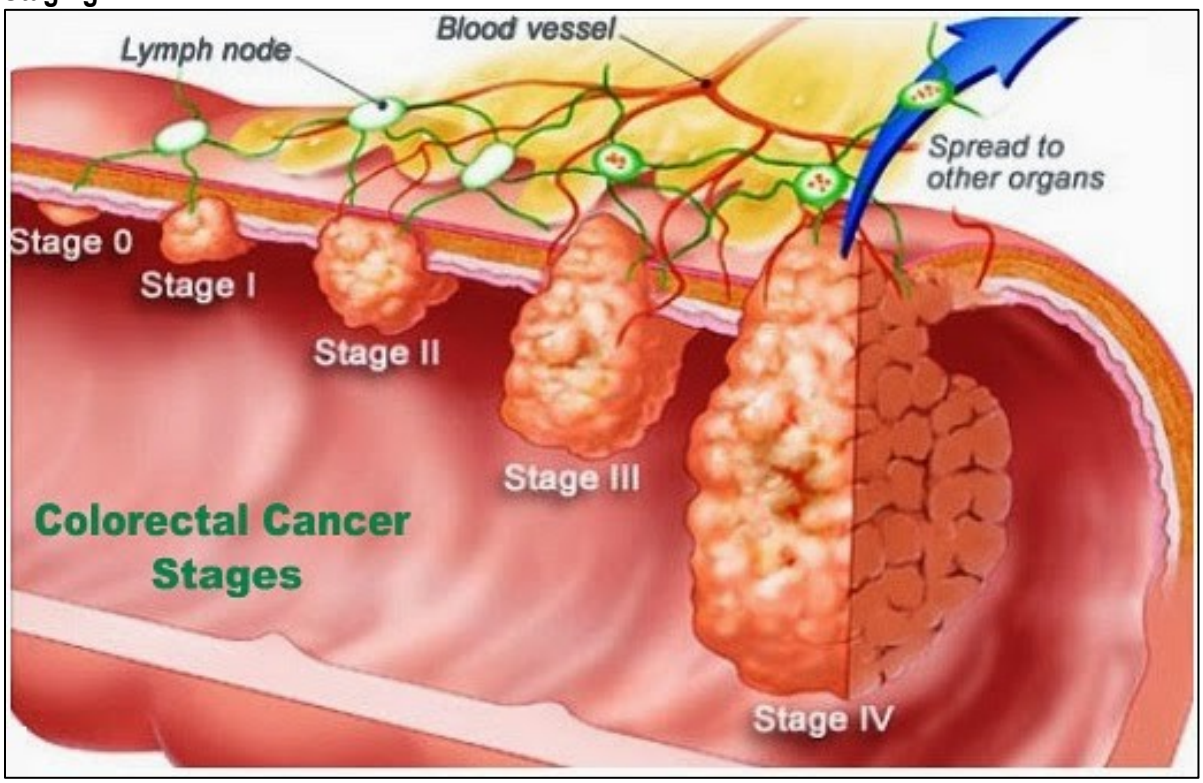
Investigations

- Total Blood counts
- Endoscopy – Colonoscopy and sigmoidoscopy
- Faecal occult blood test
- Barium enema
- Biopsy
- Imaging - X-rays, CT or CAT scan, Ultrasound, MRI
- Position emission tomography (PET) scan

Spread

- Metastasis to the liver and peritoneum

Staging



Lesson 10: DISORDERS OF THE ANO-RECTAL REGION

Learning Objectives

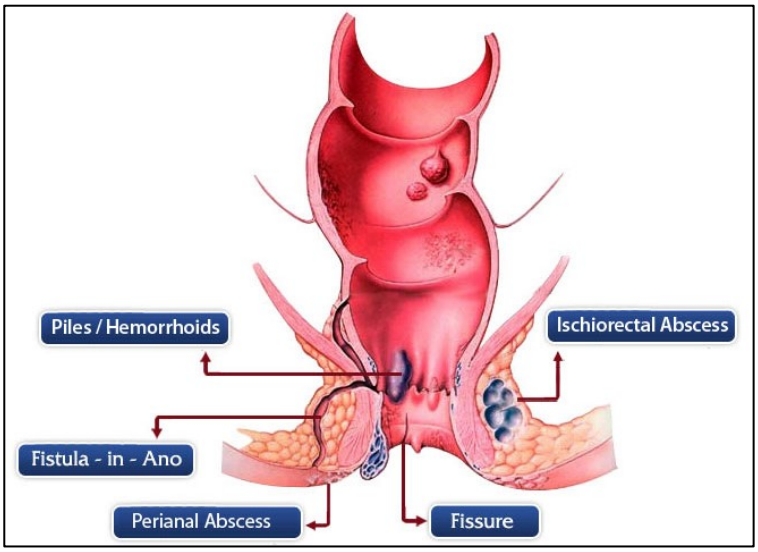
At the end of the lesson the learner will be able to: -

1) Describe the structure and function of the anus, rectum and peritoneum
2) Discuss the pathology of disorders of the anus, rectum and peritoneum
3) Investigate disorders of the anus, rectum and peritoneum

1.0 NORMAL STRUCTURE

Describe the structure of the anus and rectum

2.0 ANAL DISORDERS



3.0 FISSURE IN ANO

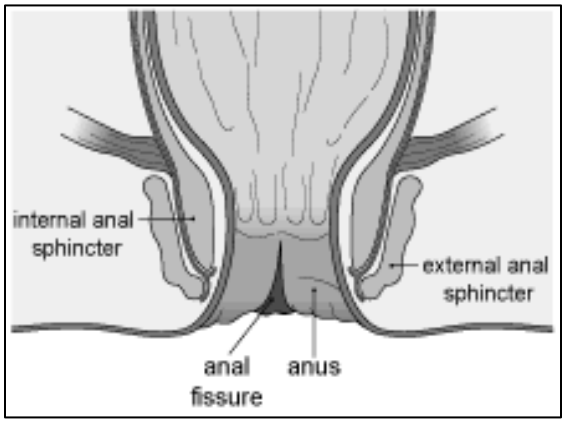
Introduction and Definition

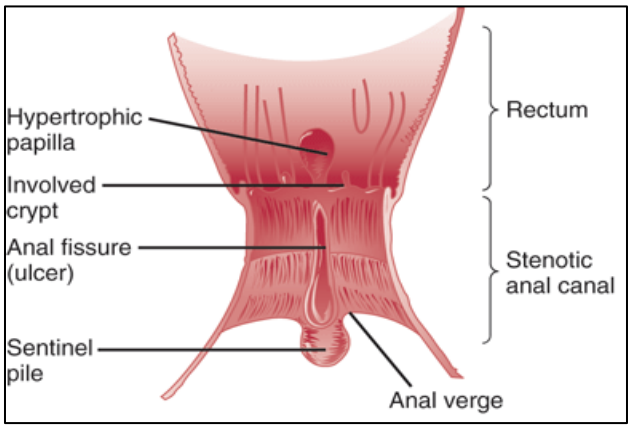
- An anal fissure or rectal fissure is a break, cut or tear in the skin of the anal canal
- Common condition of the anus and anal canal
- Affect men and women equally and both the young and the old
- Usually cause pain during bowel movements that often is severe
- Most common cause of rectal bleeding in infancy

Anatomy

- Anal fissures occur in the specialized tissue that lines the anus and anal canal called anoderm at a line just inside the anus (anal verge or inter-sphincteric groove)
- Unlike skin, anoderm has no hairs, sweat glands, or sebaceous (oil) glands and contains a larger number of somatic sensory nerves that sense light touch and pain
- Abundance of nerves explains why anal fissures are so painful
- Hairless, gland-less, extremely sensitive anoderm continues for the entire length of anal canal until it meets the demarcating line for the rectum, called the dentate line
- Most common location is the midline posteriorly in the anal canal (part of the anus nearest the spine) because of the configuration of the muscle that surrounds the anus
- When fissures occur in locations other than the midline posteriorly or anteriorly trauma is the likely cause

Diagram 10.1: Anatomy of the Rectum and Anus





Predisposing factors

- Infancy; Aging; Constipation; Childbirth; Crohn's disease

Causes of anal fissures

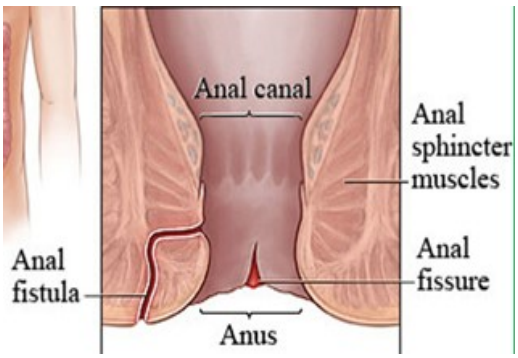
Class	Examples
Traumatic	• Bowel movement; Hard stool/constipation; Repeated episodes of diarrhoea; Insertion of a rectal thermometer, enema tip, endoscope, or ultrasound probe; During childbirth
Non-traumatic	• Anal cancer; Crohn's disease; Leukaemia; Infection - TB, viral infections (CMV or herpes), syphilis, gonorrhoea, chlamydia, Chancroid, and HIV

Pathophysiology

- Most anal fissures are caused by stretching of the anal mucosa beyond its capability
- Pathophysiology depends on the causes

Clinical features

- Pain, sometimes severe, during bowel movements; Itching or irritation around the anus; A visible crack in the skin around the anus; A small lump or skin tag on the skin near the anal fissure; Rectal bleeding



Complications

1. Failure to heal – failure to heal within six weeks is considered chronic. Poor blood supply contributes to the poor healing
2. Recurrence
3. A tear that extends to surrounding muscles

4.0 ANAL FISTULA (FISTULA IN ANO)

4.1. Introduction

- Fistula is an abnormal passage from one epithelial surface to another epithelial surface
- Fistulas occur spontaneously or secondary to perirectal abscess.
- Most fistulas originate in the anal crypts at the anorectal juncture

4.2. Classification

- Inter-sphincteric
- Trans-sphincteric
- Supra-sphincteric
- Extra-sphincteric

Etiology

- Erosion of anal canal
- Extension from infection from a tear in lining in anal canal
- Infecting organism is commonly Escherichia coli
- Fistulas usually arise spontaneously or occur secondary to drainage of a perirectal abscess.
- Predisposing causes include Crohn's disease and TB.
- Most fistulas originate in the anorectal crypts; others may result from diverticulitis, tumors, or trauma.
- Fistulas in infants are congenital and are more common in boys.
- Rectovaginal fistulas may be secondary to Crohn's disease, obstetric injuries, radiotherapy, or malignancy.

Risk factors:

- Injection of internal hemorrhoids, puncture wound from eggshells or fish bones, foreign objects, enema tip injuries
- Ruptured anal hematoma
- Prolapsed internal hemorrhoid
- Acute appendicitis, salpingitis, diverticulitis
- Inflammatory bowel disease (chronic ulcerative colitis, Crohn disease)
- Previous perirectal abscess
- Radiation treatment to perineum/pelvis

Signs and symptoms

- Constant or intermittent drainage or discharge
- Firm tender perianal lump
- External anal sphincter pain during and after defecation
- Spasm of external anal sphincter during and after defecation
- Anal bleeding
- Discoloration of skin surrounding the fistula
- Fistulous opening frequently granulose or scarred
- Possible fever
- A fistula is suggested by the presence of a small external opening outside the anal verge draining mucus, pus, or fecal matter.
- A fistula is confirmed by the demonstration of an internal opening within the anal canal.
- A history of recurrent abscess followed by intermittent or constant discharge is usual.

- On inspection, one or more secondary openings can be seen, and a cordlike tract can often be palpated

Diagnostic procedures
- Proctoscopy
- Sigmoidoscopy
- Probe inserted into tract to determine its course
- Injection of dilute methylene blue into abscess cavity may be helpful in demonstrating fistula

Differential diagnosis
- Pilonidal sinus
- Perianal abscess
- Urethroperineal fistulas
- Ischiorectal abscess
- Submucous or high muscular abscess
- Pelvirectal abscess (rare)
- Rule out: Crohn disease; carcinoma; retrorectal tumors

5.0 PRURITUS ANI

Definition
- Intense chronic itching in the anal and perianal skin. Usual course – acute

Aetiology

1) **Dermatologic disorders**
   - Allergies (soap, topical anesthetics, oral antibiotics); Fistulas; Fissures; Neoplasms; Psoriasis; Eczema; Seborrheic dermatitis; Contact dermatitis

2) **Infections**
   - Pinworms and other worms; Scabies; Pediculosis; Candidiasis; Tinea

3) **Other**
   - Poor hygiene (fecal material allowed to dry on the skin); Diabetes mellitus; Chronic liver disease; Diarrheic alkalotic irritation; Trauma from scented toilet paper

Risk factors
- Overweight
- Hairy, tendency to perspire a great deal
- Anxiety-itch-anxiety cycle

Signs and symptoms
- **Primary - r**ectal itching and anal erythema
- **Secondary- s**econdary infections with yeast, fungus, and/or bacteria are possible after prolonged scratching, anal itching, anal fissures, maceration, lichenification and excoriations

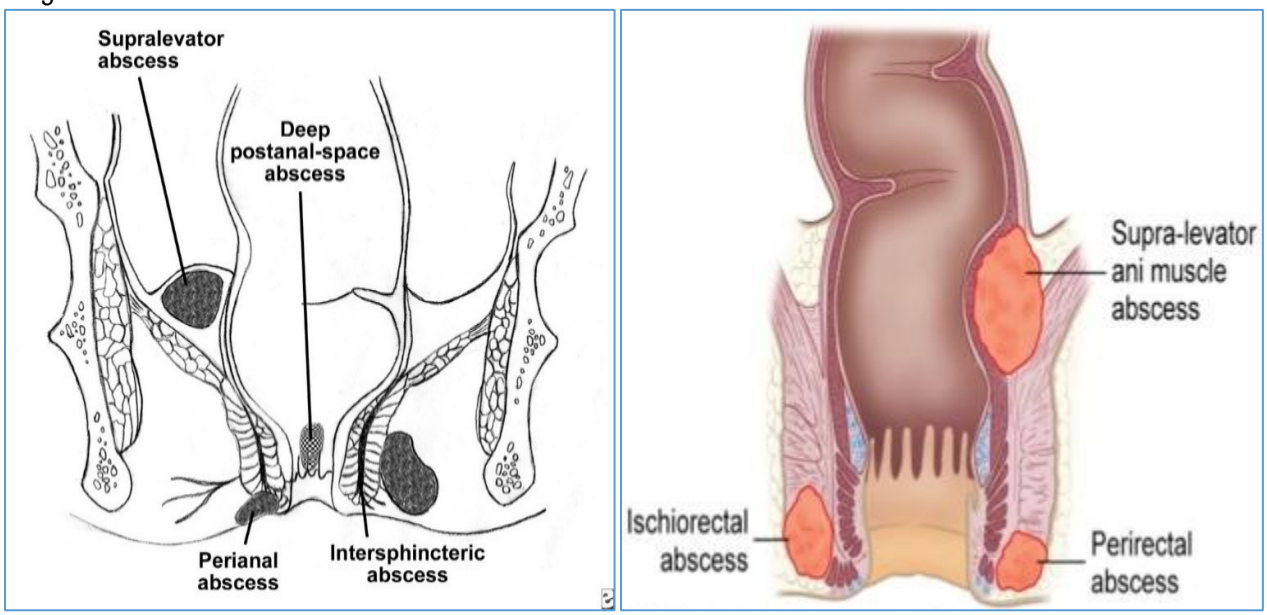
Laboratory
1) Glycosuria
2) Hyperglycemia
3) Skin scraping, yeast
4) Stool - ova plus parasites

6.0 ANORECTAL ABSCESS

Definition

- Anorectal abscess is an abscess adjacent to the anus
- Arises from an infection at one of the anal sinuses and soft tissues surrounding the anal canal, with formation of a discrete abscess cavity

Diagram 10.2: Anorectal Abscess



Causes

- Infection - Common organisms include *Escherichia coli*, *Enterococcus* species, and *Bacteroides* species

Pathophysiology

- Anal glands normally function to lubricate the anal canal
- Obstruction of anal crypts results in stasis of glandular secretions and when subsequently infected, suppuration and abscess formation within the anal gland results
- Anal abscess arises from an infection at one of the anal sinuses which leads to inflammation and abscess formation
- Pathological effects depend on the cause of the anal abscess
- Abscess typically forms in the intersphincteric space and can spread along various potential spaces.

Predisposing factors

- Anal sex; Chemotherapy drugs used to treat cancer; Diabetes; Inflammatory bowel disease (Crohn's disease and ulcerative colitis); Use of medications such as prednisone; Weakened immune system (such as from HIV/AIDS)

Clinical features

- Classic locations - perianal (60%), ischiorectal (20%), intersphincteric (5%), supralevator (4%), and submucosal (1%)
- Clinical presentation correlates with the anatomic location of the abscess

	Abscess	Description
1.	Perianal abscess	• Dull perianal discomfort (exacerbated by movement and increased perineal pressure from sitting or defecation) and pruritus • O/E - a small, erythematous, well-defined, fluctuant, subcutaneous mass near the anal orifice
2.	Ischiorectal abscess	• Systemic fevers, chills, and severe perirectal pain • Fullness consistent with the more advanced nature of this process • External signs are minimal and may include erythema, induration, or fluctuancy • Digital rectal examination (DRE), a fluctuant, indurated mass may be encountered • Optimal physical assessment of an ischiorectal abscess may require anaesthesia to alleviate patient discomfort that would otherwise limit the extent of the examination
3.	Intersphincteric abscess	• Rectal pain; Exhibit localized tenderness on DRE • Physical examination may fail to identify an intersphincteric abscess
4.	Supralevator abscesses	• Present a similar diagnostic challenge • As a result, clinical suspicion of an intersphincteric or supralevator abscess may require confirmation through computed tomography (CT) scanning, magnetic resonance imaging (MRI), or anal ultrasonography. Use of the last modality is limited to confirming the presence of an intersphincteric abscess.

Investigations
1. Pelvic CT scan,
2. MRI or trans-rectal ultrasound

Differential Diagnosis
- Tuberculosis; Squamous cell carcinoma; Adenocarcinoma; Actinomycosis; lymphogranuloma venereum; Crohn's disease; Trauma; Leukaemia; Lymphoma

Complications
- Anal fistula; Body-wide infection (sepsis); Fibrosis/scarring; Stricture; Anal incontinence; Recurrence

7.0 HAEMORRHOIDS

See CVS unit

See: Cardiovascular System Pathology

8.0 RECTAL PROLAPSE

About Rectal prolapse
1) What are the causes?
2) What are the predisposing factors?
3) Explain the pathology and clinical features

8.1. Neoplasms

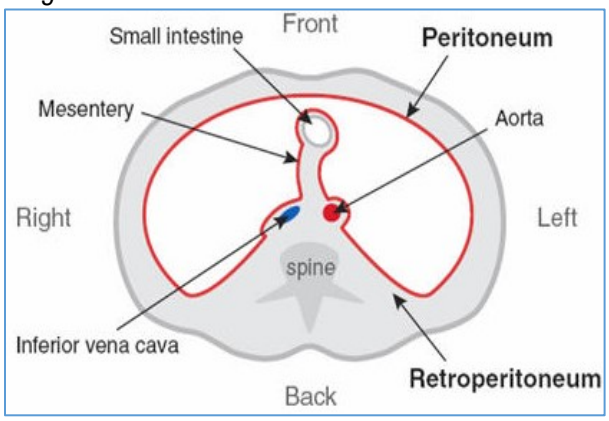
About Neoplasms of the rectum
1) What are the predisposing factors?
2) Explain the pathology and clinical features

Lesson 11: THE PERITONEUM

1.0 NORMAL STRUCTURE

- Peritoneum is the thin serous membrane that lines the walls of the abdominal and pelvic cavities and covers the abdominal and pelvic viscera
- Largest serous membrane of human body and has a rather complex arrangement
- Possesses a certain degree of mobility on the extra peritoneal fat and can be stretched to certain degree without tearing
- Composed of layer of mesothelium supported by a thin layer of connective tissue
- Has two parts
  i) Parietal peritoneum (portion that lines the abdominal and pelvic cavities)
  ii) Visceral peritoneum (covers the external surfaces of most abdominal organs, including the intestinal tract).

Diagram 11.1: Structure of the Peritoneum



Extra peritoneal tissue

- Connective tissue layer between the parietal peritoneum and the fascia lining of the abdominal and pelvic walls

Peritoneal Cavity

- Potential space between the parietal peritoneum and visceral peritoneum
- Largest cavity of human body with an enormous surface area
- Contains a small amount of serous fluid, but is otherwise empty
- Fluid lubricates the visceral peritoneum and allows the mobile viscera to glide freely on the abdominal wall and each other within the limits dictated by their attachments.
- Entire peritoneal cavity can be divided into two parts
  i) Greater Sac - main compartment, extends from the diaphragm down into the pelvis
  ii) Lesser Sac – smaller compartment that lies behind the stomach. It is in free communication with the greater sac through an oval window called the opening of the lesser sac

Abdominal structures in relation to peritoneum

i) Intraperitoneal organs
   - Stomach, first part of duodenum, jejunum, ileum, cecum, appendix, transverse colon, sigmoid colon, upper 1/3 of rectum, liver, spleen, uterus (females), Fallopian tubes (Females), ovaries (Females)

ii) Retroperitoneal Organs
- Lie behind the peritoneum and are only partially covered with visceral peritoneum
- Include second and third parts of duodenum, ascending colon, descending colon, middle 1/3 of Rectum, pancreas, kidneys, adrenal glands, proximal ureters, renal vessels, gonadal blood vessels, inferior vena cave and aorta

iii) Infraperitoneal organs
- Lie inferior to the peritoneum in the pelvis and include the lower 1/3 of rectum, urinary bladder and distal ureters

Peritoneal ligaments, omenta and mesenteries

i) Ligaments - falciform, coronary ligament, and Rt and Lt triangular ligaments
ii) Omenta
   a. Greater omentum - connects greater curvature of stomach to transverse colon
   b. Lesser omentum - suspends the lesser curvature of the stomach from the fissure of the ligamentum venosum (fibrous remnant of the ductus venosus of foetal circulation) and porta hepatis
   c. Gastrosplenic omentum - connects the stomach to the hilum of spleen
iii) Mesenteries - are two layered folds of peritoneum, which connect the parts of the intestine to the posterior abdominal wall

Peritoneal pouches, recesses, and gutters

- Peritoneum is a highly-folded membrane resulting in formation of lots of pouches, recesses and gutters
- Some of the important of them are listed below:
  i) Pouches - lesser sac and greater sac
  ii) Recesses - duodenal recesses, ceacal recesses, intersigmoid recesses
  iii) Spaces - subphrenic spaces
  iv) Gutters - paracolic gutters

2.0 FUNCTIONS OF PERITONEUM

1) Ensures that the mobile viscera glide easily on one another
2) Seals off intraperitoneal infections localizing them
3) Suspend various organs within the abdominal cavity
4) Means of conveying blood vessels, lymphatics, and nerves to these organs

3.0 DISORDERS OF THE PERITONEUM

3.1. ASCITES

Introduction

- Ascites is the accumulation of excessive volume of fluid within the peritoneal cavity (*Askitis is Greek word meaning fluid filled bag*)
- Occurs mainly due to a combination of portal hypertension and hepatocellular failure
- Usually associated with haemodilution, oedema and decreased urine output
- Presence of neutrophils suggests secondary infection and RBCs in ascitic fluid points to disseminated intra-abdominal cancer

Causes

1) Venous hypertension (congestion) –e.g.
   - portal hypertension, cirrhosis, congestive cardiac failure (CCF), Constrictive pericarditis, Hepatic venous outflow, obstruction (Budd-Chiarri syndrome/Veno-occlusive and portal vein block
2) Hypoalbuminaemia – Liver disease, Nephrotic syndrome, malnutrition, protein losing enteropathy
3) Malignant disease - secondary carcinomas, Lymphomas and leukaemia
4) Infections - TB peritonitis; fungal – candida, Cryptococcus; parasitic – strongyloides and entamoeba
5) Miscellaneous - Pancreatitis, Meig's syndrome (ovarian tumour), Myxoedema, Systemic lupus erythromatosus

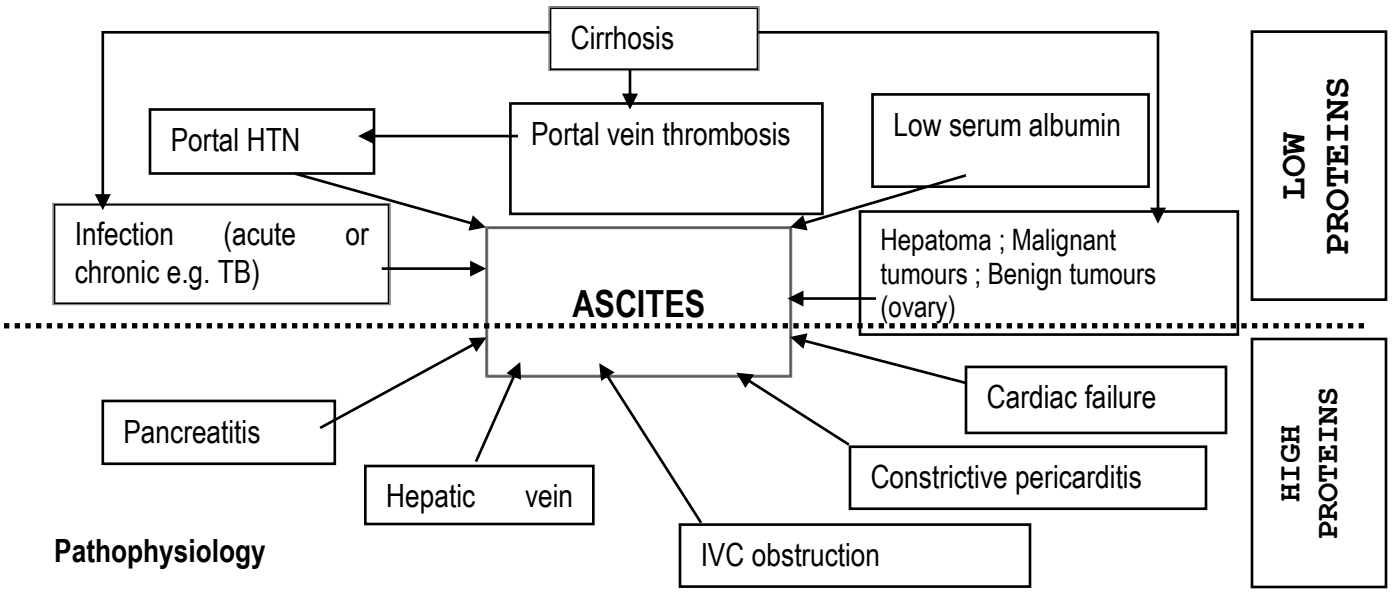
Classification

Class	Description	Causes
Transudate ascites	• Ascites protein concentration of < 25g/L • Serum ascites albumin gradient - >1.1	Cirrhosis, portal hypertension, IVC or portal vein thrombosis, portal nodes, RHF, hypoproteinaemia (nephrotic syndrome, PLE, malnutrition), myxoedema, CCF pericarditis
Exudative ascites	• Ascites protein concentration - > 25g/L • Serum ascites albumin gradient - < 1.1	Malignancy (any intra-abdominal organ e.g. colon, stomach, pancreas, liver, kidney), infections especially TB, pancreatitis, Budd-Chiari syndrome and hypothyroidism

Pathogenesis

i) Under filling theory - compensatory renal retention of sodium and water secondary to splanchnic vasodilatation.
ii) Overflow theory - primary retention of sodium and water

Diagram 10.2: Pathophysiology of Ascites



Pathophysiology

i) Sinusoidal Hypertension

- Sinusoidal hypertension e.g. portal hypertension alters Starling's forces which drives the fluid into the space of Disse

- Local HP exerted and there is increased hepatic and splanchnic production of lymph and transudation into peritoneal cavity
- Fluid not adequately removed by hepatic lymphatics thus accumulates in the cavity

ii) Hypoalbuminaemia

- Reduced OP favours fluid exudation into the cavities and allows exudation of fluid into the peritoneum across the osmotic gradient established.

iii) Percolation of Fluid

- Percolation of hepatic lymph into the peritoneal cavity occurs when normal flow of 800 – 1000 mls/day is altered as seen in cirrhosis (20L/day) or there is distortion and obstruction of hepatic sinusoids
- Hepatic lymph is rich in proteins.

iv) Renal Sodium/water retention

- Sodium is retained due to impaired renal functional renal (inability to excrete sodium)
- Reduced effective systemic blood volume due to splanchnic pooling of blood triggers mechanisms that enhance retention of fluid in the body.
- Ischemia activates the RAA system that facilitates retention of fluid in the body.
- Renal sympathetic system increases proximal convoluted tubules reabsorption of sodium and fluids.
- Increased plasma norepinephrine
- Increased ADH (AVP) due to failure of inhibition of vasopressin by prostaglandin E (from the collecting ducts).

Signs

- When ascites fluid is < 1L abdominal distension and fullness in the flanks with distortion or eversion of the umbilicus
- Hernia
- Abdominal striae
- Divarication/diastasis of the recti (separation of rectus abdominis muscles away from the midline)
- Meralgia paraethetica – tingling, formication, itching and other forms of paraesthesia in the outer side of the lower part of the thigh in the area of distribution of the femoral cutaneous nerve
- Scrotal oedema
- Right sided pleural effusion (10%)

Methods of Detection of Ascites

1. Ultrasound
2. CAT
3. Diagnostic aspiration
4. Physical examination
    a. Abdominal distension – bulging flanks that are dull to percussion with the umbilical region hyper-resonant (due to floating bowel)
    b. Fluid thrill
    c. Shifting dullness
    d. Positive Puddle sign

Staging of Ascites

Stage I Ascites demonstrable by ultrasonography
Stage II Ascites demonstrable by a fluid wave (fluid thrill)
Stage III Marked distension, spider nevi, caput medusae and emaciation
Stage IV Tense, painful distension with marked wasting

OR
Stage 1 Fluid < 500 ml of ascites fluid, splashy, floppy abdomen with fullness in flanks
Stage 2 Fluid > 500 - 5000 ml, "U" shaped, dull percussion note and shifting dullness
Stage 3 Fluid more than 5000 ml, tense abdomen with a fluid thrill

Investigations
1. Ascitic tap (paracentesis – for microscopy, protein level); Ascitic neutrophils > 250/mm^3 indicates spontaneous bacterial peritonitis
2. Abdominal U/S
3. Laparoscopy
4. Liver function tests
5. Abdominal x-rays
6. CT scan

3.2. PERITONITIS

Definition and Introduction

- Peritonitis is the inflammation of the serosal membrane that lines the abdominal cavity and the organs contained therein
- Peritoneal infections are classified as
  - Primary (i.e., from haematogenous dissemination,
  - Secondary (i.e., related to a pathologic process in a visceral organ, such as perforation or trauma, including iatrogenic trauma)
  - Tertiary (i.e., persistent or recurrent infection after adequate initial therapy)
- Primary peritonitis is most often spontaneous bacterial peritonitis (SBP) caused by chronic liver disease
- Infections in the peritoneum are further divided into generalized (peritonitis) and localized (intra-abdominal abscess)

Risk Factors
- Peritoneal dialysis
- Medical conditions - cirrhosis, appendicitis, Crohn's disease, stomach ulcers, diverticulitis and pancreatitis.

Causes
- Depends on the type, as well as location, of peritonitis
  1) Primary peritonitis - most common pathogens include Gram-negative (*E coli*, *K pneumoniae*, *Pseudomonas* spp, *Proteus* spp, and Gram-positive (*Streptococcus pneumoniae*, *Strep* spp; *Staphylococcus* sp
  2) Secondary peritonitis - cirrhosis, pancreatitis, trauma, peptic ulcer
  3) Tertiary peritonitis - immunosuppression

4) Chemical peritonitis - may be caused by irritants such as bile, blood, barium, or other substances or by transmural inflammation of visceral organs (e.g., Crohn's disease) without bacterial inoculation of the peritoneal cavity
5) Peritoneal abscess

Pathophysiology

- Infection, intra-abdominal sepsis, spillage of the contents trigger an inflammatory process
- Endotoxins produced by gram-negative bacteria lead to the release of cytokines that induce cellular and humoral cascades, resulting in cellular damage, septic shock, and multiple organ dysfunction syndrome (MODS)
- Peritonitis causes fluid to shift into the peritoneal cavity and bowel leading to severe dehydration and electrolyte imbalance
- Adult respiratory distress syndrome can develop rapidly
- Renal failure, liver failure and DIC follow

Pathology

Macroscopy	• Dull appearance • Initially scarce serous fluid or slightly turbid fluid later on, the exudate becomes creamy and evidently suppurative • May be spread to the whole peritoneum, or be walled off by the omentum and viscera
Microscopy	• Infiltration by neutrophils with fibrino-purulent exudation

Features

- Poor appetite and nausea
- Acute dull abdominal pain (that quickly turns into persistent, severe abdominal pain, worsened by any movement), tenderness, and guarding, which are exacerbated by moving the peritoneum, e.g., coughing (forced cough may be used as a test), flexing one's hips, or eliciting the Blumberg sign (rebound tenderness)
- Chills and fever
- Fluid in the abdomen
- Extreme thirst
- Not passing any urine, or passing significantly less urine than usual
- Difficulty passing gas or having a bowel movement
- Vomiting
- Sinus tachycardia
- Development of ileus paralyticus (i.e., intestinal paralysis)

Investigations

1) Urinalysis
2) Imaging studies such as X-rays and computerized tomography (CT) scans
3) Exploratory surgery
4) Paracentesis
5) Total blood counts

Complications

1. The complications of spontaneous peritonitis - hepatic encephalopathy; Hepato-renal syndrome; Sepsis
2. Complications of secondary peritonitis an abscess; Gangrenous bowel; Intraperitoneal adhesions; Septic shock

3.3 PERITONEAL CANCER

- Peritoneal cancer is a rare cancer
- It develops in peritoneal cavity

Risks of Peritoneal Cancer

- Primary peritoneal cancer is more common in women than in men.
- BRCA1 and BRCA2 genetic mutations.
- Older age

Pathophysiology

- Occurs through
  i) Dissemination from the primary tumor
  ii) Primary tumor of peritoneum
  iii) Independent origins of the primary tumor and peritoneal implants

Peritoneal Cancer Symptoms

- Abdominal discomfort or pain from gas, indigestion, pressure, swelling, bloating, or cramps
- Feeling of fullness, even after a light meal, nausea or diarrhea, constipation, frequent urination
- Loss of appetite
- Unexplained weight gain or loss
- Abnormal vaginal bleeding
- Rectal bleeding
- Shortness of breath

Investigation

1) Ultrasound - high-frequency sound waves produce a sonogram
2) CA-125 blood test
   - Measures levels of a chemical in the blood called CA-125
   - If levels are high, peritoneal or ovarian cancer is more likely present (can be high for other reasons)

Identify 10 common infections of the GIT. State the causes, pathophysiology, clinical features and investigations