Lec 5
Gastrointestinal diseases

Diseases of Esophagus ACHALASIA (CARDIOSPASM) Achalasia of the oesophagus is a neuromuscular dysfunction due to which the cardiac sphincter fails to relax during swallowing and results in progressive dysphagia and dilatation of the oesophagus (mega-oesophagus).

ETIOLOGY There is loss of intramural neurons in the wall of the oesophagus. Most cases are of primary idiopathic achalasia i.e. congenital.

Secondary achalasia may occur from some other causes which includes: Chagas' disease (an epidemic parasitosis with Trypanosoma cruzi).

MORPHOLOGIC FEATURES There is dilatation above the short contracted terminal segment of the oesophagus.

HIATUS HERNIA Hiatus hernia is the herniation or protrusion of part of the stomach through the oesophageal hiatus of the diaphragm. Oesophageal hiatal hernia is the cause of diaphragmatic hernia in 98% of cases. In symptomatic cases, especially the elderly women, the clinical features are heartburn (retrosternal burning sensation) and regurgitation of gastric juice into the mouth, both of which are worsened due to heavy work, lifting weights and excessive bending. MORPHOLOGIC FEATURES There are 3 patterns in hiatus hernia: i) Sliding or oesophago-gastric hernia is the most common, occurring in 85% of cases. ii) Rolling or para-oesophageal hernia is seen in 10% of cases. This is a true hernia in which cardiac end of the stomach rolls up para-oesophageally. iii) Mixed or transitional hernia constitutes the remaining 5% cases in which there is combination of sliding and rolling hiatus hernia.

REFLUX (PEPTIC) OESOPHAGITIS Reflux of the gastric juice is the commonest cause of oesophagitis.

PATHOGENESIS Gastro-oesophageal reflux, to an extent, may occur in normal healthy individuals after meals and in early pregnancy.

In some clinical conditions, the gastro-oesophageal reflux is excessive e.g.

i) Sliding hiatus hernia

ii) Chronic gastric and duodenal ulcers

iii) Nasogastric intubation

iv) Persistent vomiting

v) Neuropathy in alcoholics, diabetics .

Endoscopically, the demarcation between normal squamous and columnar epithelium at the junctional mucosa is lost.

BARRETT'S OESOPHAGUS This is a condition in which, following reflux esophagitis, stratified squamous epithelium of the lower esophagus is replaced by columnar epithelium (columnar metaplasia). Barrett's esophagus is a premalignant condition.

CARCINOMA OF OESOPHAGUS Carcinoma of the esophagus is diagnosed late, after symptomatic esophageal obstruction (dysphagia) has developed and the tumor has transgressed the anatomical limits of the organ. The tumor occurs more commonly in men over 50 years of age.

ETIOLOGY Following conditions and factors are implicated.

1. Diet and personal habits:

i) Heavy smoking

ii) Alcohol consumption

iii) Intake of foods contaminated with fungus

iv) Nutritional deficiency of vitamins and trace elements.

2. Oesophageal disorders:

i) Oesophagitis (especially Barrett's oesophagus in adenocarcinoma)

ii) Achalasia

iii) Hiatus hernia

iv) Diverticula .

3. Other factors:

i) Race

ii) Family history

iii) Genetic factors—predisposition with coeliac disease

MORPHOLOGIC FEATURES

Carcinoma of the oesophagus is mainly of 2 types— squamous cell (epidermoid) and adenocarcinoma

Diseases of Stomach

PYLORIC STENOSIS Hypertrophy and narrowing of the pyloric lumen occurs predominantly in male children as a congenital defect (infantile pyloric stenosis). The adult form is rarely seen, either as a result of late manifestation of mild congenital anomaly or may be acquired type due to inflammatory fibrosis or invasion by tumours. G/A & M/E There is hypertrophy as well as hyperplasia of the circular layer of muscularis in the pyloric sphincter accompanied by mild degree of fibrosis.

CLINICAL FEATURES Following clinical features may be present.

1. Vomiting, which may be projectile and occasionally contains bile or blood.
2. Visible peristalsis
3. Palpable lump
4. Constipation.
5. Loss of weight.

ACUTE PEPTIC (STRESS) ULCERS Acute peptic ulcers or stress ulcers are multiple, small mucosal erosions, seen most commonly in the stomach but occasionally involving the duodenum.

ETIOLOGY Following factors are implicated , Physiological stress as in the following:

i) Shock

ii) Severe trauma

iii) Septicaemia

vi) Drug intake (e.g. aspirin and steroids).

vii) Local irritants (e.g. alcohol, smoking, coffee etc).

G/A Acute stress ulcers are multiple (more than three ulcers in 75% of cases). They are more common anywhere in the stomach, followed in decreasing frequency by occurrence in the first part of duodenum. They may be oval or circular in shape, usually less than 1 cm in diameter.

COMPLICATIONS Acute and subacute peptic ulcers usually heal without leaving any visible scar. However, healing of chronic, larger and deeper ulcers may result in complications.

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1. Obstruction
2. Haemorrhage
3. Perforation
4. Malignant transformation

GASTRIC CARCINOMA INCIDENCE Carcinoma of the stomach comprises more than 90% of all gastric malignancies and is the leading cause of cancer-related deaths in countries where its incidence is high.

The highest incidence is between 4th to 6th decades of life.

ETIOLOGY Following factors are implicated:

1. H. pylori infection H. pylori infection of the stomach is an important risk factor for the development of gastric cancer.

Epidemiologic studies throughout world have shown that a seropositivity with H. pylori is associated with 3 to 6 times higher risk of development of gastric cancer. It may be mentioned here that similar association of H. pylori infection exists with gastric lymphomas (MALT type) as well.

2. Dietary factors Epidemiological studies suggest that dietary Factors are most significant in the etiology of gastric cancer. The evidences in support of this are multifold:

i) Occurrence of gastric cancer in the region of gastric canal (i.e. along the lesser curvature and the pyloric antrum) where irritating foods exert their maximum effect.

ii) Populations consuming certain foodstuffs have high risk of developing gastric cancer e.g. ingestion of smoked foods.
iii) Tobacco smoke and consumption of alcohol.
3. Geographical factors The higher incidence in certain geographic regions is the result of environmental influences.
4. Genetic factors Genetic influences have some role in the etiology of gastric cancer. Not more than 4% of patients of gastric cancer have a family history of this disease. Individuals with blood group A have higher tendency to develop gastric cancer.

5. Pre-malignant changes in the gastric mucosa These are:
i) Hypo- or achlorhydria in atrophic gastritis of gastric mucosa .
ii) Adenomatous (neoplastic) polyps of the stomach.
iii) Chronic gastric ulcer (ulcer-cancer).
iv) Stump carcinoma in patients who have undergone partial gastrectomy.

MORPHOLOGIC FEATURES Gastric carcinoma is most commonly located in the region of gastric canal (prepyloric region) formed by lesser curvature, pylorus and antrum. Other less common locations are the body, cardia and fundus. Pathogenetically, a sequential evolution of all gastric carcinomas from an initial stage of in situ carcinoma confined to mucosal layers called early gastric carcinoma (EGC) has been found. Accordingly, gastric carcinomas are broadly classified into 2 main groups:

I.Early gastric carcinoma (EGC).
II. Advanced gastric carcinoma, which has 5 further major gross subtypes:
i) Ulcerative carcinoma ii) Fungating (Polypoid) carcinoma
iii) Scirrhous carcinoma (Linitis plastica)
iv)Colloid (Mucoid) carcinoma v) Ulcer-cancer

Diseases of small/ large Bowel

INTESTINAL OBSTRUCTION

The causes of intestinal obstruction can be classified under the following

3 broad groups:

1. Mechanical obstruction

i) Internal obstruction (intramural and intraluminal):

a) Inflammatory strictures

(e.g. Crohn's disease)

b) Congenital stenosis, atresia

f) Gallstones, faecoliths, foreign bodies

c) Tumours

ii) External compression: a) Peritoneal adhesions b) Strangulated hernias

c) Intussusception d) Volvulus e) Intra-abdominal tumour.

2. Neurogenic obstruction It occurs due to paralytic ileus i.e. paralysis of muscularis of the intestine as a result of shock after abdominal operation or by acute peritonitis.
3. Vascular obstruction Obstruction of the superior mesenteric Artery or its branches may result in infarction causing paralysis
Out of the various causes listed above, conditions producing external compression on the bowel wall are the most common causes of intestinal obstruction (80%)

ISCHAEMIC BOWEL DISEASE (ISCHAEMIC ENTEROCOLITIS) Ischaemic lesions of the gastrointestinal tract may occur in the small intestine and/or colon; the latter is called ischaemic colitis or ischaemic enterocolitis and is commonly referred to as ischaemic bowel disease.

In either case, the cause of ischaemia is compromised mesenteric circulation. Depending upon the extent and severity of ischaemia, 3 patterns of pathologic lesions can occur:

1. Transmural infarction, characterised by full thickness involvement .
2 Mural infarction, characterised by haemorrhagic gastroenteropathy .
3. Ischaemic colitis, due to chronic colonic ischaemia causing fibrotic narrowing .

INFLAMMATORY BOWEL DISEASE (CROHN'S DISEASE AND ULCERATIVE COLITIS)

DEFINITION The term 'inflammatory bowel disease (IBD)' is commonly used to include 2 idiopathic bowel diseases having many similarities but the conditions usually have distinctive morphological appearance:

1. Crohn's disease or Regional enteritis is an idiopathic Chronic ulcerative IBD, characterised by transmural, non-caseating granulomatous inflammation, affecting most commonly the segment of terminal ileum and/or colon, though any part of the gastrointestinal tract may be involved.
2. Ulcerative colitis is an idiopathic form of acute and chronic ulceroinflammatory colitis affecting chiefly the mucosa and submucosa of the rectum and descending colon, though sometimes it may involve the entire length of the large bowel.

Both these disorders primarily affect the bowel but may have systemic involvement in the form of polyarthritis, uveitis, ankylosing spondylitis, skin lesions and hepatic involvement.

COMPLICATIONS

Crohn's disease:

1. Malabsorption 2. Fistula formation 3. Stricture formation 4. Development of malignancy lymphoma may develop more often in Crohn's disease than adenocarcinoma.

Ulcerative colitis:

1. Toxic megacolon (Fulminant colitis). 2. Perianal fistula formation may occur rarely. 3. Carcinoma may develop in long-standing cases of ulcerative colitis of more than 10 years duration. 4. Stricture formation almost never occurs in ulcerative colitis.

COELIAC SPRUE (NON-TROPICAL SPRUE, GLUTEN-SENSITIVE ENTEROPATHY, IDIOPATHIC STEATORRHOEA) This is the most important cause of primary malabsorption occurring in temperate climates. The condition is characterised by significant loss of villi in the small intestine and therefore diminished absorptive surface area. The condition occurs in 2 forms: Childhood form, seen in infants and children and is commonly referred to as coeliac disease. Adult form, seen in adolescents and early adult life and used to be called idiopathic steatorrhoea. In either case, there is genetic abnormality resulting in sensitivity to gluten (a protein) and its derivative, gliadin, present in diets such as grains of wheat, barley and rye.

The symptoms are usually relieved on elimination of gluten from the diet. The role of heredity is further supported by the observation of familial incidence and HLA association of the disease. Following hypotheses have been proposed in causing mucosal cell damage:

1. Hypersensitivity reaction as seen by gluten-stimulated antibodies.
2. Toxic effect of gluten due to inherited enzyme deficiency in the mucosal cells.

M/E There are no differences in the pathological findings in children and adults. There is variable degree of flattening of the mucosa, particularly of the upper jejunum, and to some extent of the duodenum and ileum.

APPENDICITIS

Acute inflammation of the appendix, acute appendicitis, is the most common acute abdominal condition confronting the surgeon. The condition is seen more commonly in older children and young adults, and is uncommon at the extremes of age.

ETIOPATHOGENESIS Common causes are as under:

A.ObstructiveFaecolith

B. Non-obstructive: 1. Haematogenous spread of generalized infection

2. Vascular occlusion 3. Inappropriate diet lacking roughage.

M/E The most important diagnostic histological criterion is the neutrophilic infiltration of the muscularis. In early stage, other changes besides acute inflammatory changes, are congestion and oedema of the appendiceal wall. In later stages, the mucosa is sloughed off, the wall becomes necrotic, the blood vessels may get thrombosed and there may be neutrophilic abscesses in the wall.

HIRSCHSPRUNG'S DISEASE (CONGENITAL MEGACOLON)

The term 'megacolon' is used for any form of marked dilatation of the entire colon or its segment and may occur as a congenital or acquired disorder.

Congenital form characterised by congenital absence of ganglion cells in the bowel wall (enteric neurons) is called Hirschsprung's disease. Genetically, Hirschsprung's disease is a heterogeneous disorder as under: Autosomal dominant and Autosomal recessive inheritance .

Clinically, the condition manifests shortly after birth with constipation, gaseous distension and sometimes with acute intestinal obstruction. Its has familial tendency in about 4% of cases and has predilection for development in Down's syndrome.

MORPHOLOGIC FEATURES Two types of biopsies may be done on infants suspected of having Hirschsprung's disease—full-thickness rectal biopsy, and suction biopsy that includes mucosa and submucosa.

G/A Typical case of Hirschsprung's disease shows 2 segments—a distal narrow segment that is aganglionic and a dilated proximal segment that contains normal number of ganglion cells.

HAEMORRHOIDS (PILES)

Haemorrhoids or piles are varicosities of the haemorrhoidal veins. They are called internal piles if dilatation is of superior haemorrhoidal plexus covered over by mucous membrane, and external piles if they involve inferior haemorrhoidal plexus covered over by the skin. Their possible causes include the following: 1. Portal hypertension 2. Chronic constipation 3. Cardiac failure 4. Venous stasis of pregnancy 5. Hereditary predisposition 6. Tumours of the rectum.

COLORECTAL CARCINOMA

Colorectal cancer comprises 98% of all malignant tumours of the large intestine. The incidence of carcinoma of the large intestine rises with age; average age of patients is about 60 years. Cancer in the rectum is more common in males than females in the ratio of 2:1.

ETIOLOGY A few etiological factors have been implicated:

1. Geographic variations
2. Dietary factors Diet plays a significant part in the causation of colorectal cancer: i) A low intake of vegetable fibre-diet ii) Consumption of large amounts of fatty foods iii) Excessive consumption of refined carbohydrates
3. Adenoma-carcinoma sequence There is strong evidence to suggest that colonic adenocarcinoma evolves from pre-existing adenomas, referred to as adenoma-carcinoma sequence.
4. Hereditary non-polyposis colonic cancer.
5. Other factors Presence of certain pre-existing diseases and some other factors, e.g.: i) Inflammatory bowel disease (especially ulcerative colitis).

ii) Diverticular disease for long duration.

MORPHOLOGIC FEATURES Distribution of the primary colorectal cancer reveals that about 60% of the cases occur in the rectum, followed in descending order, by sigmoid and descending colon (25%), caecum and ileocaecal valve (10%); ascending colon, hepatic and splenic flexures (5%); and quite uncommonly in the transverse colon.

M/E The appearance of right and left-sided growths is similar. About 95% of colorectal carcinomas are adenocarcinomas of varying grades of differentiation, out of which approximately 10% are mucin-secreting colloid carcinomas.

SPREAD Carcinoma of the large intestine may spread by the following routes:

1. Direct spread The tumour spreads most commonly by direct extension in both ways—circumferentially into the bowel wall as well as directly into the depth of the bowel wall.
2. Lymphatic spread Spread via lymphatics occurs rather commonly and involves, firstly the regional lymph nodeS
3. Haematogenous spread Blood spread of large bowel cancer occurs relatively late and involves the liver, lungs, brain, bones and ovary

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CLINICAL FEATURES These appear after considerable time.
i) Occult bleeding (melaena) ii) Change in bowel habits, more often in left-sided growth iii) Loss of weight iv) Loss of appetite v) Anaemia, weakness, malaise.

The most common complications are obstruction and haemorrhage; less often perforation and secondary infection may occur. Aside from the diagnostic methods like stool test for occult blood, PR examination, proctoscopy, radiographic contrast studies and CT scan, recently the role of tumour markers has been emphasised.
Of particular importance is the estimation of carcinoembryonic antigen (CEA) level which is elevated in 100% cases of metastatic colorectal cancers. CEA levels are elevated in some nonneoplastic conditions also like in ulcerative colitis, pancreatitis and alcoholic cirrhosis.

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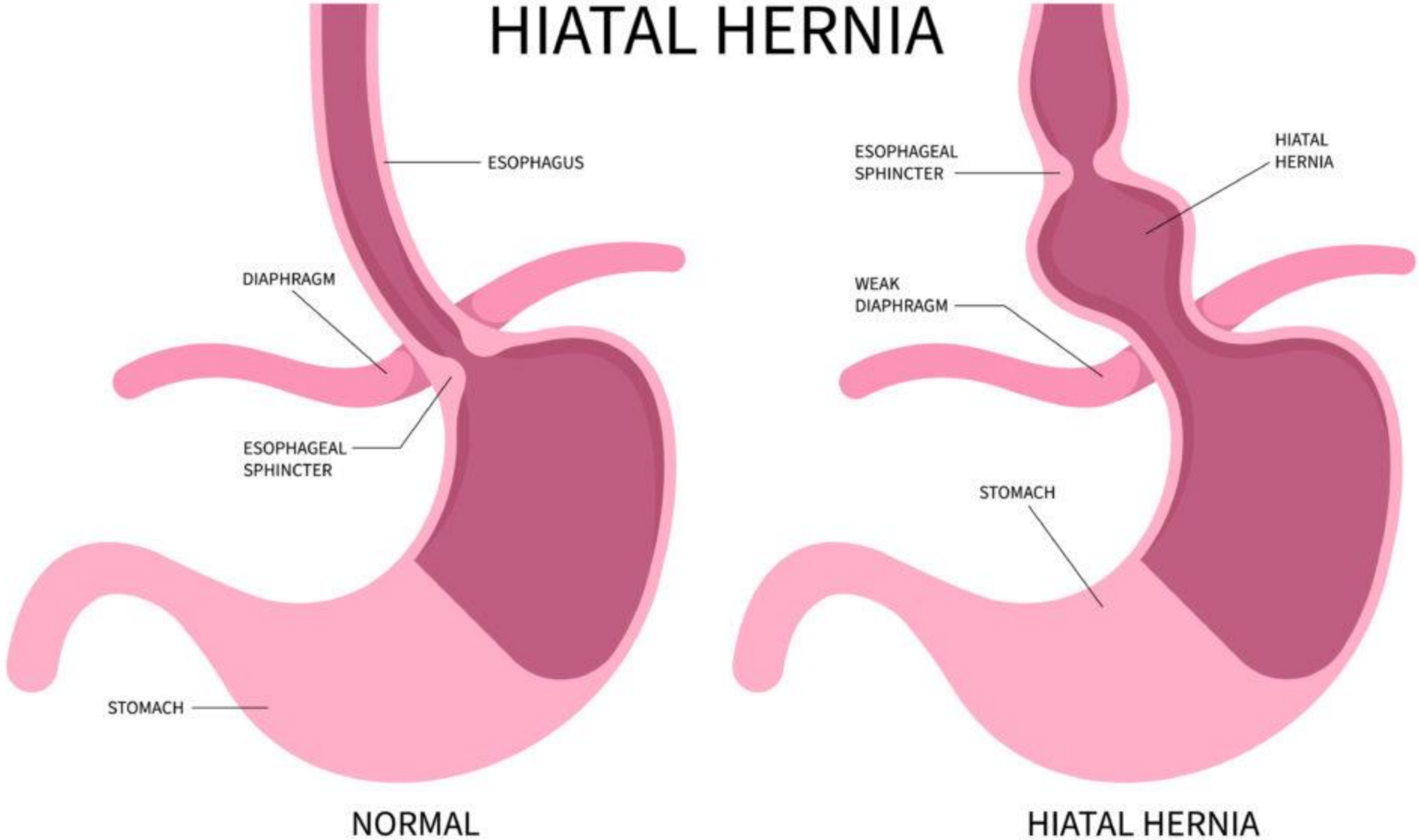
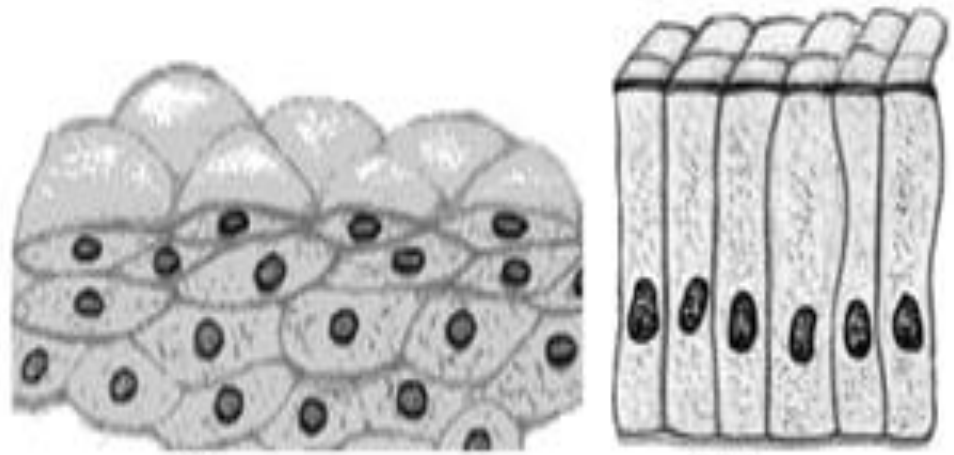
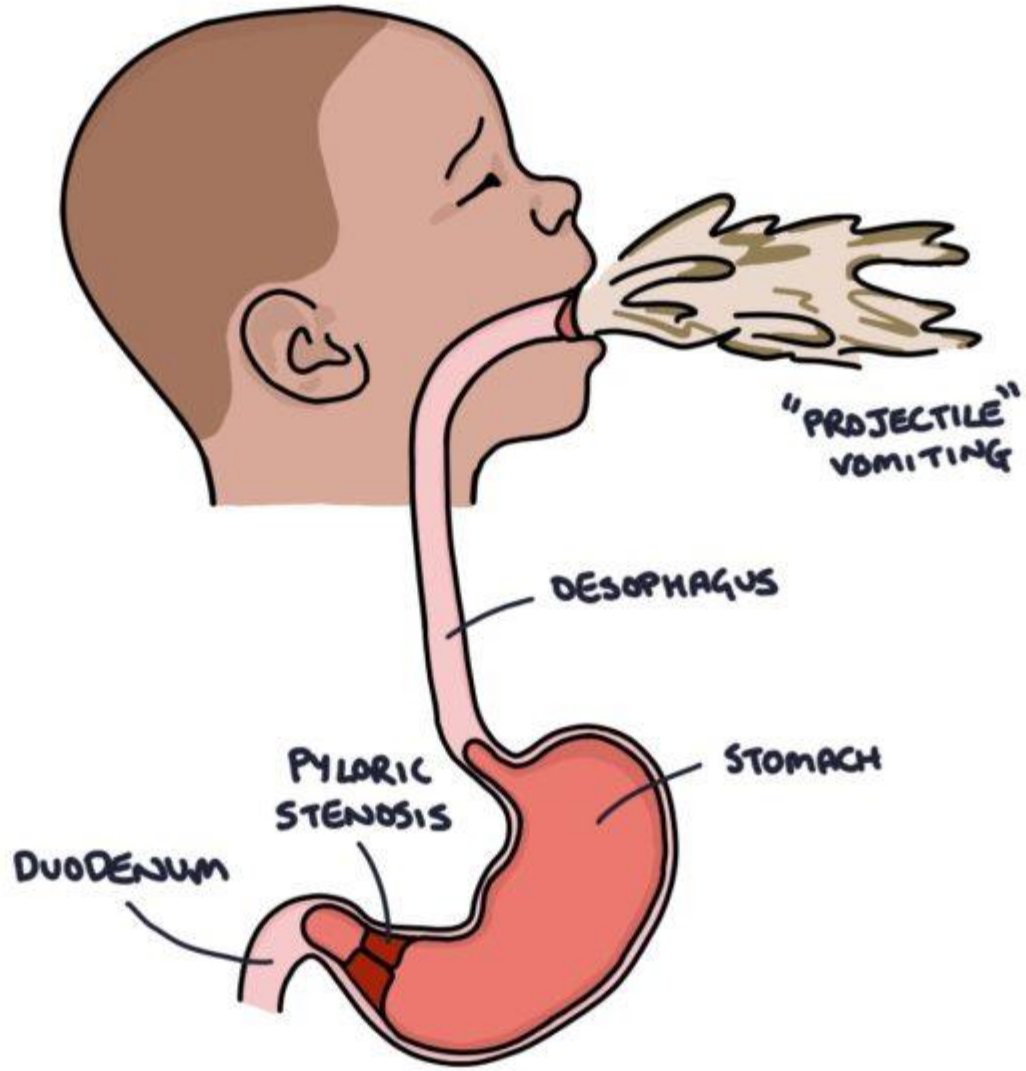
HIATAL HERNIA





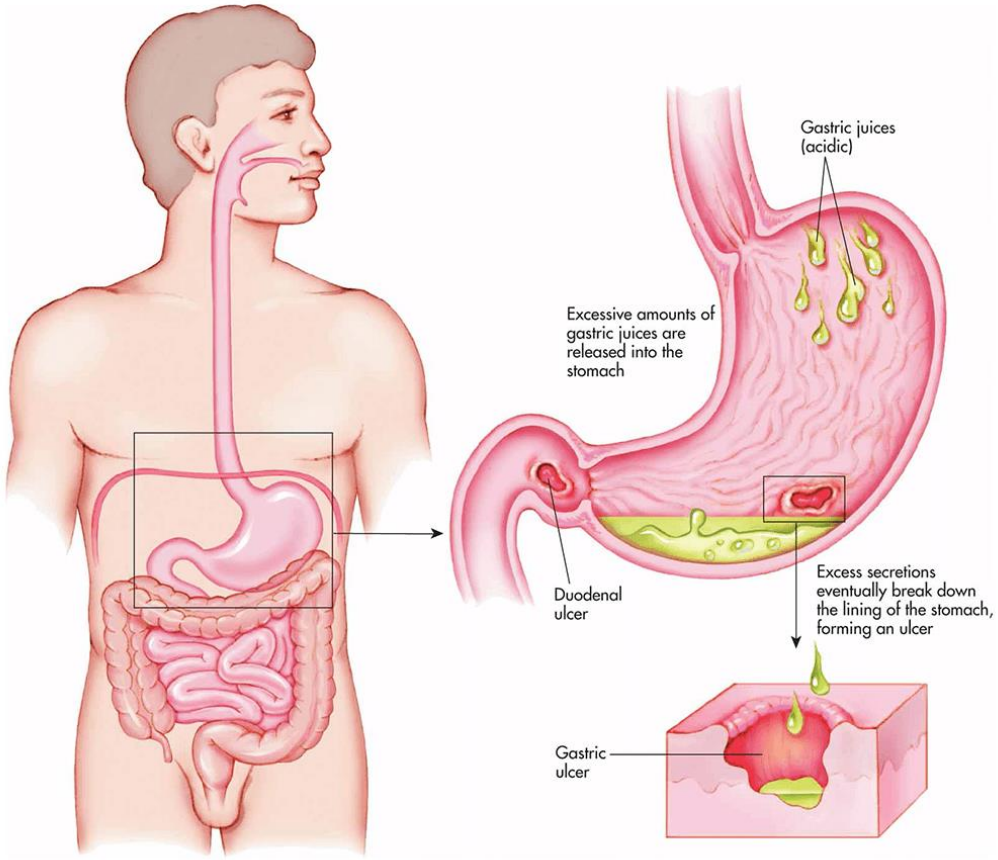
Squamous Epithelium

Columnar Epithelium



Picture of pyloric stenosis

Peptic Ulcer Disease



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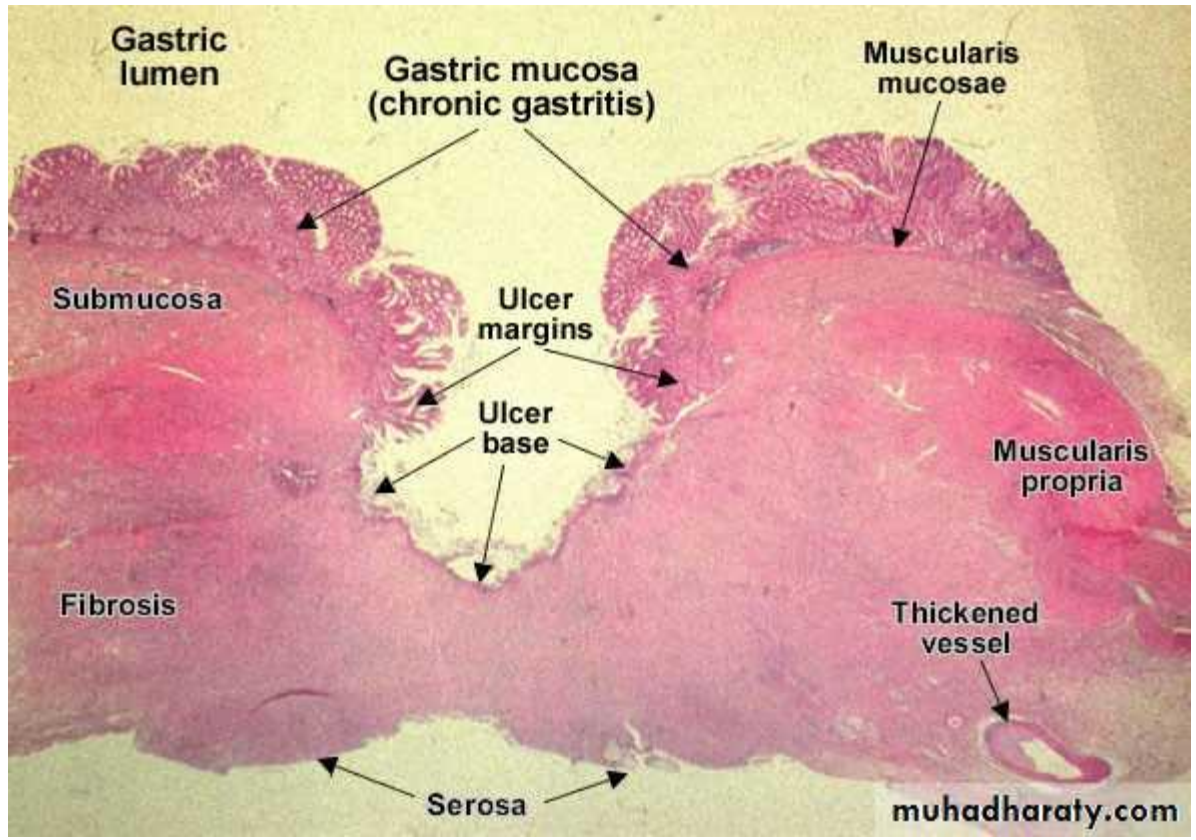
Gastric lumen
Gastric mucosa (chronic gastritis)
Muscularis mucosae
Submucosa
Ulcer margins
Ulcer base
Muscularis propria
Fibrosis
Thickened vessel
Serosa
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