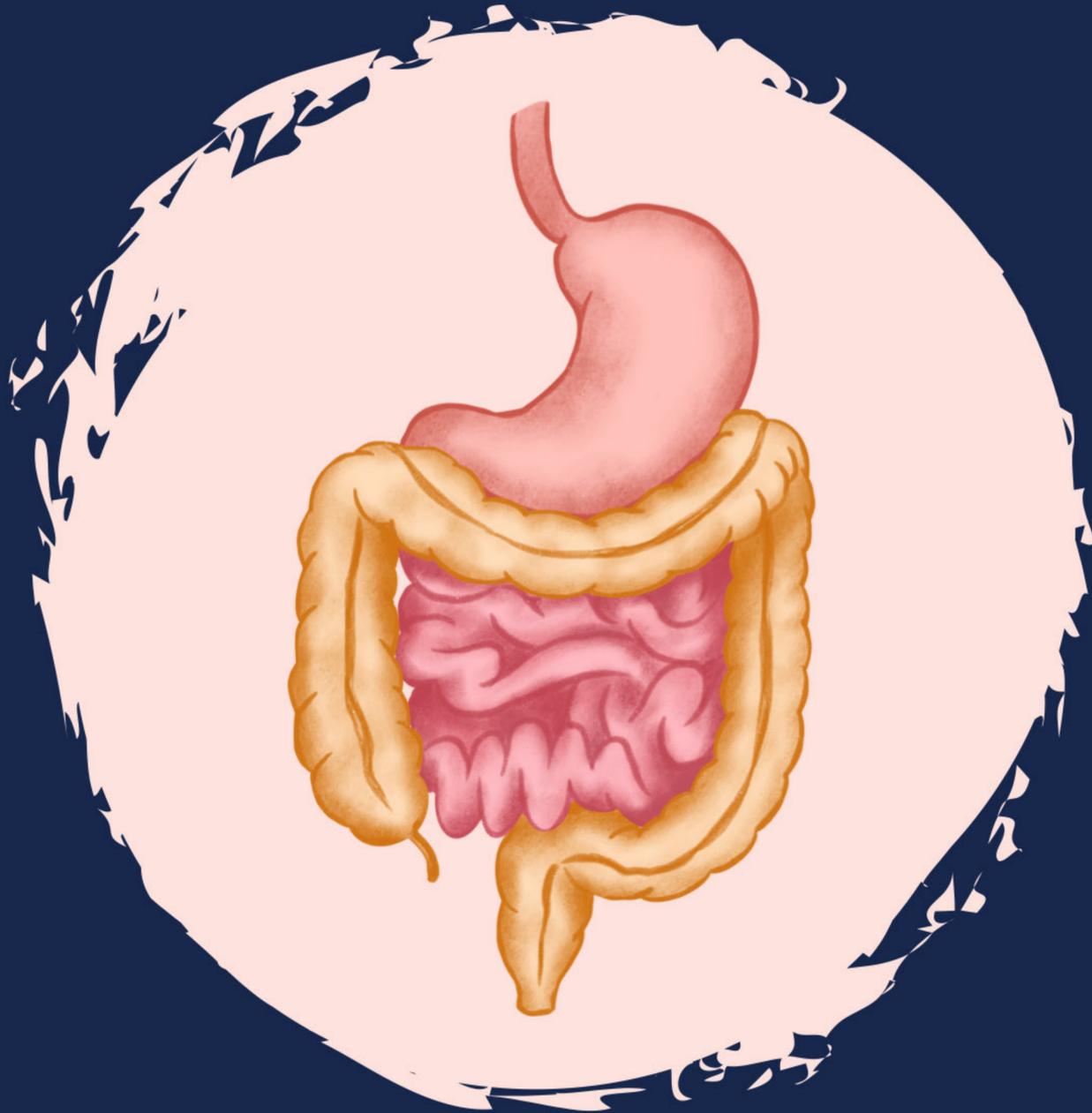


Level (3) - Semester (5)

PATHOLOGY

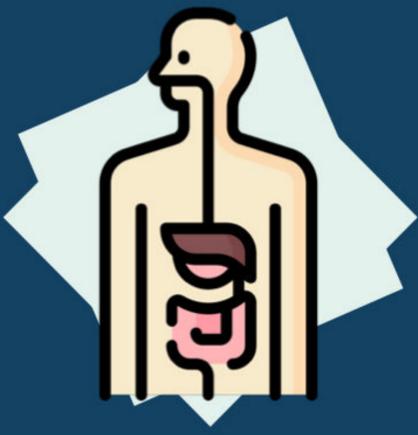


SCAN ME

GIT - Lecture (2)

Non-neoplastic diseases of
Esophagus & Stomach

DR M. YUSUF



NON-NEOPLASTIC DISEASES OF ESOPHAGUS & STOMACH

1) DISEASES OF ESOPHAGUS

ESOPHAGITIS

✓ Etiology:

Infective	Non infective
<ul style="list-style-type: none"> Herpes simplex Candidiasis 	<ul style="list-style-type: none"> Surface irritation. Reflux esophagitis Chemicals: Alcohol – Corrosives – Drugs – Smoking – Cytotoxic drugs – Radiation. Vit. A, C deficiency.

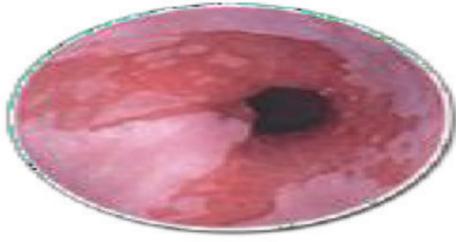
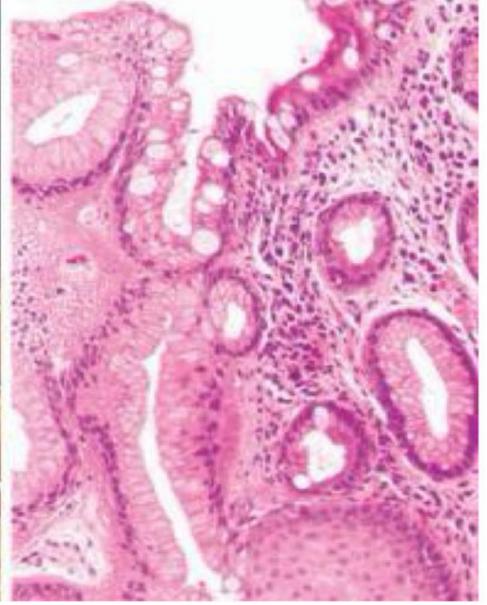
REFLUX ESOPHAGITIS

Causes	<ol style="list-style-type: none"> Incompetence of the lower esophageal sphincter. Hiatus hernia.
N/E	<ul style="list-style-type: none"> Hyperemic mucosa with superficial erosions.
M/E	<ul style="list-style-type: none"> Presence of inflammatory cells (eosinophils, neutrophils & lymphocytes) in the epithelial layer. Basal hyperplasia. Congested lamina propria
Complications	<ol style="list-style-type: none"> Barrett's esophagus (Next page) Hematemesis. Peptic ulceration. Fibrous stricture.
Figure	





BARRETT'S ESOPHAGUS

Pathology	<ul style="list-style-type: none"> Intestinal metaplasia within the esophageal squamous mucosa
Occur	<ul style="list-style-type: none"> In 10% of reflux esophagitis
Complications	<ul style="list-style-type: none"> Increased risk for development of esophageal adenocarcinoma
Figure	 <p style="text-align: center;">BARRETT'S ESOPHAGUS TISSUE</p>  

" "

ESOPHAGEAL ULCERS

- ① Traumatic ulcers: After instrumentation.
- ② Physical ulcers: After swallowing of caustic chemicals (KOH – NaOH).
- ③ Reflux esophagitis ulcers.
- ④ Peptic ulcer.
- ⑤ Tuberculous ulcers.
- ⑥ Syphilitic ulcers.
- ⑦ Malignant ulcer

" " "

ESOPHAGEAL OBSTRUCTION

☑ Cases:

Functional	<ul style="list-style-type: none"> Due to neuromuscular incoordination As in achalasia & diverticulum.
Organic	<ol style="list-style-type: none"> 1) Congenital: Esophageal atresia or Stricture. 2) Acquired: <ol style="list-style-type: none"> a. In the lumen: F.B, tumor. b. In the wall: Stricture (Congenital – Post inflammatory – Malignant) c. From outside: Goiter – Tumor – Aortic aneurysm.





IV

ACHALASIA OF CARDIA (CARDIOSPASM)

Definition	▪ Failure of relaxation or incomplete of the lower esophageal sphincter.
Etiology	▪ Idiopathic degenerative changes affecting the vagal branches that supply that part.
Effects	1) Dysphagia 2) Hypertrophy & dilatation of the esophagus. 3) Precancerous.
Figure	<p>The figure consists of two anatomical diagrams. The left diagram, labeled 'Normal', shows a cross-section of the esophagus and stomach. The esophagus is narrow, and the Lower Esophageal Sphincter (LES) is shown as a contracted ring at the junction with the stomach. The right diagram, labeled 'Achalasia Cardia', shows the esophagus significantly dilated (wider) and the LES as a thickened, contracted ring that does not relax, preventing food from passing into the stomach. Labels include 'Esophagus', 'Lower Esophageal Sphincter (LES)', and 'Stomach' in both diagrams.</p>

2) DISEASES OF STOMACH

I

GASTRITIS

Definition

- Gastric mucosal inflammatory process associated with mucosal injury.
- Could be **acute** OR **chronic**

PATHOGENESIS

 By one or more of the following:

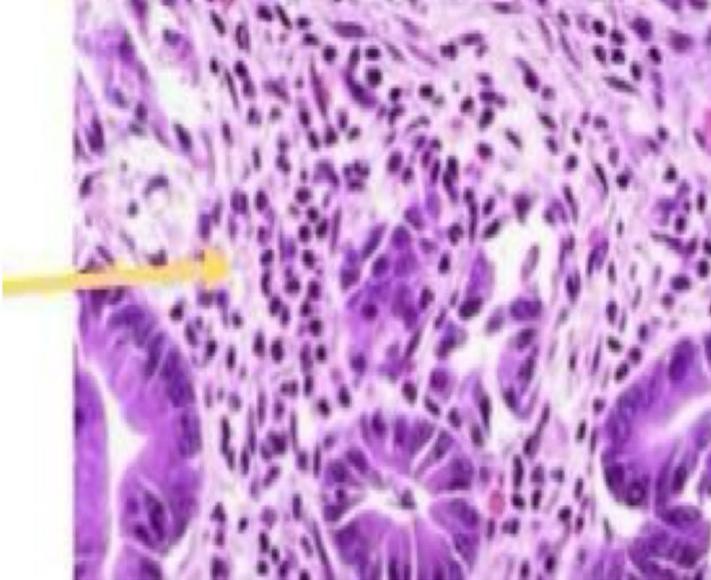
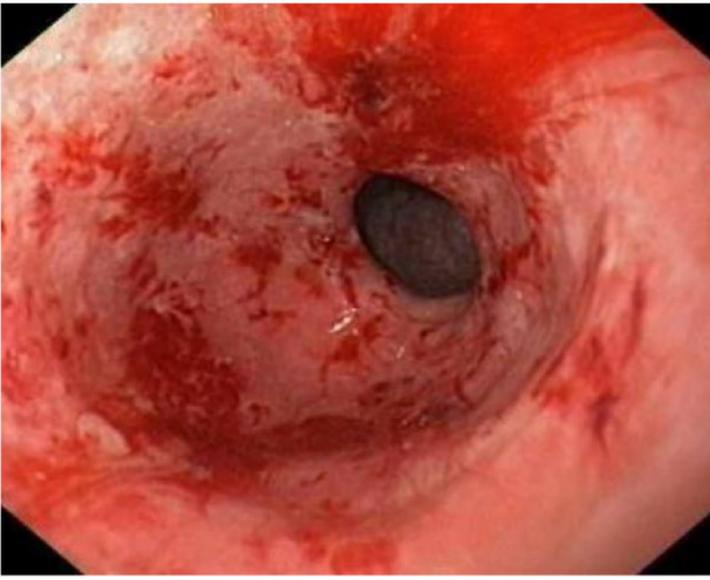
- 1) ↑↑ Acid secretion.
- 2) ↓↓ Blood flow.
- 3) ↓↓ The adherent mucous layer.
- 4) Direct damage to surface epithelium.
- 5) ↓↓ PGs secretion from the mucosa.





①

ACUTE GASTRITIS

Definition	<ul style="list-style-type: none"> Acute mucosal inflammatory process usually of sudden transient nature
Causes	<ol style="list-style-type: none"> 1) Drugs: NSAIDs - Cytotoxic chemotherapy - Cortisone. 2) Drinking: Alcohol - Coffee. 3) Irritant: Acids - Alkalies 4) Infection: As salmonella 5) Irradiation injury. 6) Shock: Due to decrease vascular perfusion. 7) Spicy food. 8) Smoking. 9) Severe stress.
N/E	<ul style="list-style-type: none"> The mucosa is: <ol style="list-style-type: none"> 1. Hyperemic 2. Edematous 3. Shows erosions (in severe cases)
M/E	<ul style="list-style-type: none"> The lamina propria shows: <ol style="list-style-type: none"> 1. Edema 2. Hyperemia 3. neutrophilic infiltration 4. Focal erosion (partial thickness defect) of surface epithelium in severe cases
Figure	<div style="display: flex; justify-content: space-around;">   </div>





②

CHRONIC GASTRITIS

Definition

- Continuous inflammation of gastric mucosa
- Associated with **fibrosis** which may lead to **gastric atrophy**.

ETIOLOGICAL TYPES

	Type A (Autoimmune gastritis)	Type B (Antral gastritis)
SITE	Affects the body 	Affects the antrum 
LEAD TO	Gland atrophy	---
PATHOGENESIS	▪ Auto antibodies against gastric parietal cells	---
ASSOCIATED WITH	Pernicious anemia	H. pylori infection

MORPHOLOGICAL FEATURES

	① Chronic superficial gastritis	② Chronic atrophic gastritis
N/E	MUCOSA	
	Edematous & Congested	Very thin & Red
M/P	LAMINA PROPRIA	
	Plasma-lymphocytic infiltrate	Dense plasma-lymphocytic cellular infiltrate
	GASTRIC GLANDS	
	Intact	Atrophy



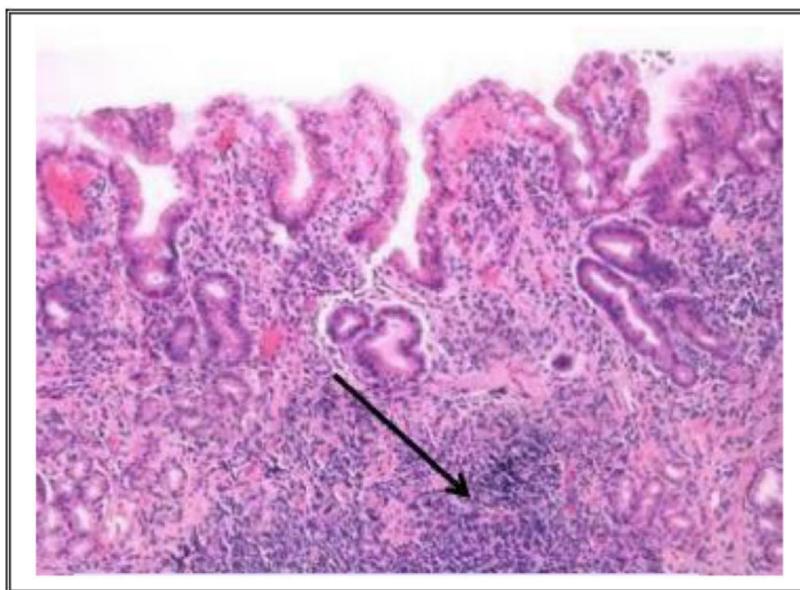


COMPLICATIONS

① Peptic ulcer

② Malignancy: Due to...

- a- Intestinal metaplasia & dysplasia
- b- H.pylori infection → Gastric carcinoma & lymphoma



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PEPTIC ULCER

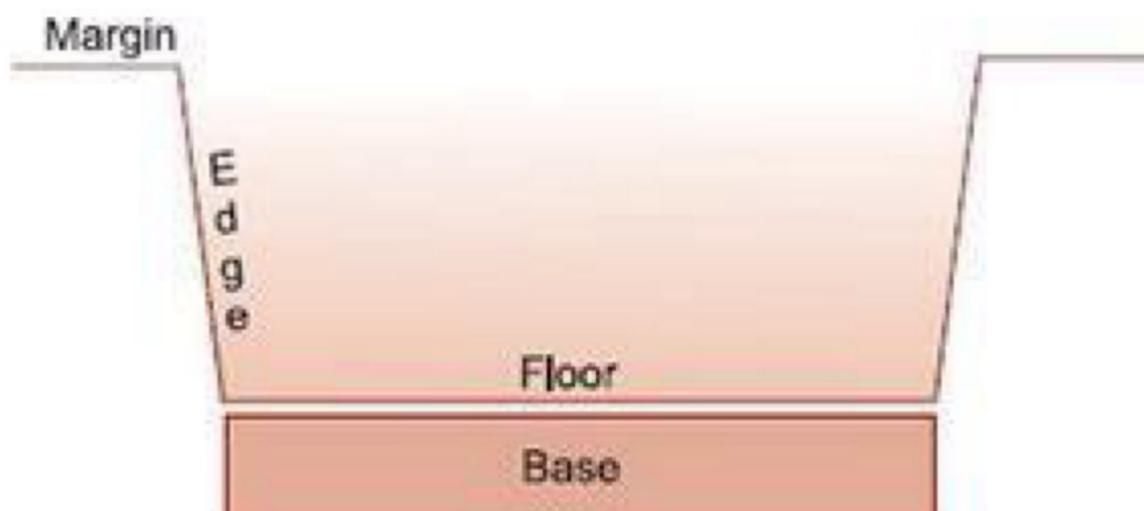
Definition

- Ulcers occurring in any part of the gastrointestinal tract exposed to the action of acidic gastric juice.

☑ Ulcer:

- **Definition:** Discontinuity in surface epithelium
- **Characters:**

Margin	▪ Line of demarcation between normal & abnormal
Floor	▪ The exposed part of an ulcer (Inspection)
Edge	▪ Part between the margin & the floor
Base	▪ the structure on which the ulcer rests (Felt on palpation)





①

ACUTE PEPTIC ULCER

ETIOLOGY

It occurs within hours in cases of:

- Stress (as shock, burn)
- Sever acute gastritis.

SITE

① Stomach

② 1st part of duodenum

MORPHOLOGICAL FEATURES

Hemorrhagic inflammation & ulcers:

Number	▪ Multiple
Size	▪ Small (< 1 cm)
Depth	▪ Superficial
Healing	▪ Heal by regeneration





②

CHRONIC PEPTIC ULCER

SITES

- ① **Duodenum:** First part.
- ② **Stomach:** Pyloric antrum on lesser curvature.
- ③ **Lower esophagus.**
- ④ **Meckel's diverticulum**
- ⑤ **Stomal ulcer** (Gastrojejunostomy)

PATIENT CHARACTERS

- ① **Age:** Usually above 20 years.
- ② **Sex:** Common in **females**.

ETIOLOGY

- ① **Genetic factors**
- ② **Hormonal factors**
 - **Zollinger Ellison Syndrome in excess gastrin** → Stimulate parietal cells → Secret acid & pepsin
- ③ **Environmental factors** (Alcohol, Smoking, Coffee)
- ⑤ **H. pylori infection:** 100% of duodenal ulcers – 75% of gastric ulcers
- ⑥ **Emotional stress.**

PATHOGENESIS

- ☑ **An imbalance between Protective & Aggressive Factors:**

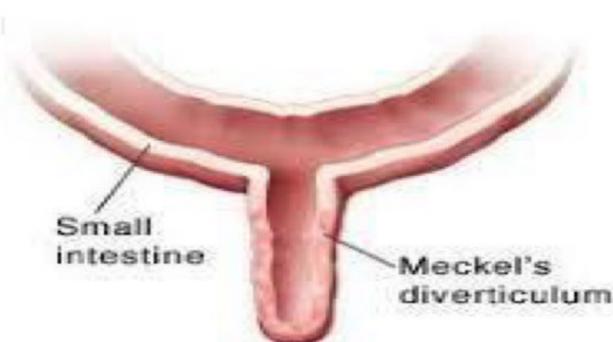
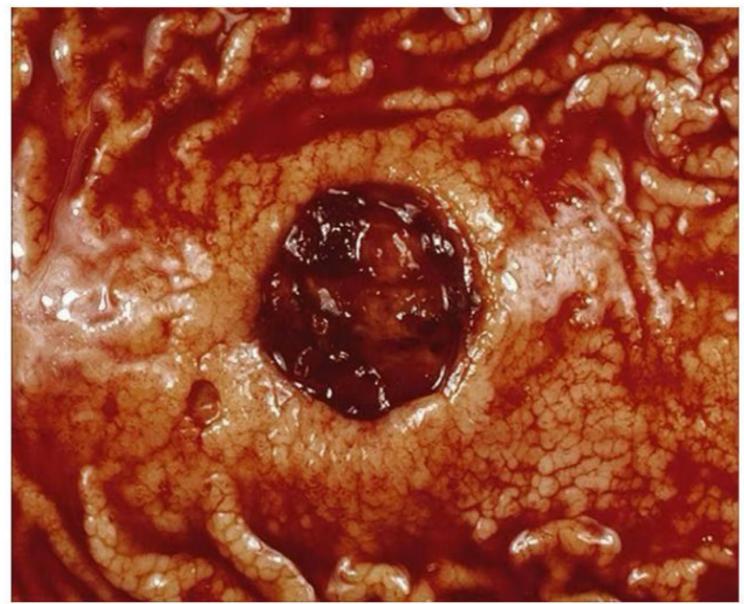
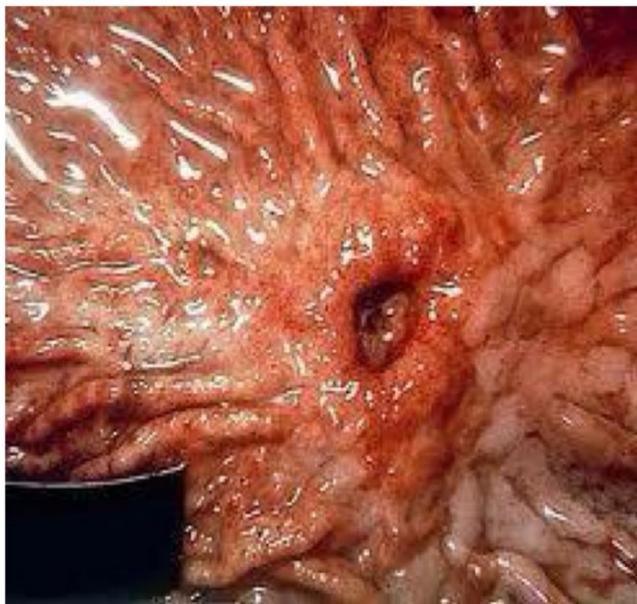
Protective factors	Hostile factors
<ol style="list-style-type: none"> 1. Mucus production 2. Bicarbonate production 3. Blood flow to mucosa 4. High epithelial cells turnover 5. Prostaglandins (PGE₂) 	<ol style="list-style-type: none"> 1. Helicobacter pylori (h.pylori) infection 2. Gastric acid secretion 3. Pepsinogen secretion 4. NSAID 5. Cigarette smoking 6. Corticosteroid use



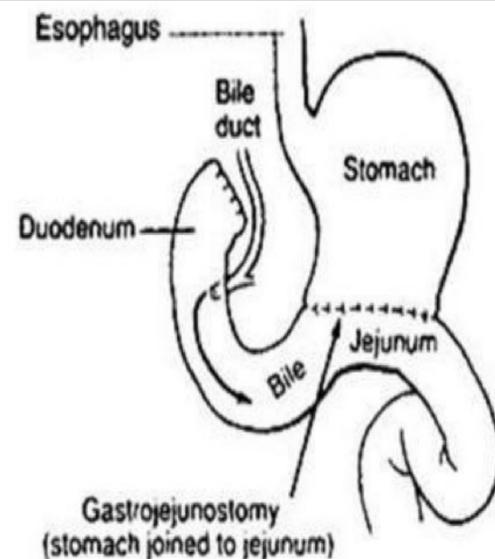


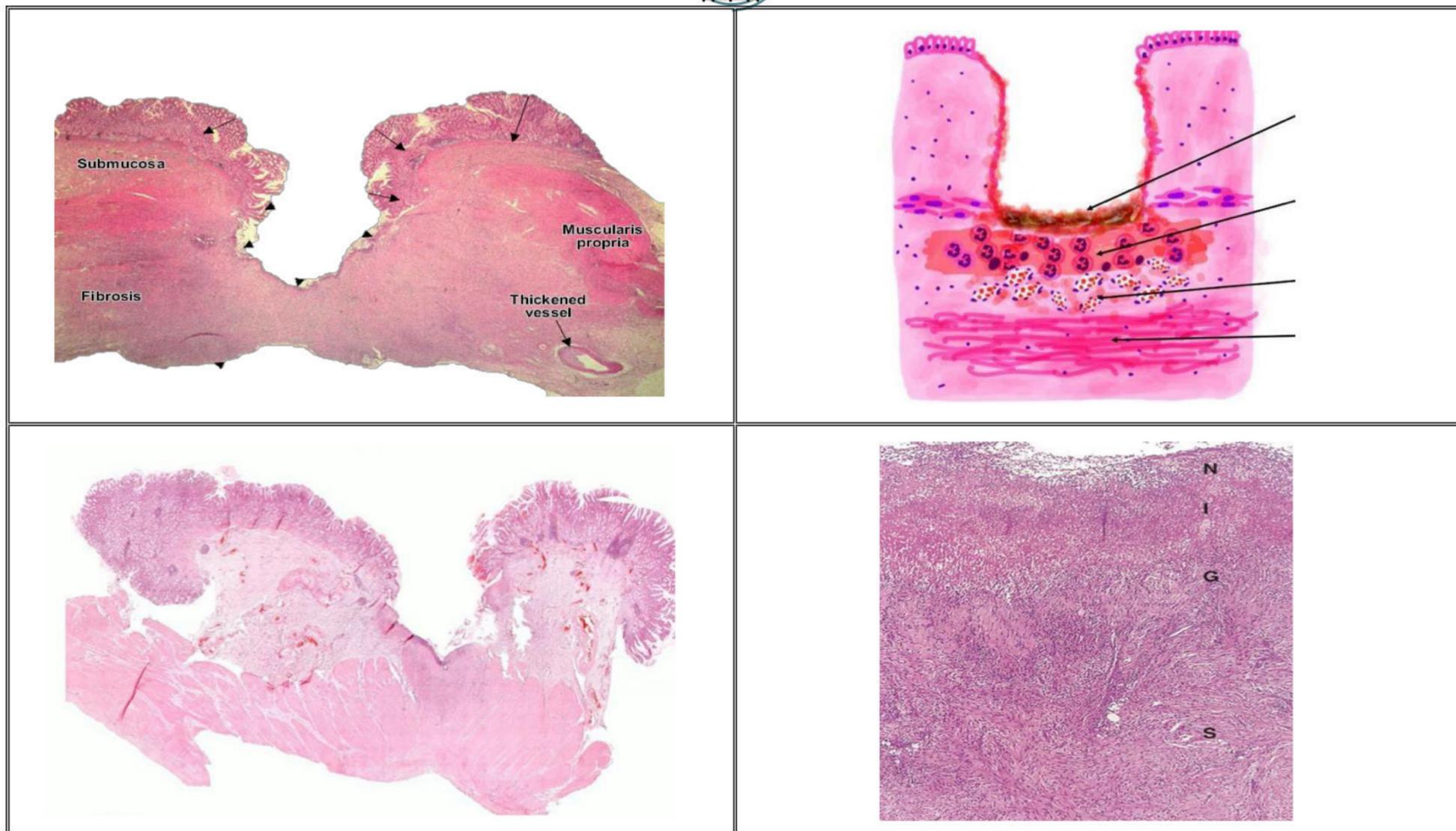
MORPHOLOGICAL FEATURES

N/E	NUMBER	<ul style="list-style-type: none"> Usually solitary well delineated lesion
	SHAPE	<ul style="list-style-type: none"> Rounded to oval
	SIZE	<ul style="list-style-type: none"> Often large (larger than 1 cm, rarely larger than 5 cm) Size does not differentiate benign from malignant ulcer
	MARGINS	<ul style="list-style-type: none"> Slightly raised because of edema
	EDGE	<ul style="list-style-type: none"> Punched out
	FLOOR	<ul style="list-style-type: none"> Smooth
	BASE	<ul style="list-style-type: none"> Thick & firm because of fibrosis
M/E	<ul style="list-style-type: none"> The base of a chronic peptic ulcer is composed of: <ol style="list-style-type: none"> A surface of Necrotic layer Acutely Inflamed layer A zone of Granulation tissue. Extensive Sclerosis (fibrosis) of the base, with extension of fibrosis into the muscle wall. 	



A Meckel's diverticulum is an abnormal pouch of tissue on the small intestine.





COMPLICATIONS

① Hemorrhage

- One of the most common complications of stomach ulcers.
- It can occur when an ulcer develops at the site of a blood vessel.

② Perforation:

- Due to erosion of the wall
- Leads to acute peritonitis & pancreatitis.

③ Fibrosis:

- Hour-glass stomach
- Stenosis causes pyloric narrowing & gastric obstruction

④ Iron deficiency anemia

⑤ Malignancy:

- Rare
- Gastric not duodenal





GASTRIC VS DUODENAL ULCER

	Gastric ulcer	Duodenal ulcer
SITE	Ulcer in the stomach	Ulcer in the duodenum
EPIGASTRIC PAIN	1-2 hours after eating	2-5 hours after eating
RELIEVED BY	Vomiting	Eating
CAN CAUSE	Hematemesis & Melena	Hematemesis & Hematochezea
ASSOCIATED WITH	Weight loss	Weight gain
RISK OF	Malignancy	Perforation

HELICOBACTER PYLORI

☑ Although the organism is not invasive:

- It induces intense inflammatory & immune response: cytokines & B-cell activation.
- Enhances gastric acid secretion
- Bacterial products such as urease, phospholipases and proteases induce epithelial injury & DNA damage.

☑ Helicobacter pylori associated disorders:

1. Chronic gastritis
2. Peptic (gastric & duodenal) ulcers
3. Gastric adenocarcinoma, intestinal type
4. Gastric lymphoma

