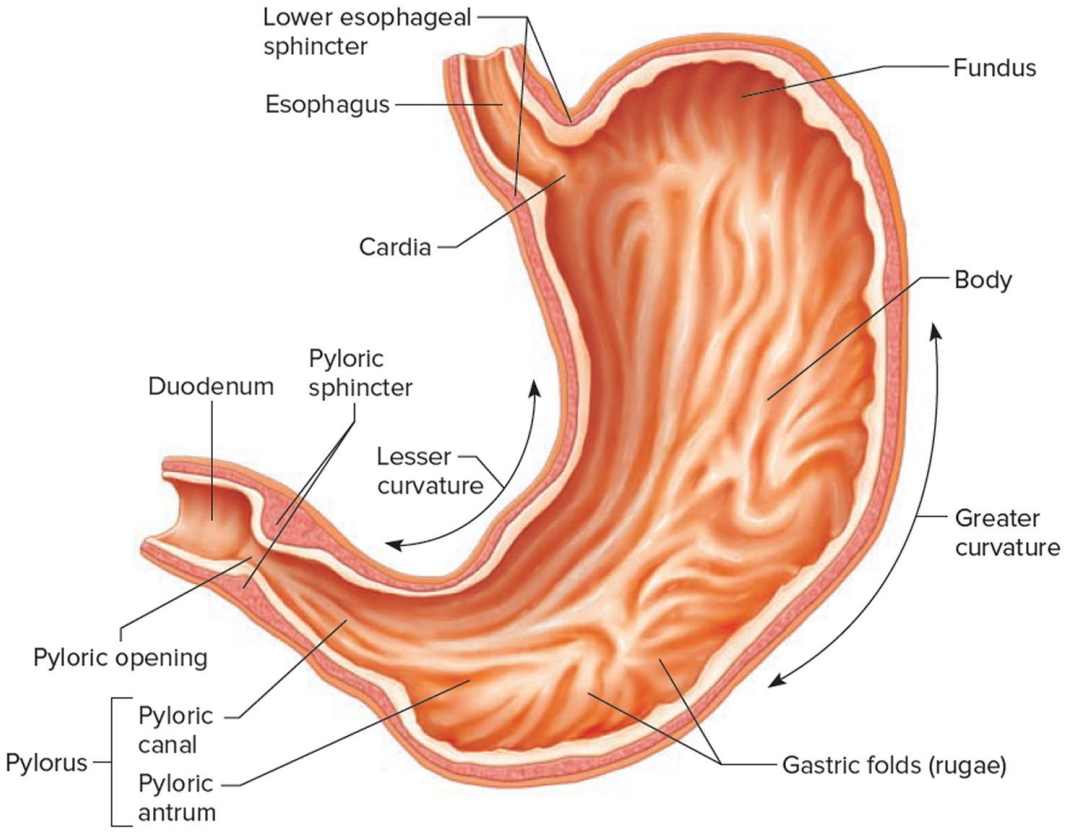
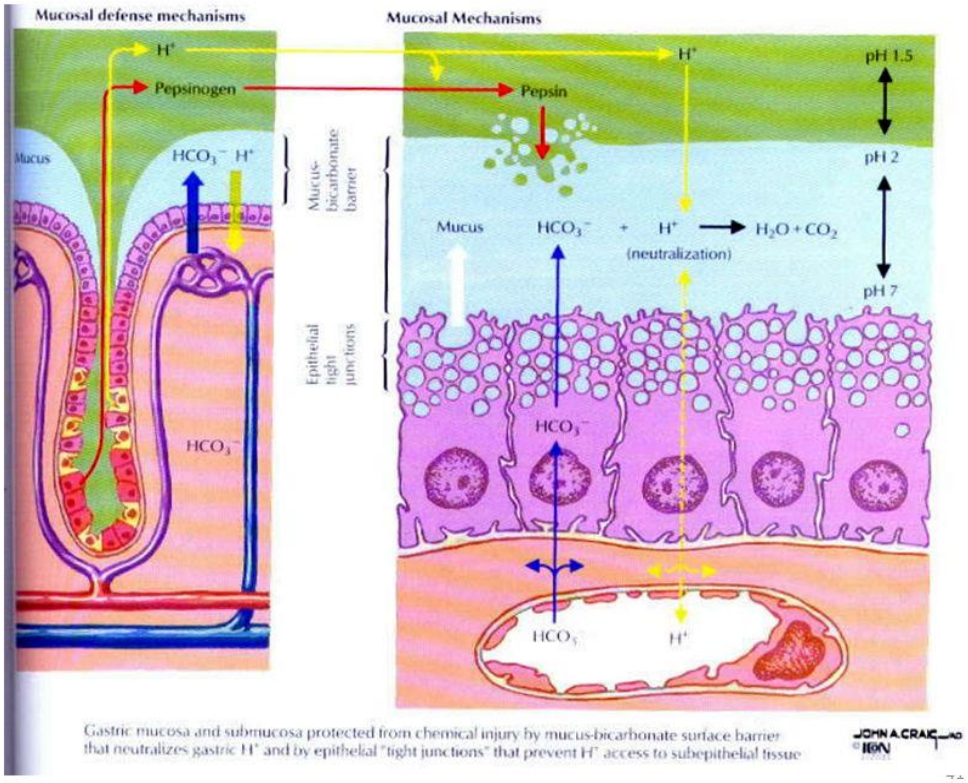
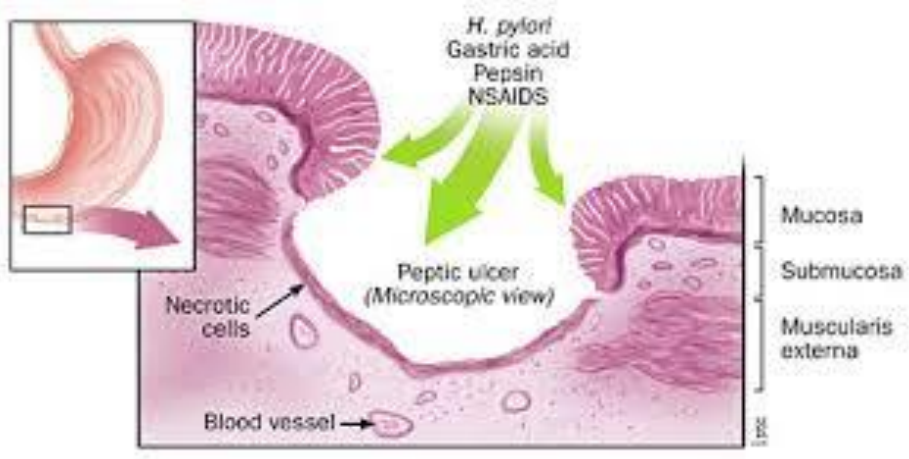

Lower esophageal sphincter
Esophagus
Cardia
Fundus
Body
Duodenum
Pyloric sphincter
Lesser curvature
Greater curvature
Pyloric opening
Pylorus
Pyloric canal
Pyloric antrum
Gastric folds (rugae)


Mucosal defense mechanisms
Mucosal Mechanisms
H+
Pepsinogen
Pepsin
pH 1.5
pH 2
pH 7
Mucus
HCO3− H+
Mucus-bicarbonate barrier
Epithelial tight junctions
HCO3−
H2O + CO2
(neutralization)
Gastric mucosa and submucosa protected from chemical injury by mucus-bicarbonate surface barrier that neutralizes gastric H+ and by epithelial "tight junctions" that prevent H+ access to subepithelial tissue

CHRONIG PEPTIC ULCER

★ **Definition:** Ulceration of mucous membrane exposed to gastric secretion by acid-pepsin digestion with **erosion of the muscle coat**.



★ **Introduction :**

- In the stomach , **HCL** is secreted due to activity of **hydrogen-potassium ATPase** (proton pump) , at the **parietal cells membrane** which present in the **fundus & body** of stomach.
- The parietal cells have **3 receptors** for stimulation :
  - **M3 receptors :**stimulated by **acetylcholine** from vagus .
  - **H2 receptors :** stimulated by **histamine** .
  - **G receptors :** stimulated by **gastrin** hormone secreted from **G cells** in the **antrum** .
- **Somatostatin** derived from D cells all over the stomach inhibit acid secretion .
- **Four different types of cells make up the gastric glands:**
  - **Mucous** cells: secrete alkaline mucous
  - **Parietal cells**: secrete HCL
  - **Chief cells** : secrete pepsinogen .

- **Endocrine cells** : secrete gastrin and somatostatine .

- **Gastro-duodenal mucosal barrier :**
  - **Definition**: factors which normally prevent digestion of gastric mucosa by acid and pepsin . These factors are :
    1- Thick **mucus** secreted by the mucosa .
    2- **Bicarbonate** secretion by the mucosa .
    3- High **regeneration** power of the mucosal cells .
    4- Rich mucosal **blood supply**.
    5- **Prostaglandins** increase mucosal blood flow , increase mucous & bicarbonate secretion and cytoprotective .

- Normally there is a **balance** between the attacking forces (acid and pepsin) on one side and the defense mechanisms (mucosal barrier) on the other side .
- **Gastric peptic ulcer** occur if there is disturbance in this balance .
- **Hyperacidity** leads to duodenal peptic ulcer and **weakness of the mucosal barrier** leads to gastric peptic ulcer .

★ **Site & Classification:**

- **An acid-pepsin secreting mucosa usually does not digest itself due to presence of gastric mucous barrier but will do so to the adjacent mucosa of the following sites :**

A) **Duodenal ulcer:** The **commonest**.

B) **Gastric ulcer:** Less common, may be one of the following types.

1) Type I: The **commonest** type (60%).

- Ulcer located on **the lesser curvature**, usually around the **incisura** angularis (**ulcer bearing area**).

- Gastric **acidity is normal or low**.
- This type is preceded by **damage** of gastric mucosal barrier leading to **chronic gastritis**.

2) Type II: (20%)

- Gastric ulcer associated with duodenal ulcer (**combined ulcers**)
  - **Duodenal** ulcer occur **first** leading to duodenal deformity & gastric stasis.

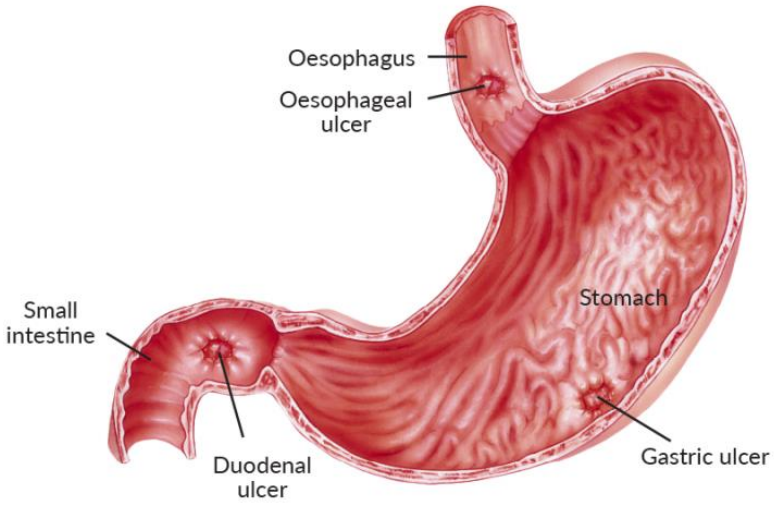
3) Type III: (20%)

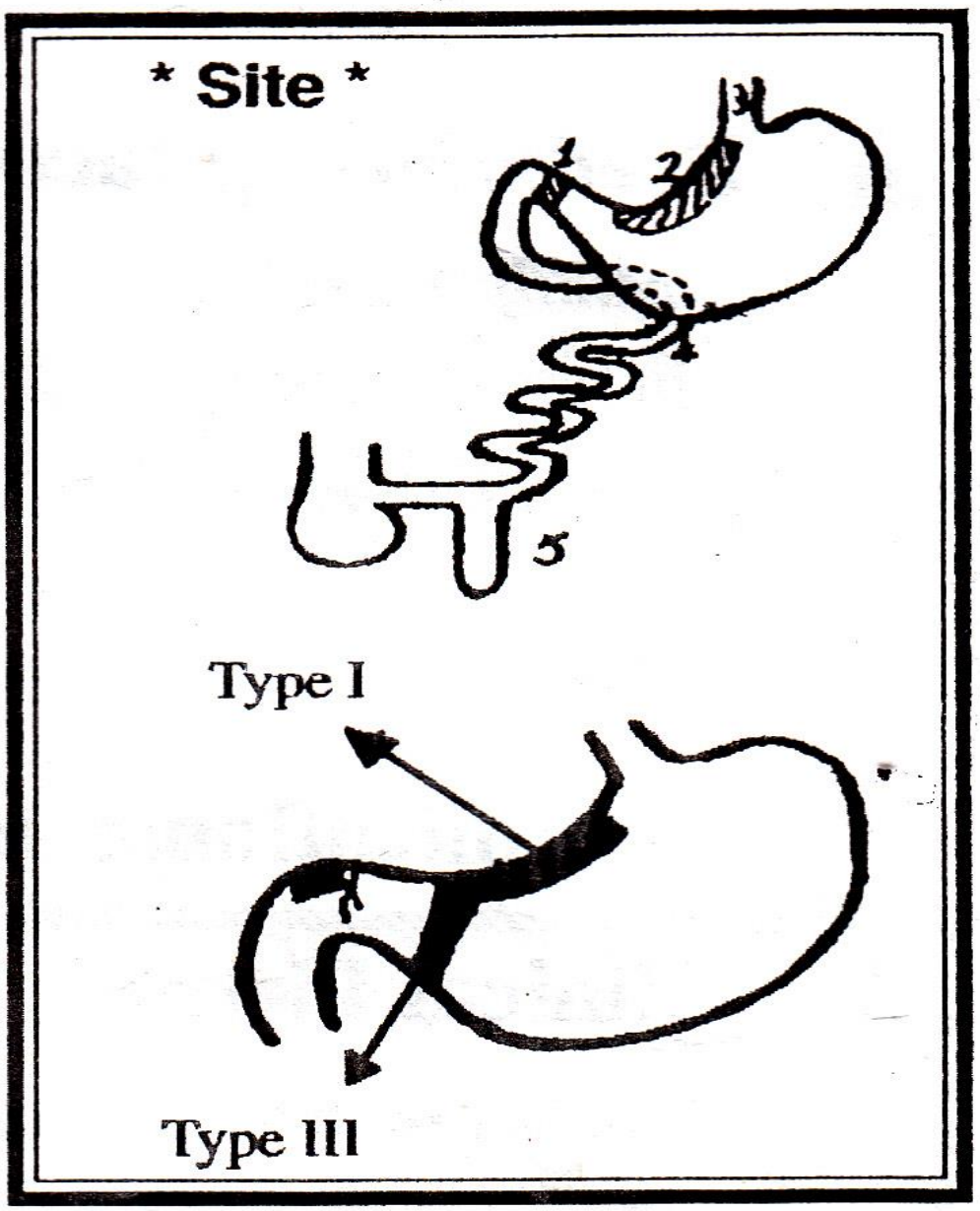
- Ulcer occur in the pyloric antrum or pyloric canal.

- There is **hypeacidity** in duodenal and type II & III gastric ulcers and uaually have the **same** aetiology , clinical picture and treatment.

C) **Rarely in:**

- **Oesophagus** due to GERD (reflux oesophagitis).
- The **jejunum** (after gastrojejunostomy or Zollinger Ellison's syndrome).
- **Meckel's** diverticulum (ectopic gastric mucosa).





Site & Classification

★ **Aetiology:** (Usually multifactorial).

A. Duodenal and type II &III Gastric ulcer	**B. Gastric ulcer type I**
▪ **Hyperacidity which may be due to one of the followings:**	▪ In long standing cases , the following factors causes **chronic gastritis → devitalization of gastric mucosa→ defective gastric mucosal barrier**
1-**Helicobacter pylori** , present in 85% of cases , produce ureae enzyme which transform urea to amonia→ increase alkalinity of the antrum → increase gastrin formation.	**1- Helicobacter Pylori** , present in 75% of cases , ➢ Helicobacter Pylori secret protease enzyme which destroy the mucous barrier.
2- Ulcerogenic drugs (aspirin & NSAIDs):	
➢ Early produce irritation of gastric mucosa	➢ Anti-prostaglandin and inhibit proliferation of mucosal cells .
3- **Irritant & fast foods** as spices, alcohol & smocking .	
➢ Early irritate gastric mucosa .	➢ produce gastritis in long standing case.
4- ***Genetic*** factor → ***Large parietal cell*** mass. It is more common with blood **group O and non-secretors** .	4- Duodeno-gastric ***reflux of bile.*** 5- ***Ischaemia*** of gastric mucosa. 6- Chemical or mechanical ***trauma***.

5- **Vagal over tone** as in nervousness and stress.

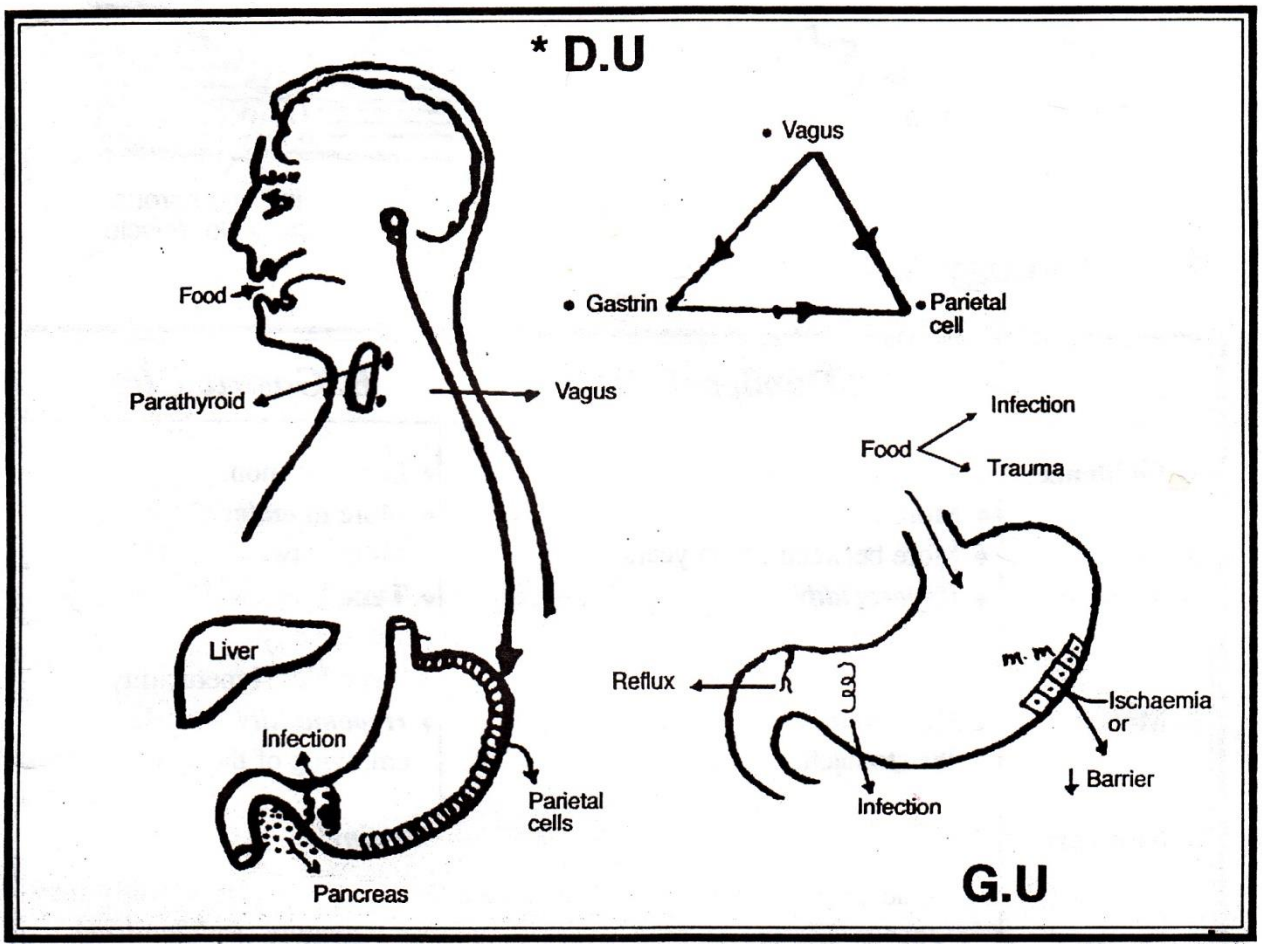
6-**Hypergastrinaemia:**

a) ***Liver diseases:*** Due to lack of inactivation of histamine & gastrin.

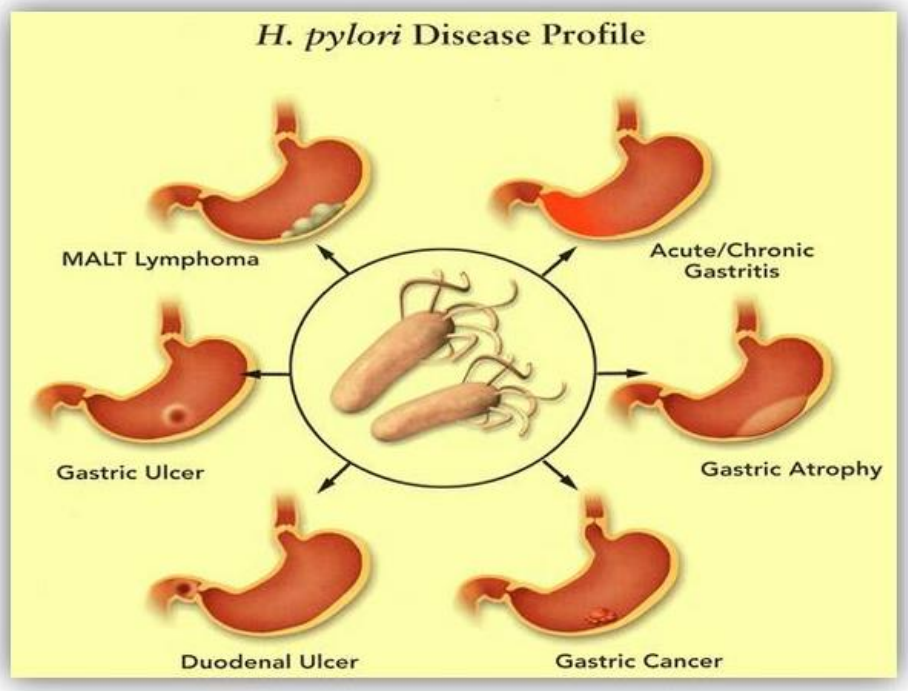
b) ***Zollinger Ellison's syndrome***: Usually due to pancreatic tumor (malignant tumor called gastrinoma) or rarely due to hyperplasia of G cells in the antrum , secreting excess gastrin.

c)***MEN type* I**: Multiple adenomas in the pituitary, pancreas & parathyroid glands .

d) ***Hyperparathyroidism :*** Hypercalcaemia stimulate excessive gastrin production .



Common causes of peptic ulcer

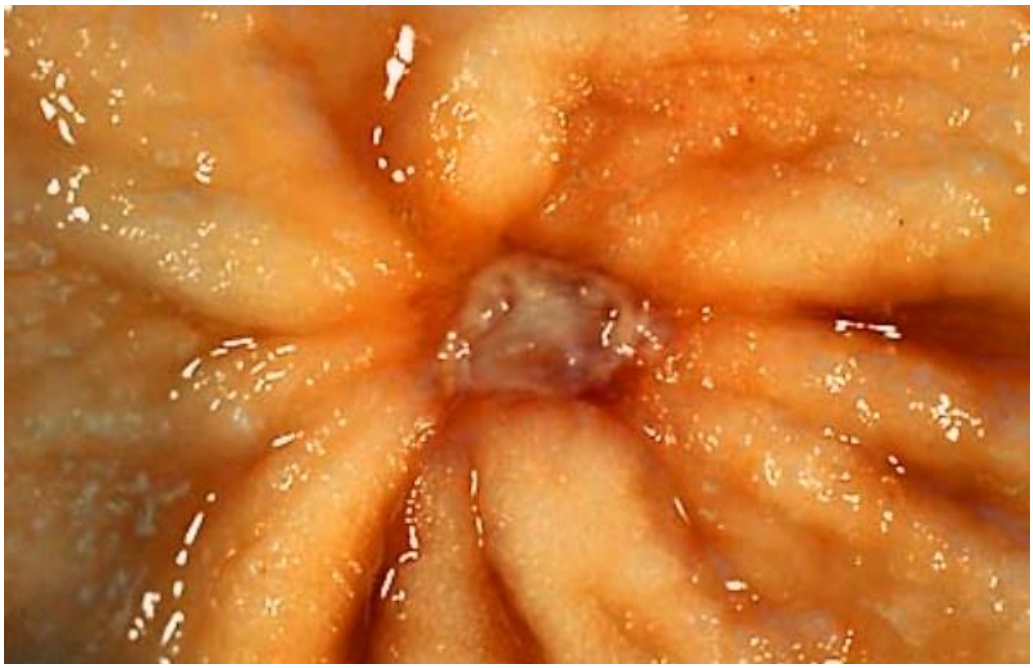


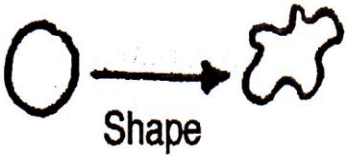
★ **Helicobacter pylori (HP) :**

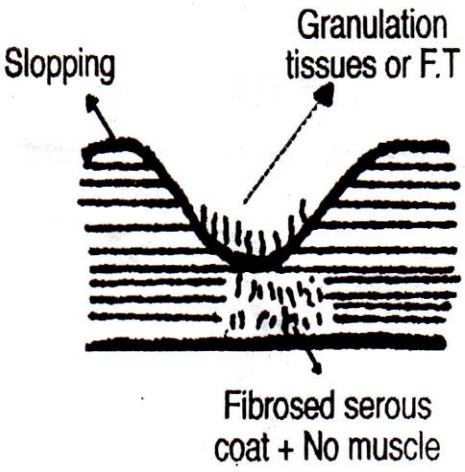
- It is the **main cause** of peptic ulcer in people not receiving NSAIDS.
- It is a gram negative, microaerophilic, **spiral** (helical) bacterium usually found in the **pyloric region** of the stomach.
- HP infection is **increasing with age** . In adults it affect **50%** of population in developed countries and **90%** in undeveloped countries .
- It may be **transmitted by** saliva or fecal contamination.
- **Pathophysiology :**
  - The organism lives in **mucus** adherent to the epithelium .
  - It produce **urease** enzyme which produce **ammonia** from urea **decreasing the acidity** around the organism to protect itself .
  - It produce **cytotoxins** , causing mucosal **inflammation** → acute and chronic **gastritis** → devitalization of gastric mucousa and defective mucous barrier + chronic **irritation** in long standing case leading to gastric **cancer** .
  - It **inhibit somatostatine** and **stimulate gastrin** secretion leading to **hyperacidity** and mucosal damage .

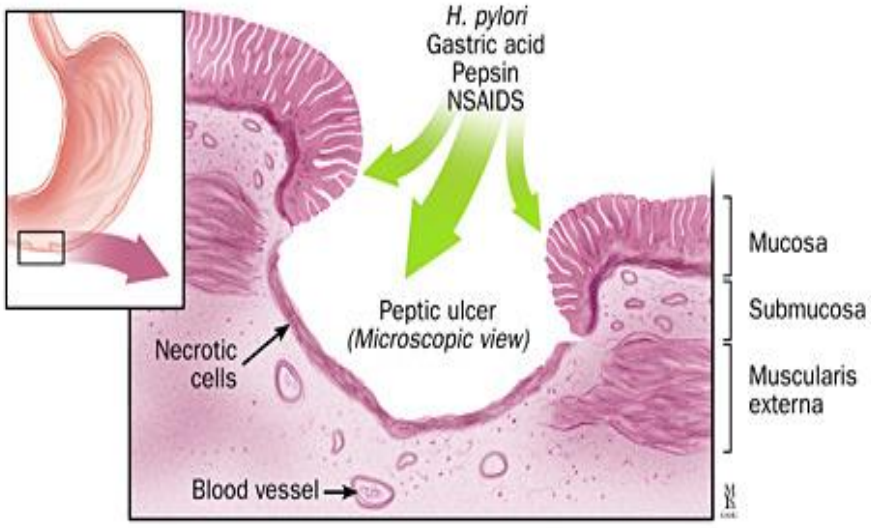
★ Pathology & Characters:

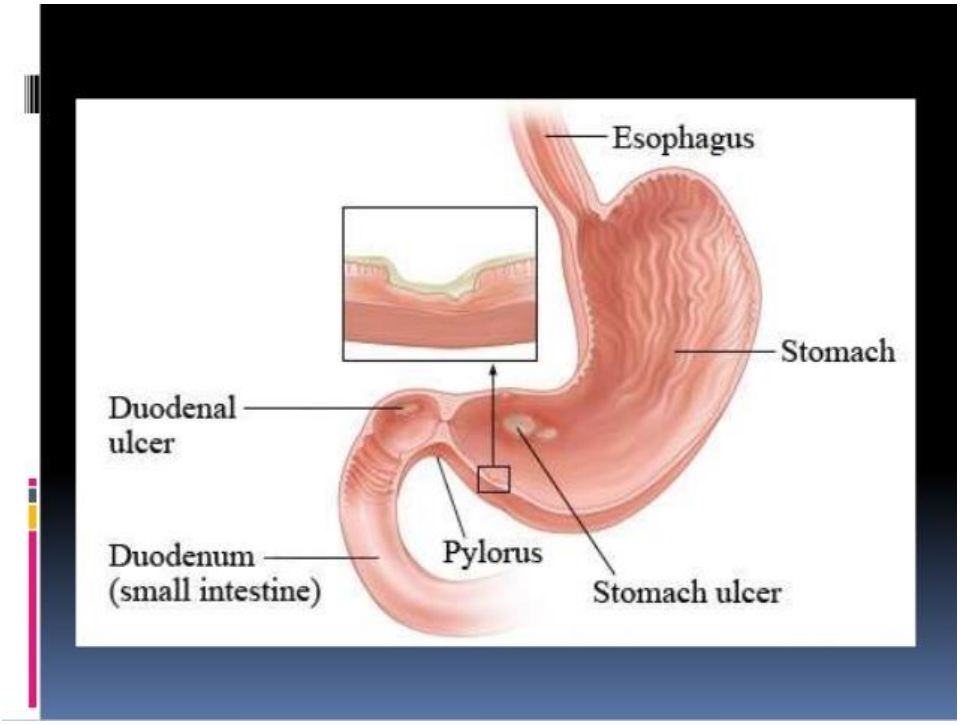
	A.Duodenal and type II &III Gastric ulcer	B. Gastric Ulcer type I
1- Incidence	▪ More common (**25** times)	▪ Less common.
2- Sex:	▪ More in **males** (5 times)	▪ More in males (2 times).
3- Age:	▪ More between 20- 40 years	▪ More above 60 years
4- Acidity:	▪ **Hyperacidity.**	▪ **Normal or Hypoacidity** in type I
5- Motility:	▪ **Hypermotility** (vagal over tone) → rapid emptying of the stomach.	▪ **Hypomotility** → delayed emptying of the stomach
6- Number:	▪ Both usually **single.** ▪ One ulcer on the anterior wall & another on the posterior wally may occur (**kissing ulcer**) or over-riding ulcer (**saddle ulcer**). ▪ One ulcer in the stomach & another in the duodenum may occur (**combined ulcer i.e gastric ulcer type II**).	
7- Site	▪ More on the **posterior wall of 1st inch** of 1 st part of the duodenum (duodenal bulb or cap) i.e. the area of duodenum receiving most of gastric HCL . ▪ **Gastric ulcer type III:** pyloric antrum or canal.	▪ **Type I**: On lesser curvature around **incisura angularis** (**ulcer bearing area**) between the body & antrum i.e between acidic & alkaline mucosa . ➢ Any large ulcer **outside** the ulcer bearing area is suspicious for **malignancy**.
8- Size :	▪ Usually small .	▪ Usually larger .
9- Shape:	▪ **Round** or oval but it may be **irregular** due to fibrosis.	
10- Edge:	▪ It is usually **punched out** in recent ulcer or **slopping** in healed ulcer.	
11- Floor:	▪ Deeply penetrate the muscle coat & filled with friable easily bleeding **granulation tissues** during activity or **fibrous** tissues during healing .	
12- Margin	▪ Indurated with **mucosal folds radiating** around the ulcer .	
13- Base:	▪ Usually indurated **fibrosed serous coat** or eroded near by **organ** .	

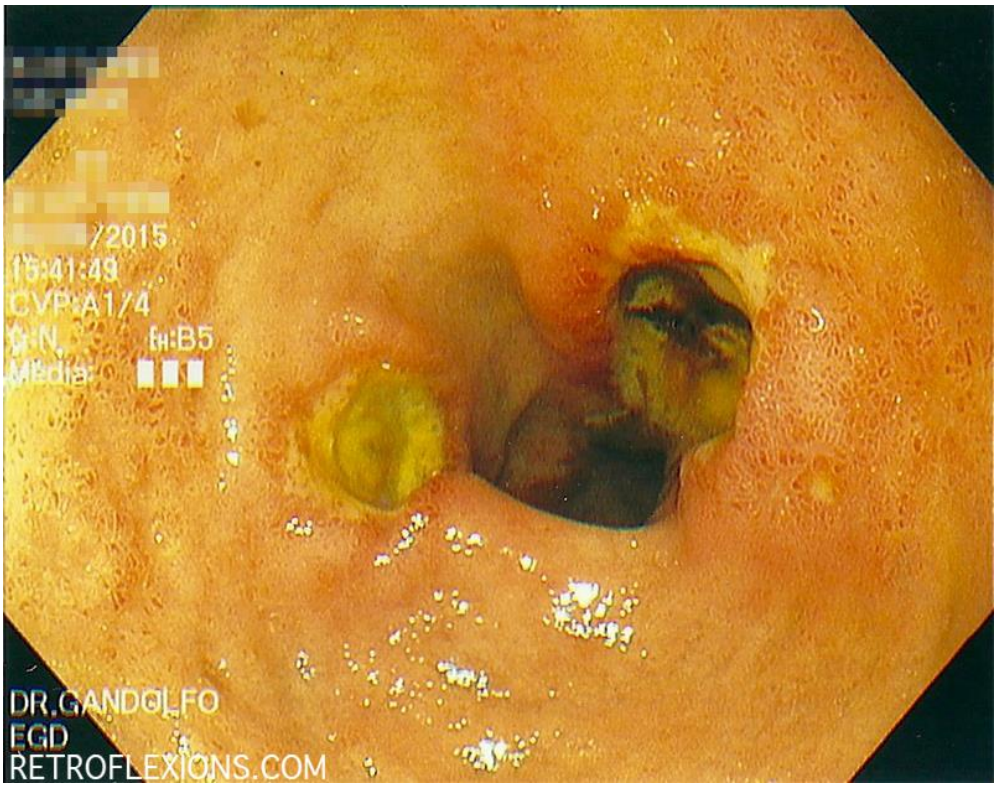



Shape

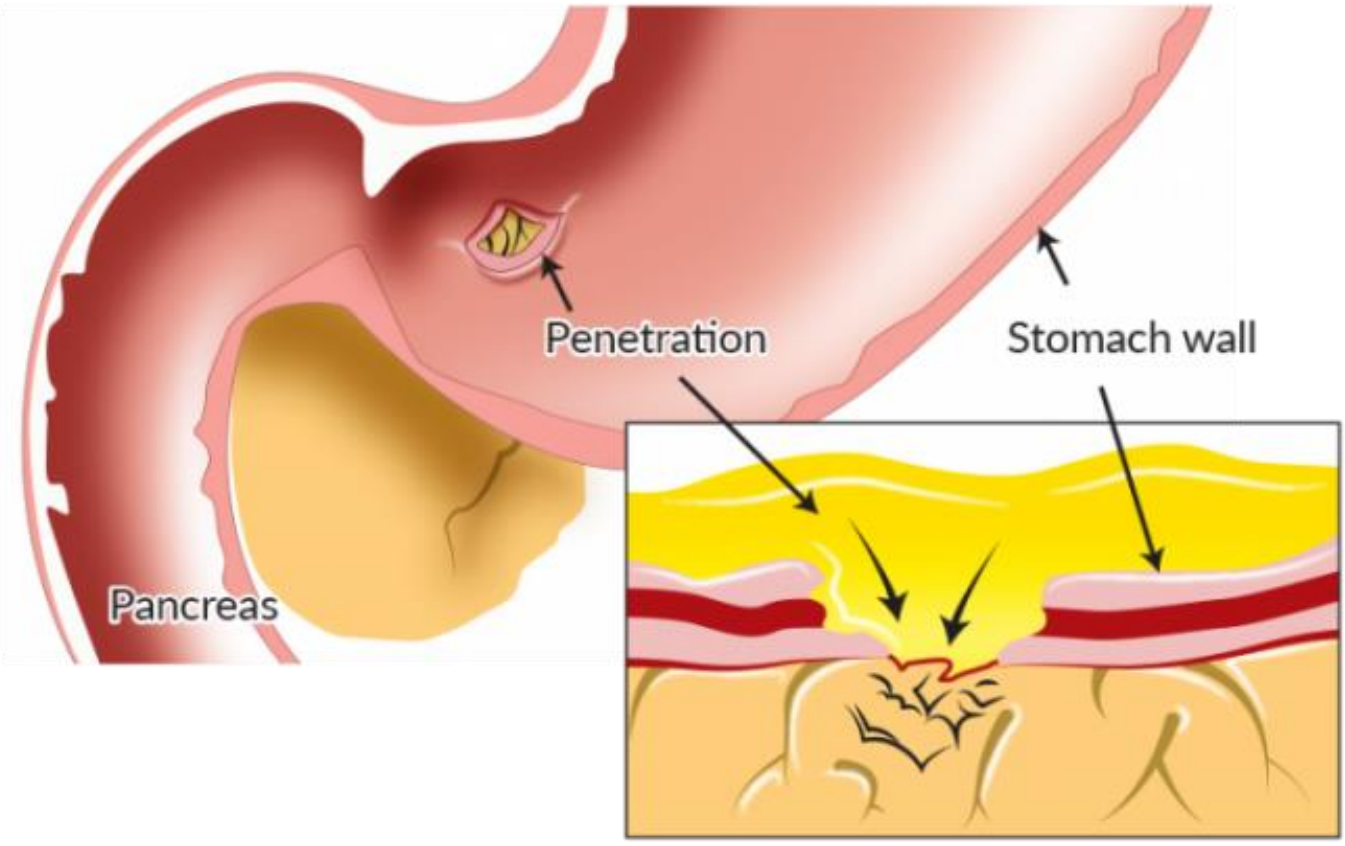

Slopping
Granulation tissues or F.T
Fibrosed serous coat + No muscle


H. pylori
Gastric acid
Pepsin
NSAIDS
Peptic ulcer
(Microscopic view)
Necrotic cells
Blood vessel
Mucosa
Submucosa
Muscularis externa





kissing ulcer of duodenum



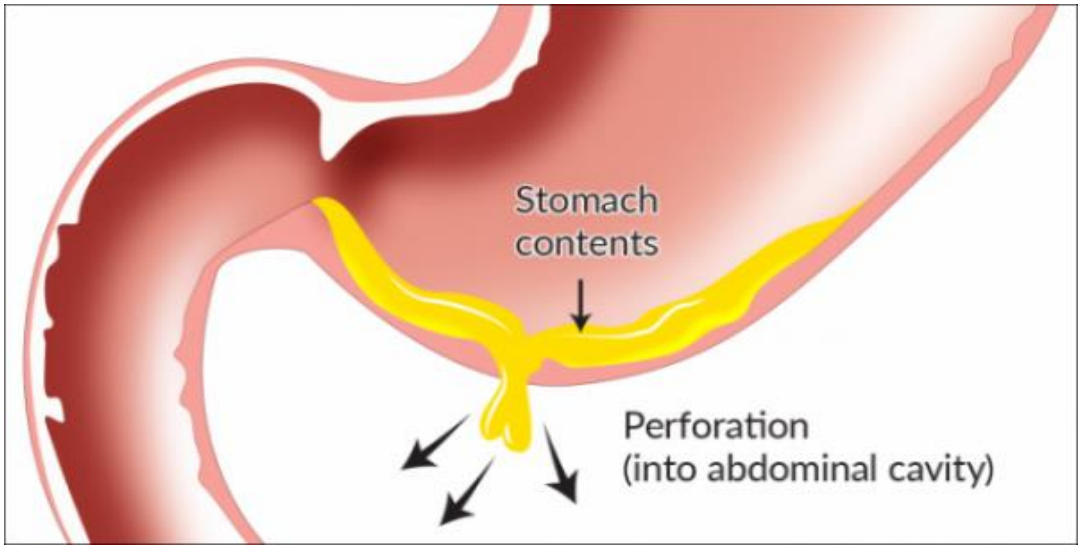
Base is eroded pancreas

★ **Complications:**

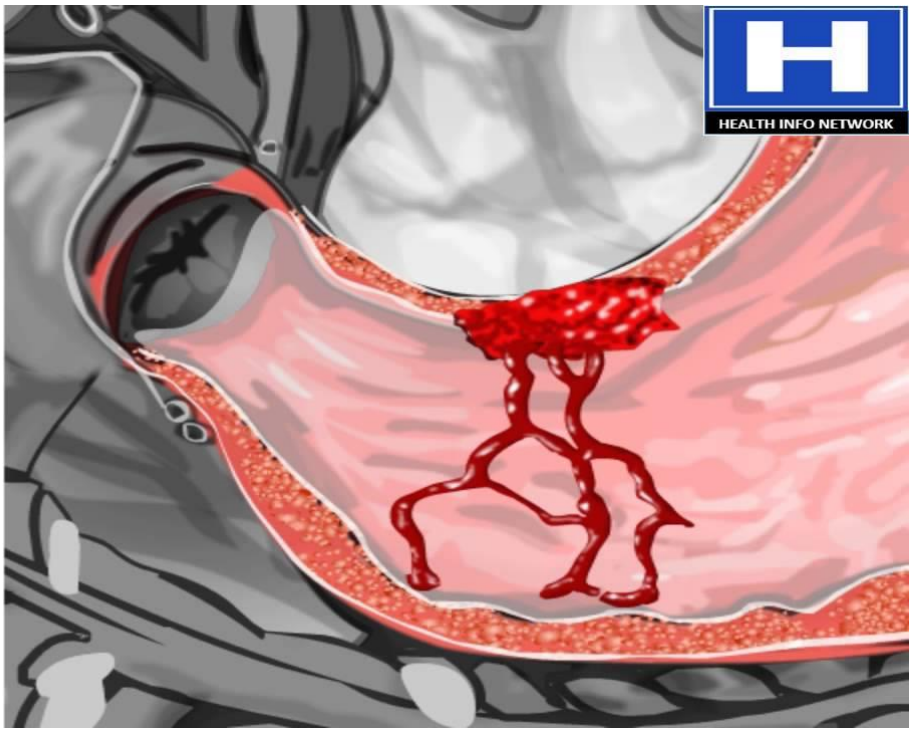
I) Acute complications : occur during ulcer activity .

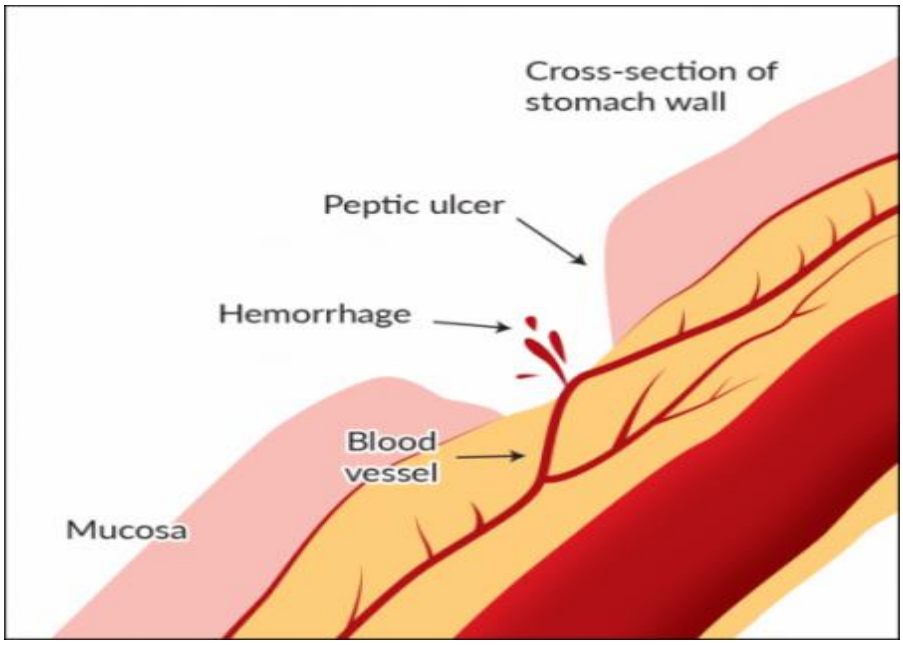
A. Acute Perforations: Occur in 5% of cases .

B. Bleeding: Occurs in 5% of cases (haematemesis, melaena or both).



Bleeding is a common sympton of ulcers.



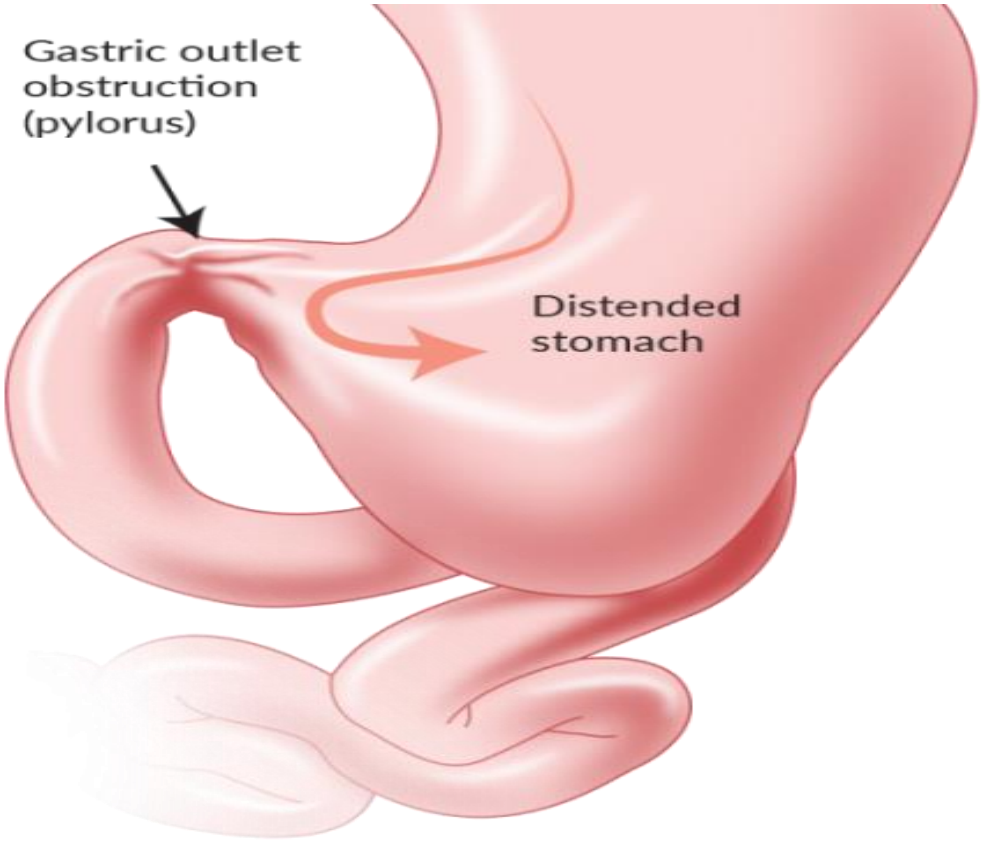


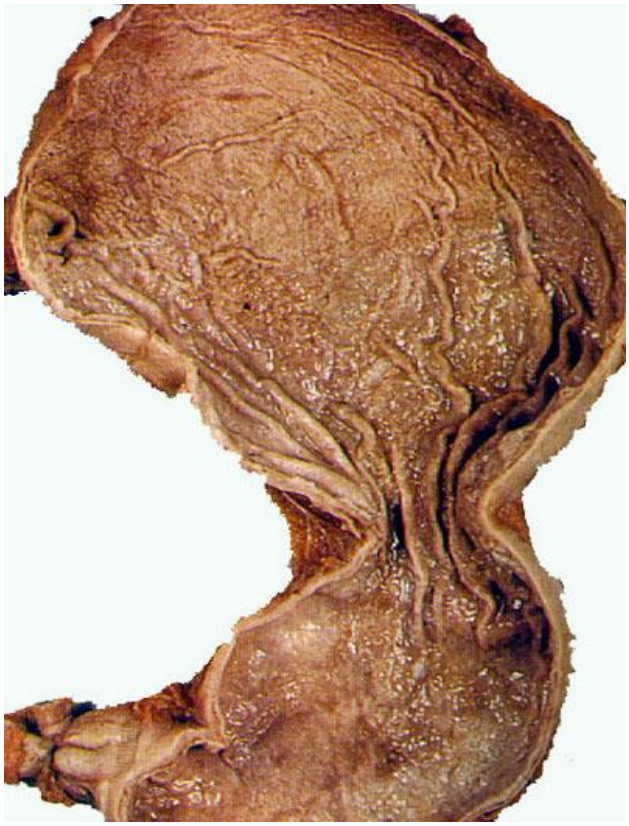
II) Chronic complications : occur insidously

1- Fibrous contracture of the ulcer : Occur in 5% of cases.

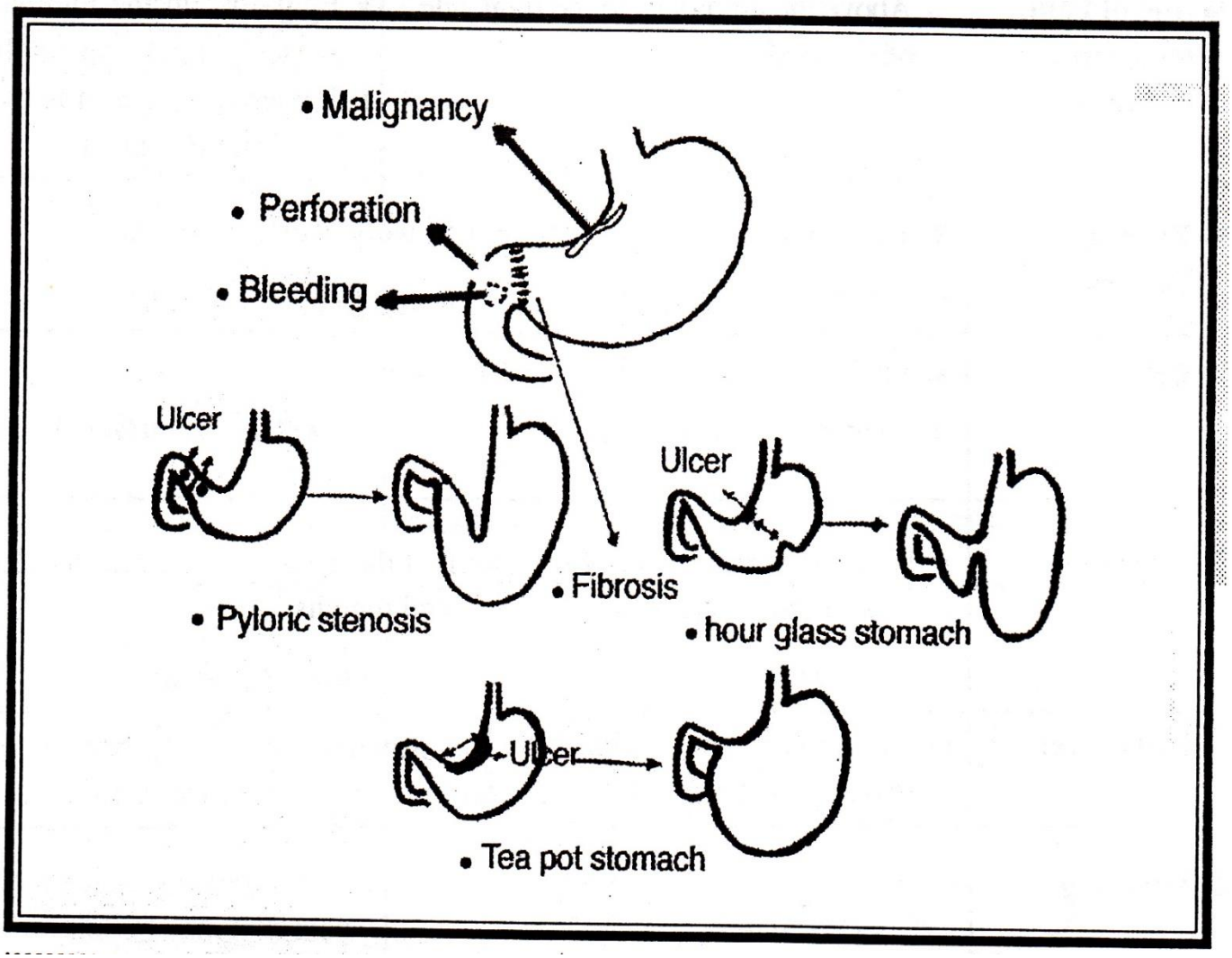
- **Duodenal or pyloric canal** ulcer → **pyloric stenosis** (gastric outlet obstruction)
- **Gastric** ulcer → **hour glass or tea pot** stomach according to the site of the ulcer.

2. Malignancy: only gastric peptic ulcer may turn malignant in less than 1% of cases .


Gastric outlet
obstruction
(pylorus)
Distended
stomach

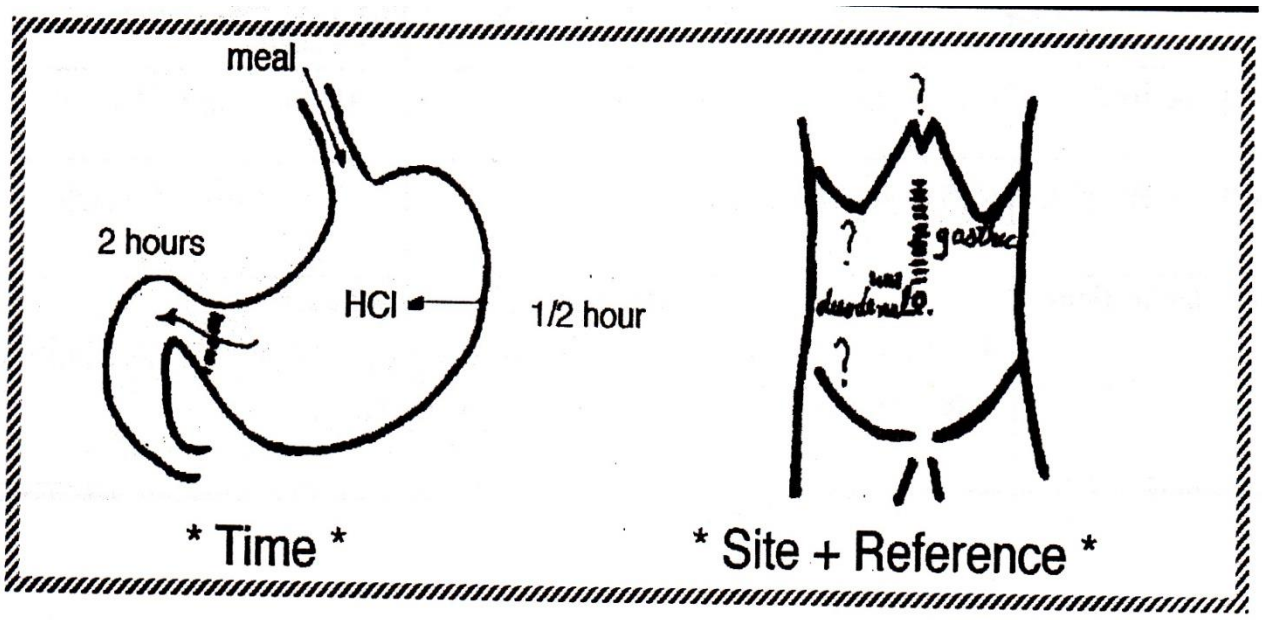


★


• Malignancy
• Perforation
• Bleeding
Ulcer
• Pyloric stenosis
• Fibrosis
Ulcer
• hour glass stomach
Ulcer
• Tea pot stomach

★ Clinical Picture : Ulcer dyspepsia.

	A. Duodenal Ulcer	B. Gastric Ulcer
Type of patient	▪ Young active high intellectual person with recent gain of weight .	▪ Old thin patient .
1- Pain	▪ It is due to contact of acid with the exposed nerve endings.	
➢ **Type:**	▪ Burning (heart burn) , stabbing, dull aching, Colic , shooting.	
➢ **Time:**	a. Starts **2 – 3 hours** after meal & persists till the next meal (**hunger** pain). b. **Nocturnal pain** is characteristic.	a. Usually starts. **½ - 1 hour** after meal. b. No pain during night.
➢ **Site of pain, tenderness:**	▪ Above the umbilicus to the right side of the midline.	▪ Around the middle line in the epigastric region .
➢ **Exciting Factors:**	▪ Irritant foods, smoking, nervousness, work, worry & weather.	
	▪ Fasting.	▪ Food.
➢ **Relieved by:**	▪ PPI , Alkalies, Antacids and physical & mental rest.	
	▪ Food (patient carry biscuits to eat when pain comes on).	▪ Fasting, vomiting & lying flat.
➢ **Periodicity:**	▪ Weeks of exacerbation of symptoms at the summit of the activity of the patient alternating with months of freedom .	
	▪ Well marked	▪ Rarely present.
➢ **Reference:**	▪ Retrosternal , back (when the ulcer penetrates posterioly into the pancreas), rarely to the right hypochondium or to the right iliac fossa.	
2- Nausea & Vomiting:	▪ Does not occur except if pyloric stenosis occurs.	▪ Very common as it relieves pain & it may be self induced.
3- Appetite:	▪ Good as eating relieves pain.	▪ Patient is afraid to eat.
4- Body weight:	▪ Usually gain weight.	▪ There is loss of weight.
5- Complications	▪ **Peptic ulcer is usully sympltomless and complications may be the first presentation.** ▪ If antacids fails to relive pain or pain becomes continuous not related to meals or loss of periodicity → possibility of complications or malignancy (In gastric ulcer only).	


meal
2 hours
HCl
1/2 hour
* Time *
* Site + Reference *