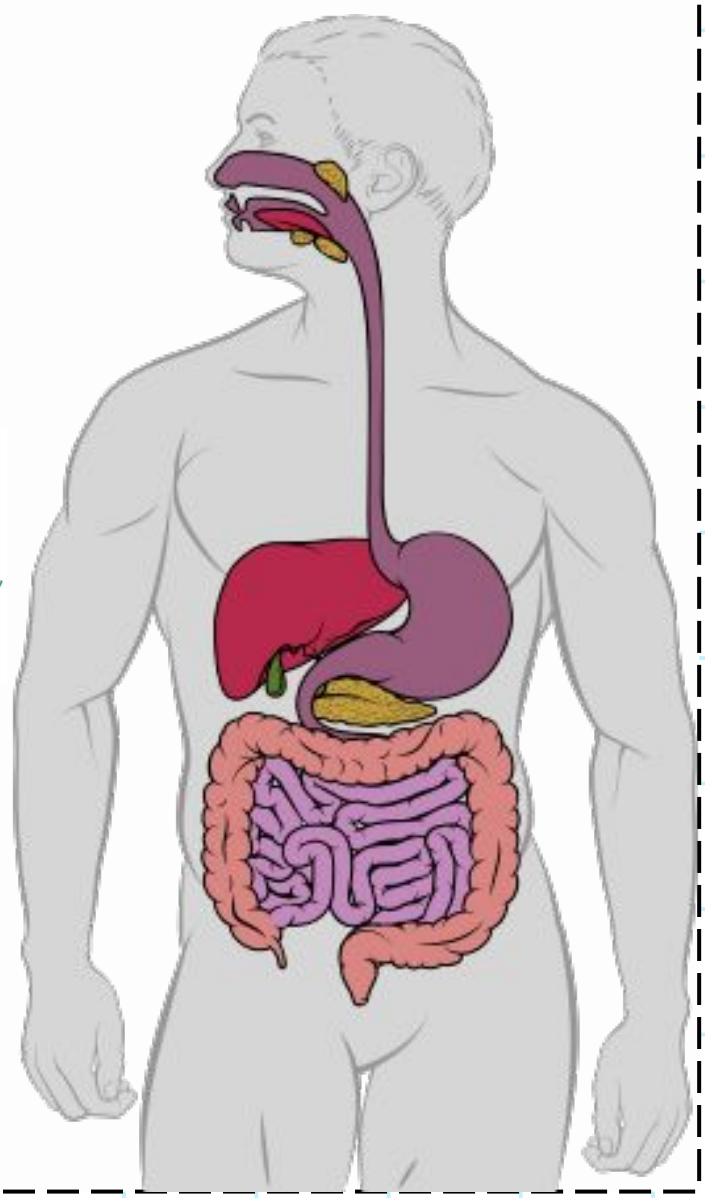
Peptic ulcers



Editing File

Color index :
Main text (black)
Female Slides (Pink)
Male Slides (Blue)
Important (Red)
Dr's note (Green)
Extra Info (Grey)

OBJECTIVES

- Deine ulcer & erosion
- Describe the pathogenesis and pathology, clinical features of both Acute gastric ulcers & chronic peptic ulcers
- Describe the complications of chronic peptic ulcers

THIS LECTURE WAS PRESENTED BY DR.MAHA ARAFAH & DR.AHMED ALHUMAIDI

IF YOU WANT TO READ THE LECTURE FROM ROBBINS

IF YOU WANT TO READ THE LECTURE FROM FIRST AID

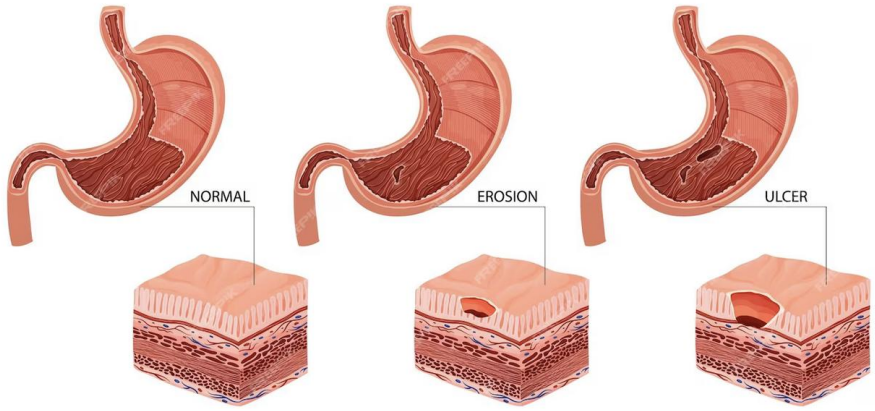
IF YOU WANT TO WATCH DR.FOUDA EXPLANATION (TILL MIN 34)

IF YOU WANT TO READ OSMOSIS SUMMARY

Introducing

Definition

- Erosion: a breach in the epithelium of the mucosa only - Only a small part-.
- Ulcer: a breach in the mucosa of the alimentary tract extending through muscularis mucosa **into** submucosa or deeper .

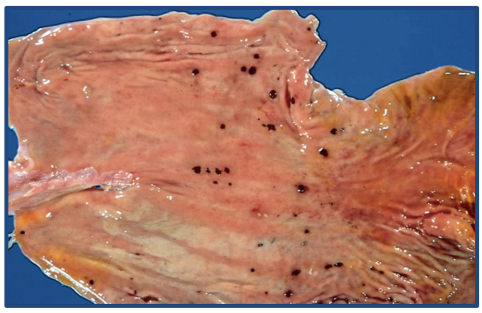


Ulcers

Acute/stress

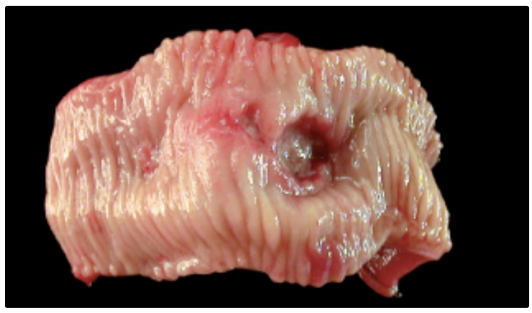
Acutely developing gastric mucosal defects that may appear after severe stress

e.g. intracerebral hemorrhage
Multiple - Small - Superficial



Chronic/peptic

Solitary/single - Deep
If I said peptic ulcer without acute or chronic, then it is chronic



Acute Peptic Ulcers

More than 75% of critically ill patients develop endoscopically visible gastric lesions during the first 3 days of their illness

Pathophysiology

Part of an acute gastritis

Acute response to an irritant chemical injury by drugs:

- NSAIDs
- Alcohol

Complication of a severe stress response

- Severe burns (Curling ulcers)
- Major trauma (stress / Cushing ulcer)
- Cerebrovascular accidents (Cushing ulcer)

A result of extreme hyperacidity

Zollinger-Ellison syndrome it's a tumor that produce more gastrin hormone = ++ Acidity.

Female Slides

01 Morphology: Range in depth very superficial lesions (erosion) deeper lesion that involve entire mucosal thickness (true ulceration)

02 Prophylaxis: Proton pump Inhibitors

03 Location: Anywhere in the stomach

04 Prognosis: Gastric mucosa can recover completely it the person does not die from the primary disease

05 Clinical feature:

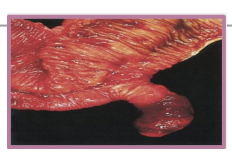
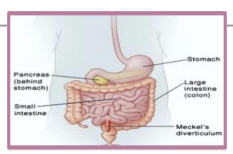
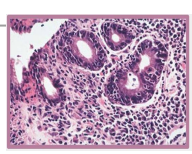
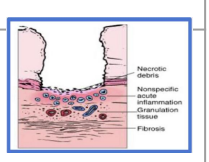
- Melena
- Hematemesis
- Pertoration
- Iron deficiency

Chronic Peptic Ulcer

- Chronic ulcers , recurring lesions that occur most often in middle-aged to older adults without obvious precipitating conditions, other than chronic gastritis.
- Often solitary

IMPORTANT

Chronic peptic ulcer

Location	Gross morphology
Gastric peptic ulcers: in stomach, mainly the interface of body + antrum at lesser curvature.	Diameter: usually <20 mm, they may reach > 100 mm - Usually 2 cm.-
98% located in first portion of duodenum or stomach. Duodenal ulcers: in the first portion of duodenum, usually within a few centimeters of pyloric valve at the anterior duodenal wall. 4:1 Ratio duodenal is 4 times more than gastric.	Duodenal ulcers: never malignant → reason for not taking a biopsy Classic Benign Peptic Ulcer (mostly): **round** to oval, shallow, clean, sharply demarcated punched-out defect, with straight walls, surrounded by hyperemia.
• Esophagus, as a result of GERD or acid secretion by ectopic gastric mucosa • Gastric mucosa within a Meckel diverticulum can result in peptic ulceration of adjacent mucosa. • Meckel diverticulum: most common congenital abnormality of small intestine caused by an incomplete obliteration of the vitelline duct. • -team 41: Simply it is normal tissue at abnormal location, so if Gastric tissue in the small intestine it will cause peptic ulceration of of adjacent mucosa. A congenital anomaly in which there is a diverticulum/in the small intestine (ilium)	Malignant Peptic Ulcer (Cancer, rare): heaped-up margins, requires biopsy.Most gastric ulcers are benign. Small percentage may be malignant, reason for biopsy
Zollinger-Ellison syndrome: **Multiple peptic ulcerations** in the stomach, duodenum, and even the jejunum	Microscopy: Base: necrotic tissue and polymorph exudate overlying inflamed granulation tissue which merges with mature fibrous (scar) tissue Neutrophils within gastric glands → active inflammation + presence of H pylori (mostly)
 	  

Chronic Peptic Ulcer

Clinical feature

-for duodenal ulcer-

IMPORTANT

Gnawing-عضه-or burning sensation.

Relieved by food or antacids -فوار-

01 **Epigastric pain** (Most common symptom).

02

03 Occurs 2-3 hours after meals

04

05 Patient awakens from pain at night

Complication

IMPORTANT

- Frank hemorrhage that will lead to iron deficiency anemia
- Penetration ،The ulcer penetrates the full thickness of duodenal wall or stomach, Progression into adherent underlying tissue (pancreas or liver).
- Perforation = peritonitis.
- Fibrous stricture in stomach: ulcers may cause pyloric stenosis
- Malignant change (extremely uncommon).

Treatment

- H.pylori eradication by antibiotics .
- Acid suppression by proton pump inhibitors or H2 blockers.

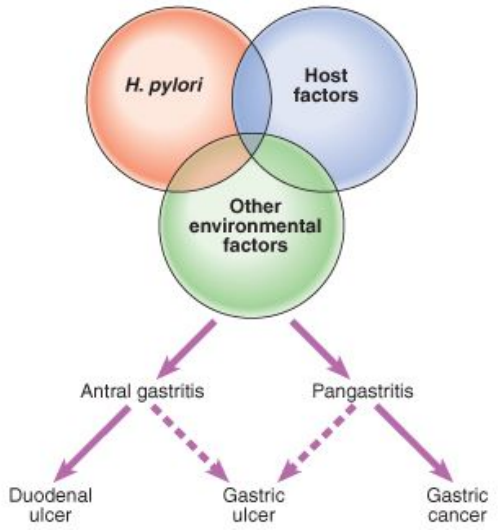
Extra table

Duodenal Ulcer	Gastric Ulcer
Food relieves pain	Food aggravates pain
Epigastric pain 2-3 hours after meal	Epigastric pain shortly after meal
Vomiting not common	Vomiting Is common
Patient awakens with pain at night	Rarely occurs at night

Chronic peptic ulcer

Pathophysiology

	Gastric Ulcers	Duodenal Ulcers
H. pylori (70% of patients)	✓ 75% of patients	✓ All patients
NSAIDs	✓	✘
Duodeno-gastric reflux (bile)	✓	✘
Hyperacidity	✘	✓

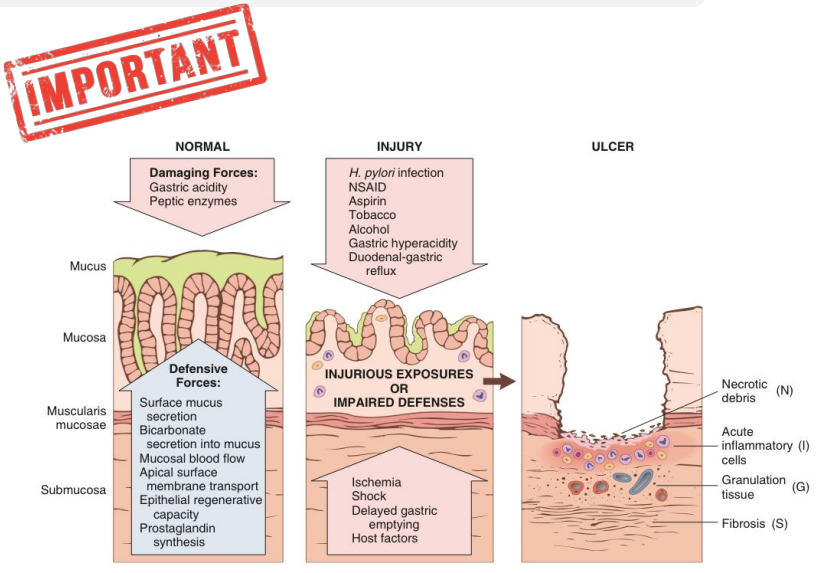


H. pylori infection of the pyloric antrum is present in nearly all patients with chronic duodenal ulcer and approximately 75% of patients with chronic gastric ulcer.

Although more than 70% of individuals with PUD are infected by *H. pylori*, fewer than 20% of *H. pylori*–infected individuals develop peptic ulcer.

Imbalance between aggressive factors & defensive factors:

Aggressive Factors	Defensive Factors
H. pylori	Mucus
Drugs (**NSAIDs**)	Bicarbonate (HCO3-)
Acid	Blood flow & Cell renewal
Pepsin	**Prostaglandins**
Bile salts	Phospholipid



Chronic gastric ulcer

Pathophysiology

The mucosal defenses against acid attack consist of:

1 The surface epithelium

A. NSAIDs (blocking the synthesis of the prostaglandins)
B. *H. pylori* infection (cytotoxins, phospholipases and urease with ammonia production)

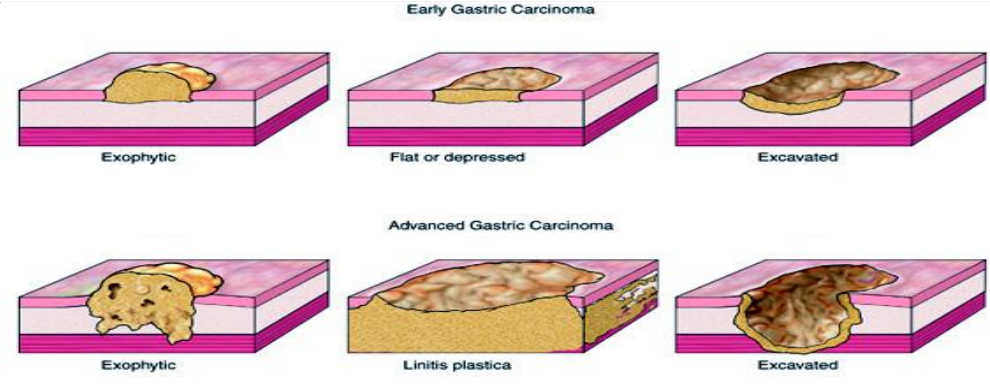
Mucus-bicarbonate barrier 2

A. Duodeno-gastric reflux (bile)

In Peptic ulcers of the stomach, breakdown of mucosal defense is much more important than excessive acid production.

Other causes:

- NSAID and aspirin stop prostaglandin synthesis
  The protective effects of prostaglandins: enhanced bicarbonate secretion and increased vascular perfusion.
- High-dose corticosteroids, which suppress prostaglandin synthesis and impair healing.
- Cigarette smoking: impairs mucosal blood flow and healing
- Chronic renal failure, and hyperparathyroidism: associated with hypercalcemia: stimulates gastrin production and therefore increases acid secretion
- Psychological stress (can increase gastric acid secretion)



Chronic peptic ulcer

Helicobacter pylori infection

H. pylori infection of gastric mucosa is present in 100% of patients with duodenal ulcer and 70% of those with gastric ulcer. H pylori infection is a major factor in the pathogenesis of peptic ulcer.

Possible mechanisms of peptic ulcer by H. pylori:

01

H. pylori does not invade the tissue. It induces an intense inflammatory and immune response and increased production of proinflammatory cytokines such as

- IL-1
- IL-6
- Tumor necrosis factor (TNF)
- and most notably IL-8. This cytokine(IL-8) is produced by the mucosal epithelial cells, and it recruits and activates neutrophils.

02

H. pylori secretes:

- **Phospholipases**: Damage surface epithelial cells.
- **Urease**: Breaks down urea to form toxic compounds such as ammonium chloride and monochloramine.

03

H. pylori enhances gastric acid secretion and impairs duodenal bicarbonate production, thus reducing luminal pH in the duodenum. This altered milieu seems to favor gastric metaplasia (the presence of gastric epithelium) in the first part of the duodenum. Such metaplastic foci provide areas for H. pylori colonization.

04

H. pylori lead to thrombotic occlusion of surface capillaries (promoted by a bacterial platelet-activating factor)and produce agents, including lipopolysaccharides, recruit inflammatory cells to the mucosa.

The chronically inflamed mucosa is more susceptible to acid-peptic injuries and peptic ulceration.

Chronic peptic ulcer

Possible mechanisms of peptic ulcer by H. pylori: (cont'd)

05 In addition, chronic inflammation of the mucosa is possibly important in the pathogenesis of gastric adenocarcinoma and a low-grade gastric lymphoma, also known as MALToma (MALT: Lymphoma of Mucosa-Associated Lymphoid Tissue)

Pathophysiology of duodenal ulcers

- Increased **production of acid** assumes more importance in the pathogenesis of duodenal ulceration.
- H. pylori-infected individuals secrete 2-6 times as much acid as non-infected controls.
- H. pylori does not colonise normal duodenal epithelium.
- H. pylori is involved in duodenal ulceration because there is **gastric metaplasia** in response to excess acid.
- Gastric metaplasia paves the way for colonisation by H. pylori.

Increased production of acid + **Helicobacter P (H. Pylori)** = **Duodenal ulcers**

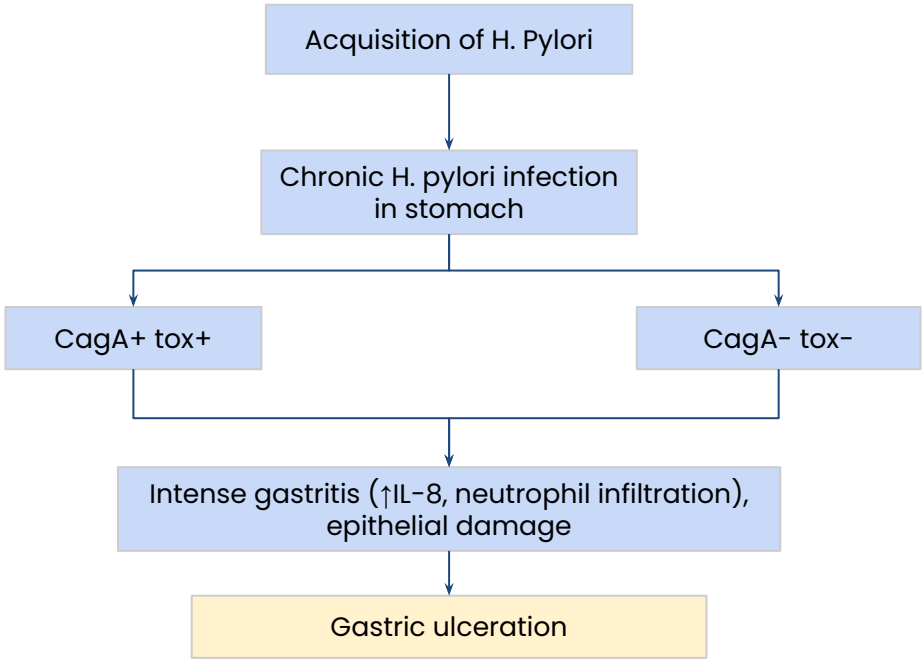
Clinical Note

Gastric ulcers occur in the alkaline-producing mucosa of the stomach, usually on or close to the lesser curvature. A chronic ulcer invades the muscular coats and, in time, involves the peritoneum so that the stomach adheres to neighboring structures. An ulcer situated on the posterior wall of the stomach may perforate into the lesser sac or become adherent to the pancreas. Erosion of the pancreas produces pain referred to the back. The splenic artery runs along the upper border of the pancreas, and erosion of this artery may produce fatal hemorrhage. A penetrating ulcer of the anterior stomach wall may result in the escape of stomach contents into the greater sac, producing diffuse peritonitis. The anterior stomach wall may, however, adhere to the liver, and the chronic ulcer may penetrate the liver substance.

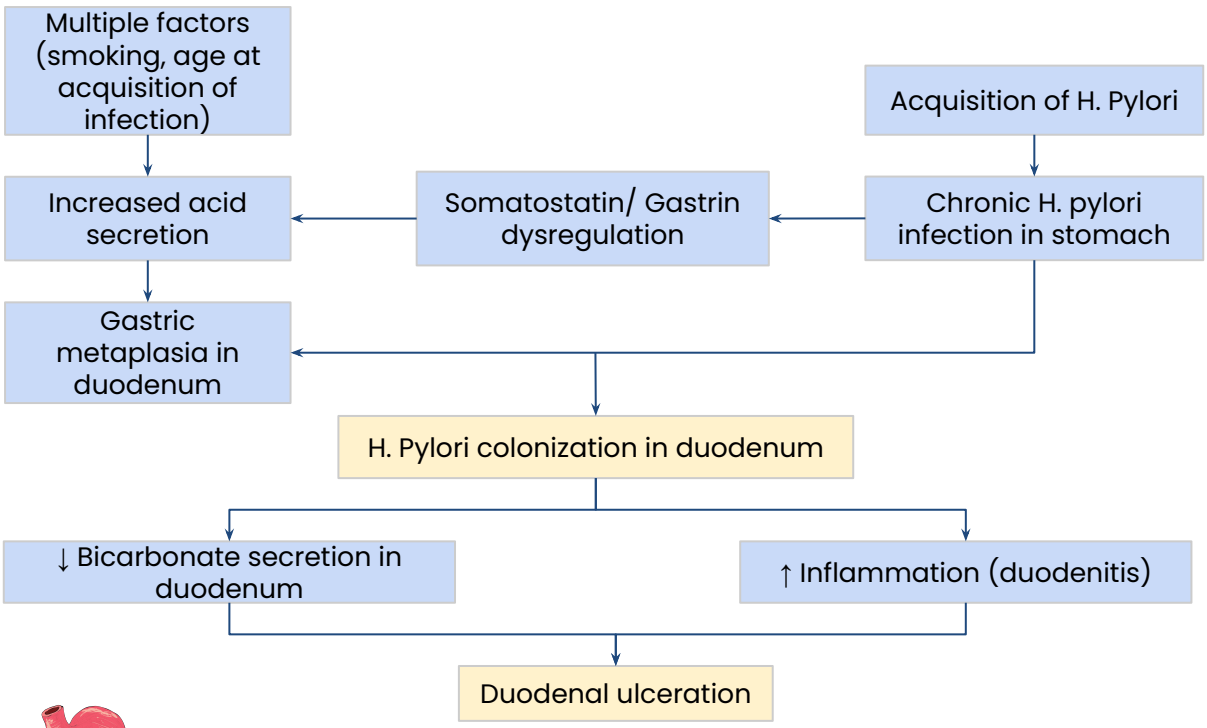
Chronic peptic ulcer

Female Slides

Pathophysiology of chronic peptic ulcers in stomach



Pathophysiology of chronic peptic ulcers in Duodenum



DR. MAHA CASE & QUESTIONS CLICK HERE

Keywords

<table>
<tr><td>Acute Peptic Ulcer</td><td>

consumption of NSAID (block PG synthesis) , Alcohol
Stress
Severe burns (Curling ulcer)
Cerebrovascular accidents (Cushing ulcer)
Zollinger-Ellison syndrome
Iron deficiency
Melena

</td></tr>
<tr><td>Chronic</td><td>

Gastric peptic ulcers: interface of body + antrum at lesser curvature.
Duodenal ulcers: first portion of duodenum (more common)
Meckel diverticulum
Benign Peptic Ulcer : sharply demarcated punched-out defect, with straight walls, surrounded by hyperemia.
Malignant Peptic Ulcer (Cancer, rare): heaped-up margins
Epigastric pain (can be seen in acute)
Dyspepsia
Radiology : free air in left dome of diaphragm
Occurs 2-3 hours after meals
Relieved by food or antacids (Especially duodenal)
iron deficiency anemia
Patient with rheumatoid arthritis (prolong NSAIDs use)
Bleeding
Aggressive Factors : H. pylori , Drugs (NSAIDs) , Acid , Pepsin , bile salt

<table>
<tr><th>Gastric Ulcers</th><th>Duodenal Ulcers</th></tr>
<tr><td>

H. pylori In 70% of patient
associated with use of NSAIDS
associated with Duodeno-gastric relux (bile)
Not associated with Hyperacidity

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H. pylori In all patient
Not associated with use of NSAIDS
Not associated with Duodeno-gastric relux (bile)
associated with Hyperacidity

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IF YOU WANT A SUMMARY CLICK HERE

YOU Vs MCQs

1

Which syndrome is characterized by increased gastrin production and multiple peptic ulcers?

A- Zollinger-Ellison syndrome

B- Cushing syndrome

C- Curling syndrome

D- Meckel diverticulum syndrome

2

Which of the following is a complication of chronic peptic ulcers?

A- Perforation

B- Iron deficiency anemia

C- Malignant change

D- all the above

3

Which of the following is NOT a defensive factor against peptic ulcers?

A- Mucus

B- Acid

C- Prostaglandins

D- Good blood flow

4

What is the main cause of peptic ulcers in patients taking NSAIDs?

A- Increased acid production

B- Decreased mucus production

C- Reduced blood flow

D- Inhibition of prostaglandin synthesis

1-A / 2-D / 3-B / 4-D

YOU Vs MCQs

5

What is the most common location for duodenal ulcers?

- A- Stomach
- B- First portion of the duodenum
- C- Pyloric valve
- D- Anterior duodenal wall

6

What is the characteristic symptom of a duodenal ulcer?

- A- Hematemesis
- B- Melena
- C- Epigastric pain relieved by food
- D- Malignant

7

Which factor is responsible for the breakdown of mucosal defense in gastric ulcers?

- A- Excessive acid production
- B- NSAIDs stoppage
- C- PPI use
- D- Duodeno-gastric reflux

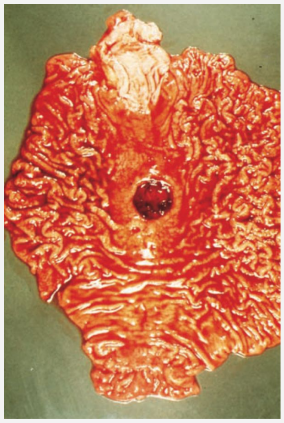
8

What is the gross feature of benign peptic ulcer?

- A- On greater curvature
- B- heaped-up margins
- C- Punched out appearance
- D- more than 2 cm diameter

5-B / 6-C / 7-D / 8-C

YOU Vs CASES

1. A 50-year-old woman with long-standing rheumatoid arthritis complains of weakness and fatigue. She states that her stools have recently become black after taking a new nonsteroidal anti-inflammatory drug (NSAID). Gastroscopy shows numerous superficial, bleeding mucosal defects. Which of the following is the most likely diagnosis?			
A.Acute erosive gastritis	B.Early gastric cancer	C.Helicobacter pylori gastritis	D.Ménétrier disease
2.A 40-year-old woman presents with a 2-month history of burning epigastric pain that usually occurs between meals. The pain can be relieved with antacids or food. The patient also reports a recent history of tarry stools. She denies taking aspirin or NSAIDs. Laboratory studies show a microcytic, hypochromic anemia (serum hemoglobin = 8.5 g/dL). Gastroscopy reveals a bleeding mucosal defect in the antrum measuring 1.5 cm in diameter. An endoscopic biopsy shows that the lesion lacks mucosal lining cells and is composed of amorphous, cellular debris and numerous neutrophils. Which of the following is the most important factor in the pathogenesis of this patient's disease?			
A.Achlorhydria	B.Autoimmunity	C.Gastrinoma	D.Helicobacter pylori infection
3.A 58-year-old woman suffers a massive stroke and expires.The stomach at autopsy is shown in the image. Prior to her death, this patient would most likely have exhibited which of the following? 			
A.Dysphagia	B.hyposecretion of gastric acid	C.Melena	D.Steatorrhea

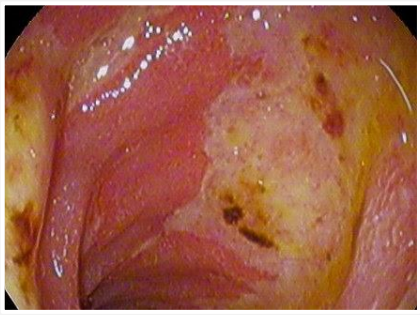
1-A / 2-D / 3-C

NEED EXPLANATION ? CLICK HERE

YOU Vs CASES

EXTRA CASES MAY REQUIRE EXTRA INFO

1.A 37-year-old man presents to the primary care physician with a several month history of burning epigastric pain approximately 3 hours after eating. The patient reports he is often woken up at night with abdominal discomfort and nausea. He does not have any previous medical problems and does not take any medications. The patient undergoes endoscopy, which demonstrates the following pathologic lesion. Which of the following is the most likely complication of this disease process?



A.Diffuse lesion with raised margins and distant metastasis	B.Postprandial pain	C.Gastric outlet obstruction due to scarring	D.Grossly thickened stomach wall

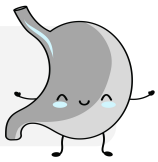
2.A 37-year-old woman presents to the primary care clinic due to three days of severe abdominal pain, bloating, and increased belching. The pain has a burning sensation and is located in the upper abdomen. It is the worst in the morning and after meals. No family members have similar symptoms. Past medical history is significant for recurrent migraines, for which the patient takes high-dose naproxen several times per week. Vital signs are unremarkable. On physical examination, there is moderate tenderness to palpation in the epigastrium. Stool antigen testing is negative for Helicobacter pylori infection. Which of the following medications should be given at this time?

A.Omeprazole	B.Calcium carbonate	C.Amoxicillin, clarithromycin, and pantoprazole	D.Calcium carbonate and tramadol for migraines

1-C / 2 - A

Pathology Team

Leader زياد العتيبي



Leader لمى العتيبي

عائشة إبراهيم	عبدالرحمن المسلّم	نورة المحيميد
الدانة عبدالله	رغد المصلح	ليان الرويلي
ريما المطيري	فيصل الشويعر	ريماز المحمود
زياد حكمي	سلطان البقمي	عروب المحمود
خالد الرشيد	عبدالله الضويحي	الجوهرة الوهيبي
لؤي الحديثي	هيا الزير	عبدالله الكودري
محمد السلامه	معاذ الحضيف	إيلاف معتبي
يوسف بادغيش	ساره العجاجي	يزيد ال طلحه
أفنان الأحمري	رند ابا الخيل	رزان السطيحي
هدى بن جدعان	منصور العتيبي	زياد السويلم
محمد العرفج	دانه المحيسن	عبدالرحمن الأحيدب
نوره المالك	دينا المهوس	عبدالمحسن الدايل
شوق الخليفة		