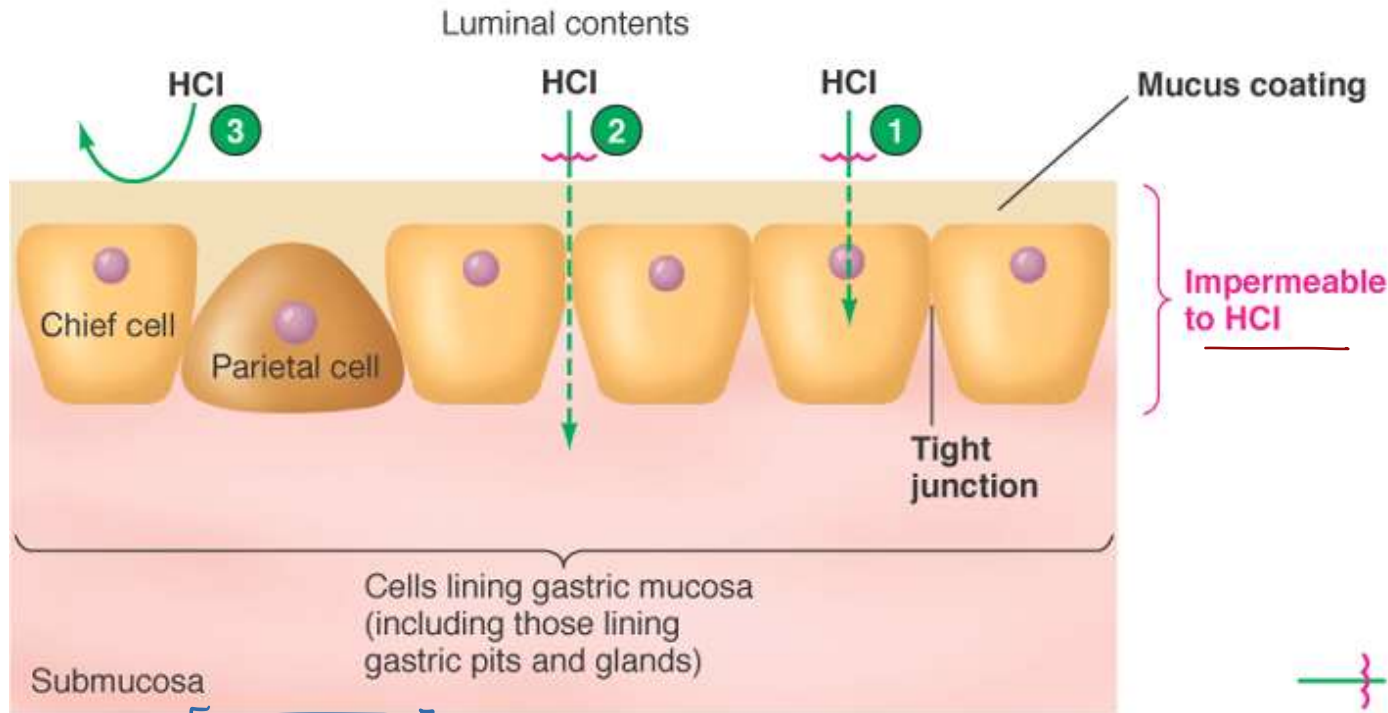


الأشياء إلى حكت المكتورة
نركز عليهم +
سنوات واسلته
فخمة *



Pathophysiology-GASTROINTESTINAL DISEASES
Faculty of Pharmaceutical Sciences
Dr. Amjaad Zuhier Alrosan, Dr. Abdelrahim Alqudah

The gastric mucosal barrier



The gastric mucosal barrier made up of the following components enables the stomach to contain acid with injuring itself:

- 1 The luminal membranes of the gastric mucosal cells are impermeable to H^+ so that HCl cannot penetrate into the cells.
- 2 The cells are joined by tight junctions that prevent HCl from penetrating between them.
- 3 A mucus coating over the gastric mucosa offers further protection.

→ } → = Passage prevented



Gastritis is an inflammation of the stomach that may occur in many forms.

➤ Acute Gastritis

The gastric mucosa ^{متورع} is inflamed and appears red and edematous. It may be ^{متقرع} ulcerated and bleeding if the mucosal barrier is severely damaged or the circulation is poor, which reduces tissue resistance.

Acute gastritis may result from:

- Infection by many types of microorganisms (e.g., bacteria and viruses).
- Allergies to foods such as shellfish or drugs.
- Ingestion of spicy or irritating foods, such as hot peppers.
- Excessive alcohol intake.
- ★ Ingestion of aspirin or other NSAIDs (especially on an empty stomach). <sup>Aspirin
Ibuprofen</sup> they inhibit COX enzyme
- Ingestion of corrosive or toxic substances.
- Radiation or chemotherapy.

Question 32 / 40

Which of the following is **CORRECT** about the effect of **NSAIDs** on **stomach**?

1. ☐ Decrease gastric acid secretion.
2. ☐ Decrease prostaglandin secretion.
3. ☐ Increase gastric acid secretion.
4. ☐ A&B
5. ☒ B&C

Next

إضافة ملاحظة

□ Acute gastritis

Signs and symptoms

فقدان الشهية

- Anorexia, nausea, vomiting.
- Hematemesis indicates ulceration and bleeding in the stomach. ← تقيؤ دموي
- Epigastric pain, cramps, or general discomfort.
- Fever and headache usually accompany infection.
- In some cases, particularly with infections, diarrhea may develop.

In persons with **severe or prolonged vomiting**, there is a danger of dehydration, electrolyte loss, and metabolic acidosis, all of which require supportive treatment.

Certain infections may **require treatment with antimicrobial drugs**.

2

Question 23 / 40

A 55-year-old man presents to the emergency with severe abdominal cramps and vomiting which have been occurring several days ago. His laboratory findings indicate a severe metabolic acidosis and reduction in electrolytes levels. Which of the following conditions do think he has?

1. ☐ Peptic ulcer
2. ☐ Duodenal ulcer
3. ☐ Heart burn
4. ☒ Initial acute gastritis
5. ☐ None of the above

Next

إضافة ملاحظة

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idiopathic

□ Chronic Gastritis

- Characterized by atrophy of the mucosa of the stomach, with loss of the secretory glands.
- The loss of the parietal cells leads to achlorhydria (absence of hydrochloric acid in the gastric secretions)
→ *secrete HCl*
- Chronic gastritis is often seen in individuals with chronic peptic ulcers, those who abuse alcohol, and the elderly. Autoimmune disorders, for example, pernicious anemia are associated with a type of chronic gastric atrophy.
عقبي
فقر دم خبيث
- Many cases are **idiopathic**.

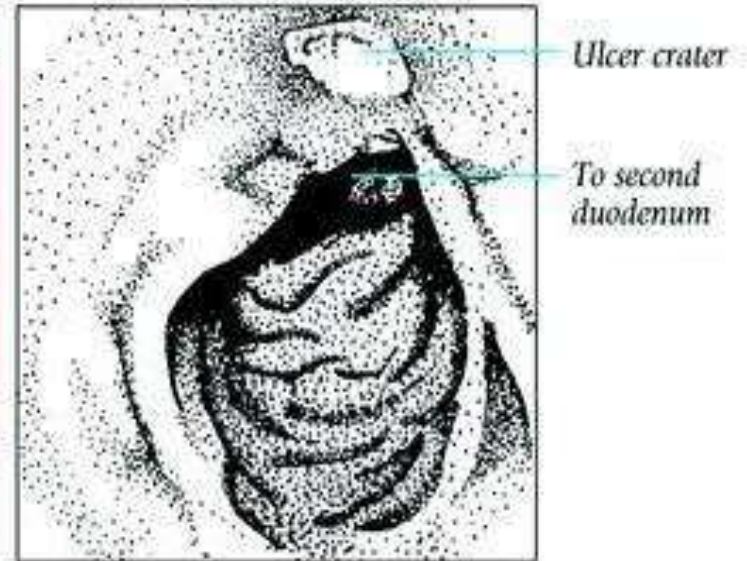
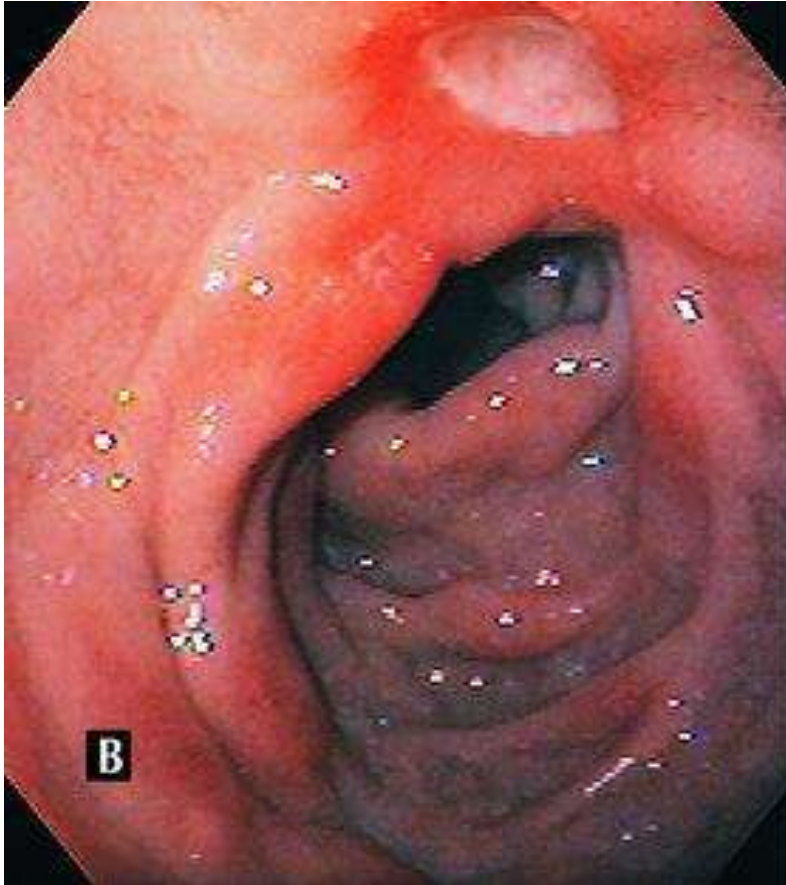
Feature	Acute Gastritis	Chronic Gastritis
Onset	Sudden	Long-term
Acid	Increased injury	Reduced secretion
Appearance	Red, swollen	Atrophic
Parietal cells	Preserved	Destroyed
Achlorhydria	Absent	Present
Bleeding	Common	Less common

Peptic Ulcer Disease



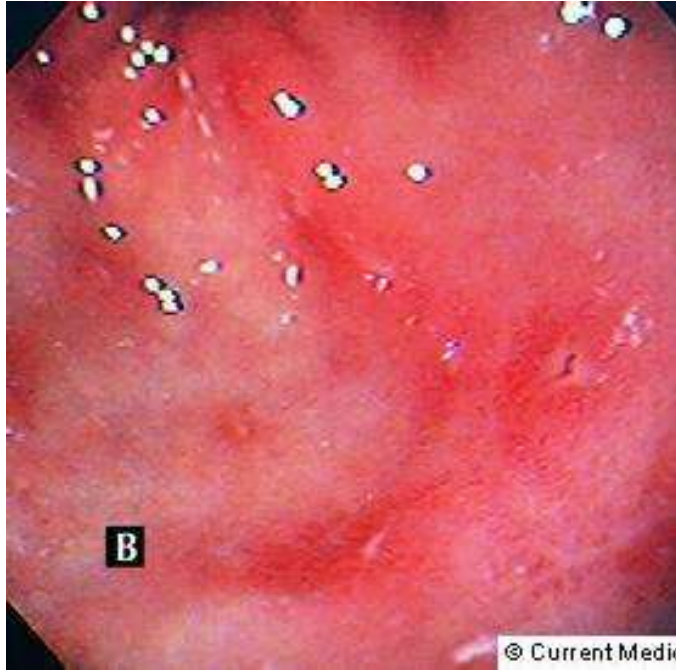
- Break in the gastrointestinal mucosa exposed to gastric acid and pepsin more than 5 mm in diameter.
greater
- Maybe acute or chronic.
- The most common forms of peptic ulcers are duodenal and gastric ulcers.
- Duodenal ulcers occur five times more common than gastric ulcers.
- Duodenal ulcers are more common in males while gastric ulcers affect equally males and females.

Duodenal ulcer



عَرَبِي

EROSION



- A break in the **GI mucosa** - Less than 5 mm in diameter - not penetrating muscularis mucosa. ✓ *not like peptic ulcer disease*
- May occur in **acid-secreting** or **none acid-secreting** mucosa.
- Peristalsis is not affected.
- Heals rapidly.

3

Question 30 / 40

Which of the following is **NOT** correct about gastric erosion?

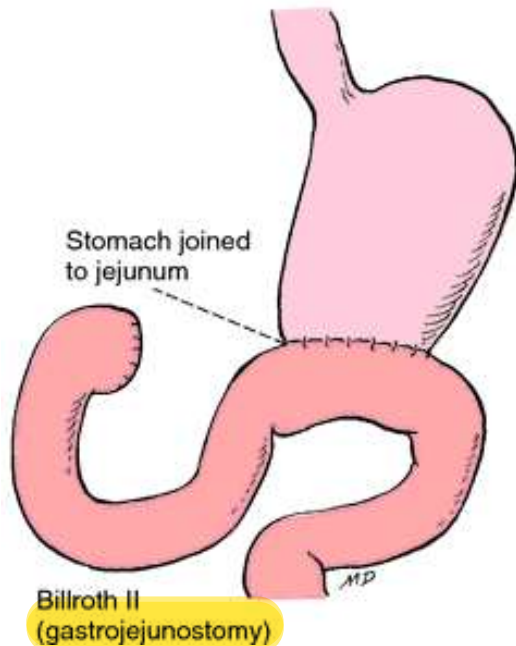
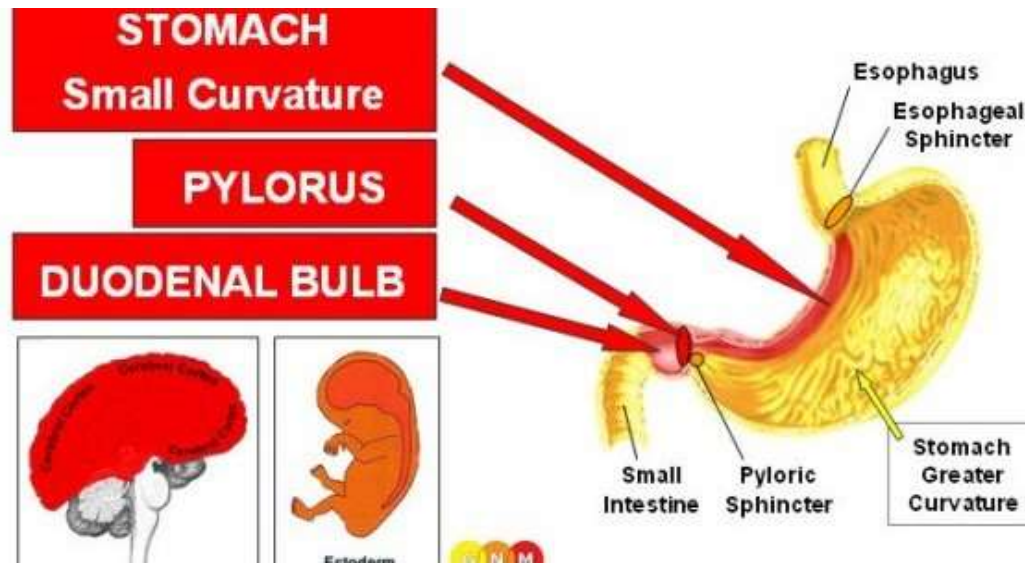
1. ☐ Is a break of less than 5 mm in diameter. ✓
2. ☒ Muscularis mucosa could be partially affected by erosion. ✗
3. ☐ Does not influence the GIT movement. ✓
4. ☐ B&C
5. ☐ None of the above

Next

Sites of PUD

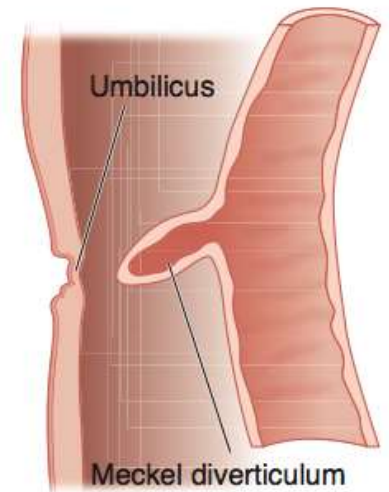
(peptic ulcer disease)

- PUD may occur in any area where acid and pepsin are present.
- Commonest sites:
 - ✓ Duodenum especially first part “duodenal bulb”.
 - ✓ Stomach especially over the lesser curve.
- Other sites:
 - ✓ Lower end of the esophagus.
 - ✓ Site of gastro -jejunal anastomosis. “surgery”
 - ✓ Opposite to Meckel’s diverticulum (congenital diverticulum, is a slight bulge in the small intestine present at birth).



surgical
connection
between the
stomach and
jejunum

Meckel diverticulum



cause or
origin of a
disease.

AETIOLOGY OF PUD

H-pyrole

1. HELICOBACTER PYLORI-ASSOCIATED ULCERS.
2. NSAID-RELATED ULCERS: (Due to inhibition of COX-1, prostaglandin-endoperoxide synthase).
on empty stomach can causes ulcer *inhibit COX-1 prostaglandin* *protective*
3. HYPERSECRETORY STATES: Zollinger-Ellison syndrome is caused by gastrin-secreting tumors in the pancreas or the upper part of your small intestine (duodenum).
4. IDIOPATHIC.

What is the primary mechanism by which NSAIDs contribute to the formation of peptic ulcers?

- A. Stimulation of parietal cells to overproduce HCl
- B. Activation of *H.pylori* colonization
- C. Inhibition of $COX - 1$ and reduction of prostaglandin synthesis
- D. Direct chemical corrosion of the mucosal epithelium



وجابت هدول وکان بدھا الفولس

activation of prostaglandin
بس کان فی خیار
هو الجواب

AETIOLOGY OF PUD

1. HELICOBACTER PYLORI-ASSOCIATED ULCERS.
2. NSAID-RELATED ULCERS: (Due to inhibition of COX-1, prostaglandin-endoperoxide synthase).
3. HYPERSECRETORY STATES: Zollinger-Ellison syndrome is caused by gastrin-secreting tumors in the pancreas or the upper part of your small intestine (duodenum).
4. IDIOPATHIC.

→ how a disease develop

Pathogenesis of PUD

IMBALANCE
BETWEEN



- Acid.
- Pepsin.

AGGRESSIVE

AND



DEFENSIVE FACTORS

- Prostaglandins.
- Mucosal blood flow.
- Mucus gel layer adherent to the mucosal surface.
- Bicarbonate.
- Tight epithelial junctions.
- Regeneration of the epithelial cell layer.
- Growth factors, eg: EGF.

epidermal growth Factor

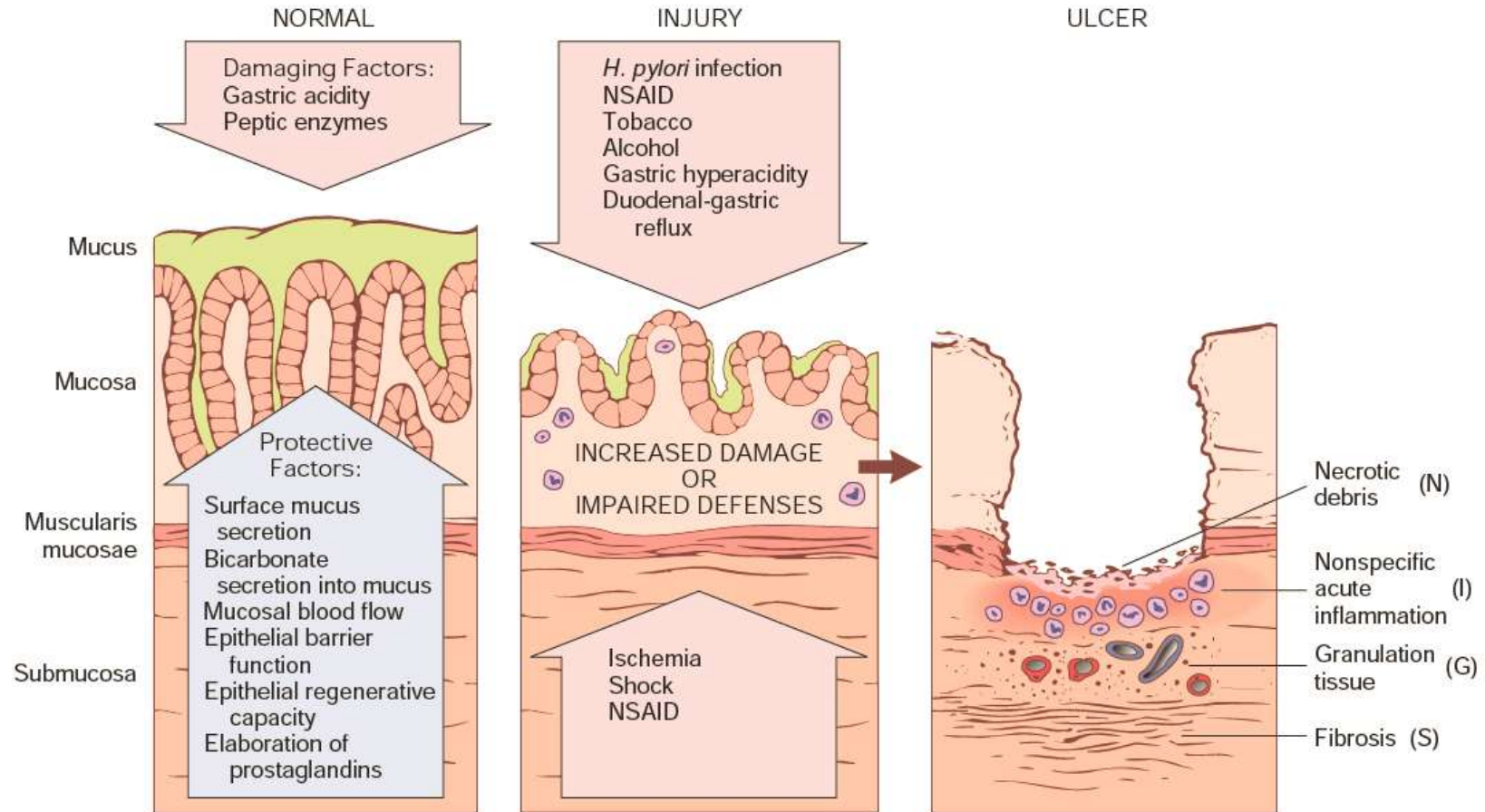


Figure 17-11 Mechanisms of gastric injury and protection. This diagram illustrates the progression from more mild forms of injury to ulceration that may occur with acute or chronic gastritis. Ulcers include layers of necrosis (N), inflammation (I), and granulation tissue (G), but a fibrotic scar (S), which takes time to develop, is only present in chronic lesions.

Epidemiology of PUD

Epidemiology

noun

the scientific study of diseases and how they are found, spread, and controlled in groups of people.

Prevalence

انتشار

Measures **existing** cases of disease and is expressed as a **proportion**

• Prevalence of about 5-10%.

- Varies in different communities.
- Higher prevalence in low socioeconomic classes and with certain diseases.

duodenum ulcer

- **DU more in males: M/F: 3:1.** ★

gastric ulcer

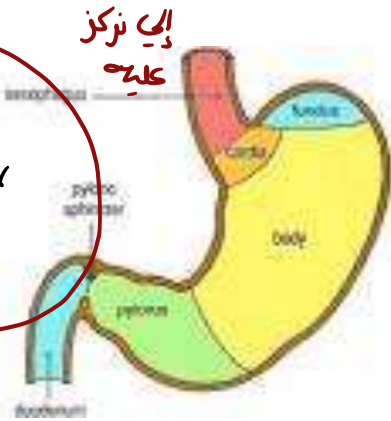
- **GU is equal in both sexes** but increases with age.

- **FAMILY HISTORY:** 3-4 increased risk.
- **CIGARETTE SMOKING:** ulceration increased.
- **EMOTIONAL DISTURBANCES and STRESS:** increase gastric acid secretion.

Fundus

Stress Ulcers

curling ulcer
- burns
cushing ulcer
- head injury
- brain surgery



- Develop in response to major physiologic stress.
- People at high risk for the development of stress ulcers include:
 1. patients with **large-surface-area burns** (*Curling's ulcer*).
 2. patients exposed to **trauma, sepsis, acute respiratory distress syndrome, severe liver failure, and major surgical procedures**.
 3. **People admitted to hospital intensive care units**.
- Location of stress ulcers is **in the fundus** and body of the stomach and are thought to result from ischemia to the mucosal tissue and alterations in the gastric mucosal barrier.

*Another form of stress ulcer, called *Cushing ulcer*: intracranial injury, operations, or tumors.
-brain

Which type of stress ulcer is specifically associated with intracranial injuries or tumors?

A. Marginal ulcer

B. Cushing ulcer

C. Curling ulcer

D. Zollinger-Ellison ulcer

4

Question 29 / 40

Which of the following is **NOT** correct about PUD?

1. ☒ Increase the blood flow to GIT increase the risk of PUD. ✗
2. ☐ Decrease EGF levels increase the risk of PUD. ✓
3. ☐ Increase pepsin secretion increases the incidence of PUD. ✓
4. ☐ Gastro jejunal anastomosis in one of the rare sites for PUD. ✓
5. ☐ All the above.

Next

ملاحظة

CLINICAL PICTURE OF PUD

- Signs and Symptoms of PUD:
 - **Epigastric pain.**
 - **Dyspepsia** (Belly pain or discomfort, Bloating, Feeling uncomfortably full after eating, Nausea, Loss of appetite, Heartburn).
 - May be asymptomatic.



Exam Tip: Symptoms alone are not enough to diagnose PUD.

Diagnosis of PUD



- ✓ Clinical picture is suggestive but not diagnostic.
- ✓ Diagnosis best by **endoscopy**.
- ✓ **Barium meal** is **less helpful** (infusion of the contrast medium **barium sulfate**, a radioopaque salt, coats the lining of the digestive tract, allowing accurate X-ray imaging of the esophagus, stomach, and small bowel).
- ✓ **Serum gastrin**; indicated if **Zollinger-Ellison syndrome** is suspected.
- ✓ Evaluation for **H. pylori** infection.
- ✓ Gastric ulcer should be biopsied to exclude malignancy.

حزاعه

درم خست

Ulcer Type	Need Biopsy?
Gastric ulcer	Yes
Duodenal ulcer	Usually no

5

Question 25 / 40

A 42-year-old male presents to the emergency with heart burn and bloating. If you are his doctor, what is the test you do to diagnose his condition?

- 1. ☐ Barium meal test is the priority in this case. x
- 2. ☐ Serum gastrin could not be helpful. x
- 3. ☐ Urea breath test should be done. ✓
- 4. ☐ Endoscopy is the best procedure. ✓
- 5. ☐ C&D

Next

إضافة ملاحظة

Which ulcer must always be biopsied?

- A. Duodenal ulcer
- B. Gastric ulcer
- C. Esophageal ulcer
- D. Stress ulcer

Answer: B. Gastric ulcer

Elevated serum gastrin levels are most suggestive of:

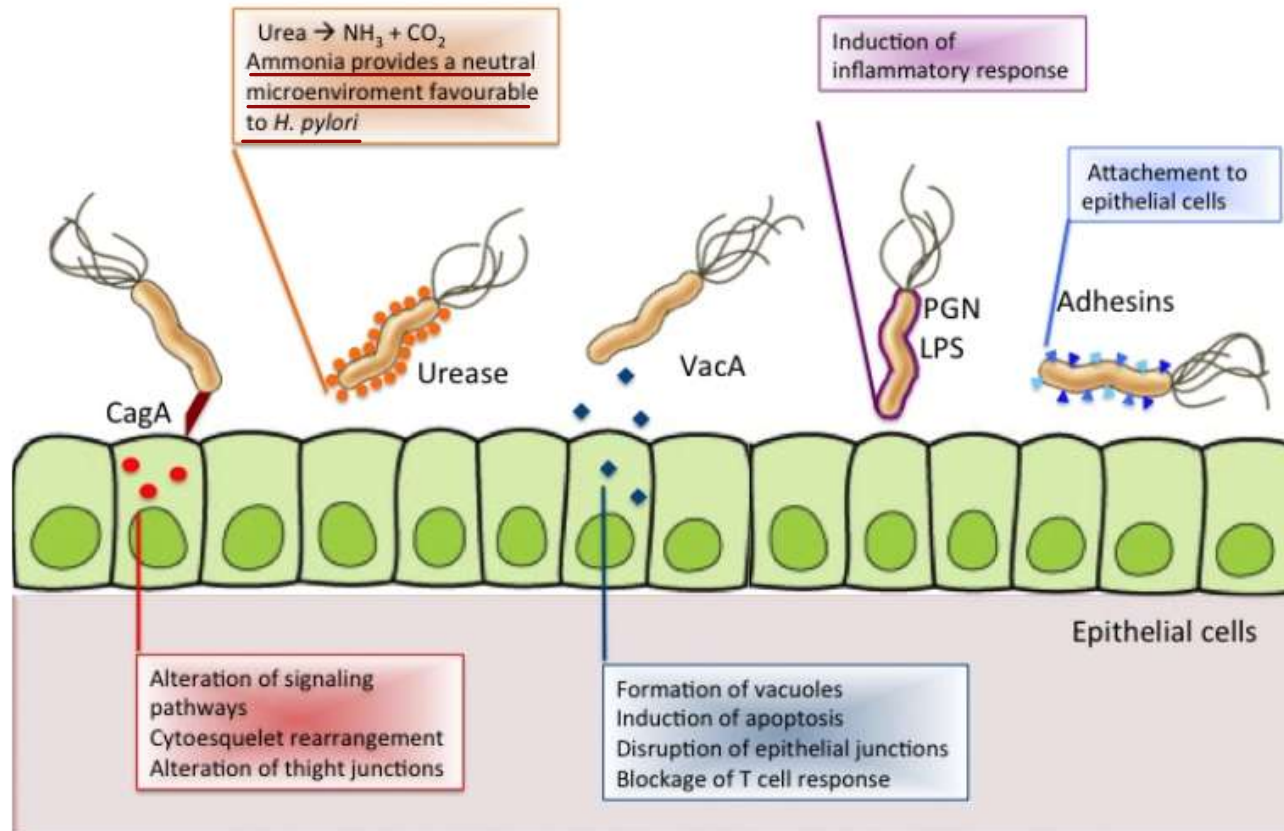
- A. GERD
- B. H. pylori infection
- C. Zollinger–Ellison syndrome
- D. Gastritis

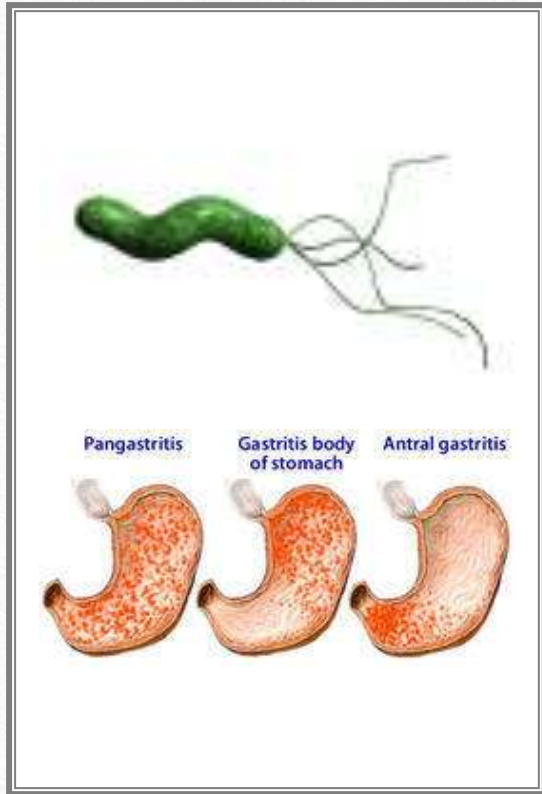
Answer: C. Zollinger–Ellison syndrome

Helicobacter pylori

H. pylori

Gastric Lumen





Helicobacter Pylori ↷

- Bacteria ^{negative}
— [Gram -ve spiral bacterium.]
— 40% of patients >60 yrs are +ve for H.pylori.
— Transmitted: possibly person to person.
— Most common cause of **antral gastritis** (antrum part is the last part of the stomach toward the duodenum).

Helicobacter Pylori

Flagella
Movement

Urease
Survival

VacA
Cell damage

CagA
Cancer risk

Gastrin stimulation
Increased acid secretion

urea
breathing
test

- Mechanism of gastric injury:
 - Breakdown of mucosal defenses. ✓
 - Increase gastrin-releasing peptide (GRP).
 - Decrease bicarbonate secretion.
 - Flagella, which allows the bacteria to be motile in viscous mucus.
 - Urease, which generates ammonia from endogenous urea and thereby elevates local gastric pH and enhances bacterial survival.
$$\text{NH}_3 + \text{CO}_2$$
 - Adhesins that enhance bacterial adherence to surface foveolar cells.
 - Toxins, such as cytotoxin-associated gene A (CagA), may be involved in disease progression (such as induction of gastric cancer).

Role of H.pylori in GI diseases

	<u>Healthy subjects</u>	20-50%
✱	<u>Chronic active gastritis</u>	100%
✱	<u>Duodenal ulcer</u>	>90%
	<u>Gastric ulcer</u>	50 - 80%
✱	<u>Gastric adenocarcinoma</u>	90%
	<u>Gastric lymphoma</u>	85%

Diagnosis of Helicobacter pylori infection

Invasive(through endoscopy):

- Gastric biopsy and staining.
- Culture of the bacterial specimen.
- Tests using urease enzyme in bacterial specimens.

Non-invasive:

- Urea breath test.
- H. pylori antibodies.
- Stool antigen.
- Salivary antigen.

Table 17-4 Complications of Peptic Ulcer Disease

Bleeding

Occurs in 15% to 20% of patients

✦ Most frequent complication

✦ May be life-threatening

Accounts for 25% of ulcer deaths

May be the first indication of an ulcer

تشقيب

Perforation *abnormal opening in an organ*

Occurs in up to 5% of patients

Accounts for two thirds of ulcer deaths

Is rarely first indication of an ulcer

Obstruction

Mostly in **chronic ulcers**

Secondary to edema or scarring

Occurs in about 2% of patients

Most often associated with pyloric channel ulcers

May occur with duodenal ulcers

Causes incapacitating, crampy abdominal pain

Can rarely cause total obstruction and intractable vomiting

TREATMENT OF PEPTIC ULCER DISEASE

- AIM OF TREATMENT:
 - RELIEVE SYMPTOMS.
 - HEAL THE ULCER.
 - PREVENT COMPLICATIONS.
 - PREVENT RECURRENCES.

جابو سؤال عن antacid drugs كان عبارة عن
مقارنة بين دوا من ال ppi ودوا من ال h2blockers
وكان ترو فولس
للامانة مافهمت شو بدو حطيتو فولس

ANTACIDS

- Rapid symptomatic relief.
- Cheap.
- Large amounts are required to heal ulcers leading to undesirable side effects.
- If taken on an empty stomach; they are effective only for 10-20 minutes.
- If taken one hour after meals, they are effective for 2-3 hours.
- Tablet preparations are less effective than suspensions.
more effective

HCl secretion by parietal cells can be stimulated by several sources:

1. **Acetylcholine (ACh)** is released by parasympathetic neurons.
2. **Gastrin** secreted by G cells. stomach
3. **Histamine**, which is a paracrine substance released by mast cells in the nearby lamina propria.

♦ Acetylcholine and gastrin stimulate parietal cells to secrete more HCl in the presence of **histamine**. In other words, **histamine acts synergistically** enhancing the effects of acetylcholine and gastrin. Receptors for all three substances are present in the plasma membrane of parietal cells.

HISTAMINE- RECEPTOR ANTAGONISTS (H2-Blockers)

- Act through blocking H₂ receptors in the parietal cells.
- Suppress nocturnal acid secretion by more than 90%.
- Suppress 24-hour acid secretion by 50-70%.
- Side effects :
 - CNS effects: **headache, mental confusion.**
 - Reversible **gynecomastia** and impotence. breast enlargement ↗ Fertility decrease
 - Interaction with drugs metabolized through hepatic cytochrome P-450 microsomal enzymes

HISTAMINE- RECEPTOR ANTAGONISTS (H2-Blockers)

ہینٹو ا ج ر Dine

CIMETIDINE

RANITIDINE

FAMOTIDINE

NIZATIDINE

H^+ PROTON PUMP INHIBITORS (PPIs)

“ most effective ”
treatment

After Giving a PPI
A PPI irreversibly blocks the proton pump.
So now:
Gastrin → Histamine → Proton pump $\times$ →
Little or no acid
The signal is still being sent, but the final
step is blocked.

Suppress acid secretion by non-competitively and irreversibly inhibiting the H^+ , K^+ - ATPase of the gastric parietal cells. *proton pump*

Inhibit over 90% of 24-hour acid secretion.

Increase secretion of gastrin usually 2-3 times the baseline with proliferation and growth of ECL cells.

استشار

No carcinoid tumors were reported occurring in men due to PPIs.

Heal 50% of DUSs by 2 weeks, 90% in 4 weeks, and almost all by 6-8 weeks.

PROTON PUMP INHIBITORS (PPIs)

PPIs (azole)

- لا تحفظ جرعات -

- Omeprazole: 10, 20 mg
- Lansoprazole: 15, 30 mg
- Pantoprazole: 20, 40 mg
- Rabeprazole: 10, 20 mg
- Esomeprazole: 20, 40 mg
- Tenatoprazole: 40 mg: longer duration of action

” مهم نعرفهم بالأمحاح “

في الجسم الي

- In vivo, sensitive to the following agents:

- amoxycillin

- tetracyclin

- clarithromycin

- Metronidazole, tinidazole

میں زخم میں
PPIs drugs

- bismuth

- PPIs مهم

- **Second line drugs:** Levofloxacin,
gatifloxacin, rifabutin

” استئصال “

Eradication therapy for H.Pylori

جابت سؤال عن دوا ما بيعالج ال H pylori
وحاطة كل الادوية الي بتعالجهم الا واحد
طبعاً فيه فخ حاطة نوع دوا من ppi

Surgery for PUD

peptic ulcer disease

- Rare after the introduction of effective therapeutic agents except for complications.

6

Question 26 / 40

Which of the following is **NOT** correct about *H. pylori*?

1. ☐ PPIs are part of its treatment. ✓
2. ☒ It dose not increase the risk of gastric carcinoma.
3. ☐ C&D
4. ☐ It could be transmitted from person to another. ✓
5. ☐ It could be diagnosed by stool antigen. ✓

Next

إضافة ملاحظة

stomach

Gastroesophageal Reflux Disease (GERD)

REFLUX ESOPHAGITIS

★ lower esophageal sphincter
Failure

- Backward movement of gastric contents into the esophagus causing heartburn (burning chest pain) resulting from recurrent mucosal injury, often worse at night, when lying supine, or after consumption of foods or drugs that diminish lower esophageal sphincter tone.
- Associated with transient relaxations of the weak or incompetent lower esophageal sphincter.

The most common cause of gastroesophageal reflux disease is:

A. Excess gastrin

B. *Helicobacter pylori*

C. Weak lower esophageal sphincter

D. Pepsin deficiency



Clinical Manifestations of GERD

➤ Heartburn:

- A. It is frequently severe,
- B. occurring 30 to 60 minutes after eating.
- C. It often is made worse by bending and lying down
- D. is relieved by sitting upright.
- E. Often, heartburn occurs during the night.



➤ Belching *expelling gas of the stomach*

➤ Chest pain (may be confused with angina).

- 1. is located in the epigastric or retrosternal area and
- 2. often radiates to the throat, shoulder, or back.

left neck shoulder



➤ Respiratory symptoms such as asthma (vagal-mediated bronchospasm), chronic cough, and laryngitis.

bronchoconstriction

- Complications, such as strictures and *Barrett esophagus*.
- **Barrett esophagus** is characterized by a reparative process in which the squamous mucosa that normally lines the esophagus gradually is replaced by abnormal columnar epithelium resembling that in the stomach or intestines. It is associated with increased risk for the development of esophageal adenocarcinoma.



FIGURE 45.4 • The presence of the tan tongues of epithelium interdigitating with the more proximal squamous epithelium is typical of Barrett esophagus. (From Rubin R., Strayer D. S. (Eds.) (2012). *Rubin's pathophysiology: Clinicopathologic foundations of medicine* (6th ed., p. 611). Philadelphia, PA: Lippincott Williams & Wilkins.)

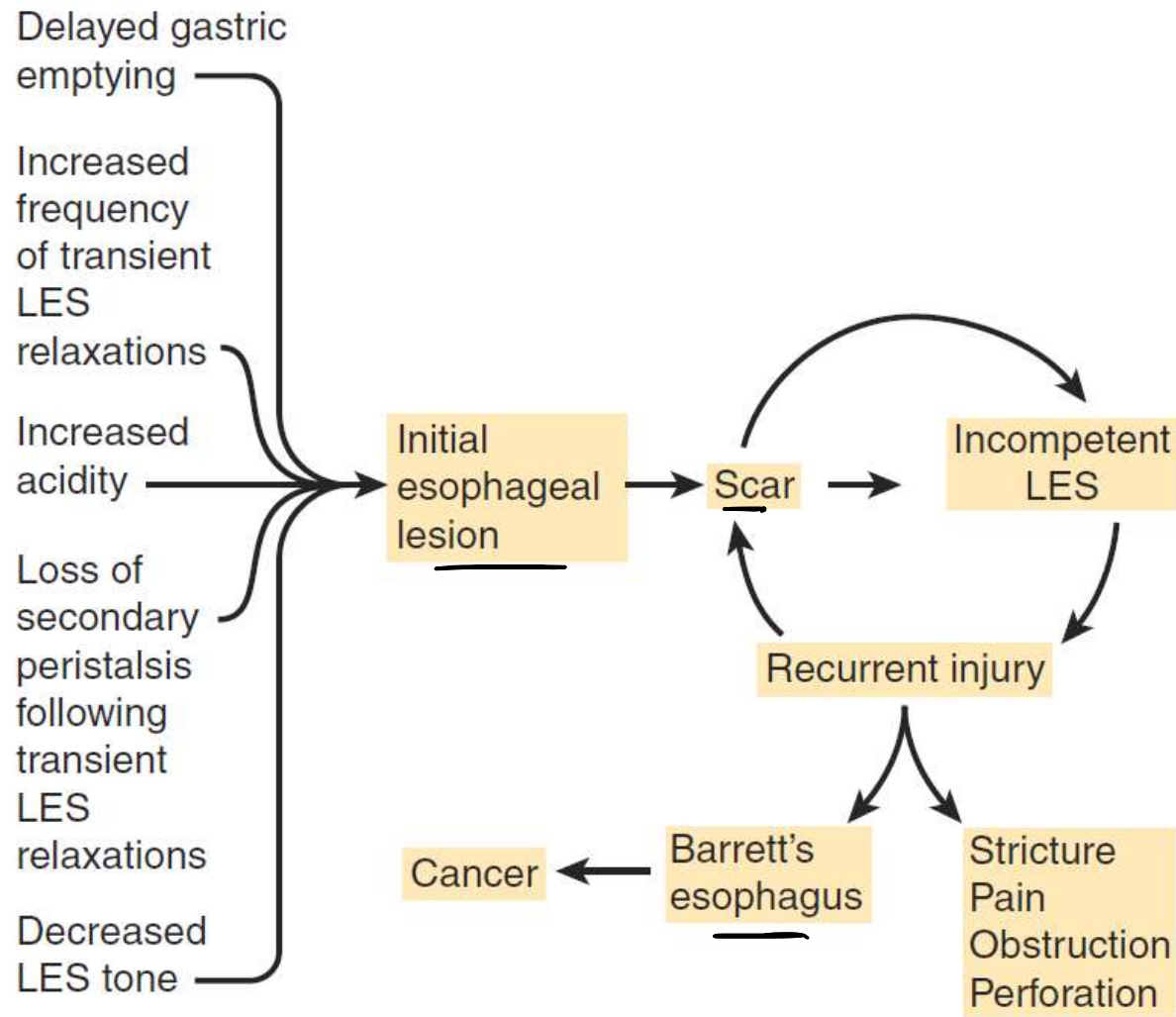


FIGURE 13-17 Pathophysiology of esophageal reflux disease.

TABLE 13-5 Modulators of lower esophageal sphincter pressure.

	Increase Pressure	Decrease Pressure
Hormones	Gastrin	Secretin
	Motilin	Cholecystokinin
	Substance P	Somatostatin
		Vasoactive intestinal peptide (VIP)
		Progesterone
Neural agents	α -adrenergic agonists	β -adrenergic agonists
	β -adrenergic antagonists	α -adrenergic antagonists
	Cholinergic agonists	Anticholinergic agents
Foods	Protein meals	Fat
		Chocolate
		Peppermint
Other	Histamine	Theophylline
	Antacids	Prostaglandins E_2 , I_2
	Metoclopramide	Serotonin

8
pie
 X

- 1" 1/2 -

Treatment

- The treatment of gastroesophageal reflux usually focuses on conservative measures.
- **Avoidance of positions and conditions** that increase gastric reflux.
- **Avoidance of large meals and foods** that reduce lower esophageal sphincter tone (e.g., caffeine, fats, chocolate), alcohol, and smoking.
- **Medication:**
 - **Antacids** or a **combination of antacids and alginic acid**. Alginic acid ^{produce foam} produces foam when it comes in contact with gastric acid; if reflux occurs, the foam rather than acid rises into the esophagus.
 - **Histamine-2 receptor (H2)-blocking antagonists**.
 - ★ • **Proton pump inhibitors**. ★
- Surgical treatment may be indicated in some people.

7

Question 2 / 40

Which of the following is **CORRECT** about GERD?

1. ☐ It increases with decreased relaxation of the lower esophageal sphincter ✕
2. ☐ Its pain is felt only in the epigastric area ✕
3. ☐ Protein meals increase the risk of it ✕
4. ☐ Alginic acid is one of the best treatments for it
5. ☐ C&D

Next

ملاحظة

Cancer of stomach

لكن ممكن يتطور ل
ورم خبيث metastasis

One of the leading causes of death in the united states.

It exhibits a male to female ratio of 2:1.

2 : 1

Risk factors for stomach cancer include:

- Genetic predisposition. ميراث مسبق
- Carcinogens in the diet (nitroso compounds found in smoked and preserved food).
- Autoimmune gastritis. متألمبات chronic gastritis
- مكاف ★ Gastric adenoma and polyps. Autoimmune disease
- Chronic infection by H.pylori act as a cofactor for some carcinomas.

polyps
ورم حميدة

Cancer of stomach

- Unfortunately, **stomach cancers** often are **asymptomatic** until late and early detection is usually difficult.
- Symptoms include:
 - Indigestion.
 - Anorexia nervosa.
 - Weight loss.
 - Epigastric pain.
 - Vomiting.
 - Abdominal mass.

Cancer of stomach

□ Diagnosis is accomplished by:

- Barium x-ray studies.
- Endoscopy and computed tomography (CT)-scan.
- Biopsy and cytologic studies of the gastric secretions. ← خزعة

□ Treatment:

- Radical subtotal gastrectomy is the treatment of choice. - إزالة جزى من المعدة -
total
- Radiation and chemotherapy may be used for palliative purposes or to control the metastatic spread of the disease. تخفيفا

8

Question 31 / 40

Which of the following is **CORRECT** about stomach cancer?

1. ☐ Nitroso compounds are risk factor for it
2. ☐ Indigestion is one of the symptoms for it
3. ☐ Cytologic study could be an approach for its diagnosis
4. ☐ Chemotherapy is a palliative approach for its treatment
5. ☒ All the above

Next

ملف ملاحظة

تَحِبُّ تَجِيبُ عَلَيْهِم
اسئلة كثير
اعمل جدول وقارن

Intestinal disorders

Irritable bowel syndrome

- It is a persistent or recurrent symptom of abdominal pain, altered bowel function, and varying complaints of flatulence, bloating, nausea and anorexia, constipation or diarrhea, and anxiety or depression.
- * The pain is relieved by defecation and associated with a change in consistency or frequency of stools.
- The pain is usually intermittent cramping in the lower abdomen. It does not interfere with sleep.
- It results from dysregulation of intestinal motor and sensory function modulated by CNS. Motility is increased as a response to stress.
- Women are more affected compared to men.

رکزوا علی مقارنت
male / women

Diagnosis:

- Continuous or recurrent symptoms of at least 12 weeks duration with two of three accompanying features: relief by defecation, onset is associated with a change in bowel frequency, and associated with a change in form of stool.
- History of lactose intolerance should be considered.
- Weight loss, anemia, fever, occult blood in the stool or nighttime symptoms, or malabsorption increases the likelihood of organic disease (any disease where observable measurable process such as inflammation and tissue damage).

Treatment

❑ No special diet is indicated but adequate fiber intake is recommended.

❑ Avoidance of offending food such as fatty and gas-producing foods, and alcohol. Caffeine-containing beverages could be beneficial.

مشروبات

❑ Pharmacological therapy includes antispasmodic and anticholinergic drugs.

Question 1 / 40

9

Which of the following is **CORRECT** about Irritable bowel syndrome?

1. ☐ It is not characterised by anxiety and depression ✕
2. ☐ Diarrhea is the most common symptom for it ✕
3. ☐ Recurrent symptoms of at least 2 weeks would be an accurate sign for it ✕
4. ☐ Mebeverine is the most common drug for its treatment ✕
5. ☒ None of the above

Next

ملاحظة

Inflammatory Bowel Disease

➤ These include:

- ☆ • Ulcerative colitis: nonspecific inflammatory bowel disease of unknown etiology that affects the mucosa of the colon and rectum.
- ☆ • Crohn's disease: nonspecific inflammatory bowel disease that may affect any segment of the GIT.
- Indeterminate colitis: 15% of patients with IBD are impossible to differentiate.

Crohn's disease and ulcerative colitis share:

- ✓ Inflammation of the bowel.
- ✓ Lack of confirming evidence of the proven causative agent.
- ✓ Pattern of familial occurrence.
- ✓ Accompanied by systemic manifestation.
- ✓ Crohn's can affect any area of GIT from the esophagus to the anus while ulcerative colitis affects the colon and rectum.
- ✓ In Crohn's, the inflammation is often transmural but ulcerative colitis is typically restricted to the mucosa.



Crohn's disease

Clinical manifestation

- They both result from the activation of inflammatory cells with the elaboration of inflammatory mediators that cause nonspecific tissue damage.
- They characterize by remission and exacerbations of diarrhea, fecal urgency, and weight loss and it may lead to intestinal obstruction as a complication.
- Systemic manifestations include axial arthritis, inflammation of the eye, skin lesions. Inflammation of the mucus membrane of the mouth (stomatitis), blood disorders (anemia and hypercoagulability) and inflammation of the bile duct. In children, it causes growth retardation.

Crohn's disease

Treatment

- Treatments focus on terminating inflammation, promoting healing, maintaining adequate nutrition, and preventing complications.
- Nutritious diet rich in calories, vitamins, and proteins is recommended, fat is better to be avoided.
- Medical therapy included:
 - **Corticosteroids** (suppress acute clinical symptoms).
 - **Metronidazole** (treats bacterial overgrowth in the intestine).
 - **Immunosuppressants** (azathioprine, 6-mercaptopurine, methotrexate and cyclosporine).
 - **Monoclonal antibodies** (anti-TNF agents) for severe disease. (Etanercept, adalimumab, infliximab).

Ulcerative colitis

Risk factors

- **Age:** commonly less than 30. *"women"*
- **Race or ethnicity:** white is at higher risk.
- **Genetic.**
- **Diet (fatty diet) or oral contraceptives** increase the risk of the disease.

أكثر عرضة لـ cancer ←

No caffeine

Ulcerative colitis

Clinical manifestation

- Marked by attacks of diarrhea that may persist for days, weeks, or months and then subside, diarrhea usually with blood.
- Anorexia, weakness, and fatigue.
- In severe disease, fever, anemia, hypovolemia and hypoalbuminemia are common.
- In children, failure to growth
- Cancer of the colon is a common complication of ulcerative colitis.

1.

Question 40 / 40

Arthritis of the axial skeleton is one of the clinical manifestations for **ulcerative colitis**?
of chohn's disease

1. ☐ True
2. ☒ False

إضافة ملاحظة

Ulcerative colitis

Treatment

- ❑ Avoid caffeine, lactose, and gas-forming food.
- ❑ Doctor may recommend high protein, a high-calorie eating plan that is low in fiber.
- ❑ Drugs including corticosteroids and TNF inhibitors can be used.
- ❑ Unlike Crohn's disease, nicotine has shown clinical and histological improvement.
good for ulcerative colitis
- ❑ Unlike Crohn's disease, UC responds to treatment by probiotic therapy.
- ❑ Iron is given to treat anemia resulting from intestinal bleeding.
- ❑ Unlike Crohn's, UC can be cured by surgical removal of the colon.

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Question 20 / 40

Which of the following is NOT correct about the difference between ulcerative colitis and crohn's disease?

1. ☐ Nicotine could be beneficial for ulcerative colitis only ✓
2. ☐ Probiotic therapy could improve both diseases ✗
3. ☐ Both diseases could be treated surgically ✗
4. ☐ B&C
5. ☐ All the above

Next

إضافة ملاحظة

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Question 35 / 40

Low fibre diet is beneficial in all intestinal disorders.

1. ☐ True
2. ☒ False

- beneficial in "ulcerative colitis"
But not in irritable bowel syndrome

Next

Which of the following treatments is specifically noted for showing clinical improvement in Ulcerative Colitis, but not in Crohn's disease?

A. TNF inhibitors

B. Corticosteroids

C. Metronidazole

D. Nicotine



Colorectal carcinoma

- Colorectal cancer affects about 1 million persons worldwide.

It peaks at 60-70 years of age in patients with:

- Family history of cancer.
- Persons with Crohn's disease or ulcerative colitis.
- Persons with familial adenomatous polyps of the colon.

- Diet including fat (increase bile salt synthesis) and refined sugar are harmful, while fiber intake (increases stool bulk and removes carcinogens) and vitamins such as vitamin A, C, and E are of great benefit in neutralizing carcinogens.

- Aspirin has shown some benefit in prevention by inhibiting COX-2 which is overexpressed in colorectal cancer.

Why is a high-fiber diet considered beneficial in reducing the risk of colorectal carcinoma?

- A. It increases stool bulk and aids in the removal of carcinogens
- B. It serves as a prebiotic to stimulate probiotic growth
- C. It suppresses the synthesis of bile salts in the liver
- D. It neutralizes bile salts to prevent them from becoming toxic

**Colorectal
carcinoma
Clinical
manifestation**

Colon cancer may present for a long period of time without symptoms.

Bleeding is the symptom that causes the person to seek medical care.

✧ Change in bowel habits, diarrhea or constipation, sense of urgency, or incomplete emptying of the bowel.

Pain usually is a late symptom.

Colorectal carcinoma ●

Diagnosis and treatment

□ Diagnosis:

- It can be detected by barium enema or colonoscopy, CT scan, pelvic magnetic resonance imaging (MRI), and ultrasonography can also be used.

□ Treatment:

- The only recognized treatment is **surgical removal**.

قبل الجراحة

- **Preoperative radiation therapy** may be used.

- **Postoperative adjuvant chemotherapy** is used.

بعد الجراحة

جابو اعراض ال **colorectal carcinoma** وانها

بتصيب عمر 60-70 سنة وانها شائعة الحدوث

للناس الي عندها polyps

وطالبين انه ايش مو من خصائص ال colorectal

carcinoma

الجواب : non لانهم كلهم مذكورين صح  