Gastrointestinal Diseases
Peptic Ulcer

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Gastroesophageal Reflux Disease (GERD)

- Typically caused by inappropriate relaxation of the low esophageal sphincter (LES) or by intragastric pressure that exceeds the LES pressure.

- Complications
  - esophagitis
  - bleeding
  - stricture formation
  - aspiration
  - Barrett esophagus
  - adenocarcinoma of the esophagus.

Gastroesophageal Reflux Disease (GERD).

- Typical Symptoms:
  - heartburn
  - Regurgitation of acid.

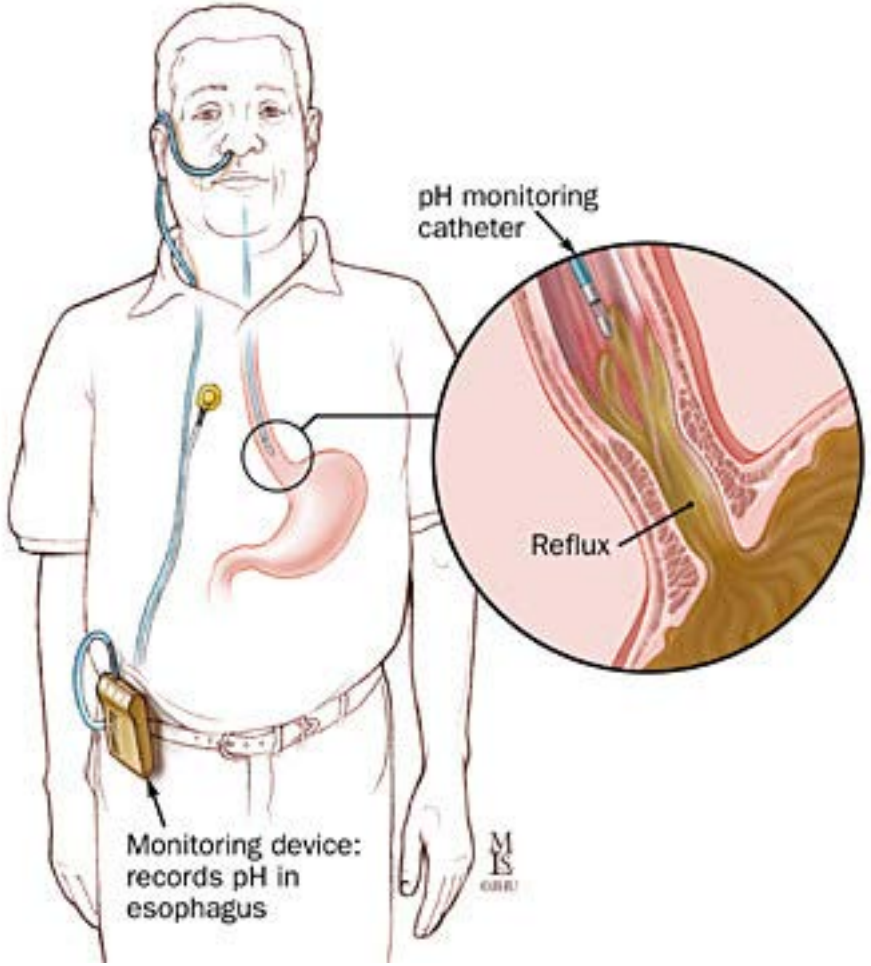
Gastroesophageal Reflux Disease (GERD).

- Atypical symptoms
  - non- cardiac chest pain
    - *Reflux is the most common cause of non- cardiac chest pain*
    - *it is imperative that cardiac status be evaluated before chest pain is attributed to reflux*
  - Asthma
    - *Asthmatic patients with coexisting reflux should receive therapy for reflux because it may improve control of respiratory symptoms.*
    - *Reflux should be considered in asthmatic patients who have postprandial or nocturnal wheezing.*
  - chronic cough
  - Hoarseness
  - enamel defects.

Gastroesophageal Reflux Disease (GERD).

- Diagnosing:
  - EGD
  - 24-hour ambulatory pH probe with impedance monitoring

  This allows a physiologic evaluation of reflux during daily activities.
  - Barium esophagography is not useful in the evaluation of reflux, since reflux of barium occurs in 25% of controls.



Gastroesophageal Reflux Disease (GERD).

➢Treatment

✓lifestyle modifications:

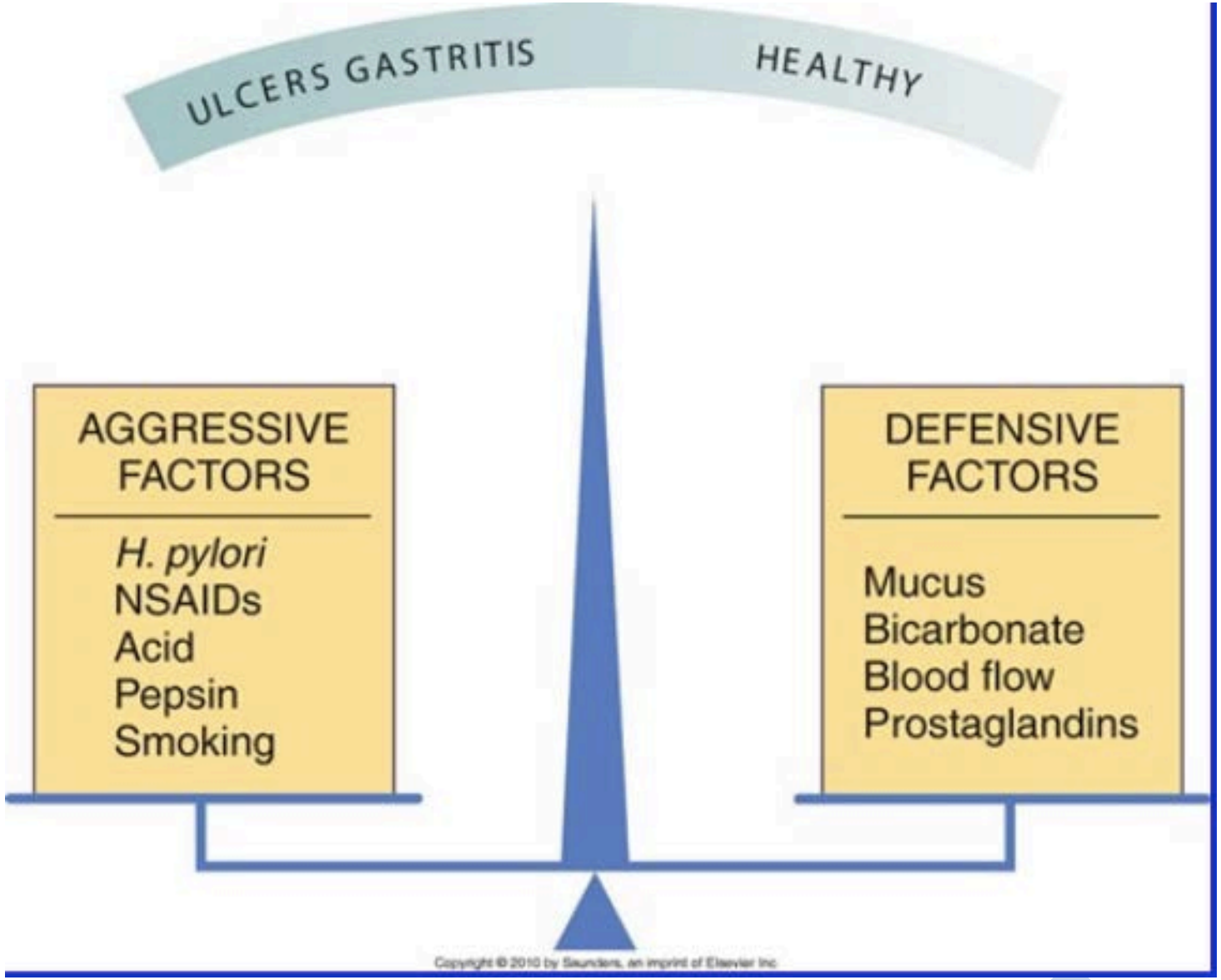
- The head of the patient's bed should be elevated 15 cm to keep the stomach lower than the esophagus,
- to not eat for 3 hours before reclining,
- lose weight if overweight,
- avoid eating foods that personally trigger symptoms (eg, fatty foods, chocolate, peppermint, citrus juices, tomato products, and coffee),
- avoid tobacco and alcohol.

✓avoid drugs that decrease LES pressure or delay gastric emptying (eg, anticholinergic agents, opioids, progesterone-containing agents, nitrates, and calcium channel blockers).

Peptic Ulcer Disease (PUD)

- PUD is ulcer formation in the lining of the upper GI tract that affects mainly the mucosal lining of the stomach, duodenum or esophagus

- Result from an imbalance between aggressive and protective mechanisms.


ULCERS GASTRITIS
HEALTHY
AGGRESSIVE FACTORS
H. pylori
NSAIDs
Acid
Pepsin
Smoking
DEFENSIVE FACTORS
Mucus
Bicarbonate
Blood flow
Prostaglandins

➢Defensive factors

- **Mucus**

Secreted cells of the GI mucosa

Forms a barrierto protect underlying cells from Acid and pepsin

- **Bicarbonate** (HCO3):

Secreted by epithelial cells of Stomac and Duodenum. coats the gastric layer and protects the cells from acids

- **Prostaglandins**:

Regulates perfusion to stomach, causes stomach cells to release mucous rich in bicarb, controls acid amounts via the parietal cells

- **Blood flow**

Poor blood flow leads to ischemia, cell injury, decrease regeneration

➢Aggressive factors

- **Helicobacter pylori, also known as H. pylori**

60%–70% of patients with PUD.Eradication of the bacterium promotes healing of the PUD and minimized recurrence of PUD

- **Nonsteroidal anti-inflammatory drugs (NSAIDs)**

Inhibit the biosynthesis of prostaglandins

Decrease blood flow, mucus, and bicarbonate

- **Gastric acid**

Causes ulcers by directly injuring cells of the GI mucosa and indirectly by activating pepsin

Increased acid alone does not increase ulcers but is a definite factor in PUD

- **Pepsin**

Proteolytic enzyme in gastric juiceSmokingDelays ulcer healing and increases risk for recurrence UD

Acid production regulation

➢Stimulators of acid production:

- acetylcholine,
- histamine,
- gastrin.

➢Inhibitors of gastric acid production

- somatostatin
- prostaglandin.

Helicobacter pylori. H. pylori

- Gram-negative bacillus that can colonize in the stomach and duodenum
- Commonly acquired through oral ingestion and transmitted among persons living in close quarters.
- Lives between epithelial cells and the mucus barrier
- Spiral-shaped which helps them invade the GI mucosa.
- Secretes urease and this breakdown UREA which produces ammonia to neutralize the acid.
- Ammonia causes more damage to the mucosal lining.
- Can remain in GI tract for decades
- Half of the world infected, but most people do not develop symptomatic peptic ulcer disease (PUD)
- *Helicobacter pylori* infection can lead to gastritis (acute to chronic), PUD, atrophic gastritis, mucosa-associated lymphoid tissue (MALT) lymphoma, or gastric malignancy.

Causes

➢3 etiologic factors:

- *Helicobacter pylori*;
- nonsteroidal anti-inflammatory drugs (NSAIDs), including aspirin;
- miscellaneous causes.

- At least 90% of peptic ulcers are due to either *H pylori* or NSAIDs.
- Miscellaneous causes include gastrinomas (Zollinger-Ellison syndrome), Crohn disease, malignancy, drugs (cocaine), and viral infections (cytomegalovirus).

Chronic Active Gastritis

- ✓The most common cause of chronic active gastritis is *H pylori* infection.
- ✓The infection is predominantly an antral- based gastritis, although gastritis throughout the gastric body may be seen.

Duodenal Ulcer

- ✓80% of patients with duodenal ulcers, *H pylori* is present.
- ✓Among *H pylori*–positive patients with a duodenal ulcer who do not receive treatment targeted at the organism, most have ulcer relapse within 1 year.
- ✓If the infection is successfully eradicated, the rate of relapse is extremely low.

Gastric Ulcer

- ✓In more than 50% of patients with gastric ulcers, *H pylori* is present.
- ✓Eradication of the bacteria decreases the relapse rate of gastric ulcers.

Chronic Gastritis

➢ Most often caused by:

- *H pylori* infection
- Autoimmune gastritis.

- *Autoimmune gastritis*

- involves the body and fundus of the stomach (not the antrum).
- In a subset of patients, atrophic gastritis develops.
- Pernicious anemia with achlorhydria and megaloblastic anemia may result.
- Antiparietal cell or anti–intrinsic factor antibodies are found in more than 90% of these patients.
- Other autoimmune diseases are often present.
- The serum gastrin level may be markedly increased (given the lack of gastric acid to provide negative feedback) and may give rise to gastric carcinoid tumors, which usually follow an indolent course in these patients.
- Peptic ulcers do not typically develop in patients with autoimmune gastritis owing to achlorhydria, but the patients are at increased risk of intestinal metaplasia and gastric adenocarcinoma.

Gastric Tumors

- ✓*H pylori* is the leading cause of gastric malignancy in the world as identified by the World Health Organization.
- ✓The gastric cancer that results from *H pylori* infection is due to a progression from chronic gastritis to atrophic gastritis, to metaplasia, to dysplasia, and eventually to gastric adenocarcinoma.

Zollinger-Ellison Syndrome

- Acid hypersecretion
- Triad of peptic ulceration, esophagitis, and diarrhea (since excess acid inactivates pancreatic lipase)
- Caused by a gastrin-producing tumor. The tumor usually is located in the “gastrinoma triangle,” which includes the head of the pancreas, duodenal wall, and distal common bile duct.
- Two-thirds of gastrinomas are malignant and can metastasize.
- One-fourth of gastrinomas are related to multiple endocrine neoplasia type 1 (MEN-1) syndrome and are associated with pituitary adenomas and hyperparathyroidism.
- Zollinger-Ellison syndrome should be considered in patients with *H pylori*–negative, NSAID-negative PUD, especially when there are
    - multiple ulcers,
    - ulcers in unusual locations (postbulbar duodenum)
    - refractory ulcers.
- Increased serum gastrin levels (>1,000 pg/mL) in patients who produce gastric acid are essentially diagnostic of gastrinoma.

Risk Factors

- Other factors that can increase susceptibility:
    - smoking
    - alcohol
    - genetics
- !!! stress and certain foods do not causes ulcers but can irritate them and prolong their healing.

Complains

- Indigestion
- Epigastric pain

Gastric Ulcers	Duodenal Ulcers
Food makes pain worst (pain 1-2 hours minutes after eating)	Pain happens when stomach empty (pain 3-4 hours after eating)
	Wake in middle of night with pain
Report of pain dull and aching	Report of pain gnawing
Upper dyspeptic syndrome – loss of appetite, nausea, vomiting	obstipation
Vomiting brings relief	
Weight loss	Weight normal
Severe: vomit blood more common	Severe: tarry, dark stool from GI bleeding

Medical history. Interview

- Ask patient when do you experience stomach pain?
- Does eating help it or make it worst?
- Do you awake with pain in the middle of the night?
- taking what medications? NSAIDS, salicylates, corticosteroids, anticoagulant…make ulcer worst)
- Any history of being diagnosed with h. pylori
- Any one in your family have it
- Smoking
- Drinking alcohol
- Caffeine products

Physical examination

- ✓Auscultation
  - Bowel sounds: hyper/hypoactive or absent
- ✓ Palpation
  - for tenderness
- ✓Percussion
  - Free gas?

Diagnosis. EGD

- ✓is the best initial test to establish the diagnosis of PUD.
- ✓active bleeding can be managed.
- ✓histologic evaluation can be performed if an ulcer has malignant features.

- ➢!!! If perforation is a concern, abdominal imaging should be the first test (endoscopy would be contraindicated).

Diagnosis. Tests for *H pylori* Infection

- ***Serology***

- Serologic testing is one of the most cost-effective, noninvasive ways to diagnose primary *H pylori* infection,
- the 1 test for *H pylori* that is not affected by medications the patient may be taking.
- It is useful only in making the initial diagnosis, however, and should not be used for eradication testing.

- ***Urea Breath Test***

- a radiolabeled dose of urea is given orally to the patient. If *H pylori* is present, the urease ac-tivity splits the urea, and radiolabeled carbon dioxide is exhaled.

- ***Stool Antigen Test***
  is simple and noninvasive.
- does not depend on disease prevalence.

- ***Rapid Urease Test***

- a biopsy specimen taken during an EGD is impregnated into agar that contains urea and a pH indicator. As the urea is split by *H pylori*–produced urease, the pH of the medium changes the color of the agar
- test depends on bacterial urease: The more organisms present, the more rapidly the test produces positive results.

- ***Histologic Examination***
  The *H pylori* organisms can be demonstrated with several specialized stains, including hematoxylin-eosin, Warthin- Starry, and immunostaining.

Treatment Goals of drug therapy

- Alleviate symptoms
- Promote healing
- Prevent complications
- Prevent recurrence
- Drugs do not alter the disease process; they create conditions conducive to healing

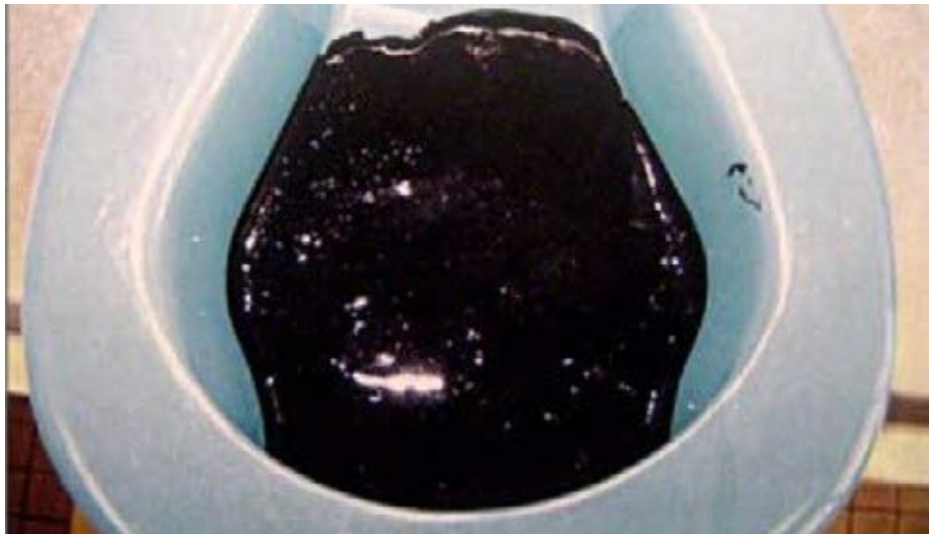
Therapy

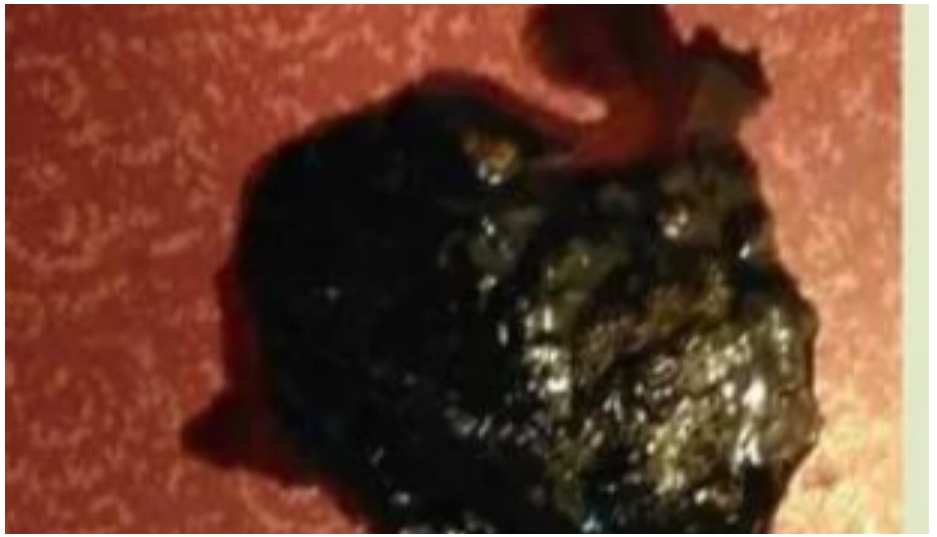
- Modify lifestyle
- Prohibiting of smoke and alcohol
- Diet

Complications

➢**GI bleeding:**

- pale skin, mucous membranes
- increased HR
- decreased BP
- bloating or mass in abdomen
- dark/tarry stool
- vomiting blood that is dark like coffee ground





Bleeding. Forrest

Stage	Characteristics	Rebleeding
I a	Jet arterial bleeding	90 %
Ib	Oozing	50 %
IIa	Visible Vessel	25 - 30 %
IIb	Adherent clot	10 - 20%
IIc	Black spot in ulcer crater	7 - 10%

Complications

- Perforation/Peritonitis:
  - severe abdominal pain with bloating,
  - vomiting
  - fever
  - increase HR and respirations
- Obstruction in pylorus: due to scarring for ulceration
  - projectile vomiting
  - vomiting with food eaten many hours ago
  - abdominal pain
  - bloating

Complications

➢Penetration

- signs of pancreatitis:
  - vomiting
  - pain with irradiation to the back
  - ↑amylase in blood, ↑diastase in urea

➢Malignization

- weight loss
- anemia
- less appetite
- changes in food habits
- ↑ESR