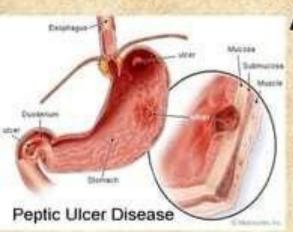


Peptic Ulcer Disease

By Aniedu, Ugochukwu



OUTLINE

- Introduction
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- Etiology/ Risk factors
- Types of PUD
- Clinical Presentation
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- · References.

INTRODUCTION

 Peptic Ulcer is a lesion in the lining (mucosa) of the digestive tract, typically in the stomach or duodenum, caused by the digestive action of pepsin and stomach acid.

Lesion may subsequently occur into the lamina propria and submucosa to cause bleeding. -Most of peptic ulcer occur either in the duodenum, or in the stomach - Ulcer may also occur in the lower esophagus due to reflexing of gastric content - Rarely in certain areas of the small intestine

PATHOPHYSIOLOGY

- Bicarbonate
- Mucus layer
- Prostaglandins
- Mucosal blood flow
- Epithelial renewal

Defensive /



- Helicobacter pylori
- NSAIDs
- Pepsins
- Bile acids
- Smoking and alcohol

Aggressive



Under normal conditions, a physiologic balance exists between gastric acid secretion and gastroduodenal mucosal defense. Mucosal injury and, thus, peptic ulcer occur when the balance between the aggressive factors and the defensive mechanisms is disrupted. Aggressive factors, such as NSAIDs, H pylori infection, alcohol, bile salts, acid, and pepsin, can alter the mucosal defense by allowing back diffusion of hydrogen ions and subsequent epithelial cell injury.

ETIOLOGY/ RISK FACTORS

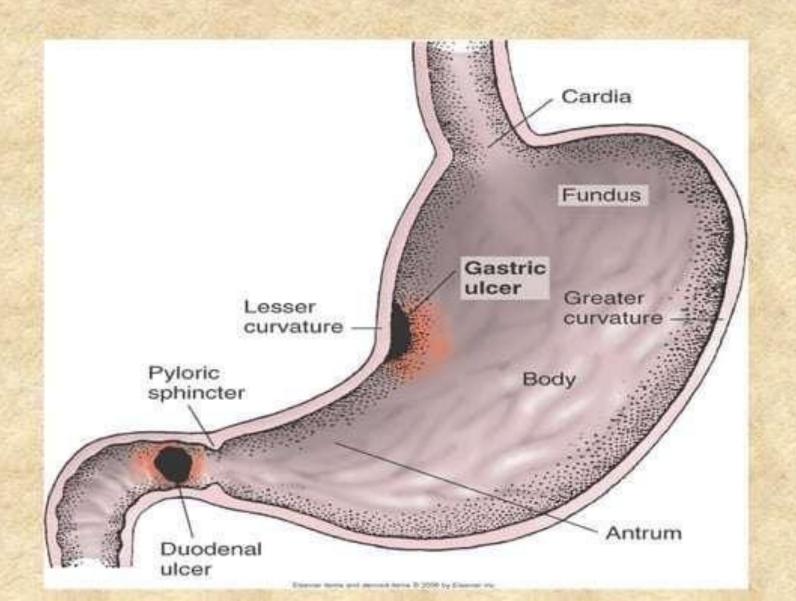
- Lifestyle
 - Smoking
 - Acidic drinks
 - Medications
- H. Pylori infection
 - 90% have this bacterium
 - Passed from person to person (fecal-oral route or oral-oral route)
- Age
 - Duodenal 30-40
 - Gastric over 50

- Gender
 - Duodenal: are increasing in older women
- Genetic factors
 - More likely if family member has Hx
- Other factors: stress can worsen but not the cause

TYPES

- GASTRIC PEPTIC ULCER
- DUODENAL PEPTIC ULCER

Gastric and Duodenal Ulcers



CLINICAL PRESENTATION

	Duodenal Ulcer	Gastric ulcer
Age	Any age specially 30-40	middle age 50-60
Sex	More in male	More in female
Occupation	Stress job eg. Manager	Same
Pain	Epigastric , discomfort	Epi. Can radiate to back
Onset	2-3 hours after eating & midnight	Immediately after eating
Agg.by	Hunger	Eating

	Duodenal Ulcer	Gastric ulcer
Relived by	Eating	Lying down or vomiting
Duration	1-2 months	Few weeks
Vomiting	Uncommon	Common(to relieve the pain)
Appetite	Good	Pt. afraid to eat
Diet	Good, eat to relieve the pain	Avoid fried food
Weight	No wt. loss	wt. Loss
Hematemesis	40%	60%
Melena	60%	40%

INVESTIGATION/ DIAGNOSTIC TEST

INVESTIGATION

Stool examination for fecal occult blood.

 Complete blood count (CBC) for decrease in blood cells.

DIAGNOSTIC TEST

- Esophagogastrodeuodenoscopy (EGD)
 - Endoscopic procedure
 - Visualizes ulcer crater
 - Ability to take tissue biopsy to R/O cancer and diagnose H. pylori
 - Upper gastrointestinal series (UGI)
 - Barium swallow
 - X-ray that visualizes structures of the upper GI tract
 - Urea Breath Testing
 - Used to detect H.pylori
 - Client drinks a carbon-enriched urea solution
 - Exhaled carbon dioxide is then measured

In all patients with "Alarming symptoms" endoscopy is required.

- ▶Dysphagia.
- Weight loss.
- ▶Vomiting.
- Anorexia.
- >Hematemesis or Melena

Complications of Peptic Ulcers

Hemorrhage

- Blood vessels damaged as ulcer erodes into the muscles of stomach or duodenal wall
- Coffee ground vomitus or occult blood in tarry stools

Perforation

- An ulcer can erode through the entire wall
- Bacteria and partially digested food spill into peritoneum=peritonitis
- Narrowing and obstruction (pyloric)
 - Swelling and scarring can cause obstruction of food leaving stomach=repeated vomiting

MANAGEMENT

LIFE STYLE MODIFICATION

HYPOSECRETORY DRUG THERAPY

- H. pylori ERADICATION THERAPY
- SURGERY

LIFE STYLE MODIFICATION





Discontinue NSAIDs



Smoking cessation



Alcohol cessation.



Stress reduction.

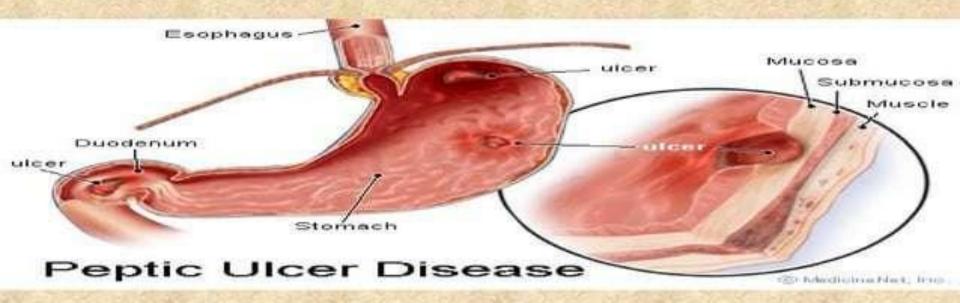
Hyposecretory Drugs

- Proton Pump Inhibitors
 - Suppress acid production
 - Prilosec, Prevacid
- H2-Receptor Antagonists
 - Block histamine-stimulated gastric secretions
 - Zantac, Pepcid, Axid
- Antacids
 - Neutralizes acid and prevents formation of pepsin (Maalox, Mylanta)
 - Give 2 hours after meals and at bedtime

- Prostaglandin Analogs
 - Reduce gastric acid and enhances mucosal resistance to injury
 - Cytotec
- Mucosal barrier fortifiers
 - Forms a protective coat
 - Carafate/Sucralfate
 - cytoprotective

H. pylori Eradication Therapy:

- Triple therapy:
 - > Proton pump inhibitor .
 - > 2 Antibiotics:
 - Metronidazole + Clarithromycin.
 - Clarithromycin + Amoxicillin.



SURGICAL TREATMENT

Indications:

- ✓ Failure of medical treatment.
- ✓ Development of complications
- ✓ High level of gastric secretion and combined duodenal and gastric ulcer.

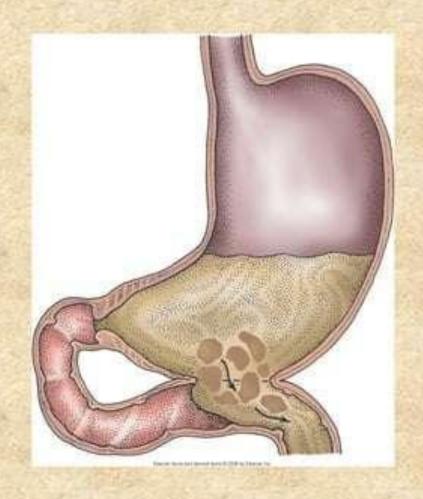
Principle:

Reduce acid and pepsin secretion.

GASTROENTEROSTOMY
 Creates a passage between the body of stomach to small intestines.

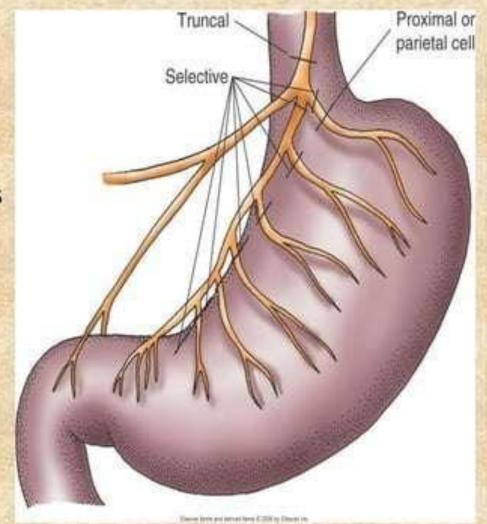
Allows regurgitation of alkaline duodenal contents into the stomach.

Keeps acid away from ulcerated area



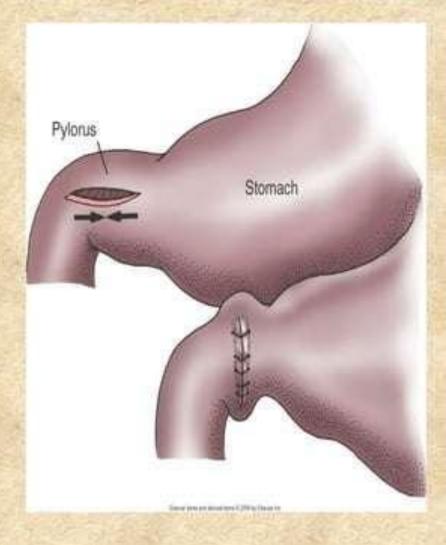
VAGOTOMY

- Cuts vagus nerve
- Eliminates acidsecretion stimulus



PYLOROPLASTY

 Widens the pylorus to guarantee stomach emptying even without vagus nerve stimulation



ANTRECTOMY/ SUBTOTAL GASTRECTOMY

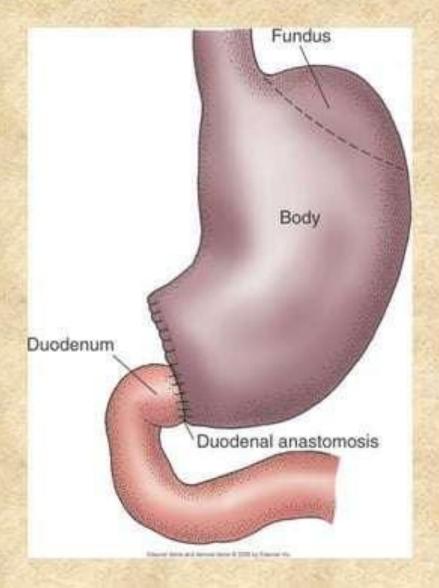
- Lower half of stomach (antrum) makes most of the acid
- Removing this portion (antrectomy) decreases acid production

SUBTOTAL GASTRECTOMY

- Removes ½ to 2/3 of stomach
- Remainder must be reattached to the rest of the bowel
 - Billroth I
 - Billroth II

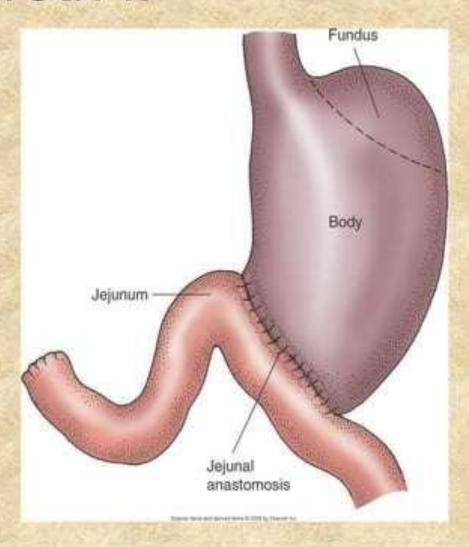
Billroth I

- Distal portion of the stomach is removed
- The remainder is anastomosed to the duodenum



Billroth II

 The lower portion of the stomach is removed and the remainder is anastomosed to the jejunum



Postoperative Care

- NG tube care and management
- Monitor for post-operative complications

Post-op Complications

Bleeding

- Occurs at the anastomosed site
- First 24 hours and post-op days 4-7

Duodenal stump leak

- Billroth II
- Severe abdominal pain
- Bile stained drainage on dressing

Gastric retention

 WILL NEED TO PUT NG TUBE BACK IN

Dumping Syndrome.

- Prevalent with sub total gastrostomies
- Early-30 minutes after meals
- Vertigo, tachycardia, syncope, sweating, pallor, palpitations
- Late 90 min-3 hours after meals
- Rx: Decrease CHO intake, Eat slowly, Avoid fluids during meals, Increase fat, Eat small, frequent meals

Anemia

 Rapid gastric empyting decreases absorption of iron

Malabsorption of fat

 Decreased acid secretions, decreased pancreatic secretions, increased upper GI mobility

Summary

- H. pylori is the most common cause of PUD and is a risk factor for gastric cancer
- H Pylori eradication reduces risk of disease recurrence
- Test-and-Treat strategy is recommended for patients with undifferentiated dyspepsia
- Intial evaluation with endoscopy is recommended for those with alarm symptoms or those failing treatment
- Optimum treatment regimens are 14d multidrug with antibiotics and acid suppressants(Triple therapy)

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