GERD and Peptic Ulcer Disease

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GERD.... Definition

- Prolonged reflux of hydrochloric acid and pepsin in esophagus, oral cavity and respiratory system
- Chronic and relapsing condition
- Lead to esophagitis and other post inflammatory conditions of intestinal and respiratory mucosa

GERD.... Epidemiology

- Developed countries
- Epidemic proportion; present in 40% of healthy population
- Adult male , over 40 year
- Low mortality



GERD.... Pathophysiology

- Dysfunction of LES(lower esophageal sphincter)
- Barrier Function: prevent reflux with mutual contraction of diaphragm
- Retain high pressure during gastric digestion

GERD... Risk Factor

- Prolonged gastric emptying
- Obesity
- Pregnancy
- Hiatal hernia
- Trauma
- Transient LES relaxation- Nocturnal and Post prandial

GERD.... Symptoms

- Main symptom of GERD is HEART BURN which is
- retrosternal burning pain
- may star in abdomen and extend into neck

Other symptoms are

Regurgitation

Globus syndrome

Respiratory symptoms – atypical symptoms connected to asthma, hoarseness and hiccough

Nausea resulting in vomiting but very uncommon

GERD.... Complications

- Chronic esophagitis Erosive changes
- Stricture leading to dysphagia
- Barrett's esophagus
- Dysplasia
- adenocarcinoma

GERD....Diagnosis

Clinical

Evaluation of symptoms ,excluding other possible cause like angina pectoris

• Endoscopic procedure

Esophagogastroscopy with biopsy

• PH metric

24 hour intraluminal monitoring

GERD... Treatment

• Non Pharmacological (life style modifications)













Other

• Elevate the head of your bed while sleeping

• Slowly eat and chew thoroughly

• Avoid trigger food that is fried and fatty food

GERD... Treatment

Medication

- Proton pump inhibitors
- H2 antagonist
- Antacid
- Surgical and endoscopic procedue



Introduction

Erosion of GI mucosa resulting from digestive action of HCl and pepsin Site

- Lower esophagus
- Stomach
- Duodenum
- 10% of men, 4% of women

Definition

• Peptic ulcer is hole or open sore in the lining of the stomach, duodenum(beginning of small intestine) or esophagus

Introduction

Peptic refers to pepsin: a stomach enzyme that breaks down protein

An ulcer occurs when the lining of these organs is corroded by digestive juices secreted by stomach



Types

Acute

- Superficial erosion
- Minimal erosion

Chronic

- Muscular wall erosion with formation of fibrous tissue
- Present continuously for many months or intermittently

Etiology

• While acid is still considered significant in ulcer formation , the leading cause of ulcer disease is currently believed to be infection of the stomach by bacteria called

"Helicobacter pyloridus (H pylori)



Stress increase HCL secretion.

Stress Ulcer



Nonsteriodal anti-inflammatory drugs

• Chronic use of NSAID is another cause of ulcer formation

Prostaglandins are substances which are important in helping the gut lining resist corrosive acid damage. NSAIDS inhibit prostaglandin secretion



Smoking

- Cigarette smoking is and important cofactor of ulcer formation and ulcer treatment failure.
- Cigarette smoking increase the risk of complications.
- Nicotine is ganglionic stimulant so it stimulates vagal nerve and increase HCL secretion through acetylcholine.



others

- Alcohol
- Coffee
- Caffeine
- Colas
- Spicy food













Types of Peptic Ulcer

- Duodenal
- Gastric
- Esophageal
- Zollinger Ellison syndrome(severe peptic ulcer +gastric hyperacidity,gastrinoma: islet cell tumor of pancreas)
- Curling's Ulcer

Gastric Ulcers

Characterized by

- A normal to low secretion of gastric acid
- Back diffusion of acid is greater (chronic)
- Critical pathologic process is amount of acid able to penetrate mucosal barrier
- H pylori is present in 50% to 70%
- Drugs --- Aspirin, corticosteroids, N SAIDs, reserpine, Chronic alcohol abuse, chronic gastritis

Duodenal Ulcers

- Between ages of 35 to 45 years
- Account for 80% of all peptic ulcers
- Associated with \uparrow HCl acid secretion
- H.pylori associated in 90-95% of cases
- Diseases with ↑risk of duodenal ulcers

COPD, cirrhosis of liver, chronic pancreatitis, hyperparathyroidism, chronic renal failure

Clinical Features

- Common to have no pain or other symptoms
 - Gastric and duodenal mucosa not rich in sensory pain fibers
 - Duodenal ulcer pain
 - Burning, cramp like
 - Gastric ulcer pain
 - Burning, gaseous

Complications

- 3 major complications
 - Hemorrhage
 - Perforation
 - Gastric outlet obstruction
- Initially treated conservatively
- May require surgery at any time during course of therapy



Diagnostic Studies

- Endoscopy procedure
 - Determines degree of ulcer healing after treatment
 - Tissue specimens can be obtained to identify H. pylori and to rule out gastric cancer
- Tests for H.pylori
 - Noninvasive tests
 - Serum or whole blood antibody tests
 - Immunoglobin G (I g G)
 - Urea breath test
 - C 14 breath test
 - Invasive tests
 - Biopsy of stomach
 - Rapid urease test

- Barium contrast studies
 - Widely used
- X- ray studies
 - Ineffective in differentiating a peptic ulcer from a malignant tumor
- Gastric analysis
- Lab analysis

Drug Therapy

- Antacids
- H₂ receptor blockers
- PPIs
- Antibiotics
- Anticholinergics
- Cytoproctective therapy

Histamine receptor blocks (H₂ R blockers)

- \succ Used to manage peptic ulcer disease
- \geq Block action of histamine on H₂ receptors
 - \checkmark HCl acid secretion
 - $\boldsymbol{\downarrow}$ conversion of pepsinogen to pepsin
 - ↑ ulcer healing

Proton pump inhibitors

Block ATPase en zyme that is important for secretion of HCl acid

Antibiotic therapy

- Eradicate H. pylori infection
- No single agents have been effective in eliminating H. pylori

Differential Diagnosis

- Stomach perforation surgical emergency, rigid abdomen, rebound tenderness
- UGI bleed melena or hematemesis
- Gastric Cancer needs EGD to view the lesion and for histologic exam of the biopsy specimens
- Gastritis negative physical exam, consistent burning epigastric pain
- GERD/Esophagitis epigastric pain after eating and improves with antacids. Negative physical exams
- Duodenal Ulcer pain is nocturnal and occurs after 30 minutes to 2 hours after eating when stomach is empty. Relieved with food or antacid intake.

