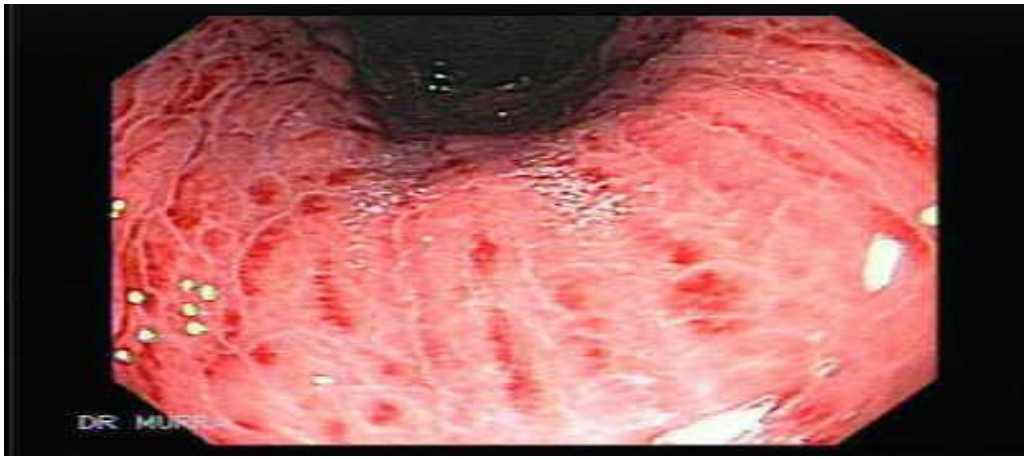
Gastritis

د- مشتاق وتوت



*Definition: •

inflammation of the gastric or stomach mucosa • with mucosal injury.

Gastritis may be •

*acute, lasting several hours to a few days, •

*or chronic, resulting from repeated exposure to • irritating agents

*or recurring episodes of acute gastritis •

* male to female ratio 1/1 •

*causes: •

I- acute gastritis: •

*drugs: aspirin, NSAIDs, iron preparation •

*H. pylori •

*alcohol •

*stress: burns, CNS trauma. •

*bile reflux •

*viral infections: CMV, HS •

*radiation •

II- chronic non-specific gastritis: •

*HP infection •

*autoimmune (perniciuos an) •

*post-gastrectomy •

III- chronic specific gastritis: •

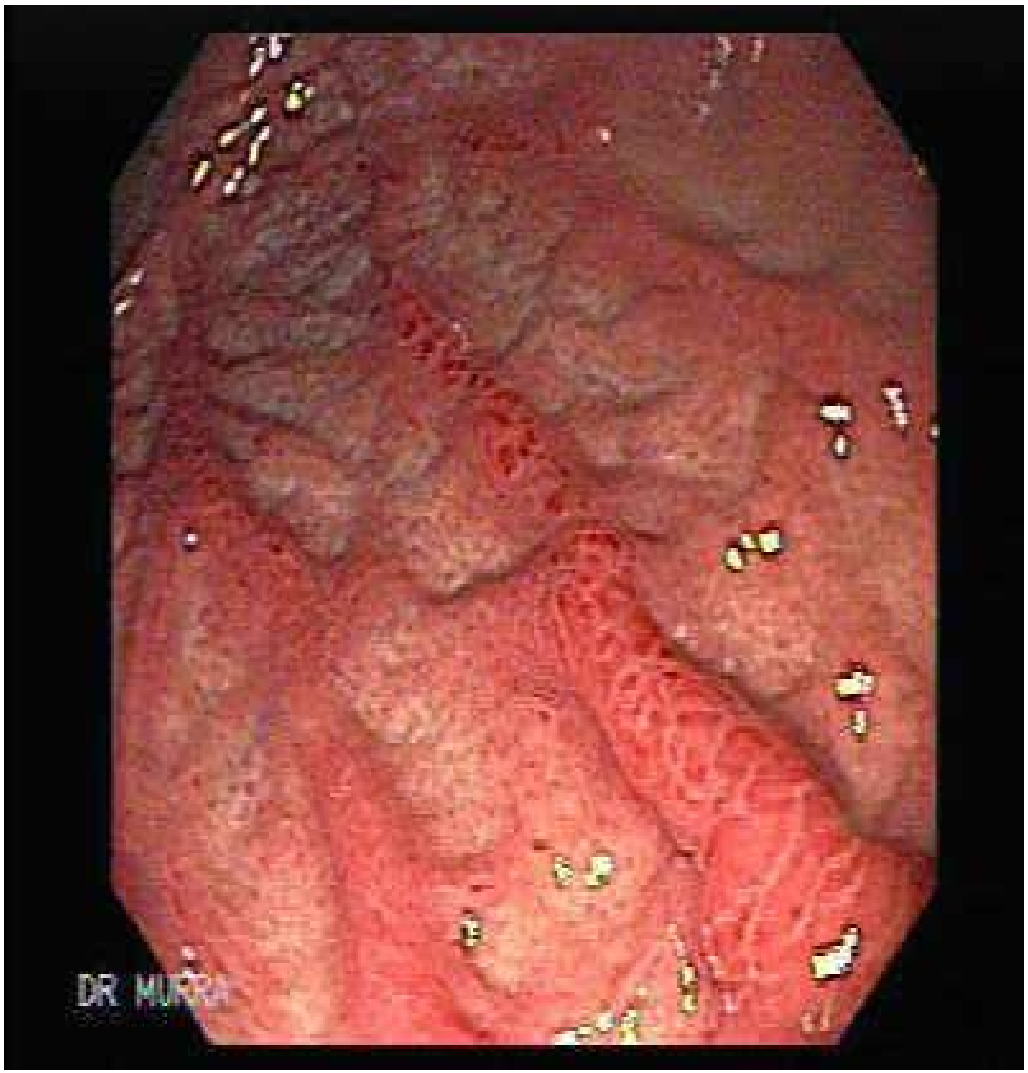
*infection, CMV, TB •

*Crohn's disease •

*sarcoidosis, GVHD •

*idiopathic, granulomatous G •

•



Acute gastitis

*definition: an acute mucosal inflammatory process, usually of a transient nature.

*pathophysiology: inflammatory mucosal changes, with neutrophils predomenant.

*symptoms:

1- asymptomatic.

2- dyspepsia, anorexia, nausea, vomiting & hematemesis or malena.

* Diagnosis: •

1- clinically, no need investigations •

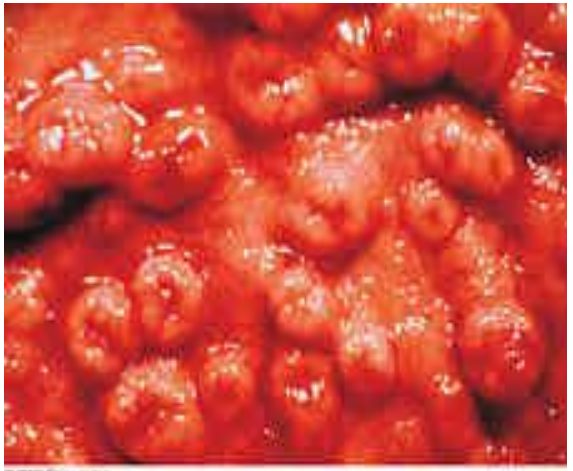
2- may need endoscopy & biopsy to exclude • PU or cancer.

*Treatment: •

1- treat underlying cause •

2- symptomatic therapy with antacids, PPIs & • antiemetics (e.g. metoclopramide)

Chronic gastritis



*Definition: •

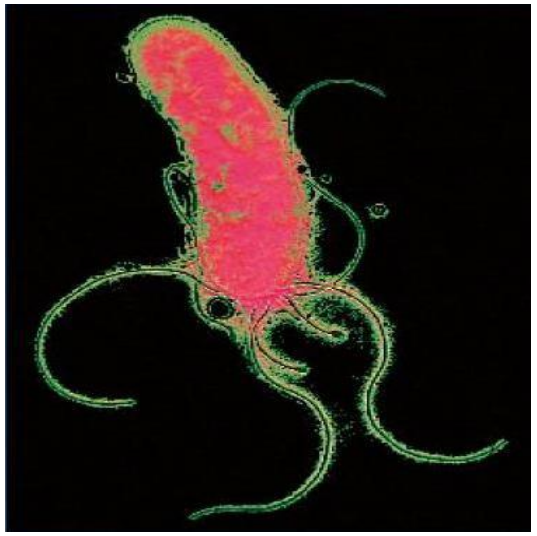
prolonged inflammation of the stomach. •

*Pathophysiology: chararctrised by •

mononuclear cell infiltrate especially • lymphocyte and maccrophages of gastric mucosa.

•

Some types of Ch Gastritis



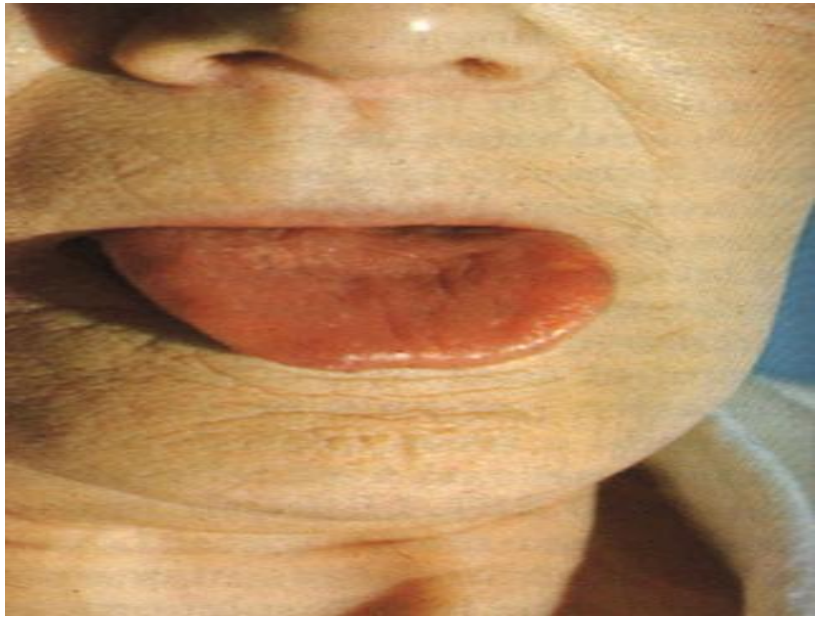
1- ch g due to HP infection:

*is the most common cause.

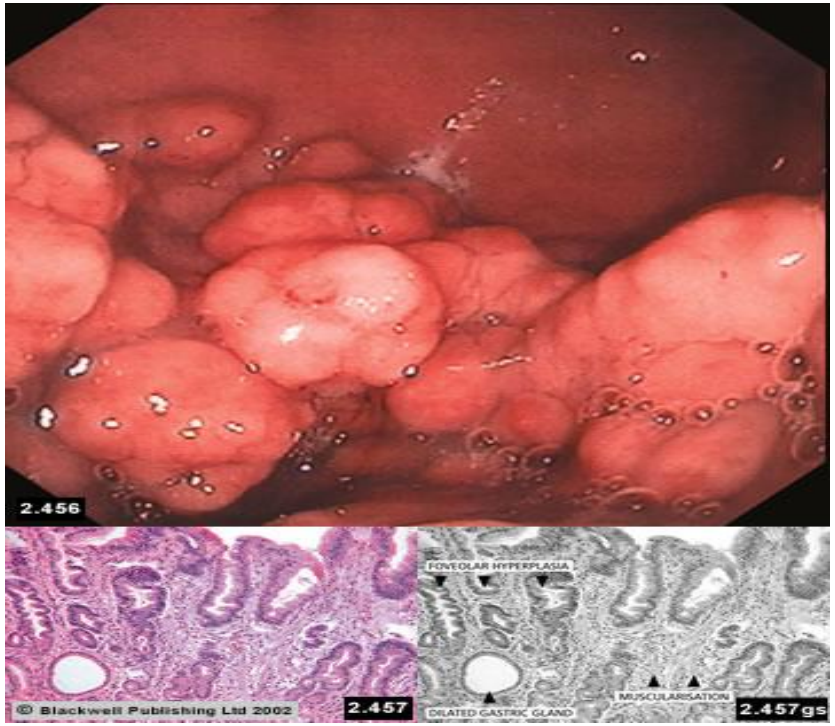
*lymphocytes & plasma cells.

* poor correlation between clinical & endoscopic or pathological findings.

*most pt are asymptomatic & managed by modifying patient's diet& do not require treatment , but if pt develop dyspepsia may benefit from HP eradication.



- 2- autoimmune ch gastritis:
- *it results from autoimmune activity againast parietal cells.
- *involves the body of stomach &
- spares the antrum, characterized by diffuse chronic inflammation, atrophy & loss of fundic glands, intestinal metaplasia & some times hyperplasia of enterochromaffin like cells(ECL).
- *Ab to parietal cell & intrinsic factor may be present.
- *may be severe lead to pernicious anemia.



3- Menetrier's disease: :

*disease of middle & old age.

*the gastric pits are elongated & tortuous, with replacement of the parietal & chief cells by mucus-secreting cells.

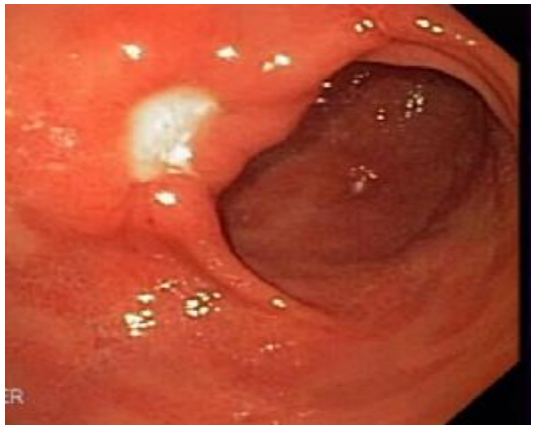
* mostly involve the body & fundus.

*most pt are hypochlorhydric.

*ass with protein losing enteropathy.

*treated with antisecretry drugs or partial gastrectomy.

Peptic ulcer disease (PUD)

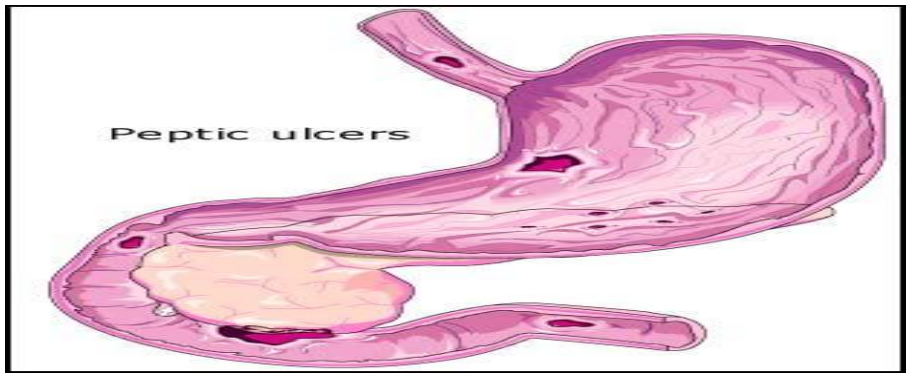


*Definition: •

break in the gastrointestinal mucosa exposed to gastric • acid and pepsin, which penetrate the muscularis mucosa, more than 5 mm in diameter may be acute or chronic.

In the acute ulcer, there is no evidence of fibrosis. •

While in erosion, there is no penetration of muscularis • mucosa & less than 5 mm.



*Site: •

()lower oesophagus •

() stomach ()duodenum •

()Jejunum after surgical anastomosis to the stomach •

()rarely in the ileum adjacent to a Meckel's diverticulum. •

Gastric & Duodenal ulcer

introduction:

* it's more common in developing countries& Higher prevalence in low socioeconomic classes
*the lifetime risk for developing a peptic ulcer is approximately 10%
*family history: 3-4 increased risk .
*male/female ratio: DU (5:1 to 2:1)
GU (2:1 or less)
*DU usually occur in 30-50 years of age while
*GU usually occur in 50-70 years of age.
* the MR : approximately 1 death per 100,000 cases.
*The hospitalization rate is approximately 30 patients per 100,000 cases.
*ch GU is usually single; 90% on the lesser curve within the antrum or at the junction bet body & antral mucosa.
*ch DU is more than one DU in 10-15% of cases, occur usually in the 1st part of duodenum, 50% on the ant wall .
* GU & DU may coexist in 10% of patients.

- *Causes*
- *1- H pylori* infection
- 2- Nonsteroidal anti-inflammatory drugs : The gastric mucosa protects itself from gastric acid with a layer of mucus, the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclooxygenase 1 (*cox-1*), which is essential for the production of these prostaglandins
- 3-Smoking: more with GU & less extent to DU.
- 4-Severe physiologic stress
  - Burns
  - CNS trauma
  - Surgery
- 5-Severe medical illnessHypersecretory states (uncommon)
  - Gastrinoma (Zollinger-Ellison syndrome) or multiple endocrine neoplasia (MEN-I)
  - Antral G cell hyperplasia
  - Systemic mastocytosis
  - Basophilic leukemias

6-Diseases associated with an increased risk of PUD include cirrhosis, chronic obstructive pulmonary disease, renal failure, and organ transplantation.

7- Additional rare, miscellaneous causes include radiation-induced or chemotherapy

-

pathophysiology

H pylori: •

*is G –ve & spiral, •

*Has multiple flagella at one end •

() make it motile •

()allowing it to burrow to live deep •

Beneath the mucus layer •

*has following antigens: •

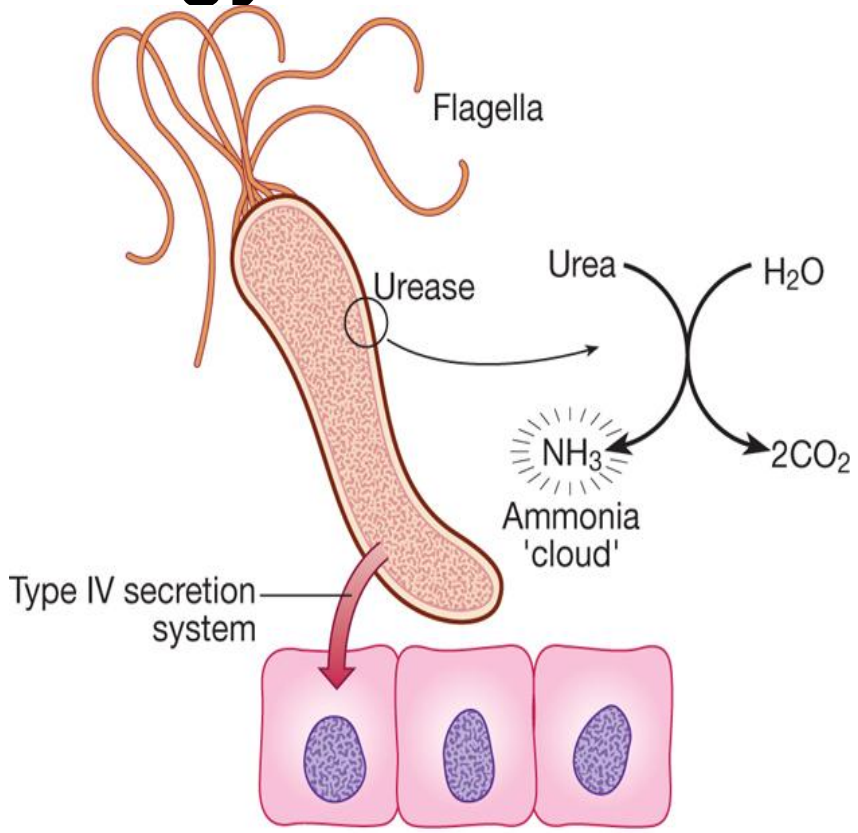
1- adhesin molecule (BabA) which •

Bind to Lewis b antigen on epith •

Cells to buffer acidity of stomach •

By urease enzyme to raise PH. •

•



Other factors

- Vacuolating cytotoxin (vacA)
- Cytotoxin-associated gene (cagA)
- Adhesins (BabA)
- Outer inflammatory protein A (oipA)

Colledge et al: Davidson's Principles and Practice of Medicine, 21st Edition

2- cytotoxin-associated gene (cag A):

Provoke a local inflammatory response in the underlying epithelium by cell replication & apoptosis.

3- vacuolating cytotoxin (vac A) :

*Increase cell permeability,

*efflux of micronutrients from epithelium

*induction of apoptosis

* suppression of local immune cell activity.

4- outer inflammatory protien A(oip A)

* in developed countries, HP infection raise with the age, 50% of people over the age of 50.

While in developing countries, affect up to 90% of the adult population.

*HP infections are probably acquired in childhood, the vast majority of colonized people remain healthy & asymptomatic.

*HP responsible for 90% of DU & 70 % of GU

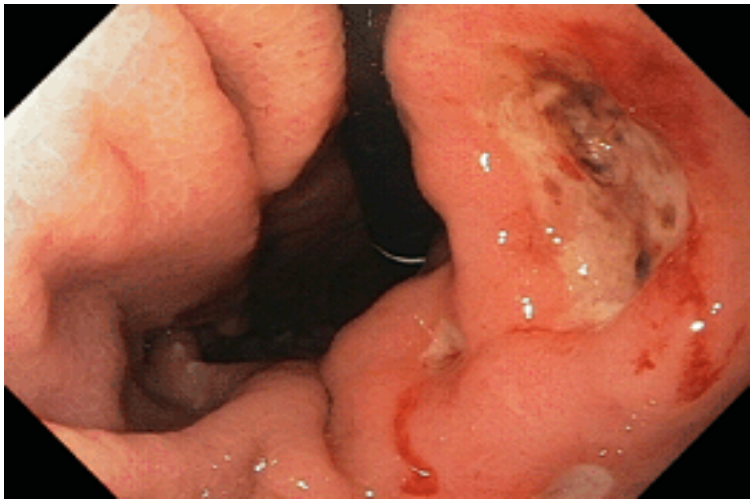
*HP spread by person to person via

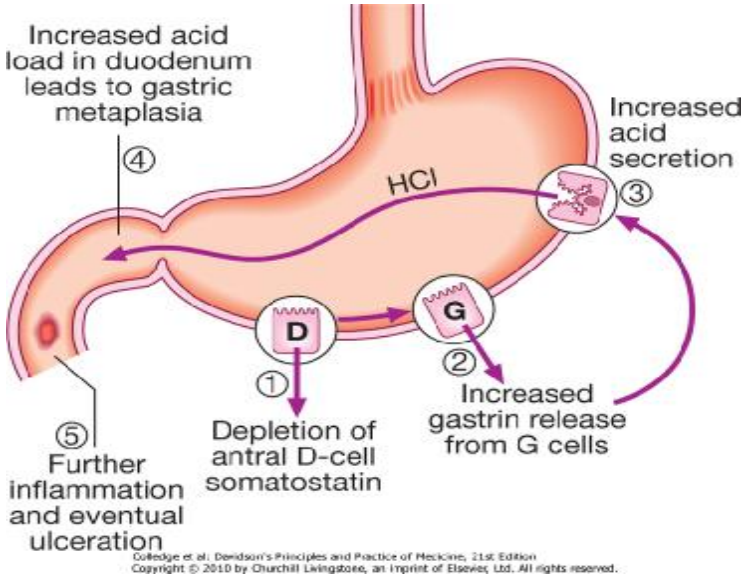
gastric reflux ate or vomit.

HP & DU

*It's exclusively colonizes gastric type epithelium & is only found in the duodenum in association with patches of gastric metaplasia, which produce DU by:

- 1- depletion of antral D-cell(somatostatin)
- 2- increase gastrin release from G cells
- 3- increased acid secretion by parietal cells
- 4- increased acid load in duodenum leads to gastric metaplasia
- 5- further inflamation & ulceration





HP & GU

- *the role of HP in the pathogenesis of GU is less clear but it probably acts by:
- 1- reducing gastric mucosal resistance to attack from acid & pepsin.
- 2- cause pan gastritis leading to gastric atrophy & hypochlorhydria.
- 3- this hypochlorhydria allows bacteria to proliferate within the stomach, which may produce mutagenic nitrites from dietary nitrates predispose to gastric ca.
- * also the effects of HP are more complex, which occur probably because impaired mucosal defense resulting from a combination of HP , NSAID, & smoking.

Role of H.pylori in GI diseases

Healthy subjects 20-50%

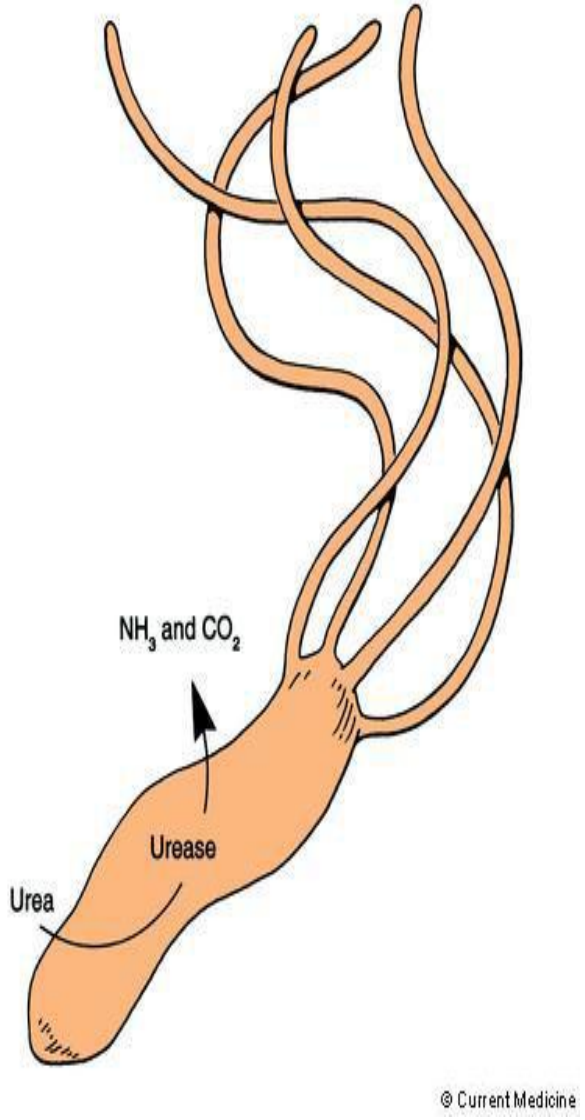
Chronic active gastritis 100%

Duodenal ulcer >90% •

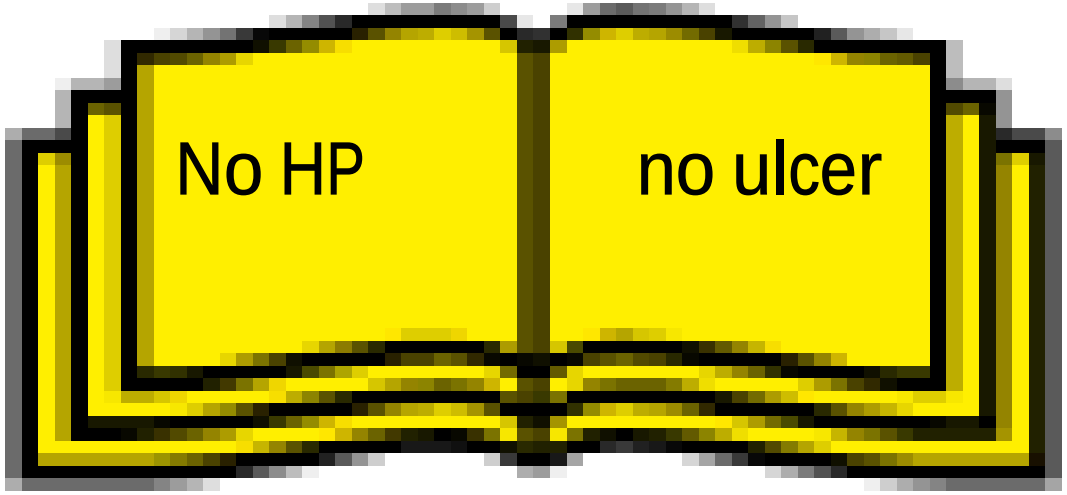
Gastric ulcer 70 % •

Gastric adenocarcinoma 90%

Gastric lymphoma 85%



Old statement



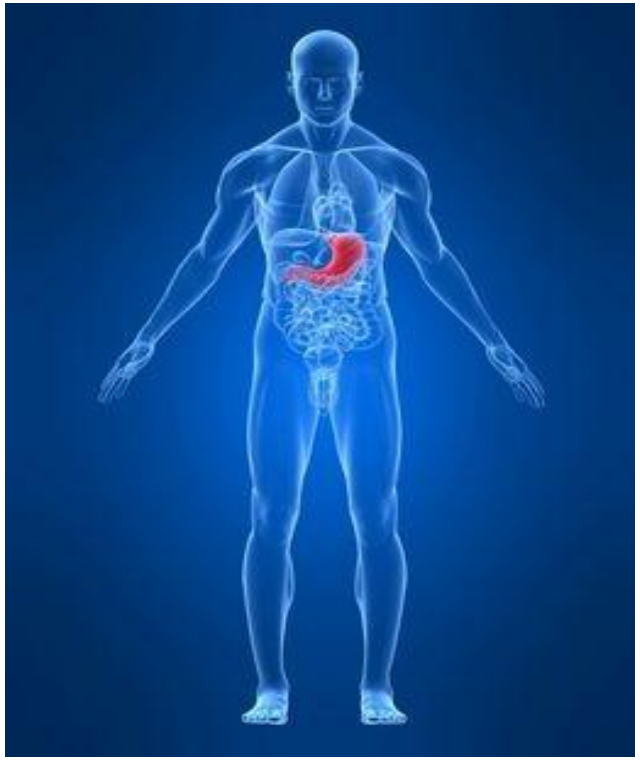
New statment

Clinical features

()abdominal pain: •
- *recurrent. •
- *epigastric •
- *relation to food •
- *episodic •
- *hunger pain •
- *night pain •

()dyspepsia, heartburn, water brash. •
()nausea & anorexia •
()vomiting (40%) •
()silent as anemia from undetected blood loss •
()cx as hematemesis or malena or perforation •
()the diagnostic value of individual symptoms for PUD is poor •

Clinical notes

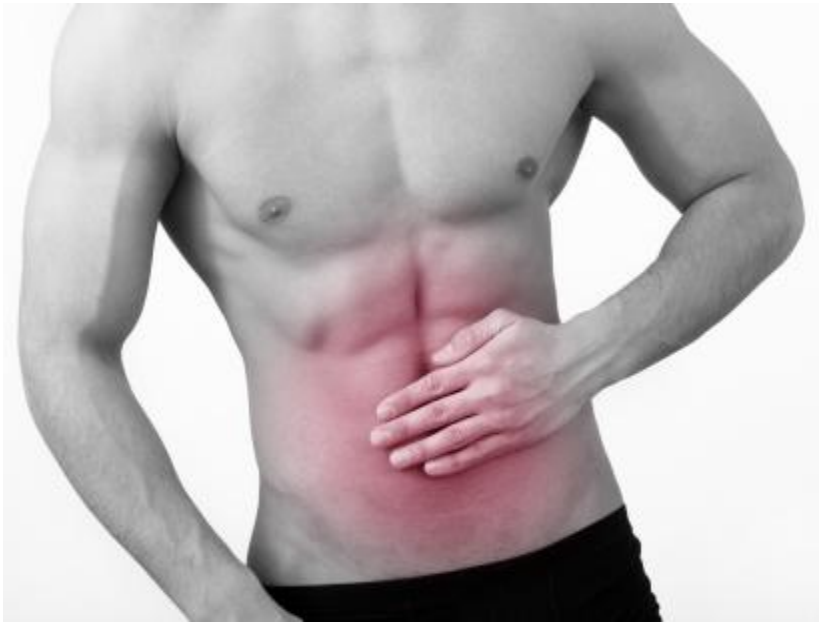


History •

()Patients typically present with abdominal pain •
that has the following characteristics: •
*Epigastric to left upper quadrant –
*Frequently described as burning –
*May radiate to the back –
*Usually occurs 1-5 hours after meals –
*May be relieved by food, antacids (duodenal), –
or vomiting (gastric) –

“()Alarm features" that warrant prompt gastroenterology referral are following: •
*Bleeding or anemia –
*Early satiety –
*Unexplained weight loss –
*Progressive dysphagia or odynophagia –
*Recurrent vomiting –
*Family history of GI cancer –

()NSAID-induced gastritis or ulcers may be silent. •
()Sudden onset of symptoms may indicate perforation. •
()Gastritis may present as bleeding, which is more likely in elderly patients. •
()Symptoms consistent with anemia (eg, fatigue, dyspnea) may manifest. •



Physical

*Epigastric tenderness is present and usually mild.

*Bowel sounds are typically normal.

*Perform a rectal examination and Hemoccult testing.

*Signs of peritonitis may be present with perforation.

*A succussion splash may be present with gastric outlet obstruction

Compare and Contrast the symptoms of Duodenal and Gastric Ulcers

Duodenum U:

-Gnawing or burning upper abd pain 1-3 hrs after meals

Worse pain when stomach empty

Bleeding occurs with deep erosion

Hematemesis –

Melena –

Gastric U:

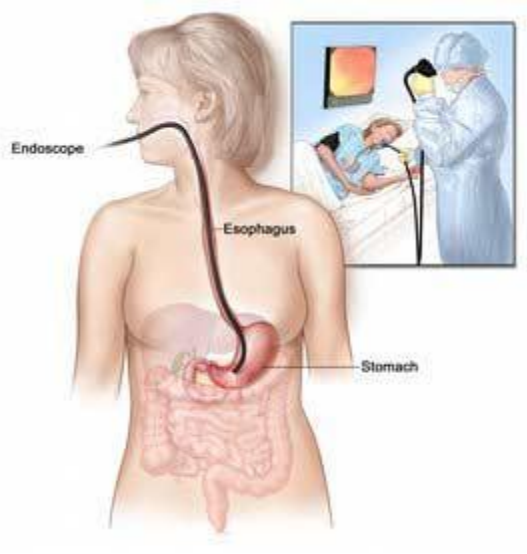
-Relieved by food but pain may persist even after eating

Anorexia, wt loss, vomiting

Infrequent or absent remissions

Small % become cancerous

Severe ulcers may erode through stomach wall



*investigations: •

Endoscopy is the preferred investigation.

*in GU , may occasionally malignant & therefore must always be biopsied & follow up. •

* investigations for HP are: •

()non-invasive: serology •

urea breath test •

fecal antigen test •

()invasive: histology •

rapid urea's test •

microbiological culture (gold standard) •