Gastritis
peptic ulcer

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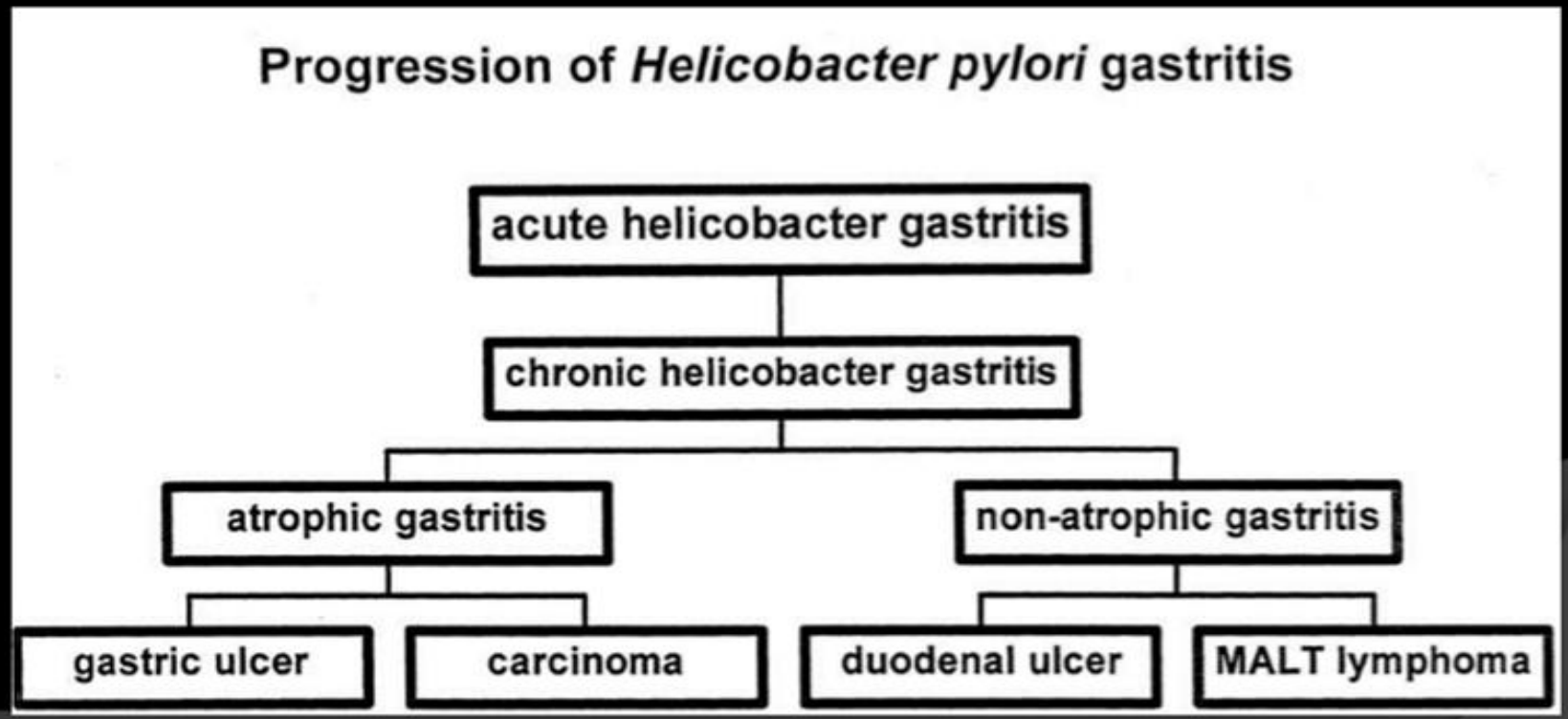
FKUMM

DEFINITION

- **GASTRITIS**
  - used to denote inflammation associated with mucosal injury
  - Histological term that needs biopsy to be confirmed
  - Due to infectious agents (such as Helicobacter pylori), autoimmune and hypersensitivity reactions.
- **GASTROPATHY**
  - Epithelial cell damage and regeneration without associated inflammation
  - Just according to gross appearance in endoscopy or radiology
  - Caused by irritants such as drugs (eg, nonsteroidal anti-inflammatory and alcohol), bile reflux, hypovolemia, and chronic congestion.

Etiology

- Drugs (NSAID, herbal)
- Helicobacter Pylori
- Bile acid



Classification

- Acute
  - Short term inflammation
  - Histology: neurophilic infiltrate
- Chronic
  - Long standing forms
  - Histology : mononuclear cell infiltrate especially lymphocyte and maccrophages

Pathophysiology

The mechanisms of mucosal injury in gastritis are thought to be an imbalance of

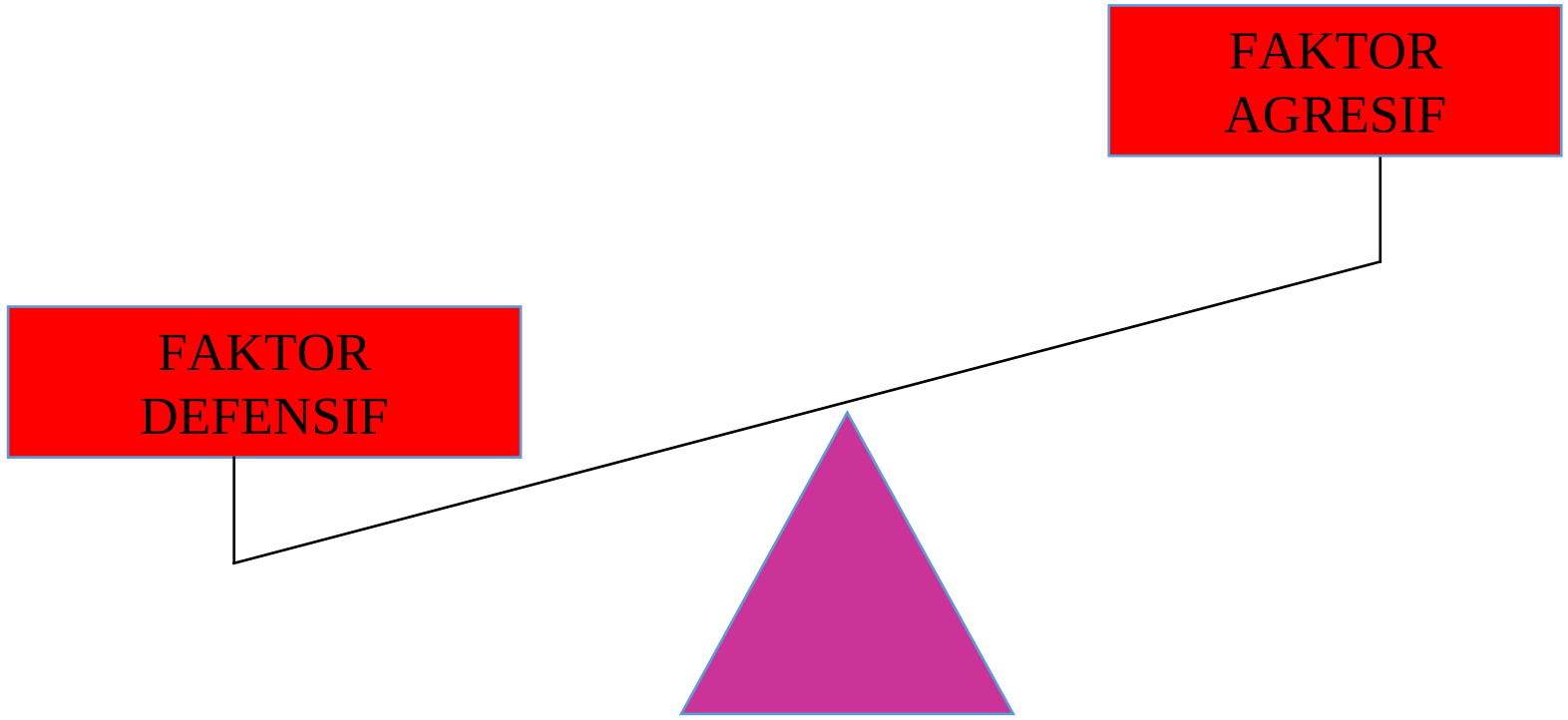
aggressive factors

- acid production or pepsin

and **defensive factors**

- mucus production
- bicarbonate
- and blood flow

“IMBALANCE”



Robbins.Pathology Anatomy

Patients typically present with abdominal pain that has the following characteristics

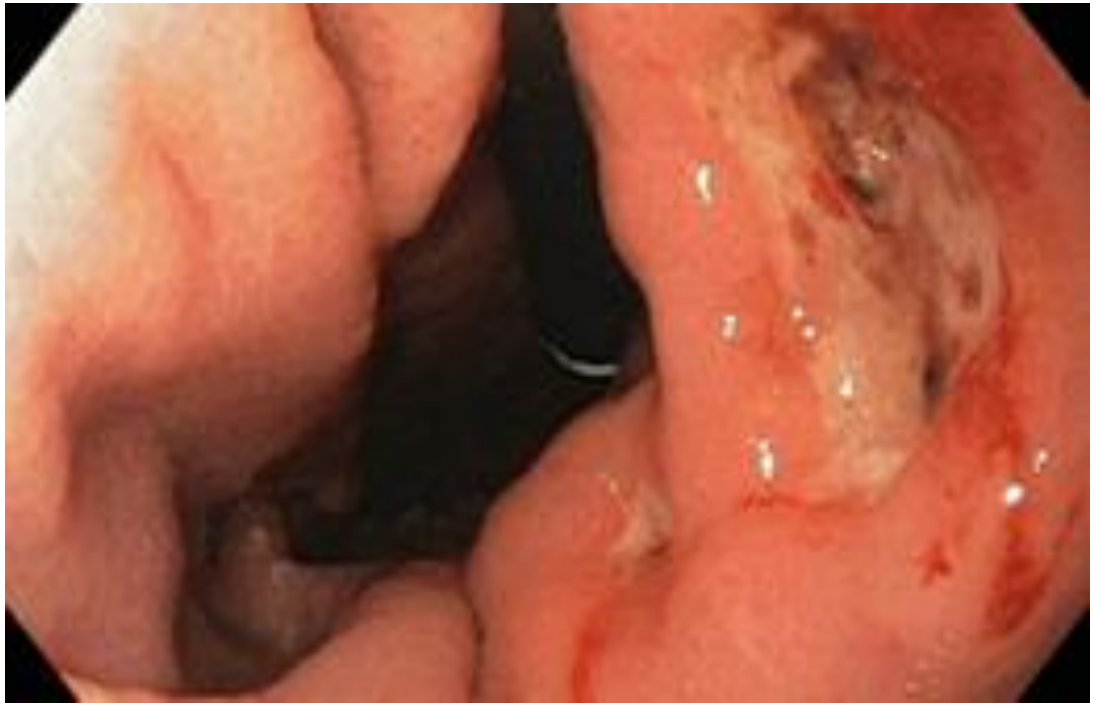
- Epigastric to left upper quadrant
- Frequently described as burning
- May radiate to the back
- Usually occurs 1-5 hours after meals
- May be relieved by food, antacids (duodenal), or vomiting

Treatment

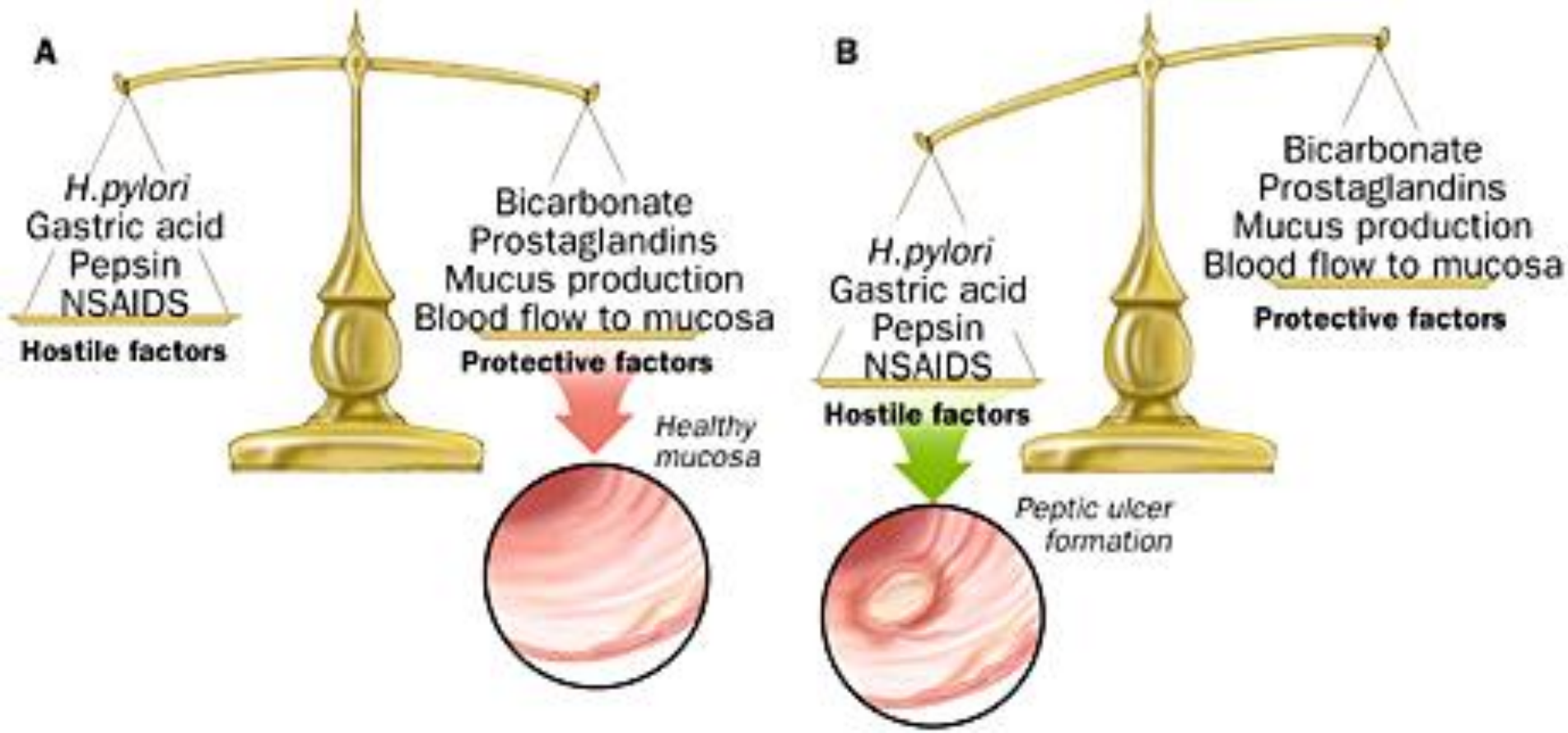
- Drugs:
  - Antasida
  - Antagonis reseptor H2
  - Proton pump inhibitor
  - Antikolinergic
  - Sitoprotektor sukralfat dan rebamipid
  - Prostaglandin

Peptic ulcer

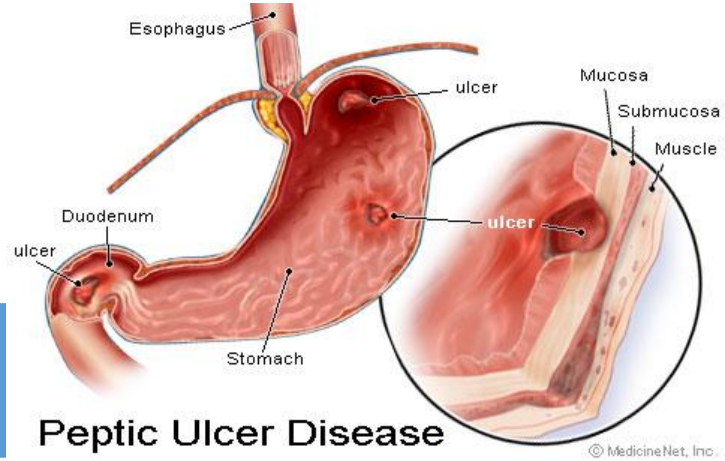
- A break in superficial epithelial cells penetrating down to muscularis mucosa



Pathogenesis PUD



Duodenal vs Gastric



	DUODENAL	GASTRIC
INCIDENCE	More common	Less common
ANATOMY	First part of duodenum – anterior wall	Lesser curvature of stomach
DURATION	Acute or chronic	Chronic
MALIGNANCY	Rare	Benign or malignant

Duodenal VS gastric

Duodenal ulcers

- Duodenal sites are 4x as common as gastric sites
- Most common in middle age
  - peak 30-50 years
- Male to female ratio—4:1
- Genetic link: 3x more common in 1st degree relatives
- More common in patients with blood group O
- Associated with increased serum pepsinogen
- H. pylori infection common
  - up to 95%
- Smoking is twice as common

Gastric ulcers

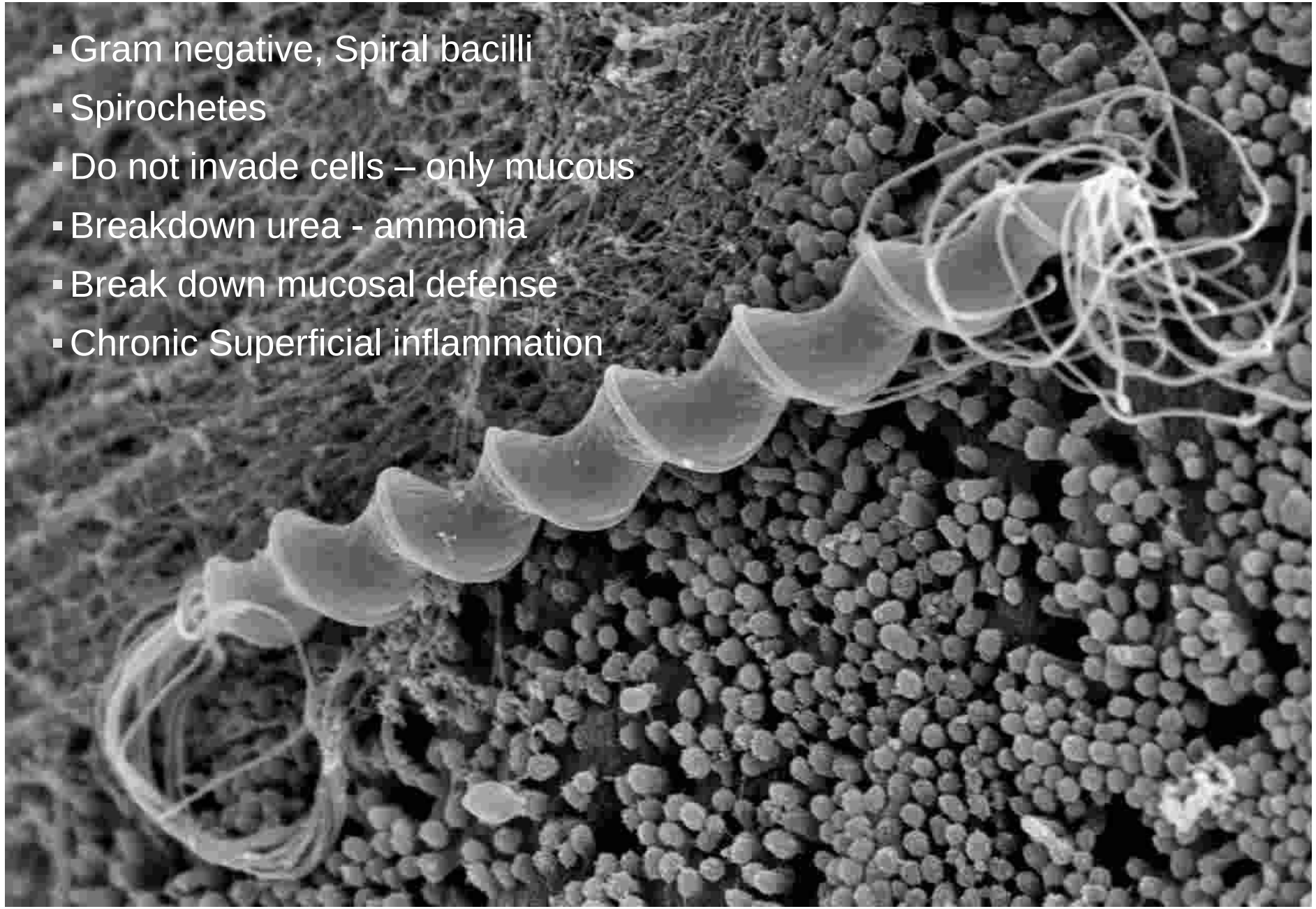
- Common in late middle age
  - incidence increases with age
- Male to female ratio—2:1
- More common in patients with blood group A
- Use of NSAIDs - associated with a three - to four-fold increase in risk of gastric ulcer
- Less related to H. pylori than duodenal ulcers – about 80%
- 10 - 20% of patients with a gastric ulcer have a concomitant duodenal ulcer

Risk factors

- HELICOBACTER PYLORI
- Non Steroidal Anti-inflammatory Drugs
- Steroid therapy
- Smoking
- Excess alcohol intake
- Genetic factors
- Zollinger Ellison syndrome – rare syndrome caused by gastrin-secreting tumour
- Blood group O
- Hyperparathyroidism

H Pylori

- Urease producing, gram negative bacillus
- Developing countries
- Infection increases with age
- Infects mucosa of stomach → inflammatory response → gastritis → increased gastrin secretion → gastric metaplasia → damage to mucosa → ulceration
- Increased risk of developing gastric adenocarcinoma


Gram negative, Spiral bacilli
Spirochetes
Do not invade cells – only mucous
Breakdown urea - ammonia
Break down mucosal defense
Chronic Superficial inflammation

Symptoms of PUD (Peptic Ulcer Disease)

- Pain—"gnawing", "aching", or "burning"
  - Duodenal ulcers: occurs 1-3 hours after a meal and may awaken patient from sleep. Pain is relieved by food, antacids, or vomiting.
  - Gastric ulcers: food may exacerbate the pain while vomiting relieves it.
- Nausea, vomiting, belching, dyspepsia, bloating, chest discomfort, anorexia, hematemesis, &/or melena may also occur.
  - Nausea, vomiting, & weight loss more common with Gastric ulcers

ALARM signs for epigastric pain

- Chronic GI bleeding
- Iron-deficiency anaemia
- Progressive unintentional weight loss
- Progressive dysphagia
- Persistent vomiting
- Epigastric mass
- Patients aged 55 years and older with unexplained and persistent recent- onset dyspepsia alone

PUD DIAGNOSIS

- Endoscopy
- Barium meal – contrast x-ray
- Biopsy – bacteria & malignancy
- H.Pylori:
  - Endoscopy cytology
  - Biopsy – Special stains
  - Culture - difficult
  - Urease Breath test.
  - Stool antigen test

- Drugs used
- to treat PUD
  - **Antimicrobial**
  - **Agents**

  - Amoxicillin
  - Bismuth
  - Clarithromycin
  - Metronidazole
  - Tetracycline

  - **H2-Histamine**
  - **R Blockade**

  - Cimetidine
  - Famotidine
  - Nizatidine
  - Ranitidine

  - **Prostaglandins**

  - Misoprostol

- **PPI**

- Lansoprazole
- Omeprazole

- **Antimuscarinic**
- **Agents**

- Pirenzepine

- **Antacids**

- Al(OH)3
- [Mg(OH)2]
- CaCO3

- **Mucosal**
- **Protective**
- **Agents**

- Bismuth
- Sulcralfate

Treatment Plan: H. Pylori

- Medications: Triple therapy for 14 days is considered the treatment of choice.
  - Proton Pump Inhibitor + clarithromycin and amoxicillin
    - Omeprazole (Prilosec): 20 mg PO bid for 14 d **or**

      Lansoprazole (Prevacid): 30 mg PO bid for 14 d **or**

      Rabeprazole (Aciphex): 20 mg PO bid for 14 d **or**

      Esomeprazole (Nexium): 40 mg PO qd for 14 d **plus**

      Clarithromycin (Biaxin): 500 mg PO bid for 14 **and**

      Amoxicillin (Amoxil): 1 g PO bid for 14 d
    - Can substitute Flagyl 500 mg PO bid for 14 d if allergic to PCN
  - In the setting of an active ulcer, continue qd proton pump inhibitor therapy for additional 2 weeks.

Treatment Plan: Not H. Pylori

- Medications—treat with Proton Pump Inhibitors or H2 receptor antagonists to assist ulcer healing
  - H2: Tagament, Pepcid, Axid, or Zantac for up to 8 weeks
  - PPI: Prilosec, Prevacid, Nexium, Protonix, or Aciphex for 4-8 weeks.

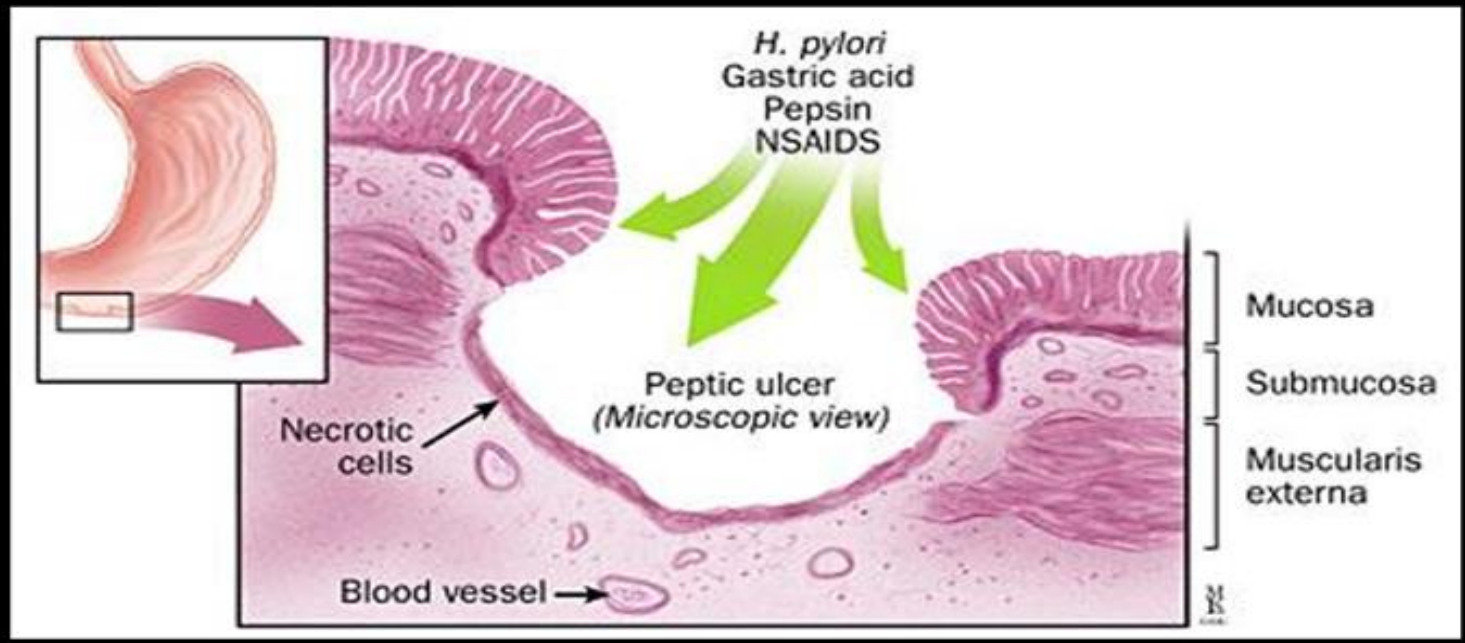
Lifestyle Changes

- Discontinue NSAIDs and use Acetaminophen for pain control if possible.
- Acid suppression--Antacids
- Smoking cessation
- No dietary restrictions unless certain foods are associated with problems.
- Alcohol in moderation
  - Men under 65: 2 drinks/day
  - Men over 65 and all women: 1 drink/day
- Stress reduction

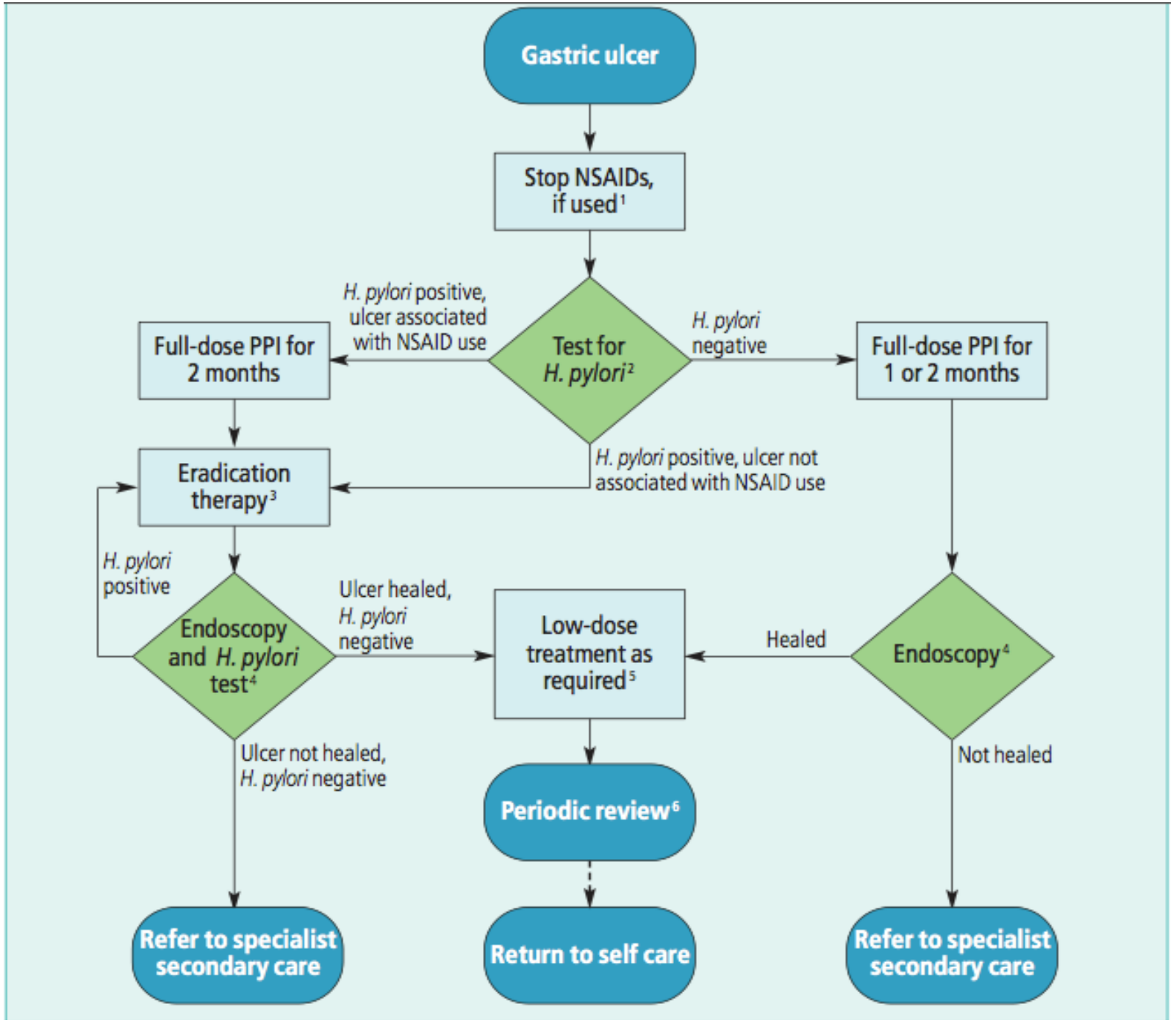
Complications

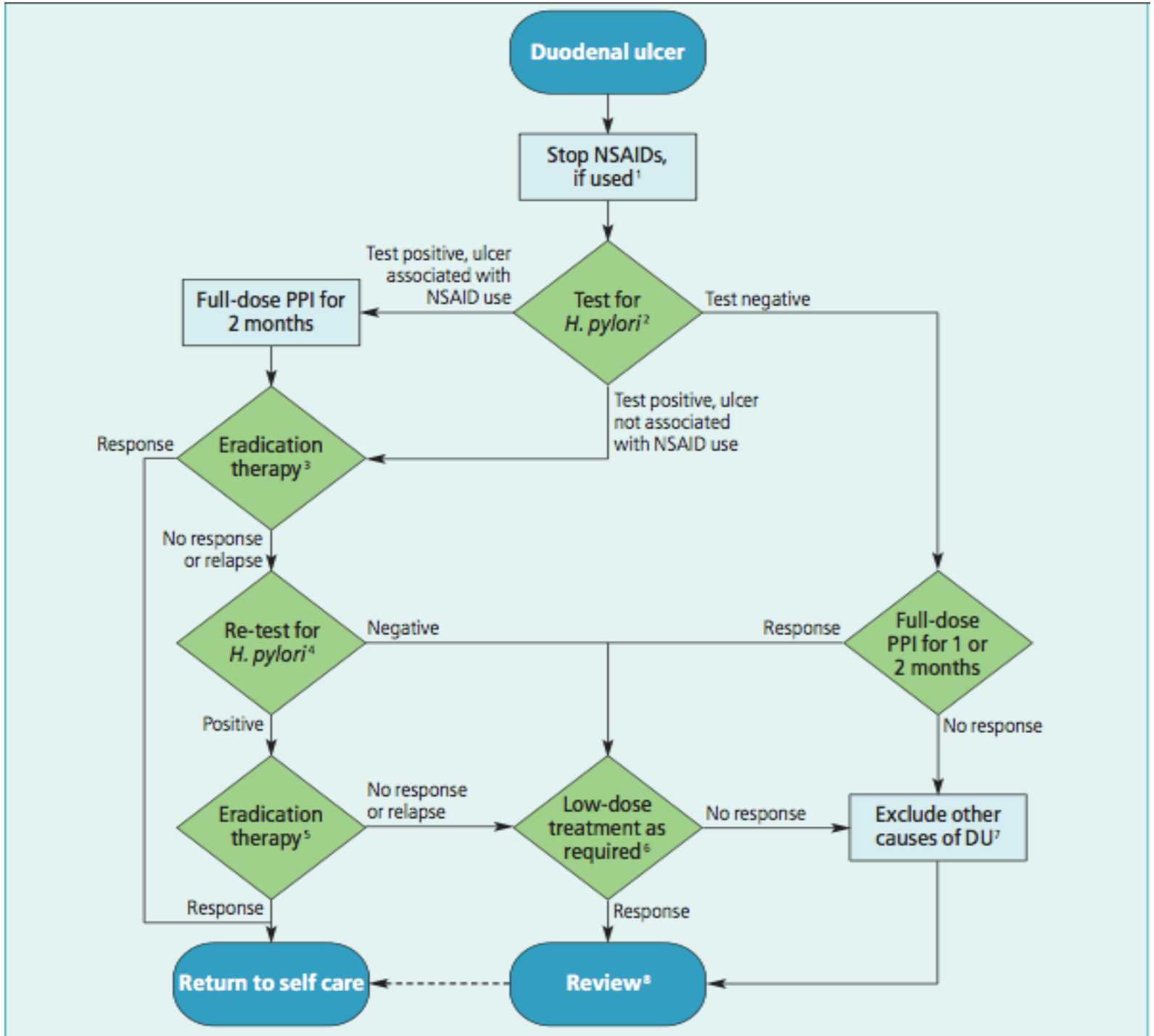
- Perforation & Penetration—into pancreas, liver and retroperitoneal space
- Peritonitis
- Bowel obstruction, Gastric outflow obstruction, & Pyloric stenosis
- Bleeding--occurs in 25% to 33% of cases and accounts for 25% of ulcer deaths.
- Gastric CA

Schematic of peptic ulcer



Forrest classification


Gastric ulcer
Stop NSAIDs, if used[1]
Test for H. pylori[2]
H. pylori positive, ulcer associated with NSAID use
H. pylori negative
Full-dose PPI for 2 months
Full-dose PPI for 1 or 2 months
H. pylori positive, ulcer not associated with NSAID use
Eradication therapy[3]
H. pylori positive
Endoscopy and H. pylori test[4]
Ulcer healed, H. pylori negative
Low-dose treatment as required[5]
Healed
Endoscopy[4]
Ulcer not healed, H. pylori negative
Not healed
Periodic review[6]
Refer to specialist secondary care
Return to self care
Refer to specialist secondary care


Duodenal ulcer
Stop NSAIDs, if used[1]
Test for H. pylori[2]
Test positive, ulcer associated with NSAID use
Full-dose PPI for 2 months
Test negative
Test positive, ulcer not associated with NSAID use
Response
Eradication therapy[3]
No response or relapse
Re-test for H. pylori[4]
Negative
Response
Full-dose PPI for 1 or 2 months
Positive
No response
Eradication therapy[5]
No response or relapse
Low-dose treatment as required[6]
No response
Exclude other causes of DU[7]
Response
Response
Return to self care
Review[8]