यह कार्यक्रम स्वास्थ्य विभाग और राज्य स्वास्थ्य एवं परिवार कल्याण संस्थान (SIHFW), उत्तर प्रदेश की पहल पर उत्तर प्रदेश टेक्निकल सपोर्ट यूनिट (UPTSU) के सहयोग से हो रहा है।

Principal Secretary, Medical Health & Family Welfare, U.P.
Shri Partha Sarthi Sen Sharma

Director Administration & Director, SIHFW
Dr. Rajaganapathy R

एपिसोड 14

स्वास्थ्य विभाग की पहल

शुक्रवार की शाम, डाक्टर्स के नाम

प्रदेश के जाने-माने चिकित्सकों से सीधे जुड़ें और उनके अनुभवों का लाभ उठाएँ

दिनांक : 14 जून, 2024 | समय : सांय 6:00 बजे से 7:30 बजे तक

— वेबकास्ट का विषय —

पेप्टिक अल्सर रोग
(Peptic Ulcer Disease)

वक्ता

डॉ. अजय कुमार पटवा

(MBBS, MD, DM: Gastro-SGPGI)

प्रोफेसर, यकृत एवं पित्त रोग प्रभाग,
मेडिसिन विभाग, किंग जॉर्ज मेडिकल यूनिवर्सिटी,
लखनऊ, उत्तर प्रदेश

आयोजक

राज्य स्वास्थ्य एवं परिवार कल्याण संस्थान (SIHFW)
इंदिरा नगर, लखनऊ, उत्तर प्रदेश

YouTube LIVE STREAMING

PEPTIC ULCER DISEASE

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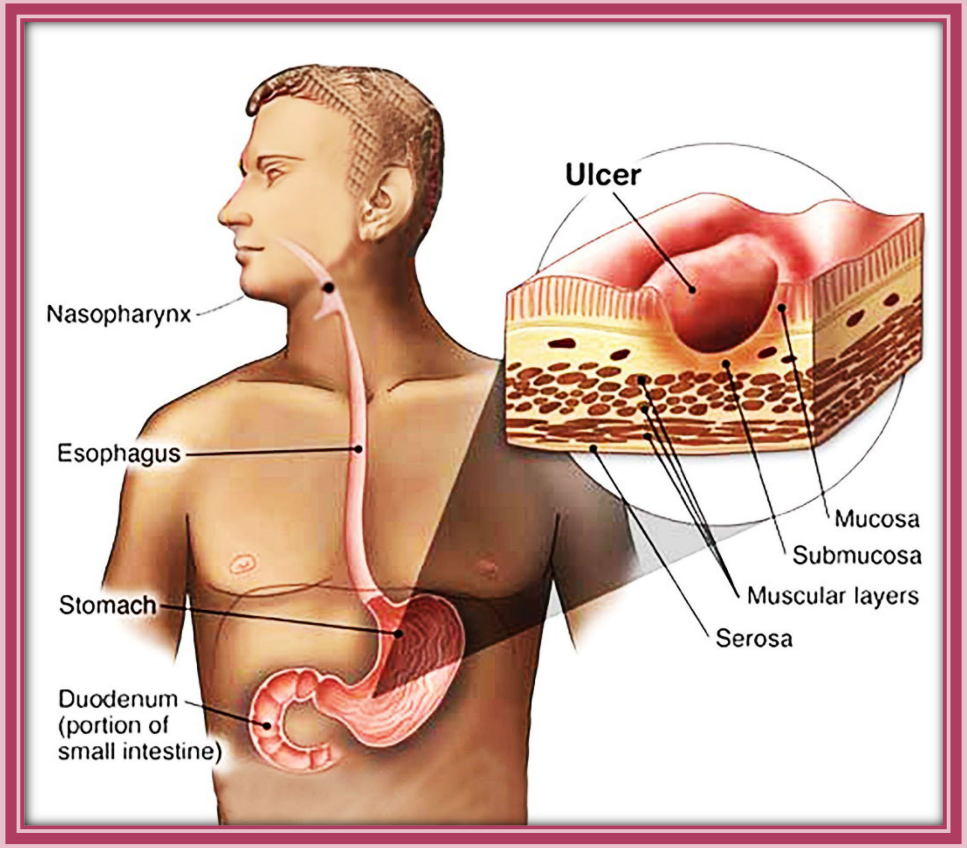
LEARNING OBJECTIVES

- Definition and epidemiology
- Etiopathogenesis
- Clinical features
- Complications
- Management
- Algorithm for the management of undiagnosed PUD
- Case scenario
- Summary

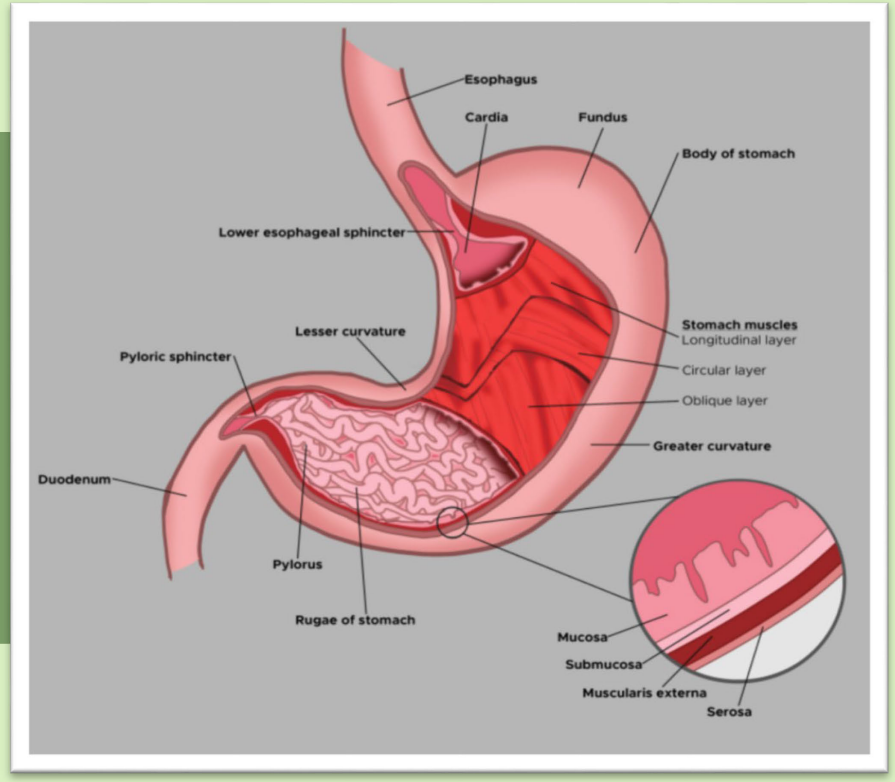


DEFINITION AND EPIDEMIOLOGY

- Ulcers are defined as breaks in the mucosal surface **≥5 mm** in size.
- An erosion is a break less than 5 mm.
- A peptic ulcer is defined as disruption of the mucosal integrity of the stomach and/or duodenum leading to a local defect or excavation due to active inflammation.
- Prevalence=8.4% in US
- Decreasing with time

HISTOLOGY OF STOMACH

The gastric epithelial lining consists of rugae that contain microscopic gastric pits, each branching into four or five gastric glands.



HISTOLOGY OF STOMACH

75% of gastric glands are found within the oxyntic mucosa and contain mucous neck, parietal, chief, endocrine, enterochromaffin, and enterochromaffin-like (ECL) cells

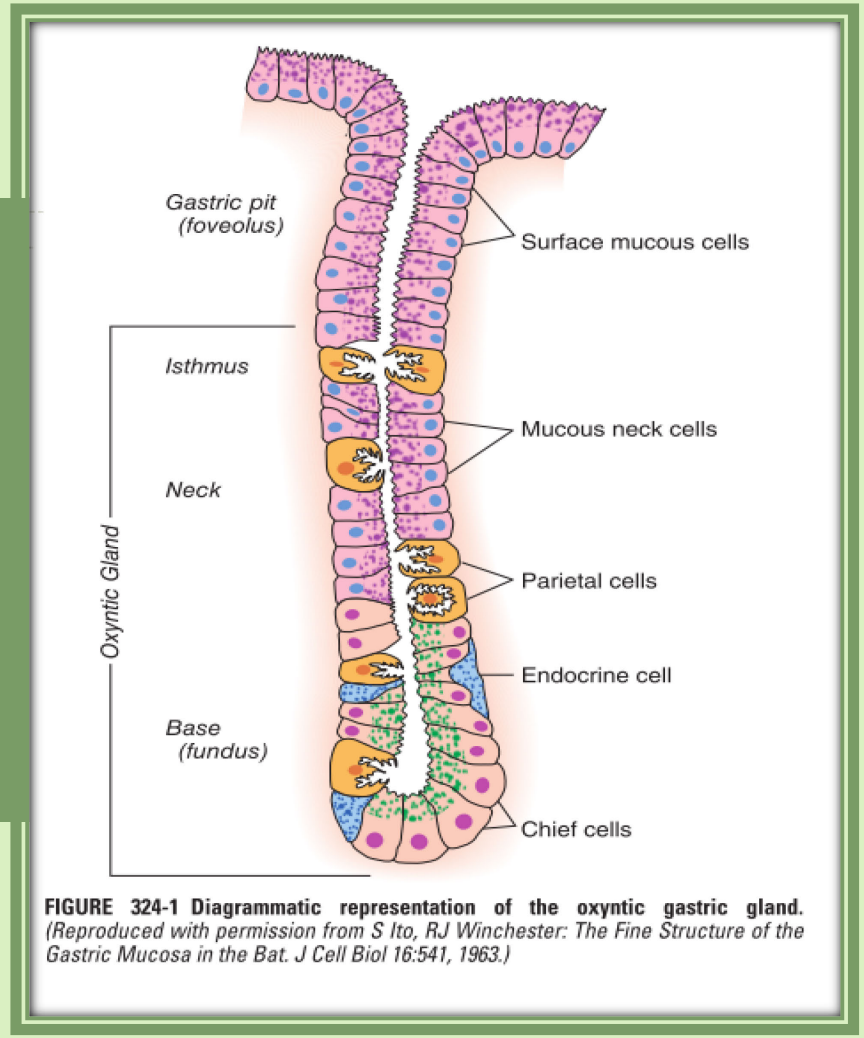


FIGURE 324-1 Diagrammatic representation of the oxyntic gastric gland. *(Reproduced with permission from S Ito, RJ Winchester: The Fine Structure of the Gastric Mucosa in the Bat. J Cell Biol 16:541, 1963.)*

HISTOLOGY OF STOMACH

The parietal cell, also known as the oxyntic cell, is usually found in the neck or isthmus or in the oxyntic gland. It is responsible for secretion of acid, intrinsic factor and IL-11.

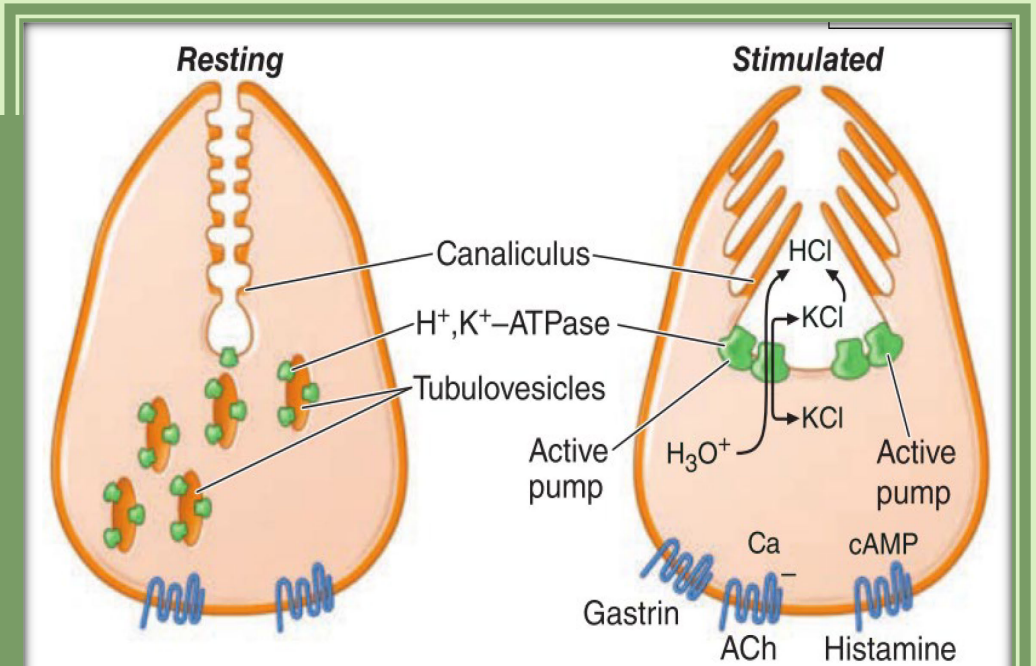
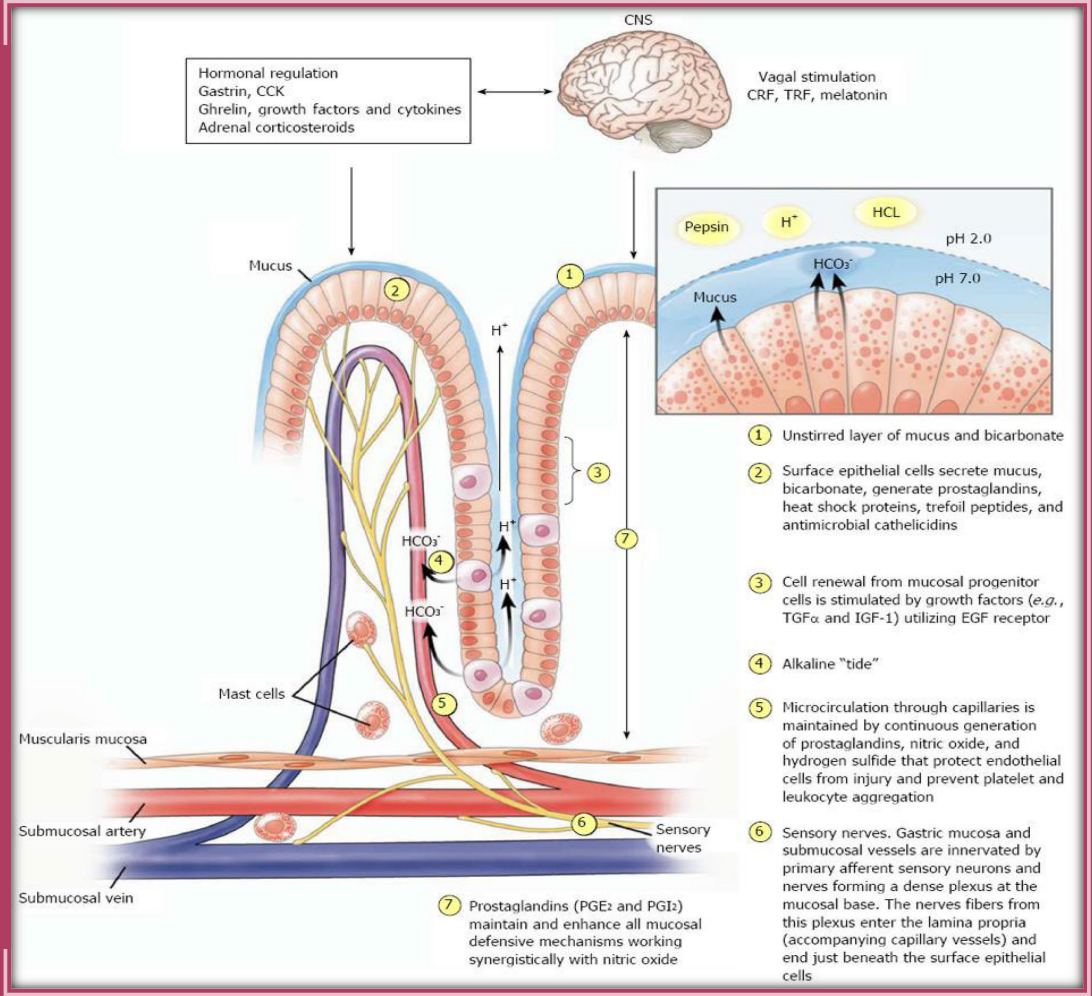


FIGURE 324-2 Gastric parietal cell undergoing transformation after secretagogue-mediated stimulation. cAMP, cyclic adenosine monophosphate. *(Reproduced with permission from SJ Hersey, G Sachs: Gastric acid secretion. Am Physiol Soc 75:155, 1995.)*

MUCOSAL DEFENSE MECHANISM

- Three level barrier –
  - Pre-epithelial (mucus-bicarbonate-phospholipid layer)
  - Epithelial (Surface epithelial cells and intercellular tight junctions)
  - Subepithelial (microvascular system within the gastric submucosal layer)
- Prostaglandins & nitric oxide play a central role in gastric epithelial defence/ repair.



PATHOGENESIS

Ulcer develops when

- Protective factors↓
- Damaging factors ↑

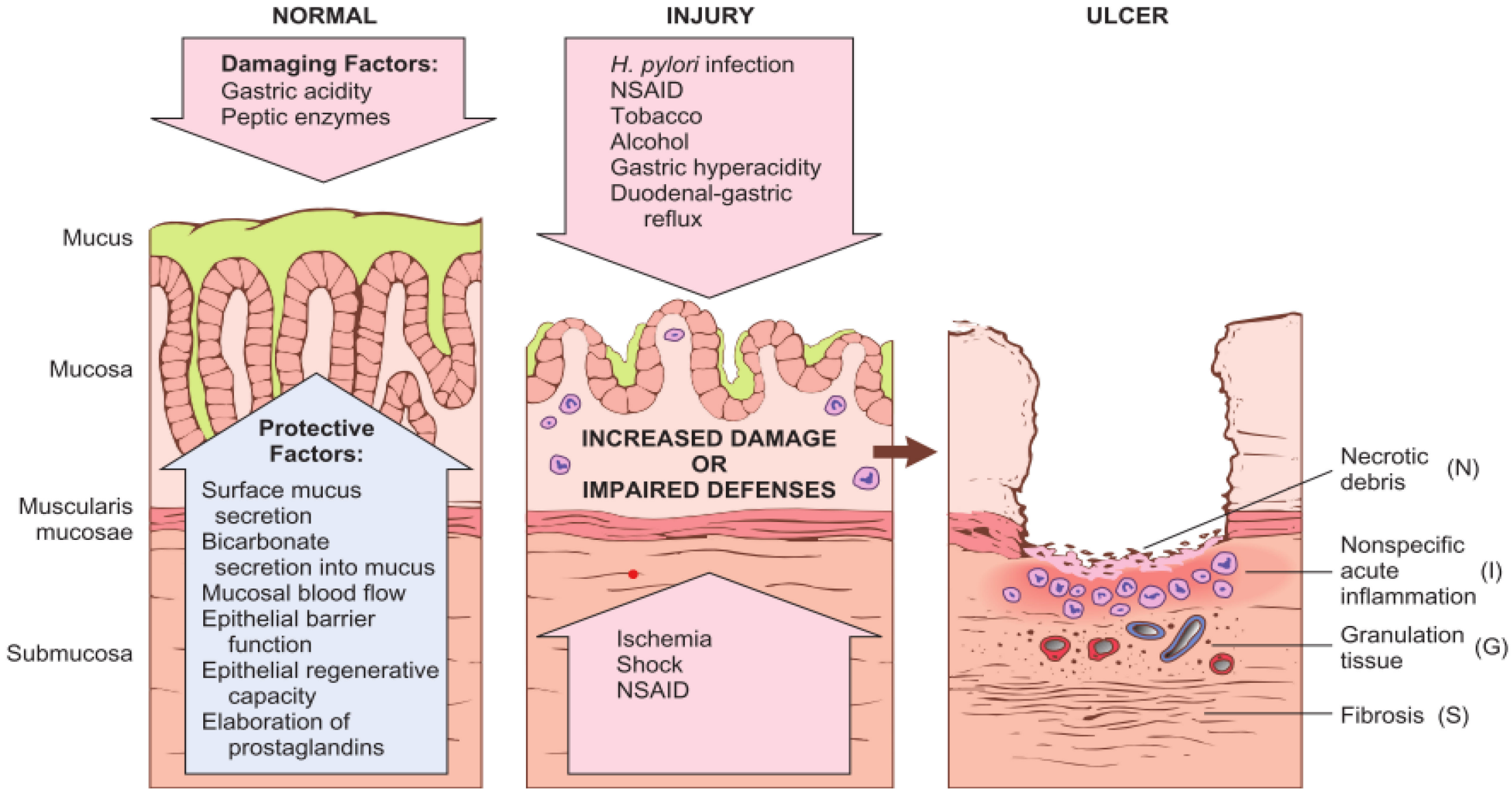


Figure 17-11 Mechanisms of gastric injury and protection. This diagram illustrates the progression from more mild forms of injury to ulceration that may occur with acute or chronic gastritis. Ulcers include layers of necrosis (N), inflammation (I), and granulation tissue (G), but a fibrotic scar (S), which takes time to develop, is only present in chronic lesions.

ETIOLOGY

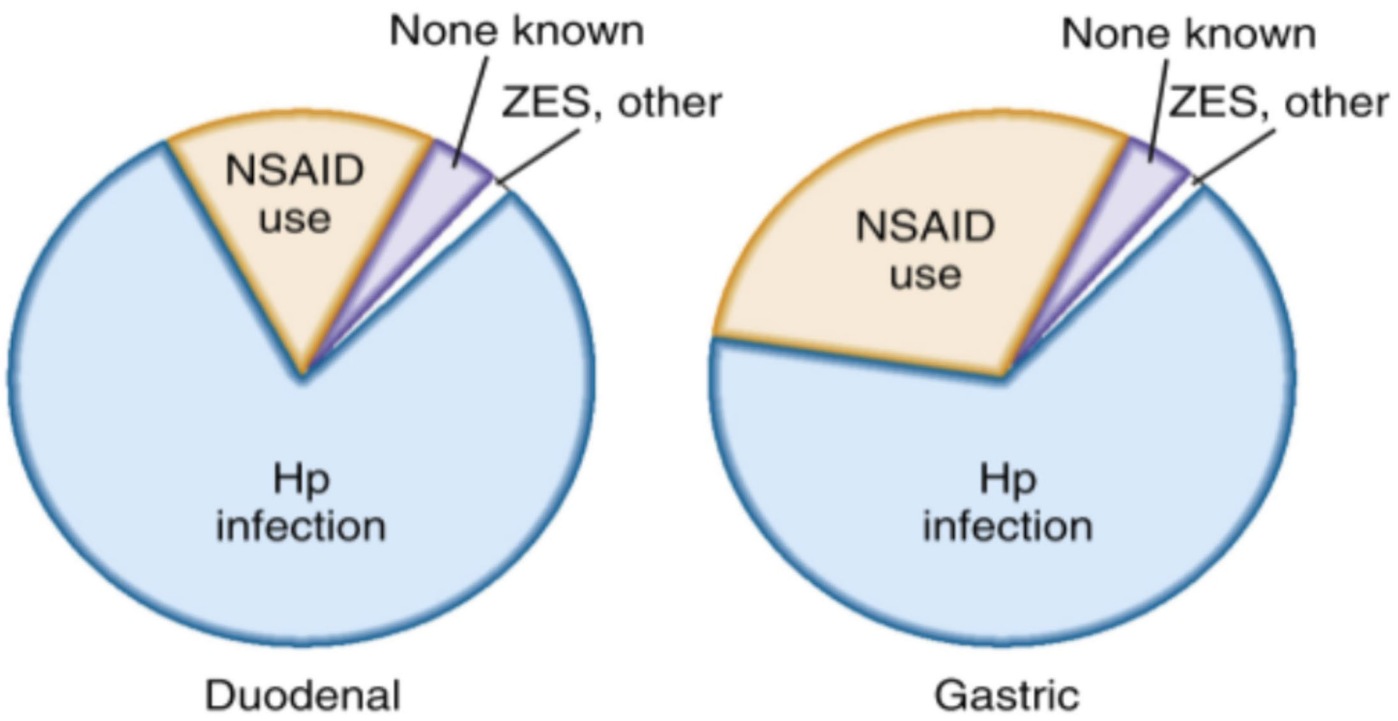


Fig. 53.1 Pie charts depicting conditions associated with PUD. The percentages shown are rough approximations based on studies from Western countries. The relative contributions of Hp infection and NSAID use to peptic ulcer vary considerably among different populations and, within populations, vary with age and socioeconomic status. Also, the separation depicted in this figure is somewhat artificial because NSAID use and Hp infection often coexist.

TABLE 324-1 Causes of Ulcers Not Caused by *Helicobacter pylori* and NSAIDs

Pathogenesis of Non-Hp and Non-NSAID Ulcer Disease
Infection
Cytomegalovirus
Herpes simplex virus
Helicobacter heilmannii
Drug/Toxin
Bisphosphonates
Chemotherapy
Clopidogrel
Crack cocaine
Glucocorticoids (when combined with NSAIDs)
Mycophenolate mofetil
Potassium chloride
Miscellaneous
Basophilia in myeloproliferative disease
Duodenal obstruction (e.g., annular pancreas)
Infiltrating disease
Ischemia
Radiation therapy
Eosinophilic infiltration
Sarcoidosis
Crohn's disease
Idiopathic hypersecretory state

Abbreviations: Hp, *H. pylori*; NSAIDs, nonsteroidal anti-inflammatory drugs.

ASSOCIATIONS WITH PUD

Disorders/ factors with **strong** association to PUD :

- Advanced age
- Chronic pulmonary disease
- Chronic renal failure
- Cirrhosis
- Nephrolithiasis
- α1 antitrypsin deficiency
- Systemic mastocytosis

Disorders/ factors with **possible** association to PUD :

- Hyperparathyroidism
- Coronary artery disease
- Polycythemia vera
- Chronic pancreatitis
- Former alcohol use
- Obesity
- African- American race
- Three or more doctor visits in a year

HELICOBACTER PYLORI

- In developing parts of the world, 80% of the population may be infected by the age of 20.
- H. pylori infection is virtually always associated with a chronic active gastritis.
- Only 10–15% of infected individuals develop frank peptic ulceration.
- H. pylori is present in only 30–60% of individuals with GUs and 50–70% of patients with DUs.
- Result of H. pylori infection –
  - Gastritis
  - Peptic Ulcer Disease
  - Gastric MALT lymphoma
  - Gastric cancer

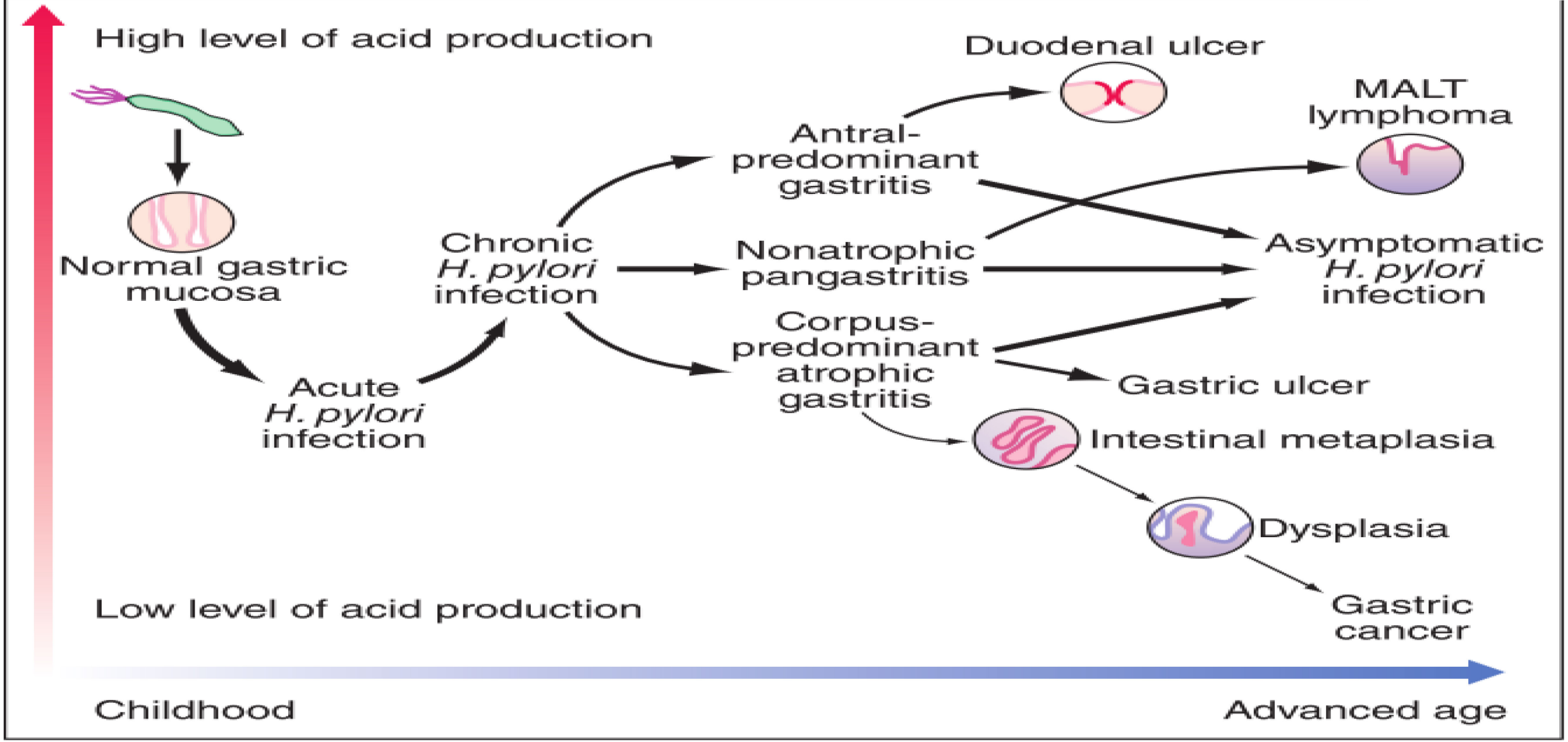


FIGURE 324-8 Natural history of *H. pylori* infection. MALT, mucosal-associated lymphoid tissue. *(From S Suerbaum, P Michetti: Helicobacter pylori infection. N Engl J Med 347:1175, 2002. Copyright © 2002 Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society.)*

NSAID INDUCED PEPTIC ULCER

- Aspirin is increasingly used on regular basis for the prevention of cardiovascular events.
- NSAIDs users who also take aspirin are at an especially high risk for complications.
- NSAIDs damage the gastric and duodenal mucosa by suppression of prostaglandin synthesis.
- Hp infection increases the risk of PUD in patients receiving low- dose aspirin.
- Even **75 mg/d of aspirin** may lead to serious GI ulceration; thus, no dose of NSAID is completely safe. In fact, the incidence of mucosal injury (ulcers and erosions) in patients taking low-dose aspirin (75–325 mg) has been estimated to range from 8% - 60%.

TABLE 53.1 Risk Factors for NSAID Ulcers*

Risk factor	Risk ratio
History of complicated ulcer	13.5
Use of multiple NSAIDs (including aspirin, COX-2 inhibitors)	9
Use of high doses of NSAIDs	7
Use of an anticoagulant	6.4
History of an uncomplicated ulcer	6.1
Age >70 years	5.6
Hp infection	3.5
Use of a glucocorticoid	2.2

*Not all NSAIDs pose the same risk.

CLINICAL FEATURES

- **History:**
  - **Abdominal pain** is common to many GI disorders, including DU or GU, therefore it has a poor predictive value for the presence of either DU or GU.
  - Two-thirds of patients with PUD do not have abdominal pain, and up to 87% of patients with NSAID-induced mucosal disease can present with a complication (bleeding, perforation, and obstruction) without antecedent symptoms.
  - Epigastric pain described as a **burning or gnawing discomfort**.
  - The typical pain pattern in **DU** occurs **90 min to 3 h after a meal** and is frequently relieved by antacids or food.
  - Pain pattern in **GU** patients may be different from that in DU patients, where **discomfort may actually be precipitated by food.**
  - **Pain that awakes the patient from sleep** (between midnight and 3 a.m.) is the most discriminating symptom, with two thirds of DU patients describing this complaint.
  - Elderly patients are less likely to have abdominal pain as a manifestation of PUD and may instead present with a complication such as ulcer bleeding or perforation.
  - **Nausea and weight loss** occur more commonly in Gastric Ulcer patients.

- **Ulcer complication is predicted by** –
  - Variation in the intensity or distribution of the abdominal pain
  - onset of associated symptoms such as nausea and/or vomiting
- **Penetrating Ulcer:**
  - Dyspepsia that becomes constant
  - no longer relieved by food or antacids
  - radiates to the back
- **Perforation:**
  - Sudden onset of severe, generalized abdominal pain
  - gastric outlet obstruction –
  - Pain worsening with meals
  - Early satiety
  - Nausea
  - vomiting of undigested food
- **Bleeding:**
  - Tarry stools or coffee-ground emesis

PHYSICAL EXAMINATION

- **Epigastric tenderness.**
- Tachycardia and orthostasis suggest **active GI blood loss.**
- Severely tender, board-like abdomen suggests a **perforation.**
- Presence of a **succussion splash** indicates retained fluid in the stomach, suggesting **gastric outlet obstruction**.

PEPTIC ULCER RELATED COMPLICATIONS

1. Gastrointestinal bleeding
2. Perforation
3. Gastric outlet obstruction

- **Gastrointestinal Bleeding**
  - **Most common** complication observed in PUD
  - Bleeding and complications of ulcer disease occur more often in individuals **>60 years** of age
  - Greater than 50% of patients with ulcer-related hemorrhage bleed **without any preceding warning signs or symptoms.**

- **Perforation**
  - estimated 30-day **mortality of >20%**
  - Acute abdominal pain, tachycardia, and abdominal rigidity compose the **classic triad** associated with this complication.
  - **Penetration** is a form of perforation in which the ulcer bed tunnels into an adjacent organ.
  - DUs tend to penetrate posteriorly into the **pancreas**, leading to pancreatitis, whereas GUs tend to penetrate into the **left hepatic lobe**.

- **Gastric outlet obstruction**
  - **Least common** ulcer-related complication
  - **Relative obstruction** - secondary to ulcer related inflammation and edema in the peripyloric and duodenal region
  - **Fixed, mechanical obstruction** - secondary to scar formation in the peripyloric areas. This requires endoscopic (balloon dilation with or without placement of a biodegradable stent) or surgical intervention with a stricturoplasty or gastrojejunostomy.
  - **Signs and symptoms**
    - New onset of early satiety
    - Nausea
    - Vomiting
    - Increase of postprandial abdominal pain
    - Weight loss

BOX 53.1 Alarm Features in Patients With UGI Symptoms*

Age older than 55 years with new-onset dyspepsia
Family history of UGI cancer
GI bleeding, acute or chronic, including unexplained iron deficiency
Jaundice
Left supraclavicular lymphadenopathy (Virchow node)
Palpable abdominal mass
Persistent vomiting
Progressive dysphagia
Unintended weight loss

*These features should prompt EGD and often other testing to establish a definitive diagnosis (see Chapter 14).

DIAGNOSTIC EVALUATION

Endoscopy:

- Most sensitive and specific approach for examining the upper GI tract
- Advantages:
  - Direct visualization of the mucosa
  - Photographic documentation of a mucosal defect
  - Facilitates tissue biopsy to rule out malignancy (GU) or H. pylori
  - Helpful in identifying lesions too small to detect by radiographic examination
  - To determine if an ulcer is a source of blood loss

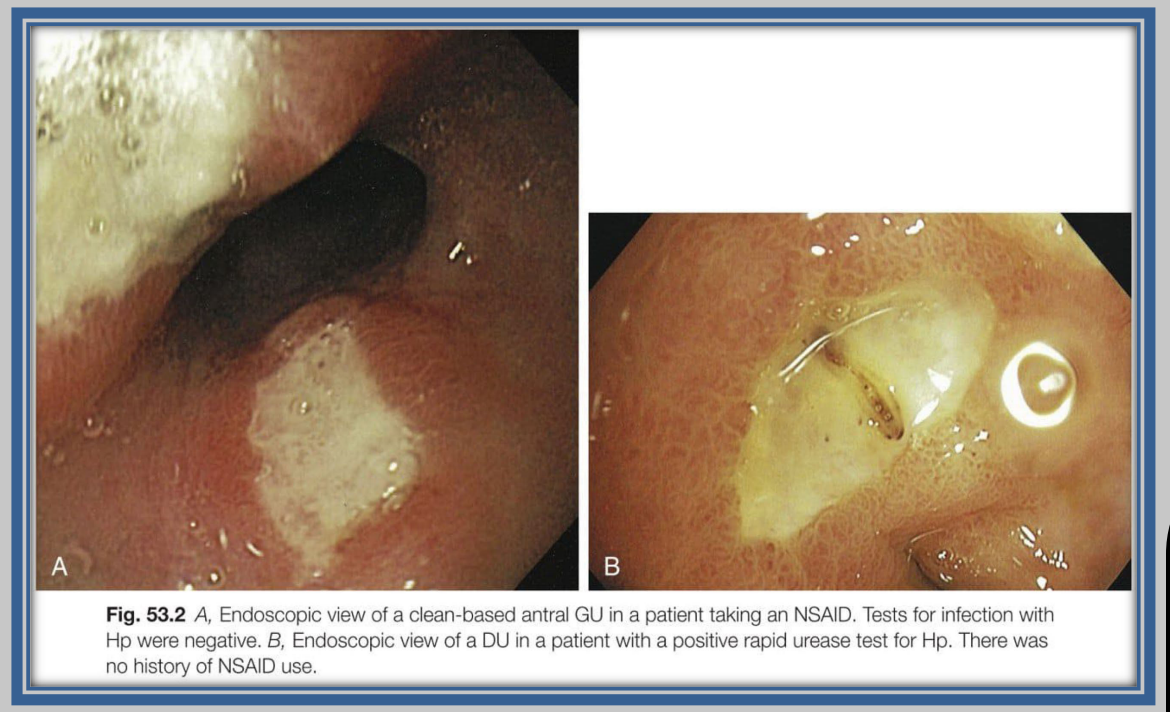


Fig. 53.2 *A*, Endoscopic view of a clean-based antral GU in a patient taking an NSAID. Tests for infection with Hp were negative. *B*, Endoscopic view of a DU in a patient with a positive rapid urease test for Hp. There was no history of NSAID use.

- **<u>Barium studies</u>:**
  - Barium studies of the proximal GI tract are **rarely used** as a first test for documenting an ulcer.
  - The sensitivity of older single-contrast barium meals for detecting a DU is as high as 80%, with a double-contrast study providing detection rates as high as 90%.
  - A benign GU also appears as a discrete crater with radiating mucosal folds originating from the ulcer margin
  - **Ulcers >3 cm** in size or those associated with a mass are more often **malignant.**

METHODS FOR DIAGNOSIS OF H.PYLORI

TABLE 324-2 Tests for Detection of *Helicobacter pylori*		
TEST	**SENSITIVITY/ SPECIFICITY, %**	**COMMENTS**
Invasive (Endoscopy/Biopsy Required)		
Rapid urease	80–95/95–100	Simple, false negative with recent use of PPIs, antibiotics, or bismuth compounds
Histology	80–90/>95	Requires pathology processing and staining; provides histologic information
Culture	—/—	Time-consuming, expensive, dependent on experience; allows determination of antibiotic susceptibility
Noninvasive		
Serology	>80/>90	Inexpensive, convenient; not useful for early follow-up
Urea breath test	>90/>90	Simple, rapid; useful for early follow-up; false negatives with recent therapy (see rapid urease test); exposure to low-dose radiation with ^{14}C test
Stool antigen	>90/>90	Inexpensive, convenient

MANAGEMENT OF PEPTIC ULCER

- Goals of therapy:
  - Acid suppression
  - Eradication of H.pylori
  - Therapy/ prevention of NSAID induced disease

TABLE 324-3 Drugs Used in the Treatment of Peptic Ulcer Disease

DRUG TYPE/MECHANISM	EXAMPLES	DOSE
Acid-Suppressing Drugs		
Antacids	Mylanta, Maalox, Tums, Gaviscon	100–140 meq/L 1 and 3 h after meals and hs
H_2 receptor antagonists	Cimetidine	400 mg bid
	Ranitidine	300 mg hs
	Famotidine	40 mg hs
	Nizatidine	300 mg hs
Proton pump inhibitors	Omeprazole	20 mg/d
	Lansoprazole	30 mg/d
	Rabeprazole	20 mg/d
	Pantoprazole	40 mg/d
	Esomeprazole	20 mg/d
	Dexlansoprazole	30 mg/d
Mucosal Protective Agents		
Sucralfate	Sucralfate	1 g qid
Prostaglandin analogue	Misoprostol	200 µg qid
Bismuth-containing compounds	Bismuth subsalicylate (BSS)	See anti–*H. pylori* regimens (Table 324-4)

Abbreviation: hs, at bedtime (*hora somni*).

PROTON PUMP INHIBITORS (PPIS)

- Most potent acid inhibitory agents available.
- Inhibition of H+K+-ATPase (proton pump).
- PPIs are most effective if they are administered immediately before meals.
- **Agents :**
  - Omeprazole
  - Esomeprazole
  - Lansoprazole
  - Rabeprazole
  - Pantoprazole
  - Dexlansoprazole

- **Side effects :**
  - Higher incidence of community-acquired pneumonia
  - Community- and hospital-acquired **Clostridium difficile– associated disease**
  - Risk of spontaneous bacterial peritonitis in cirrhotics
  - Diarrhea
  - Collagenous colitis particularly with lansoprazole
  - Development of **hip fractures** in older women
  - Development of iron, vitamin B12 and magnesium deficiency
  - Negative effect on the **antiplatlet effect of clopidogrel**
  - **Acute interstitial nephritis**

H2 RECEPTOR ANTAGONISTS

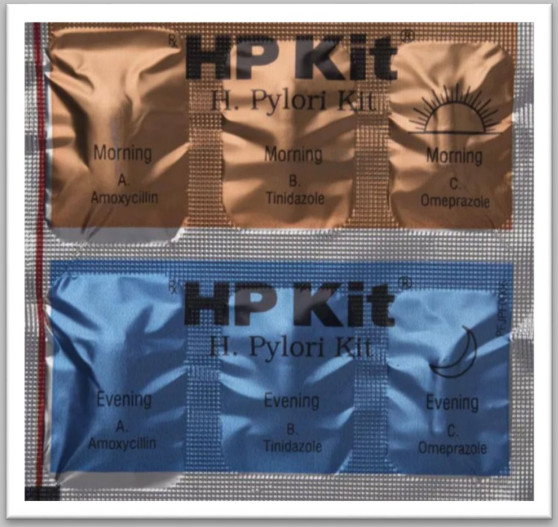
- **Role**: Used where PPI cannot be used.
- **Agents**:
  - Cimetidine
  - Ranitidine
  - Famotidine
  - Nizatidine
- **Side effects**:
  - Antiandrogenic side effects resulting in reversible gynecomastia and impotence (Cimetidine)
  - Inhibit cytochrome P450 (Cimetidine)
  - Elevated levels of serum aminotransferases, creatinine, and serum prolactin (rare, seen with cimetidine)

ANTACIDS

- **Role** : They are now rarely, if ever, used as the primary therapeutic agent. But they are often used by patients for symptomatic relief of dyspepsia.
- **Agents** :
  - Aluminum hydroxide
  - Magnesium hydroxide
  - Calcium carbonate
  - Sodium bicarbonate
- **Side effects** –
  - Constipation (Aluminum hydroxide)
  - Loose stools (Magnesium hydroxide)
  - Milk-alkali syndrome (Calcium carbonate)
  - Alkalosis (Sodium bicarbonate)

INDICATIONS OF THERAPY OF H.PYLORI

- **Documented Peptic Ulcer Disease.**
- Hp test positive in areas of **moderate- to- high Hp prevalence**
- Hp test positive in individuals **aged <60 years** with uninvestigated dyspepsia
- First-degree relatives of family members with **gastric cancer**
- Patients with **previous gastric neoplasm** treated by endoscopic or subtotal resection
- Individuals with a risk of gastritis (**severe pangastritis or body-predominant gastritis**) or severe atrophy
- Patients with **gastric acid inhibition for >1 year**
- Individuals with strong environmental risk factors for gastric cancer (heavy smoking; high exposure to dust, coal, quartz, or cement; and/or work in quarries)
- Patients with **unexplained iron deficiency anemia**
- **Idiopathic thrombocytopenic purpura**



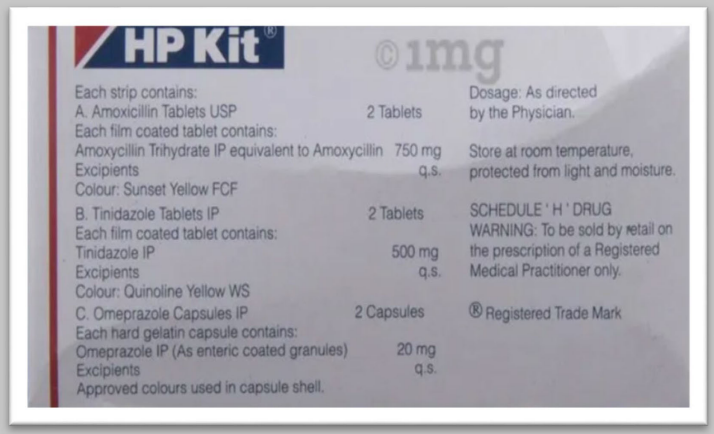


TABLE 52.7 Summary of First-Line Treatment Regimens as Recommended in the 2017 ACG Clinical Guideline on the Treatment of Hp Infection

First-line Regimen	Components	Duration (days)	Recommendation	Level of Evidence	Comments
Clarithromycin triple therapy	PPI, clarithromycin 500 mg, and amoxicillin 1000 mg, each twice daily (or, if penicillin allergic, metronidazole 500 mg 3 times daily in place of amoxicillin)	14	Conditional	Low (For duration, moderate)	Avoid in patients with prior macrolide exposure. Avoid in areas where local clarithromycin resistance rate is ≥15%.
Bismuth-based quadruple therapy	PPI twice daily, bismuth subcitrate or subsalicylate 4 times daily, tetracycline 500 mg 4 times daily, and metronidazole 250 to 500 mg 3 or 4 times daily	10-14	Strong	Low	Particularly recommended for patients with prior macrolide exposure or proven penicillin allergy
Concomitant therapy	PPI, clarithromycin 500 mg, and amoxicillin 1000 mg, and a nitroimidazole 500 mg, each twice daily	10-14	Strong	Very low	Nitroimidazole may be metronidazole or tinidazole
Sequential therapy	PPI and amoxicillin 1000 mg, both twice daily	5-7	Conditional	Low (For duration, very low)	Nitroimidazole may be metronidazole or tinidazole
	PPI, clarithromycin 500 mg, and a nitroimidazole 500 mg, each twice daily	5-7			
Hybrid therapy	PPI and amoxicillin 1000 mg, both twice daily	7	Conditional	Low (For duration, very low)	Nitroimidazole may be metronidazole or tinidazole
	PPI, clarithromycin 500 mg, amoxicillin 1000 mg, and a nitroimidazole 500 mg, each twice daily	7			
Levofloxacin triple therapy	PPI twice daily, levofloxacin 500 mg once daily, and amoxicillin 1000 mg twice daily.	10-14	Conditional	Low (For duration, very low)	—
Levofloxacin sequential therapy	PPI and amoxicillin 1000 mg, each twice daily.	5-7	Conditional	Low (For duration, very low)	Nitroimidazole may be metronidazole or tinidazole
	PPI and amoxicillin 1000 mg, each twice daily, levofloxacin 500 mg once daily, and a nitroimidazole 500 mg twice daily	5-7			

Adapted from Checchi S, Montanaro A, Pasqui L, et al. L-thyroxine requirement in patients with autoimmune hypothyroidism and parietal cell antibodies. J Clin Endocrinol Metab 2008;93:465-9.

MANAGEMENT OF BLEEDING

- Risk stratification done by
  - Rockall scoring system
  - Glasgow Blatchford score (GBS) (Preferred)

SEVERITY ASSESSMENT: ROCKALL SCORING SYSTEM FOR UPPER GI BLEEDING

ABBCDE

Variable	Points			
	0	1	2	3
Age (yr)	<60	60-79	≥80	—
Pulse rate (beats/min)	<100	≥100	—	—
Systolic blood pressure (mm Hg)	Normal	≥100	<100	—
Comorbidity	None	—	Ischemic heart disease, cardiac failure, other major illness	Renal failure, hepatic failure, metastatic cancer
Diagnosis	Mallory-Weiss tear or no lesion observed	All other benign diagnoses	Malignant lesions	—
Endoscopic stigmata of recent hemorrhage	No stigmata or dark spot in ulcer base	—	Blood in upper GI tract, adherent clot, visible vessel, active bleeding	—

SEVERITY ASSESSMENT: ROCKALL SCORING SYSTEM FOR UPPER GI BLEEDING

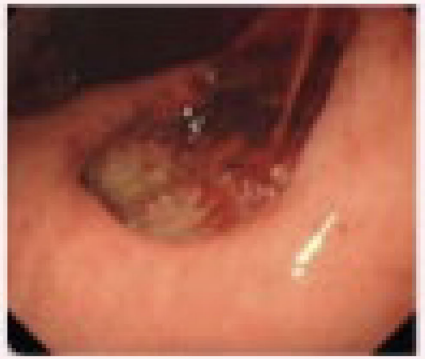
Total Score	Frequency (% of Total)	Rebleeding Rate (%)	Mortality Rate (%)
0	4.9	4.9	0
1	9.5	3.4	0
2	11.4	5.3	0.2
3	15.0	11.2	2.9
4	17.9	14.1	5.3
5	15.3	24.1	10.8
6	10.6	32.9	17.3
7	9.0	43.8	27.0
≥8	6.4	41.8	41.1

TABLE 48-1 Glasgow-Blatchford Score

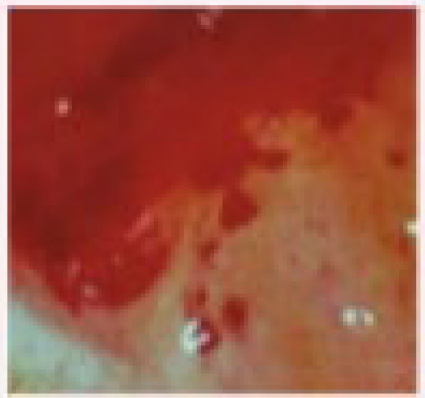
RISK FACTORS AT ADMISSION	SCORE
Blood urea nitrogen (mg/dL)	
18.2 to <22.4	2
22.4 to <28.0	3
28.0 to <70.0	4
≥70.0	6
Hemoglobin (g/dL)	
12.0 to <13.0 (men); 10.0 to <12.0 (women)	1
10.0 to <12.0 (men)	3
<10.0	6
Systolic blood pressure (mmHg)	
100–109	1
90–99	2
<90	3
Heart rate (beats per minute)	
≥100	1
Melena	1
Syncope	2
Hepatic disease	2
Cardiac failure	2

FORREST AND FINLAYSON'S ENDOSCOPIC CLASSIFICATION OF ULCERS

Acute Hemorrhage

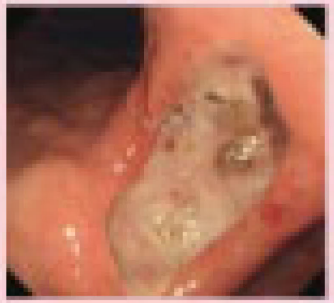


1a
Active Spurting
Rebleeding Risk:
60 to 100%

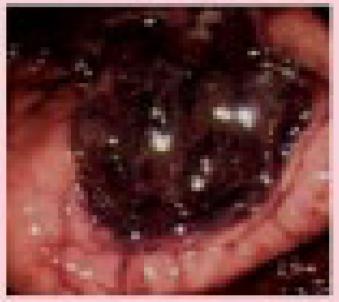


1b
Active Oozing
Rebleeding Risk:
50%

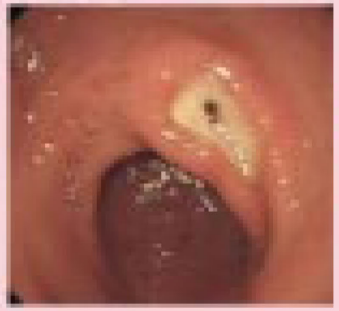
Signs of Recent Hemorrhage



IIa
Non-Bleeding Visible Vessel
Rebleeding Risk:
40 to 50%

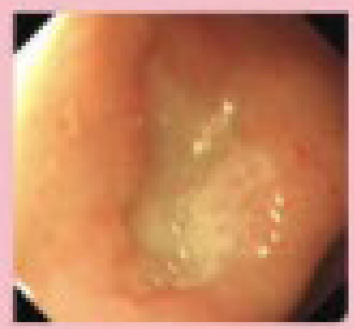


IIb
Adherent Clot
Rebleeding Risk:
20 to 30%



IIc
Flat Spot in Ulcer Base
Rebleeding Risk:
7 to 10%

Lesions without Active Bleeding



III
Clean-Based Ulcer
Rebleeding Risk:
3 to 5%

Initial risk stratification and triage

Very-Low Clinical Risk

- Risk score[a] indicates 0-1% false negatives for transfusion, hemostatic intervention, or death
  - Glasgow-Blatchford Score 0-1

Not Very-Low Clinical Risk

- Risk score indicates >1% false negatives for transfusion, hemostatic intervention, or death
  - Glasgow-Blatchford Score ≥2

Discharge from emergency department with outpatient management if no other reason for hospitalization

Admit to hospital or observation unit

Pre-endoscopic management

- Resuscitation, attention to active comorbidities
- RBC transfusion if hemoglobin <7 g/dL
- Suggest erythromycin 250mg infusion 30-90 minutes before upper endoscopy
- No recommendation for or against proton pump inhibitors

Endoscopy

Upper endoscopy within 24 hours of presentation

Low-risk endoscopic findings

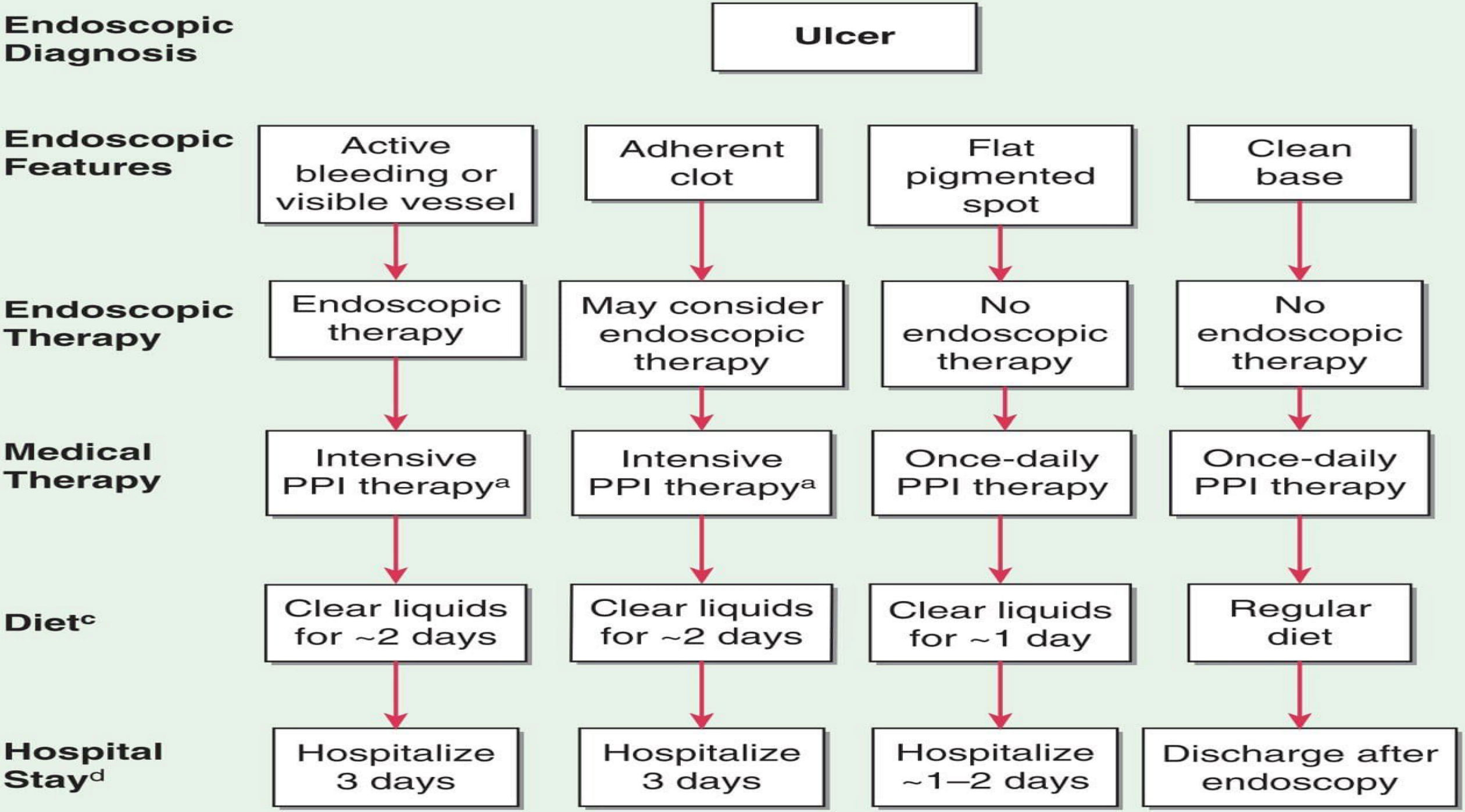
- e.g., clean-based ulcer, nonbleeding Mallory-Weiss tear, erosions

Discharge patient if stable vital signs and hemoglobin, and no other reason for hospitalization

Non-low-risk endoscopic findings

- e.g., ulcer with stigmata of hemorrhage, varices, neoplasm, Dieulafoy lesion

Patient remains in hospital


Endoscopic Diagnosis
Ulcer
Endoscopic Features
Active bleeding or visible vessel
Adherent clot
Flat pigmented spot
Clean base
Endoscopic Therapy
Endoscopic therapy
May consider endoscopic therapy
No endoscopic therapy
No endoscopic therapy
Medical Therapy
Intensive PPI therapy[a]
Intensive PPI therapy[a]
Once-daily PPI therapy
Once-daily PPI therapy
Diet[c]
Clear liquids for ~2 days
Clear liquids for ~2 days
Clear liquids for ~1 day
Regular diet
Hospital Stay[d]
Hospitalize 3 days
Hospitalize 3 days
Hospitalize ~1–2 days
Discharge after endoscopy

SURGICAL THERAPY IN PUD

- Specific operations for duodenal ulcers:
  - vagotomy and drainage (by pyloroplasty, gastroduodenostomy, or gastrojejunostomy),
  - highly selective vagotomy (which does not require a drainage procedure)
  - vagotomy with antrectomy (Billroth I and Billroth II)
- Specific Operations for Gastric Ulcers:
  - Antrectomy (including the ulcer) with a Billroth I anastomosis is the treatment of choice for an antral ulcer
  - Subtotal gastrectomy with a Roux-en-Y esophagogastrojejunostomy (Csendes' procedure)
  - Antrectomy
  - Intraoperative ulcer biopsy, and vagotomy (Kelling-Madlener procedure)

ALGORITHM FOR THE MANAGEMENT OF UNDIAGNOSED PUD

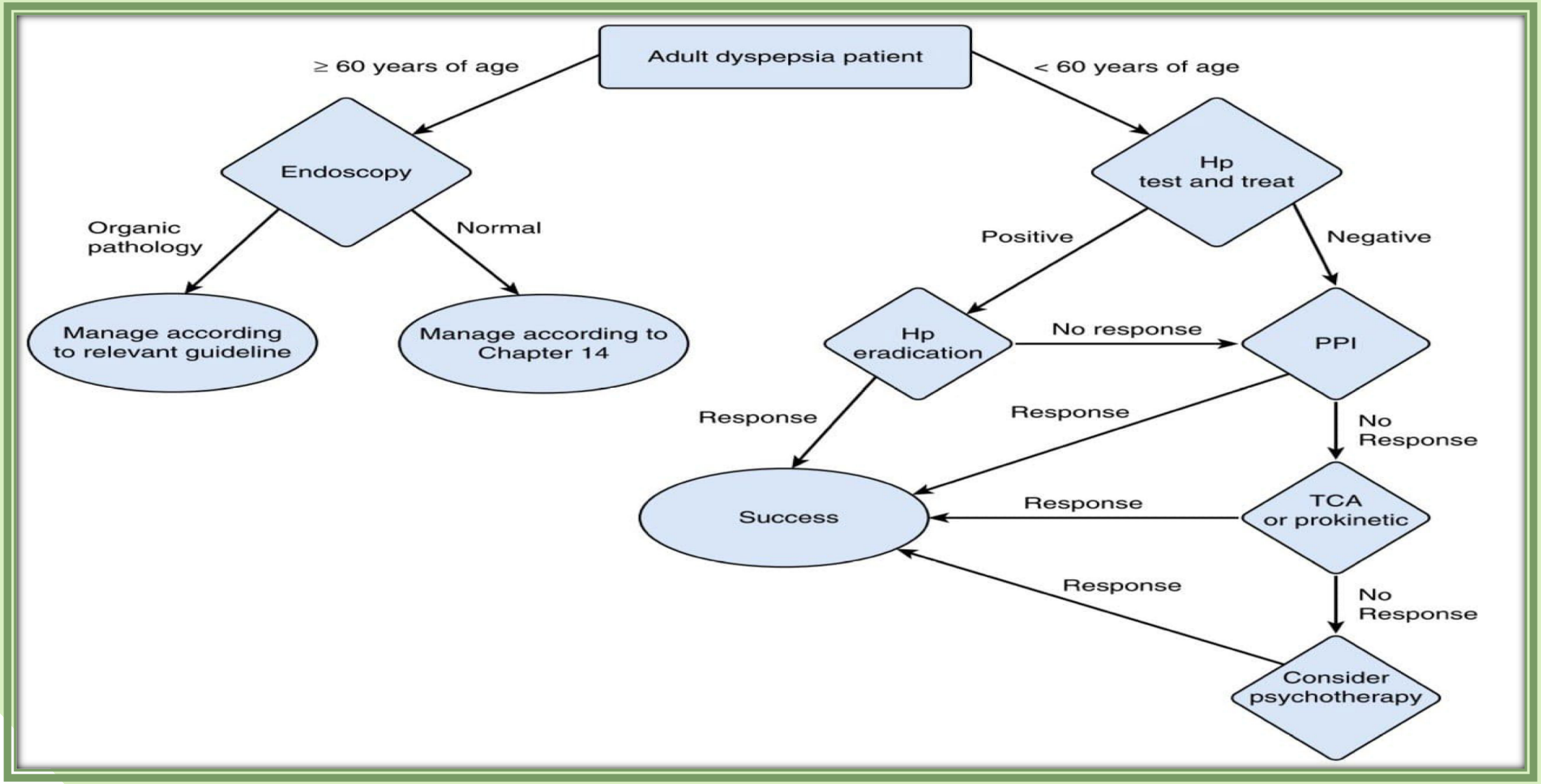


TABLE 53.2 Recommendations for Reducing the Risk of NSAID Ulcers as a Function of GI and Cardiovascular Risk

	GI Risk*		
	Low	**Moderate**	**High**
Low CV risk	NSAID at the lowest effective dose	NSAID plus a PPI, or celecoxib alone	Celecoxib plus a PPI
High CV risk†	Naproxen or celecoxib, plus a PPI	Naproxen or celecoxib, plus a PPI	Celecoxib plus a PPI if simple analgesics failed

**Low GI risk* denotes no risk factors (see Table 53.1); *moderate GI risk* denotes 1 or 2 risk factors; *high GI risk* denotes ≥3 risk factors, prior complicated ulcer, or concomitant use of low-dose aspirin or anticoagulants. All patients with a history of ulcer who require NSAIDs should be tested for Hp, and if infection is present, eradication therapy should be given (see Chapter 52).

†*High CV risk* denotes the requirement for prophylactic low-dose aspirin for primary or secondary prevention of serious CV events.

CV, cardiovascular.

CASE SCENARIO

1. A 45 years old male without any comorbid illness, with history of daily alcohol intake for 15 years presented with vomiting of red coloured blood for 1 day. He takes on and off painkiller for headache. Examination showed feeble pulse, rate 110/minute, BP 100/60 mmHg and mild pallor. Abdominal examination was normal.

- What is the most probable diagnosis in this case?

A. Esophageal varices

B. Duodenal ulcer

C. Mallory Weis tear

D. Acute upper GI bleed

INVESTIGATION

- A 45 years old male without any comorbid illness, with history of daily alcohol intake for 15 years presented with vomiting of red coloured blood for 1 day. He takes on and off painkiller for headache. Examination showed feeble pulse, rate 110/minute, BP 100/60 mmHg and mild pallor. Abdominal examination was normal.
- What is the most useful investigation in this case?

A. Endoscopy
B. Ultrasound
C. CT abdomen
D. Barium meal

TREATMENT

- A 45 years old male without any comorbid illness, with history of daily alcohol intake for 15 years presented with vomiting of red coloured blood for 1 day. Examination showed feeble pulse, rate 110/minute, BP 100/60 mmHg and mild pallor. Abdominal examination was normal. Upper GI endoscopy shows large ulcer at antrum of stomach with overlying dark red adherent blood clot. Rapid urease test was positive for H. pylori.

- What should be the next step?

A. Oral PPI and send home

B. Admission in emergency for monitoring and further investigation

C. PRBC transfusion

D. Refer to higher centre

SUPPORTIVE AND SPECIFIC TREATMENT

- A 45 years old male without any comorbid illness, with history of daily alcohol intake for 15 years presented with vomiting of red coloured blood for 1 day. Examination showed feeble pulse, rate 110/minute, BP 100/60 mmHg and mild pallor. Abdominal examination was normal. Upper GI endoscopy shows large ulcer at antrum of stomach with overlying dark red adherent blood clot. Rapid urease test was positive for H. pylori. Patient was admitted in emergency. Vitals remained normal except tachycardia. Intravenous fluid was started.

- What should be the next best management step?

A. IV PPI infusion with endoscopic therapy

B. IV PPI infusion only with no endoscopic therapy

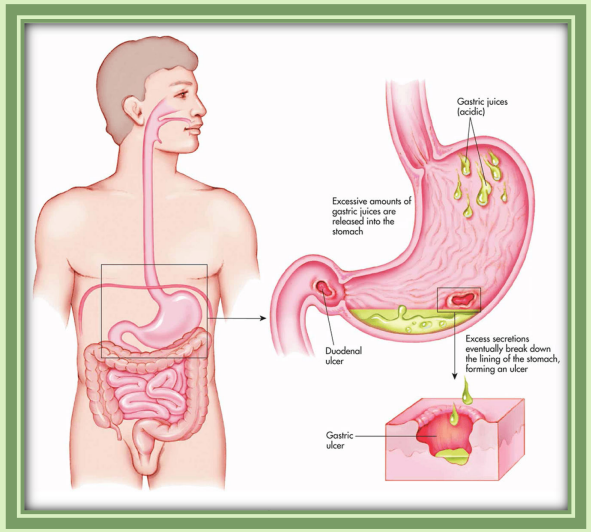
C. Vasoactive drugs plus band ligation

D. Anti H. pylori therapy

SUMMARY

- Peptic ulcer disease is a syndrome of clinical scenario caused by ulceration in stomach and duodenum
- Its prevalence is decreasing due to better understanding of pathogenesis, early diagnosis and treatment
- NSAIDs and H. pylori are two main culprits
- Early diagnosis can be made by history and endoscopy
- Treatment involves symptomatic treatment, acid suppression, H pylori eradication and managing complications

Thank You


Gastric juices (acidic)
Excessive amounts of gastric juices are released into the stomach
Duodenal ulcer
Excess secretions eventually break down the lining of the stomach, forming an ulcer
Gastric ulcer