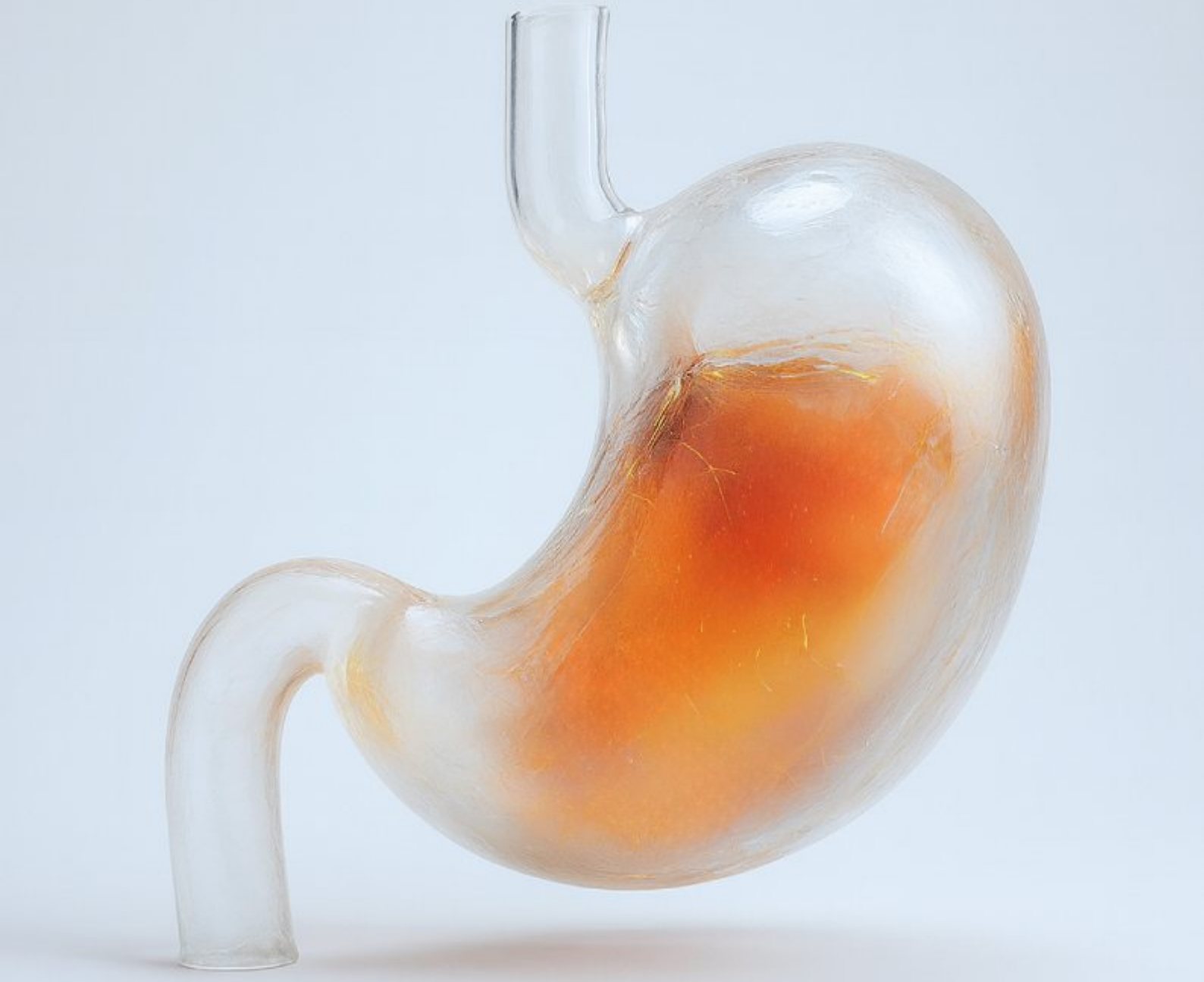
ACID PEPTIC DISEASE

National Standard Treatment Guideline



Ministry of Health
Republic of Maldives

ADB

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Japan Fund for Prosperous and Resilient Asia and the Pacific

From the People of Japan

World Health Organization
Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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ACID PEPTIC ULCER DISEASE

QUICK REFERENCE GUIDE

Acid-peptic disease (APD) is mucosal injury caused by gastric acid and pepsin affecting the esophagus, stomach, or duodenum. The spectrum spans gastroesophageal reflux disease (GERD), gastritis/gastropathy, and peptic ulcer disease (PUD), with functional dyspepsia, Rome IV symptoms without structural disease often overlapping.

Causes, Risk factors & Triggers

- **Infectious:** H. pylori.
- **Drugs:** nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, corticosteroids + NSAIDs, bisphosphonates, potassium chloride, doxycycline, iron, selective serotonin reuptake inhibitors (SSRIs) with NSAIDs, anticoagulants.
- **Physiologic/other:** acid hypersecretion (e.g., Zollinger-Ellison syndrome), stress illness, diabetes (gastroparesis), tobacco, alcohol, spicy/fatty/acidic foods, late meals, obesity (for reflux).

Evaluation for Diagnosis

Clinical features: Epigastric pain/burning, post-prandial fullness, early satiety, heartburn, regurgitation, nausea, bloating.

Alarm features: gastrointestinal bleeding, anemia, weight loss, progressive dysphagia, persistent vomiting, palpable mass, jaundice.

Physical examination: Vitals, pallor, dehydration, abdominal tenderness, peritonitis signs, stool color (melena).

Laboratory investigations: Complete blood count (CBC), renal and liver panel if ill/ bleeding, iron studies if anemia. **Noninvasive H. *pylori testing***: urea breath test (UBT) or stool antigen. Avoid antibiotics for 4 weeks and proton-pump inhibitors (PPIs) for ~2 weeks before testing when possible.

Confirmation of diagnosis

- **Upper endoscopy (esophagogastroduodenoscopy, EGD):** for alarm features, age ≥55 with new symptoms, failure of therapy, suspected ulcer complications.
- **Biopsy:** gastric ulcer margins; test for H. pylori if not already done.

Classification / severity assessment

- **Uncomplicated APD:** no bleeding, perforation, or obstruction.
- **Complicated APD:** upper gastrointestinal bleeding, perforation, gastric outlet obstruction.
- **Ulcer size:** giant/complex >2 cm.
- **Treatment response:** responsive vs refractory/non-healing at expected time points (duodenal ulcer persistent symptoms at 4 weeks; gastric ulcer not healed by 8-12 weeks).

Differential Diagnosis

- Functional dyspepsia, GERD, biliary colic/ cholecystitis, pancreatitis, acute coronary syndrome (inferior wall), gastroparesis, eosinophilic esophagitis, celiac disease, gastric malignancy, drug-induced dyspepsia.

Management Goals & principles

- Heal mucosa, relieve symptoms, prevent complications and recurrence, minimize unnecessary long-term PPI exposure, eradicate H. pylori when present, and address modifiable risks (NSAIDs, smoking, alcohol).

Approach to management

1. **Stratify:** alarm features/complications - urgent endoscopy and resuscitation as needed.
2. **Test-and-treat *H. pylori*** when indicated; confirm cure.
3. **Acid suppression:** short, effective course; correct timing (PPIs 30-60 min before meals).
5. **Review co-medications:** stop or protect when NSAIDs/antithrombotics are essential.
6. **Plan step-down** after control; lowest effective dose or on-demand.
7. **Follow up:** 4-8 weeks to assess response and adjust.

Non-Pharmacological interventions

- Smaller meals; avoid late-night eating; avoid lying down for 2-3 hours after meals.
- Elevate head of bed 6-8 inches for nocturnal reflux.
- Identify personal trigger foods; reduce tobacco and alcohol.
- In high-prevalence settings, promote hygiene and safe water to reduce *H. pylori* transmission.
- Use a simple symptom/medication diary (paper or phone notes).
- Low-resource: focused counseling, OTC antacids for brief rescue, prioritize UBT/ stool antigen for *H. pylori* where endoscopy access is limited.

Pharmacological therapy

Symptom control (typical adult doses)

Drug class	Generic name	Dose & route	Duration	Key cautions
Proton-pump inhibitor (PPI)	Omeprazole / Pantoprazole / Esomeprazole	20-40 mg orally once daily (before breakfast); consider twice daily for refractory cases	4-8 weeks	Long-term risks: hypomagnesemia, vitamin B12 deficiency, infections, fractures, renal injury - use lowest effective dose
Potassium-competitive acid blocker (PCAB)	Vonoprazan	10-20 mg orally once daily	4-8 weeks or per regimen	Potent acid suppression; drug interactions - follow local guidance
Histamine-2 receptor antagonist (H2RA)	Famotidine	20 mg orally twice daily or 40 mg at night	4-8 weeks / on-demand	Tachyphylaxis with continuous use; renal dose adjustment
Antacids/alginate	Calcium carbonate; alginate-antacid	Per label, as needed	PRN (as needed)	Avoid chronic overuse; separate from some meds

H. pylori eradication regimen (tailor to resistance and local guidelines)

- **Bismuth quadruple (14 days):** PPI 12 hourly +Bismuth subsalicylate 300 mg QID+Tetracycline 500 mg QID +Metronidazole 500 mg 8 hourly
- **Concomitant (14 days):** PPI standard dose BID + amoxicillin 1000 mg BID + clarithromycin 500 mg BID + metronidazole or tinidazole 500 mg BID

- **PCAB-based regimens:** vonoprazan with amoxicillin ± clarithromycin per local guidance.
- **Test-of-cure:** UBT or stool antigen ≥4 weeks after therapy; off PPI ~2 weeks if feasible.

Gastroprotection when NSAIDs are essential

- Continue PPI or PCAB during NSAID therapy; consider switching to COX-2 selective agent plus PPI in high-risk patients.

Assessment of response, review, and adjustment

- **4-8 week review**: symptom control, adherence, correct PPI timing, co-medications, lifestyle.
- **Step-down** after healing: reduce PPI dose (e.g., 40 → 20 mg), switch to on-demand, or trial histamine-2 receptor antagonist.
- **Before step-up/step-down, check**: adherence (pill counts/refills), ongoing NSAIDs/aspirin, smoking/alcohol, atypical etiologies (e.g., Zollinger-Ellison syndrome).
- **Gastric ulcer**: repeat endoscopy at 8-12 weeks if symptoms persist or if high-risk features/uncertain etiology; biopsy any persistent ulcer.
- **Duodenal ulcer**: no routine repeat endoscopy unless symptoms persist at 4 weeks or recur.

Referral for specialist consultation (tiered)

- **Immediate hospital/tertiary care**: hematemesis/melena with instability, perforation/peritonitis, gastric outlet obstruction, severe anemia/syncope.
- **Early gastroenterology (secondary/ tertiary)**: alarm features; non-healing gastric ulcer at 8-12 weeks; duodenal ulcer with persistent symptoms at 4 weeks or recurrence; giant/complex ulcers (>2 cm); recurrent bleeding; suspected malignancy; refractory symptoms despite optimized therapy; need for advanced testing (pH-impedance, endoscopic ultrasound, gastric emptying).
- **Endocrinology/oncology**: confirmed/ suspected Zollinger-Ellison syndrome; proven gastric cancer for staging and multidisciplinary care.

Complications

- Upper gastrointestinal bleeding, perforation, penetration, gastric outlet obstruction, strictures, iron-deficiency anemia, malignancy in non-healing gastric ulcers.

Patient education

Objectives

- Build understanding of disease and triggers; ensure correct medication use; reduce risk behaviors; recognize red flags; support step-down when stable.

Instructions

- Take PPIs 30-60 minutes before meals; complete any H. pylori regimen and return for test-of-cure.
- Avoid or minimize NSAIDs; if essential, use protection and discuss dosing.
- Eat smaller meals; avoid lying down for 2-3 hours after eating; elevate the head of the bed for night symptoms.
- Stop smoking; limit alcohol; track personal triggers in a brief diary.
- **Red flags - seek care now:** vomiting blood, black stools, severe or sudden abdominal pain, persistent vomiting, new/progressive dysphagia, unintentional weight loss, fainting.
- Keep follow-ups; ask about lowering to the smallest effective dose once controlled.

ACID PEPTIC DISEASE

INTRODUCTION

Acid Peptic Disease (APD) refers to a spectrum of disorders and includes gastritis, gastric ulcer, duodenal ulcer, and gastro-esophageal reflux disease (GERD), in which the gastrointestinal mucosa is damaged by acid and pepsin. Global peptic-ulcer prevalence and incidence have fallen since 1990, yet lifetime risk remains ~5-10%, and millions of new cases still occur each year. Helicobacter pylori drive much of the burden, with high prevalence across Southeast Asia and some of the world's highest PUD mortality rates in parts of the region. Maldives data are limited; H. pylori is likely common given South Asian estimates. APD is usually controlled by acid suppression and H. pylori eradication, but inconsistent care leads to both under-treatment and unnecessary long-term proton-pump inhibitor use with safety risks. Standardized protocols improve cure rates, curb resistance, cut recurrences, and use resources better.

SCOPE OF THE GUIDELINES

These guidelines applies to all patients with suspected or confirmed acid peptic disease. Guides evaluation, diagnosis, risk stratification, treatment, and follow-up with an emphasis on early detection and evidence-based care, including when to escalate or refer for endoscopy or specialist input. Surgical management is outside the scope and should follow specialized protocols.

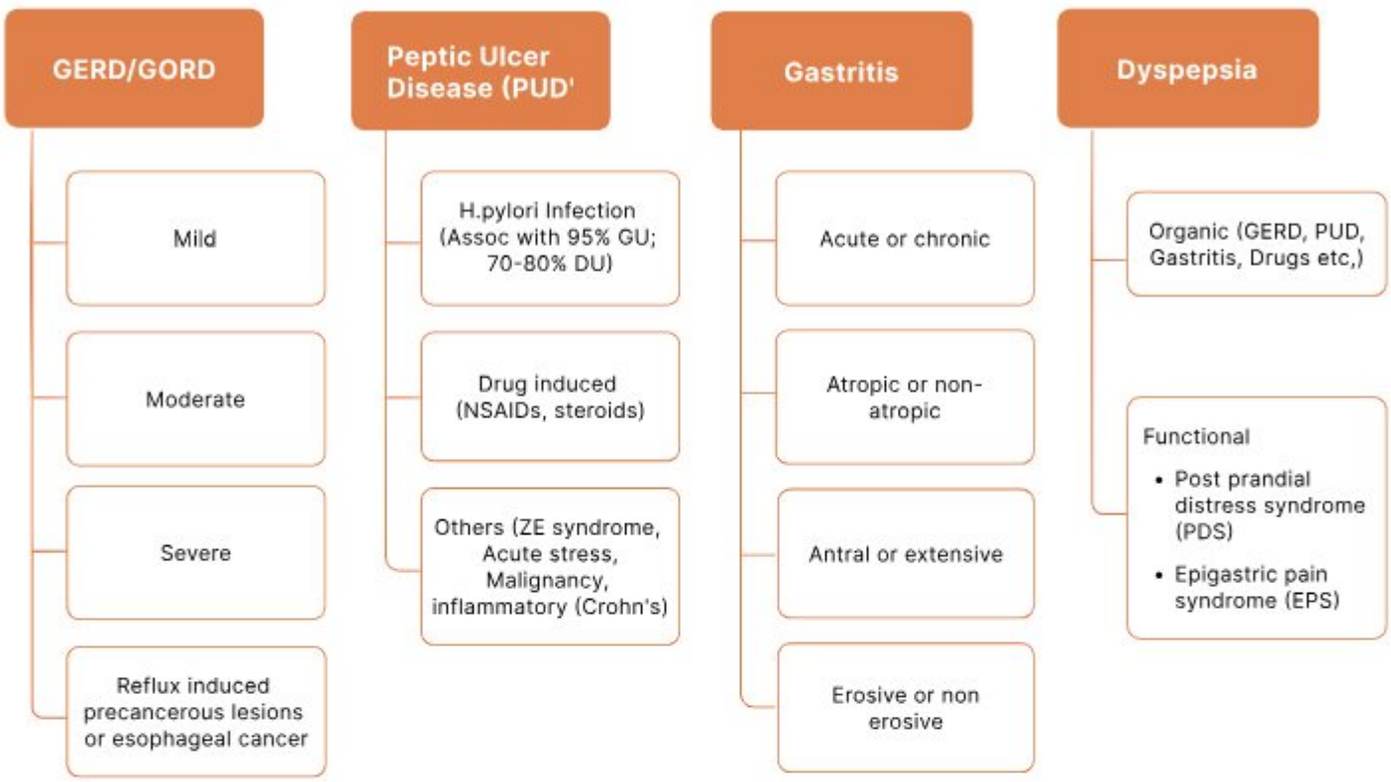
Intended users

Primary care physicians, general practitioners, internists, gastroenterologists, nurses, dietitians, allied health professionals, and health program managers. Offers simplified, standardized protocols with clear referral criteria and practical interim management to maintain continuity of care and reduce delays, even with limited infrastructure and specialists.

Use across levels of care

- **Primary care:** Identify APD early, do the initial work-up, start basic therapy, give diet and lifestyle advice, recognize alarm features, and refer promptly.
- **Secondary care:** Perform targeted diagnostics including selective endoscopy, optimize medical therapy, and manage complications.
- **Tertiary care:** Provide advanced diagnostics and manage refractory or complex disease.

TYPES OF ACID PEPTIC DISEASE



DEFINITIONS

- **Acid peptic disease (APD):** An umbrella term for conditions in which gastric acid/ pepsin injure the upper GI mucosa - principally gastritis, peptic ulcer disease (gastric and duodenal ulcers), and gastro-esophageal reflux disease. (Merck Manuals)
- **Dyspepsia:** Upper abdominal symptoms such as epigastric pain/burning, early satiety, or post-prandial fullness; “functional dyspepsia” meets Rome IV criteria with no structural disease. (British Society of Gastroenterology 2022)
- **Gastritis:** Histologic inflammation of the gastric mucosa (acute or chronic), classified by cause, distribution, and histology (for example, atrophic gastritis).(AGA 2021)
- **Gastric ulcer:** A peptic ulcer located in the stomach: a mucosal break >5 mm penetrating through the muscularis mucosae. (BMJ Best Practice)
- **Duodenal ulcer**: A peptic ulcer in the duodenum: a mucosal break >5 mm with depth to at least the muscularis mucosa. (BMJ Best Practice)

  For a lesion to be considered a peptic ulcer (either gastric or duodenal), it must penetrate through the muscularis mucosae. If it stops at or above this layer, it is an erosion.

- **Gastro-esophageal reflux disease (GERD):** Symptoms and/or mucosal injury caused by abnormal reflux of gastric contents into the esophagus (and beyond). (ACG 2023)
- **Non-erosive reflux disease (NERD):** A GERD phenotype with typical reflux symptoms and no endoscopic mucosal breaks; diagnosis is supported by reflux monitoring per Lyon Consensus 2.0. (ACG 2023)
- **Eosinophilic esophagitis (EoE):** Chronic, immune-mediated esophageal disease defined by symptoms of esophageal dysfunction and ≥15 eosinophils per high-power field on esophageal biopsy after excluding other causes.
- **Gastroparesis:** Symptoms consistent with delayed gastric emptying (early satiety, post-prandial fullness, nausea, vomiting, upper abdominal pain) with objective evidence of delayed emptying and no mechanical obstruction.

RISK FACTORS & TRIGGERS

Identifying and addressing these factors is crucial to prevent onset or recurrence:

Table . Causes & Risk Factors of Acid-Peptic Disorders

Disorder	Lifestyle & Behaviors	Dietary / Environmental Triggers	Associated Conditions & Agents	Medications & Drugs	Other Risk Factors
Dyspepsia (organic)	Smoking; Alcohol use; NSAID use	Fatty meals; Spicy foods; Carbonated drinks; Caffeine	Peptic ulcer; GERD; Gastric or esophageal cancer; Pancreatic/ biliary disorders; Food/drug intolerance; Other systemic diseases	NSAIDs; Bisphosphonates; Potassium chloride	Advanced age; H. pylori infection; Psychological stress
Dyspepsia (functional)	Stress/ anxiety; Irregular eating patterns; Sedentary habits	Fatty meals; Spicy foods; Coffee; Alcohol; Carbonated drinks	Genetic predisposition; H. pylori infection; Mucosal inflammation; Psychosocial factors; Delayed gastric emptying; Hypersensitivity to distension; Altered duodenal sensitivity; Autonomic dysregulation; Dysbiosis	NSAIDs (occasionally trigger symptoms)	-

GERD/NERD	Obesity; Pregnancy (NERD); Smoking; Heavy lifting; Overeating; Eating just before bedtime	High-fat foods; Citrus fruits; Tomato products; Carbonated beverages; Alcohol; Chocolate; Peppermint; Onion; Caffeine	Diabetes; Asthma; COPD; Scleroderma; Rheumatoid arthritis; Esophageal motility disorders; Hiatus hernia	Anticholinergics; SSRI antidepressants; Inhaled bronchodilators; Oral contraceptives; Corticosteroids; Bisphosphonates	Age > 50; Family history; Sedentary lifestyle; Sleep disturbances
Gastritis	Excessive alcohol intake; Smoking; Extreme stress	Spicy foods; High salt intake; Caffeine; Contaminated food/ water (H. pylori)	Infections (bacterial, viral); Autoimmune gastritis; Chronic bile reflux; Pernicious anemia	NSAIDs (aspirin, ibuprofen) & other OTC pain/fever medicines	Major surgery; Traumatic injury or burns
Gastric ulcer	Smoking; heavy alcohol use; physiological stress (ICU, burns)	H. pylori-contaminated food/ water; high-salt diet	**H. pylori;** NSAID injury; stress-related mucosal disease; gastric malignancy (rare cause)	NSAIDs/aspirin; corticosteroids (with NSAIDs); anticoagulants/ antiplatelets	Age >60; prior ulcer/bleed; serious comorbidity
Duodenal ulcer	Smoking; irregular meals; night eating	H. pylori; high acid load; caffeine (symptom trigger)	**H. pylori** (strongest link); Zollinger-Ellison syndrome	NSAIDs/aspirin (less common than gastric)	Younger to middle age; O blood group (weak association); family history
Eosinophilic esophagitis (EoE)	Rapid eating; poor chewing	Food allergens (milk, wheat, egg, soy, nuts, seafood); aeroallergens/pollen	Atopic diseases (asthma, allergic rhinitis, atopic dermatitis); family history of EoE	PPI may help PPI-responsive EoE; avoid repeated dilation without therapy	Male sex; young age; fibrostenotic disease with delay in diagnosis
Gastroparesis	Poor glycemic control; large meals; low activity after meals	High-fat and high-fiber foods; carbonation; alcohol	Diabetes (type 1/2); post-viral; post-surgical vagal injury; connective-tissue disease; hypothyroidism; neurologic disorders	Opioids; GLP-1 receptor agonists; anticholinergics; tricyclics; dopamine agonists; calcium-channel blockers	Female sex; long diabetes duration; prior gastric surgery

EVALUATION FOR DIAGNOSIS

Suspected disorder	Symptom pattern (timing vs meals)	Exam focus	Distinguish from / pitfalls	First-line tests if no alarms
Peptic ulcer - duodenal	Epigastric gnawing/ burning; improves with food; recurs 2-3 h post-meal or at night	Epigastric tenderness	Biliary colic; ischemic heart disease; functional pain	*H. pylori* test (stool Ag or urea breath); CBC if chronic symptoms
Peptic ulcer - gastric	Epigastric pain worsens with food; early satiety, nausea	Weight, signs of anemia	Gastric cancer in older pts; NSAID gastritis	*H. pylori* test; CBC
Gastritis	Diffuse epigastric discomfort; nausea, anorexia, early satiety	Dehydration; epigastric tenderness	PUD; pancreatitis; MI (atypical)	*H. pylori* test; CBC; consider LFTs/lipase if severe pain
GERD (incl. NERD)	Heartburn, acid regurgitation; worse after large/fatty meals or lying flat; chest pain mimicking angina	Oropharyngeal/dental enamel changes; BMI; hiatal hernia risk	Cardiac ischemia; eosinophilic esophagitis; pill esophagitis	None needed initially; ECG if chest-pain risk; consider *H. pylori* only if dyspeptic features
Extra-esophageal reflux	Chronic cough, hoarseness, asthma-like symptoms, globus; often minimal heartburn	ENT/oral exam; lung exam	Post-nasal drip, asthma, vocal cord dysfunction	Consider empiric PPI if typical reflux coexists; reflux monitosring if persistent
Functional dyspepsia	Chronic post-prandial fullness, early satiety, epigastric pain/ burning without structural disease	Anxiety/stress screening; BMI	PUD, GERD, biliary disease, gastroparesis	Noninvasive *H. pylori* test; basic labs (CBC)
Gastroparesis	Early satiety, post-prandial fullness, nausea/vomiting of undigested food hours after eating	Volume status; signs of neuropathy (diabetes)	Gastric outlet obstruction; eating disorders	Exclude obstruction; gastric emptying study if available
Biliary-type pain (colic)	Severe episodic RUQ/ epigastric pain after fatty meals; may radiate to right shoulder	RUQ tenderness; Murphy sign	PUD; pancreatitis; ACS	LFTs; lipase; RUQ ultrasound

Alarm (red-flag) features - Urgent endoscopy and targeted work-up

- Unintentional weight loss ≥5% in 3-6 months
- Persistent vomiting (esp. with blood)
- Dysphagia or odynophagia
- Gastrointestinal bleeding (hematemesis/melena)

- Iron-deficiency (microcytic) anemia
- New-onset symptoms at age ≥55 years
- Palpable epigastric mass or supraclavicular lymphadenopathy
- Family history of upper GI malignancy

Note:

- GERD is dominated by heartburn and reflux symptoms, especially post-prandial and nocturnal. Extraesophageal reflux (EER) occurs when stomach contents travel above the upper esophageal sphincter and irritate structures in the throat, airway, or mouth. Patients often complain of chronic cough, hoarseness, throat clearing, a sensation of a lump in the throat (globus), or wheezing. Others may experience sour or bitter taste, burning in the throat, recurrent sore throat, or dental enamel erosion. Unlike classic GERD, heartburn may be absent; instead, clinicians should suspect EER when these atypical symptoms persist without another clear cause.
- PUD pain is more "ulcer-pattern," with duodenal ulcers relieved by eating and gastric ulcers aggravated by it.
- Gastritis often presents with vague burning and nausea, sometimes asymptomatic until bleeding occurs.
- Functional dyspepsia shows non-specific epigastric discomfort, post-prandial fullness, and early satiety without structural disease.
- Gastroparesis is characterized by delayed gastric emptying leading to early satiety, nausea/vomiting of undigested food, and marked bloating, often without obstruction.

CONFIRMATION OF DIAGNOSIS & SEVERITY ASSESSMENT

- Endoscopy: gold standard for visualization, biopsy (to exclude malignancy), and for guiding therapy if alarm signs are present or symptoms persist despite empiric therapy.
- *H. pylori* testing: non-invasive urea breath test or stool antigen recommended before initiating eradication therapy; if endoscopy is performed, rapid urease test or histology can be used.
- Laboratory tests: full blood count (to detect anemia), stool occult blood (if bleeding suspected).
- *Barium swallow* in case when EGD is contraindicated.
- Complete blood work, liver function, and levels of amylase and lipase.
- Serum gastrin is ordered if Zollinger-Ellison syndrome is suspected.

Functional dyspepsia- Rome IV diagnostic criteria

All must be met

- One or more: bothersome postprandial fullness, early satiation, epigastric pain, or epigastric burning.
- Symptom duration: present for the last 3 months with onset ≥6 months before diagnosis.
- No structural disease to explain symptoms (normal evaluation/endoscopy when indicated).

Subtypes and frequency thresholds

Subtype	Required symptoms	Frequency
Postprandial Distress Syndrome (PDS)	Bothersome postprandial fullness and/or early satiation	≥3 days/week
Epigastric Pain Syndrome (EPS)	Epigastric pain and/or epigastric burning (not limited to post-meal)	≥1 day/week

Supportive features (not required): upper abdominal bloating, nausea, excessive belching; overlap with GERD or IBS may occur.

Classification of GERD based on severity

Stage	Severity	Key Clinical Features
1	**Mild**	Occasional (<1×/week) heartburn and regurgitation
2	**Moderate**	Heartburn and regurgitation several times per week
3	**Severe**	Frequent heartburn & regurgitation, dysphagia, cough, voice changes, non-cardiac chest pain, sleep disturbance, nocturnal symptoms
4	**Reflux-induced precancerous lesions or esophageal cancer**	All Stage 3 features **plus** endoscopic evidence of strictures, Barrett's esophagus, or frank esophageal carcinoma

Note: *Based on endoscopic and histopathological appearance, GERD phenotypes include*: Non-erosive reflux disease (NERD), Erosive esophagitis (EE), Barrett's esophagus (BE)

DIFFERENTIAL DIAGNOSIS

Condition	How it differs from APD	Key clues on history/exam	First tests to order	Initial management	Refer/urgent when
Biliary colic / cholecystitis	Right upper quadrant pain, not epigastric burnings	Post-fatty meals; Murphy sign; fever in cholecystitis	LFTs, ultrasound hepatobiliary	Analgesia; antibiotics if cholecystitis	Persistent pain, fever, jaundice, sepsis
Pancreatitis	Inflammation, not mucosal acid injury	Severe epigastric pain radiating to back; worse supine; nausea/ vomiting	Serum lipase/ amylase; ultrasound ± CT	IV fluids, analgesia, NPO	Immediate if suspected
Acute coronary syndrome (inferior MI)	Cardiac ischemia mimicking "gastric" pain	Exertional pain, diaphoresis, dyspnea, risk factors	ECG, troponin	Antiplatelet/ACS protocol	Emergency if suspicion
Mesenteric ischemia	Ischemic gut pain	Disproportionate pain vs exam; AF, vascular disease	Lactate, CT angiography	Resuscitate, urgent vascular/ surgical	Emergency
Gastric malignancy	Ulcer-like pain but non-healing and systemic signs	Weight loss, anemia, early satiety, age >55	Upper GI endoscopy with biopsies; CT for staging	Oncologic pathway; nutrition, PPI for symptoms	Urgent if suspected
Eosinophilic esophagitis	Immune-mediated esophageal disease	Solid-food dysphagia, atopy; young adults	Endoscopy with biopsies (eosinophils)	PPI, topical steroids, diet therapy	Food impaction or refractory dysphagia
Gastritis (autoimmune/ atrophic)	Hypochlorhydria, not acid excess	B12 deficiency, thyroid disease	B12, antibodies; endoscopy with biopsies	H. pylori eradication if present; B12	Dysplasia/ intestinal metaplasia on biopsy

Gastroparesis	Delayed emptying, not acid injury	Early satiety, post-prandial fullness, vomiting; diabetes	Gastric emptying study after structural exclusion	Gluten-free diet; nutrition	Weight loss/ dehydration/ refractory vomiting
Peptic ulcer complications (bleed, perforation, GOO)	Consequences rather than simple APD	Hematemesis/ melena, peritonitis, persistent vomiting	CBC, CMP; urgent endoscopy; upright CXR/CT	Resuscitate, IV PPI; endoscopic/ surgical care	Emergency
Celiac disease	Malabsorption presenting as dyspepsia	Bloating, diarrhea, weight loss, anemia	tTG-IgA, total IgA; duodenal biopsy	Gluten-free diet; nutrition	Refractory disease or severe malnutrition
IBS-D/M	Pain linked to stool form/ frequency	Relief after defecation; alternating habits	Rome IV; limited labs to exclude red flags	Diet (low FODMAP), antispasmodics	Alarms (bleeding, weight loss, anemia)
Helicobacter pylori -negative, NSAID-negative ulcer	Consider hypersecretory states	Multiple/ refractory ulcers, diarrhea	Gastrin with low gastric pH; imaging for gastrinoma	High-dose acid suppression; specialist work-up	Sp

Approach to the evaluation and management of dyspepsia in adults

STEP-WISE INITIAL EVALUATION OF DYSPEPSIA IN PATIENTS WITHOUT ALARM FEATURES

Confirm Absence of Alarm Signs

- Ensure no weight loss, persistent vomiting, gastrointestinal bleeding, anemia, dysphagia, or age ≥ 55 with new symptoms.

1

Comprehensive History

- **Symptom Characterization:** Timing (relation to meals, nocturnal vs. daytime), location (epigastric vs. substernal), quality, duration, and radiation.
- **Associated Symptoms:** Heartburn or regurgitation (suggests GERD), nausea/vomiting (consider gastroparesis), post-prandial fullness (functional dyspepsia), or biliary-type pain.
- **Medication & Risk Factor Review:** NSAIDs/aspirin use, smoking, alcohol, prior ulcer history, and comorbidities (eg, diabetes).
- **Lifestyle & Dietary Habits:** Meal size/frequency, trigger foods, timing of eating relative to bedtime.

2

Focused Physical Examination

- **Abdominal Exam:** Inspect for distension or cachexia; auscultate for succussion splash; palpate for epigastric or RUQ tenderness; perform Carnett's test if abdominal wall pain suspected.
- **General & Systems Exam:** Check vital signs for orthostasis; look for pallor, jaundice, spider angiomata, lymphadenopathy (e.g., Virchow's node), and signs of chronic disease.

3

Non-invasive Testing ("Test-and-Treat")

- **H. pylori Screening:** Urea breath test or stool antigen in regions with ≥10% prevalence.
- **Laboratory Workup:** CBC (anemia), LFTs if risk factors for liver disease.

4

Empiric Acid- Suppressive Trial

- **PPI Therapy:** Prescribe omeprazole 20-40 mg once daily (30-60 minutes before breakfast) for 4-8 weeks.
- **Lifestyle Advice:** Small, frequent meal; avoid trigger foods; don't lie down for 2-3 hours after eating; elevate head of bed.

5

Reassessment at 4-8 Weeks

- **Symptom Resolution:** If improved, consider stepping down to on-demand PPI or H_2RA.
- **Persistent Symptoms:** If no relief, proceed to endoscopy or further testing (pH-impedance imaging) despite initial low risk.

6

MANAGEMENT GOALS

Clear goals align clinician and patient expectations, enable objective assessment of treatment success, and support shared decision-making.

1. Rapid symptom relief
2. Healing of mucosal erosions/ulcers
3. Eradication of *H. pylori* where present
4. Prevention of recurrence and complications

MANAGEMENT PRINCIPLES

- Lifestyle and diet: Identify and avoid personal triggers (spicy, fatty, caffeinated, alcohol), stop smoking, maintain healthy weight, avoid late-night meals.
- Acid suppression: Use proton-pump inhibitors first line; H2 receptor antagonists if PPI not tolerated; antacids for short-term relief.
- H. pylori: Test and treat with guideline-recommended regimens when infection is confirmed.
- Address causes: For NSAID-related disease, stop or switch the culprit drug and add gastroprotection.
- Follow-up: Review symptoms, adherence, and adverse effects; step down, stop, or adjust therapy based on response.
- Refer early if red flags or refractory: Bleeding, weight loss, dysphagia, persistent vomiting, anemia, suspected perforation or malignancy.

NON-PHARMACOLOGICAL INTERVENTIONS

Non-pharmacological measures form the foundation of long-term acid-peptic disease management by targeting modifiable risk factors and helping sustain remission.

Intervention	How to do it	Notes
Stop smoking; cut alcohol	Smoking cessation program; limit alcohol to rare/ none	Avoid binge drinking; healing is faster when both are addressed
Avoid or minimize NSAIDs/aspirin	Use alternatives (acetaminophen/paracetamol if suitable); if essential, co-prescribe a PPI	Reassess need regularly; highest risk with prior ulcer/bleed
Meal pattern and triggers	Smaller, more frequent meals; avoid personal triggers; no food within 2-3 hours of bedtime; Large/spicy/fatty/acidic foods and late meals worsen symptoms	Keep a symptom-food diary to individualize

Weight management	Aim for gradual weight loss; increase physical activity as obesity raises intra-abdominal pressure and reflux	Even 5-10% weight loss can reduce GERD symptoms
Head-of-bed elevation	Raise head of bed 15-20 cm (6-8 in) with blocks or wedge to reduce nocturnal reflux events	Extra pillows don't work; use a wedge or bed risers
Stress reduction	Relaxation, mindfulness, yoga; regular sleep as stress can increase acid and pain perception	Pair with other measures; set a daily 10-15 min routine

PHARMACOLOGICAL THERAPY

1. Rule out danger

- If alarm features present → urgent endoscopy and targeted work-up (don't start empiric therapy alone).

2. Baseline intervention for everyone

- Stop or minimize NSAIDs/aspirin; add PPI prophylaxis if any analgesic must continue.
- Lifestyle bundle: trigger control, weight loss if overweight, no late meals, head-of-bed elevation, stop smoking, cut alcohol.

3. Empiric acid suppression (no alarms)

- PPI once daily for 4-8 weeks (e.g., omeprazole 20-40 mg OD Or esomeprazole 20-40 mg, Or pantoprazole 40 mg, Or rabeprazole 20 mg, Or lansoprazole 30 mg) for rapid symptom relief and mucosal healing. (for details see table below).
- If PPI contraindicated/unavailable, H2 Receptor Antagonists (H2RA e.g., ranitidine 150 mg BID or 300 mg HS) for 4-8 weeks; recognize lower potency and possible tachyphylaxis.

4. Test for *H. pylori* (who to test)

- All dyspeptic patients <55 years without alarms, and anyone with endoscopically confirmed peptic ulcer.
- Use stool antigen or urea breath test (hold PPI 2 weeks beforehand if feasible).
- If positive, treat (see below). If negative, continue Step 3 and reassess.

5. *H. pylori* eradication (first-line and proofs)

- Treat for 14 days (local resistance patterns guide choice).
- Confirm cure ≥4 weeks after antibiotics and ≥2 weeks off PPI with stool antigen or breath test.

6. NSAID-induced ulcer

- Stop NSAID if possible. If it must continue: COX-2 selective + daily PPI and treat 4-8 weeks for healing.
- High-risk patients (prior ulcer/bleed, age >65, anticoagulant/antiplatelet use) need ongoing PPI prophylaxis while NSAID persists.

7. Stress-ulcer prophylaxis (ICU only)

- If mechanical ventilation or coagulopathy/major risk → IV PPI or H2RA during the risk period only; discontinue when risk resolves.

8. Reassessment at 4-8 weeks

- If symptoms resolved: step down (see Step 8).
- If persistent/recurrent: confirm adherence, review triggers/meds, ensure *H. pylori* status, consider endoscopy.

9. Step down / long-term control

- Aim to minimize PPI exposure: use lowest effective dose, on-demand PPI, or switch to H2RA/antacids for intermittent symptoms.
- Keep indefinite low-dose PPI only in high-risk (complicated ulcer history, chronic NSAID/antiplatelet need, severe erosive esophagitis, Barrett's with symptoms).

10. Refractory or recurrent disease (after adherence and *H. pylori* cure confirmed)

- Consider bismuth quadruple if standard triple failed; or levofloxacin-based triple, rifabutin-based triple, high-dose PPI + amoxicillin dual, or vonoprazan-based regimens where available.
- Re-scope to exclude missed pathology (malignancy, ZE syndrome, bile reflux, EoE).

11. Special case: Zollinger-Ellison (hypersecretory)

- High-dose PPI (e.g., omeprazole 60-120 mg/day in divided doses).
- Specialist-led localization and surgical/oncologic management of gastrinoma.

12. Adjuncts (useful but not curative alone)

- Antacids/alginates: rapid, short-lived relief for breakthrough symptoms.
- Sucralfate: 1 g QID for 4-8 weeks on an empty stomach (1 h before meals and HS); separate other medicines by ≥2 h; constipation common; caution in severe renal impairment.
- Prokinetics: reserve for documented delayed emptying (e.g., gastroparesis).

Extraesophageal reflux (EER)

1. Ask every GERD patient about extraesophageal symptoms including chronic cough, hoarseness, asthma, and dental erosions to assess whether reflux may contribute.
2. Manage EER with a multidisciplinary team, incorporating otolaryngology, pulmonology, dentistry, and speech pathology input. Consider non-GI diagnostic results (bronchoscopy, laryngoscopy, thoracic imaging) when evaluating reflux as an EER cause.
3. No single test definitively links GER to EER. Diagnosis should rest on clinical judgment, symptom patterns, response to reflux therapy, endoscopy findings, and reflux monitoring.
4. In patients with suspected EER but without typical heartburn, consider objective reflux testing (pH or pH-impedance monitoring) before empiric PPI therapy. If typical GERD symptoms are present, a single-dose PPI trial (up to twice daily) is reasonable.
5. Symptom improvement on a PPI trial does not confirm acid reflux as the sole mechanism; non-acidic reflux and placebo effects also play roles.
6. For suspected EER patients who do not improve after a 12-week PPI trial, proceed to objective reflux testing rather than switching acid-suppressive agents.
7. Tailor initial testing to the clinical scenario. Options include upper endoscopy and ambulatory reflux monitoring off therapy for those without prior testing, or on therapy for refractory cases to evaluate breakthrough reflux.
8. In GERD patients with confirmed abnormal reflux but persistent EER symptoms on high-dose PPIs, use pH-impedance monitoring on therapy to differentiate ongoing acid versus non-acid reflux and adjust management accordingly.
9. Offer adjunctive therapies - lifestyle modification (weight loss, head-of-bed elevation, dietary changes), alginate-containing antacids, external esophageal sphincter devices, neuromodulators, or cognitive-behavioral therapy to relieve EER pones predicts poor surgical results and should inform shared decision-making.
   symptoms not fully responsive to PPIs.

   Before anti-reflux surgery for EER, confirm objectively documented reflux and discuss expected outcomes. Lack of PPI response predicts poor surgical results and should inform shared decision-making.

Functional dyspepsia

- Management focuses on symptom relief and may include dietary modifications (small, low-fat meals; avoidance of trigger foods), reassurance, and a trial of acid suppression (PPI or H2receptor-antagonist for 4-8 weeks).
- In patients with predominant post-prandial distress, a prokinetic agent (e.g., metoclopramide or domperidone) can be added. (For details see Table below)

- Psychological therapies (e.g., low-dose tricyclic antidepressants, gut-directed hypnotherapy) have shown benefit in refractory cases by targeting visceral hypersensitivity and brain-gut dysregulation.
- Regular follow-up ensures that alarm features have not emerged, and treatment is stepped up or down based on response.

H. pylori Eradication: Key Considerations

Step 0. Confirm indication

- Positive noninvasive test (urea breath test or stool antigen) or endoscopic diagnosis with testing.
- Treat dyspepsia/ulcer/bleed, MALT lymphoma, early gastric cancer post-resection, or strong family history/risk.

Step 1. Prepare the patient

- Explain the need for near100% adherence.
- PPI timing: 30-60 minutes before breakfast (and dinner if BID).
- Review drug allergies, prior macrolide/quinolone exposure, pregnancy, and drug interactions (warfarin, theophylline, methotrexate, tacrolimus).
- Pause antisecretory meds for testing/cure checks when scheduled (see Step 6).

Step 2. Choose first-line regimen by local resistance and allergy

- Use 14 days in all cases.
- Prefer bismuth quadruple where clarithromycin resistance is ≥15-20% or unknown.
- Use clarithromycin triple only where local clarithromycin resistance is low and there's no macrolide exposure.

Recommended regimens (adults)

Table . Helicobacter pylori Eradication Regimens

Regimen	Components	Duration	Expected Eradication Rate	Remarks
Clarithromycin Triple Therapy	PPI (e.g. omeprazole 20 mg) BD Clarithromycin 500 mg BD Amoxicillin 1 g 12 hourly (or metronidazole 400 mg 8 hourly if penicillin-allergic)	14 days	70-80 %	Only if local clarithromycin resistance is low and no prior macrolide exposure; otherwise avoid due to failures.

Bismuth Quadruple Therapy (BQT)	PPI 12 hourly Bismuth subsalicylate 300 mg QID Tetracycline 500 mg QID Metronidazole 500 mg 8 hourly	10-14 days	80-90 %; 14-day BQT slightly better eradication rates and compliance.	Preferred where clarithromycin resistance is common, or with penicillin allergy. Avoid alcohol with metronidazole. Ensure tetracycline availability; do not substitute doxycycline unless guideline-directed.
Concomitant (Non-bismuth) Quad	PPI 12 hourly Amoxicillin 1 g 12 hourly Clarithromycin 500 mg 12 hourly Metronidazole 500 mg 12 hourly	10-14 days	85-90 %; 14-day course is preferred due to superior eradication rates compared to 10-day regimens	First-line option if macrolide resistance is not very high and no prior macrolide use.
Symptom diary	Days 1-5: PPI twice daily + Amoxicillin 1 g 12 hourly Days 6-10: PPI BD + Clarithromycin 500 mg 12 hourly + Metronidazole 500 mg 12 hourly	10 days	80-88 %	It was initially superior to 7-day triple therapy, but its efficacy has plateaued in recent years due to rising resistance
Hybrid Therapy	Days 1-7: PPI BD + Amoxicillin 1 g 12 hourly Days 8-14: PPI BD + Amoxicillin 1 g 12 hourly + Clarithromycin 500 mg BD + Metronidazole 500 mg 12 hourly	14 days	88-95 %	Combines the strengths of sequential and concomitant approaches, showing excellent eradication rates and better tolerability than concomitant therapy

PPI "standard doses (BID)": omeprazole 20 mg, esomeprazole 20 mg, pantoprazole 40 mg, rabeprazole 20 mg, lansoprazole 30 mg. In rapid CYP2C19 metabolizers or severe disease, consider higher PPI intensity.

Step 3. Support adherence and acid control

- **Use simple AM/PM schedules;** provide a one-page checklist.
- **Manage expected effects:** metallic taste, nausea, dark stools (bismuth), photosensitivity (tetracycline).
- **Counsel:** no alcohol with metronidazole; separate tetracycline from dairy/iron by ≥2 hours; sun protection with tetracycline.

Step 4. Special scenarios

- **Active/complicated ulcer or bleed:** treat H. pylori as above and continue PPI beyond eradication course (e.g., 8 weeks for gastric ulcer).
- **NSAID users:** stop NSAID if possible; otherwise continue daily PPI prophylaxis during ongoing NSAID/antiplatelet therapy.
- **Pregnancy:** defer eradication if possible; avoid tetracycline, bismuth subsalicylate, and quinolones.

Step 5. If first-line fails (salvage)

- Verify true failure (see Step 6), then switch drug classes; do not repeat the same macrolide/quinolone.
- Common options (14 days):
  - **Bismuth quadruple** (if not used first).
  - **Levofloxacin triple:** PPI BID + levofloxacin 500 mg OD + amoxicillin 1 g BID (avoid where fluoroquinolone resistance is high; monitor for tendinopathy/QT).
  - **High-dose dual:** PPI at high frequency (e.g., esomeprazole 40 mg QID) + amoxicillin 750 mg-1 g QID.
  - **Rifabutin triple:** PPI BID + amoxicillin 1 g BID + rifabutin 150 mg BID (watch for myelotoxicity; check CBC).
  - **Vonoprazan-based rescue** where available. (Vonoprazan 20 mg BID + amoxicillin 1000 mg TID Or Vonoprazan 20 mg BID + amoxicillin 1000 mg BID + clarithromycin 500 mg BID for 14 days)
- Where endoscopy is available, culture/AST-guided therapy is preferred after failure.

Step 6. Test of cure (mandatory)

- **Timing:** ≥4 weeks after finishing antibiotics and ≥2 weeks after stopping PPI/ bismuth.
- **Tests:** Urea breath test or monoclonal stool antigen.
- If positive - treat with an effective, class-switched salvage regimen.

Step 7. After eradication

- Step down/stop PPI if no separate indication (e.g., severe esophagitis, chronic NSAID).
- Educate on NSAID avoidance, smoking cessation, and alarm symptoms.
- Recheck for recurrence only if symptoms return or there is ongoing risk.

Safety Notes:

- **Tetracycline:** avoid in pregnancy/children <8; photosensitivity.
- **Metronidazole/tinidazole:** avoid alcohol during and 48-72 h after.
- **Levofloxacin:** tendon rupture, neuropathy, QT prolongation-avoid with known risks.
- **Rifabutin:** neutropenia/uveitis; drug interactions (CYP).
- **Bismuth subsalicylate:** salicylate caution (anticoagulants, kids with viral illness).

ASSESSMENT OF RESPONSE

Objective monitoring prevents overtreatment and identifies non-responders early.

Component	Rationale	When	Key details / criteria
Symptom diary	Record patient-reported pain scores	Daily for 2-4 weeks	Use a simple 0-10 scale; note triggers, meals, and nocturnal pain
Endoscopic reassessment (general)	Repeat endoscopy only if needed	If symptoms persist or alarm features develop	Alarm features: GI bleeding, anemia, weight loss, progressive dysphagia, persistent vomiting, palpable mass, jaundice
Duodenal ulcer (DU) - repeat endoscopy	Not routinely required after treatment	Only if symptoms persist at 4 weeks or recur	Otherwise no surveillance endoscopy needed
Gastric ulcer (GU) - surveillance endoscopy	Perform surveillance endoscopy with biopsies if ulcer persists	After 8-12 weeks of antisecretory therapy	Do if any ONE of: symptoms despite therapy; unclear etiology; giant GU (>2 cm); biopsies not done or inadequate at index endoscopy; suspicious features at index endoscopy (mass, elevated irregular borders, abnormal adjacent folds); initial bleeding ulcer with signs of continued bleeding; risk factors for gastric cancer
H. pylori test-of-cure	Confirm eradication	≥4 weeks after completing therapy	Use urea breath test or stool antigen test; ensure no antibiotics for 4 weeks and no PPI for 2 weeks before testing if possible
Reinfection / recurrence prevention	Address modifiable risks and counsel	Ongoing	Avoid NSAIDs or co-prescribe PPI if NSAIDs are essential; hygiene counseling; in high-prevalence settings, consider testing/treating close contacts

Review, follow-up and adjustment of treatment

Component	Rationale	When	Key details / criteria
4-8 week review	Confirm symptom resolution; consider stepping down proton-pump inhibitor (PPI) to the lowest effective dose	After 4-8 weeks of full-dose PPI and healing	If stable, reduce daily dose (e.g., 40 → 20 mg) or trial on-demand use; arrange follow-up to reassess control
Maintenance therapy	Continue long-term acid suppression only for high-risk patients	Ongoing, after initial control	High-risk: chronic nonsteroidal anti-inflammatory drug (NSAID) use, prior complicated ulcer (bleeding, perforation, obstruction)

Rationale for reviews	De-escalate safely and limit chronic PPI exposure; detect complications and need for specialist input	At each step change	Use reviews to check healing, adverse effects, and ongoing indications
Before step-up / step-down: Adherence	Verify correct use (timing before meals, dose, duration); check pill counts / pharmacy refills	Before changing therapy	Non-adherence can mimic treatment failure; fix this first
Before step-up / step-down: Lifestyle / co-meds	Reassess NSAID/aspirin use, smoking, alcohol	Before changing therapy	Minimize or stop ulcerogenic agents; add gastroprotection if NSAIDs essential
Before step-up / step-down: Refractory causes	Exclude uncommon etiologies	If inadequate response or atypical features	Consider Zollinger-Ellison syndrome, malignancy; investigate per clinical judgment
PPI long-term risks (monitoring plan)	Use the lowest effective dose; monitor for adverse effects	If >8-12 we	

PROGNOSIS & PROGRESSION

- Most patients do well. Uncomplicated acid-peptic disease heals with short courses of acid suppression and appropriate *H. pylori* therapy.
- *H. pylori* eradication changes the trajectory: it prevents ulcer relapse and markedly reduces bleeding and need for long-term medication.
- Recurrence is mainly driven by ongoing NSAID/aspirin use, smoking, poor adherence, or persistent *H. pylori*. Removing these risks improves long-term control.
- Duodenal ulcers usually heal faster than gastric ulcers; gastric ulcers need follow-up to document healing and to exclude malignancy.
- After an upper-GI bleed or perforation, early mortality and rebleeding risk depend on age, comorbidities, shock, and endoscopic stigmata; timely endoscopy and PPI therapy improve outcomes.
- Reinfection with *H. pylori* is uncommon in low-prevalence settings but can occur; test-of-cure and, where appropriate, household testing reduce relapse.
- Many patients can step down to the lowest effective dose or on-demand therapy once healed, limiting long-term PPI adverse effects.
- Special situations (Zollinger-Ellison syndrome, giant/complex ulcers, anticoagulation) warrant specialist care; prognosis then hinges on underlying disease control.
- With education, adherence, and periodic review, quality of life is good and serious complications are uncommon.

COMPLICATIONS

Complication	Pathophysiology)	Key clinical clues	How to confirm	Initial management	Urgency / referral	Sequelae / prevention
Upper GI bleeding (hematemesis /melena)	Ulcer erodes into vessel (e.g., gastroduodenal artery in posterior DU)	Hematemesis, melena, syncope, tachycardia, hypotension	CBC, BUN/Cr; urgent upper GI endoscopy for stigmata of bleed	Resuscitate (IV access, fluids, blood as needed), IV PPI bolus + infusion, endoscopic hemostasis (clips/thermal/ injection)	Emergency; admit. GI endoscopy urgently; surgery/ interventional radiology if refractory	Rebleed, anemia. Prevent with H. pylori eradication, avoid NSAIDs/ anticoagulants when possible, PPI co-therapy if needed
Perforation with peritonitis	Full-thickness ulcer, often anterior DU	Sudden severe epigastric pain → generalized rigidity, shoulder tip pain, sepsis	Upright CXR/ AXR: free air; CT abdomen if uncertain	Resuscitate, IV antibiotics, IV PPI, NPO; surgical consult	Emergency surgical evaluation	Intra-abdominal infection, abscess. Prevent by early diagnosis/ treatment of ulcers; avoid NSAIDs, smoking
Penetration (into pancreas/ liver)	Ulcer burrows into adjacent organ without free perforation	Persistent, severe, often posterior pain radiating to back; refractory to PPIs	CT abdomen; endoscopy may show deep ulcer	IV PPI, analgesia; manage complications of involved organ; GI/ surgical input	Urgent specialist referral	Chronic pain, pancreatitis. Prevent as above
Gastric outlet obstruction	Edema and spasm acutely; chronic scarring/ stricture in pylorus/ duodenal bulb	Early satiety, post-prandial vomiting of undigested food, weight loss, metabolic alkalosis	NG aspirate >200 mL; hypochloremic alkalosis; endoscopy after decompressi on; CT to exclude malignancy	NG decompressi on, IV fluids/ electrolytes, IV PPI; endoscopic balloon dilatation if benign	Urgent GI referral; surgery if refractory or malignant	Recurrent obstruction, malnutrition. Prevent with H. pylori eradication, stop NSAIDs
Recurrent/ refractory ulcer	Persistent H. pylori, ongoing NSAIDs, hypersecretion (e.g., Zollinger-Ellison), smoking, poor adherence	Symptoms despite adequate therapy; non-healing on follow-up	Test-of-cure for H. pylori; medication review; fasting gastrin with low gastric pH if suspected ZES; repeat endoscopy	Optimize PPI timing/dose or switch (consider PCAB where appropriate), eradicate H. pylori, stop NSAIDs, address smoking	Refer to GI if non-healing at 8-12 weeks (GU) or persistent DU symptoms at 4 weeks/ recurrence	Chronic pain, complications above. Prevention as listed

Iron-deficiency anemia	Chronic microscopic blood loss	Fatigue, pallor; microcytic indices	CBC, iron studies; endoscopy to source	Treat source ulcer; iron repletion	Routine-urgent depending on Hb	Functional decline. Prevent viaeradication and NSAID avoidance
Gastric ulcer harboring malignancy	Some GU are cancer at presentation; H. pylori and chronic gastritis predispose	Weight loss, anemia, early satiety; non-healing or same-site recurrent GU; rolled/irregular edges	Endoscopy with targeted biopsies and repeat biopsies if non-healing; EUS/CT for staging	Oncologic pathway after confirmation; nutrition, PPI for symptoms	Urgent GI/ oncology referral	Cancer progression. Prevention: timely biopsy and surveillance per guideline
Stricture/ stenosis (benign)	Fibrosis from chronic ulceration	Progressive vomiting, dehydration, electrolyte imbalance	Endoscopy ± contrast study	Endoscopic balloon dilatation; PPI; treat H. pylori	GI referral; surgery if refractory	Recurrence. Prevent by early ulcer control
Fistula (rare)	Penetrating ulcer creates abnormal tract (e.g., gastrocolic)	Intractable diarrhea, foul belching, weight loss	CT with contrast; endoscopy	Stabilize, nutrition support, definitive surgical repair	Specialist referral	Malnutrition. Prevent via early management
Post-ulcer functional dyspepsia/ quality-of-life impact	Visceral hypersensitivity					

REFERRAL FOR SPECIALIST CONSULTATION

While the majority of patients with acid-peptic disease respond to standard medical therapy, early referral to specialist care - gastroenterology, surgical, or endocrinology should be considered in specific scenarios.

Immediate hospital referral (red flags)

- Hematemesis, melena, or syncope
- Hemodynamic instability or orthostasis
- Suspected perforation or peritonitis
- Gastric outlet obstruction with intractable vomiting
- Severe anemia or rapid hemoglobin drop
- Unable to tolerate oral intake or high aspiration risk

Early gastroenterology referral

- Alarm features: unintentional weight loss, progressive dysphagia, persistent vomiting, iron-deficiency anemia, palpable mass, jaundice
- Non-healing ulcer: gastric ulcer not healed after 8-12 weeks of antisecretory therapy, duodenal ulcer with persistent symptoms after 4 weeks or recurrence
- Complex or "giant" ulcer: size >2 cm, irregular or undermined margins, penetrating ulcers
- Recurrent or refractory symptoms despite optimized PPI timing and confirmed adherence
- Positive *H. pylori* with treatment failure or positive test-of-cure after appropriate regimen
- Need for advanced diagnostics: pH-impedance, gastric emptying studies, endoscopic ultrasound, targeted re-biopsy

Surgical referral (with GI involvement)

- Recurrent bleeding requiring repeated endoscopic therapy or transfusions
- Free perforation or contained perforation not amenable to endoscopic management
- Fixed gastric outlet obstruction not relieved endoscopically
- Suspicion of malignancy requiring oncologic resection

Endocrinology/oncology referral

- Suspected or confirmed Zollinger-Ellison syndrome: elevated fasting gastrin with low gastric pH, need for tumor localization and systemic management
- Proven or strongly suspected gastric malignancy for staging and multidisciplinary care

High-risk or special populations

- Ongoing NSAID, antiplatelet, or anticoagulant use with prior ulcer or bleed
- Significant comorbidity increasing complication risk: cirrhosis, chronic kidney disease, severe cardiopulmonary disease
- Pregnancy with refractory or complicated disease
- Pediatric patients with alarm features or suspected complications
- Post-surgery recurrence after prior ulcer surgery

Before referral (optimize first when safe)

- Confirm adherence and correct PPI timing: 30-60 minutes before meals
- Stop NSAIDs or add gastroprotection if essential
- Test-and-treat H. pylori and plan test-of-cure at ≥4 weeks post-therapy
- Review alcohol, tobacco, and other ulcerogenic drugs

What to include in the referral

- Symptom timeline and red flags, prior endoscopy and pathology reports
- *H. pylori* testing and treatment details, test-of-cure result. Refer non-responders.
- Current and prior acid-suppression regimens with dosing and adherence checks
- CBC, iron studies, renal and liver function if available
- Antithrombotic and NSAID exposure, relevant comorbidities
- Documentation quality drives care: always attach endoscopy images/reports, biopsy details, exact drug doses/timing, and hemodynamic trends.

HEALTH PROMOTION & PREVENTION

Goals are to reduce risk factors, eradicate H. pylori, prevent complications and relapse.

Primary prevention (reduce first-ever disease)

- Limit NSAIDs/aspirin; use the lowest dose, shortest duration; avoid dual NSAID use.
- If analgesia needed: try acetaminophen/paracetamol first; add PPI only when NSAIDs are essential.

- Stop smoking; limit alcohol.
- Meal habits: smaller meals, avoid late dinners; don't lie down for 2-3 hours after eating; reduce known triggers.
- Weight reduction for reflux-predominant symptoms; elevate head of bed for nocturnal reflux.
- Hygiene and safe water in high-prevalence areas to curb H. pylori transmission.

Secondary prevention (early detection and recurrence control)

- Test-and-treat H. pylori when indicated; confirm test-of-cure ≥4 weeks post-therapy.
- Educate on correct PPI timing (30-60 min before meals) and full course adherence.
- Review and deprescribe unnecessary long-term PPIs; step down to the lowest effective dose or on-demand use once controlled.
- Avoid concurrent ulcerogenic drugs (high-dose steroids, SSRIs + NSAID, anticoagulants) where possible; add gastroprotection when combinations are unavoidable.

Tertiary prevention (prevent complications)

- Early endoscopy for alarm features: bleeding, anemia, weight loss, persistent vomiting, dysphagia.
- In chronic NSAID/antithrombotic users with prior ulcer/bleed: continuous PPI co-therapy and regular review.
- Vaccinate per general schedules and manage comorbidities (CKD/CLD) to lower bleed risk (indirect support).

PATIENT EDUCATION

Explain to patients in plain language

- Symptoms come from stomach acid injuring the stomach/duodenum or causing reflux into the lower esophagus.
- Medicines and habits work together: drugs heal; lifestyle prevents flares and relapse.
- After healing, many can step down to the lowest effective dose under medical advice.

Do's

- Take medicines exactly as prescribed. For proton-pump inhibitors (PPIs), take 30-60 minutes before breakfast (and before dinner if twice daily).
- Complete the full H. pylori regimen if prescribed; return for test-of-cure as advised.
- Keep a simple diary: pain time, meals, meds, and possible triggers.
- Eat smaller, more frequent meals; finish dinner 2-3 hours before lying down.
- Elevate the head of the bed 6-8 inches to reduce night symptoms.
- Choose non-irritating foods; identify and avoid your personal triggers.
- Stop smoking; limit alcohol.
- Use safer pain options if possible; if you must take NSAIDs/aspirin, discuss protection (e.g., PPI) with your clinician.
- Keep follow-up appointments; ask about stepping down to on-demand or lower-dose therapy once controlled.
- Caregivers: help with medication timing, meal planning, and spotting warning signs.

Don'ts

- Don't skip doses or stop early because you "feel better."
- Don't lie down soon after eating or eat late-night heavy meals.
- Don't rely on frequent NSAIDs without medical advice.
- Don't smoke "just a little" even small amounts can worsen symptoms.
- Don't ignore persistent or worsening symptoms while on treatment.
- Don't self-start long-term PPIs without a plan to review and step down.

Red flags - seek care now

- Vomiting blood, black or maroon stools.
- Severe or sudden abdominal pain, fainting, or signs of shock.
- New or progressive trouble swallowing, persistent vomiting.
- Unintentional weight loss or unexplained anemia.

Follow-up plan (agree with your clinician)

- Symptom check at 4-8 weeks; confirm control and discuss dose reduction.
- Test-of-cure for H. pylori when due.
- Periodic review of long-term PPI need and side-effect risks.

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