



Review Article

An Integrated Approach to Peptic Ulcer Disease: Diet, Lifestyle Modifications and Pharmacotherapy

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Peptic ulcer disease (PUD) remains a significant global health problem despite considerable advances in diagnosis and treatment. It is characterized by mucosal ulceration of the stomach or duodenum resulting from an imbalance between aggressive factors, such as gastric acid, pepsin, *Helicobacter pylori* infection, and nonsteroidal anti-inflammatory drugs (NSAIDs), and protective mechanisms of the gastroduodenal mucosa. Although the prevalence of *H. pylori*-associated ulcers has declined in several regions due to improved sanitation and eradication therapies, NSAID-induced ulcers and ulcer-related complications continue to contribute substantially to morbidity and healthcare costs. Traditional management of PUD has focused predominantly on pharmacological interventions, including proton pump inhibitors (PPIs), histamine-2 receptor antagonists (H₂RAs), mucosal protective agents, and *H. pylori* eradication regimens. However, increasing evidence indicates that dietary habits, lifestyle factors, and patient education play a crucial role in ulcer prevention, symptom control, mucosal healing, and reduction of recurrence. Dietary modifications such as regular meal patterns, avoidance of irritant foods, adequate intake of fruits, vegetables, antioxidants, and probiotics, along with lifestyle measures including smoking cessation, moderation of alcohol consumption, stress management, and avoidance of unnecessary NSAID use, can complement standard medical therapy and improve clinical outcomes. Recent advances, including potassium-competitive acid blockers, microbiome-based interventions, and personalized treatment approaches, further support the need for a comprehensive management strategy. Therefore, this review aims to provide an integrated overview of the epidemiology, pathophysiology, dietary interventions, lifestyle modifications, and current pharmacotherapeutic approaches in PUD, highlighting the importance of a multidisciplinary and patient-centered approach for effective management and prevention of disease recurrence.

Keywords: Peptic ulcer disease, *Helicobacter pylori*, diet, lifestyle modification, proton pump inhibitors, pharmacotherapy, nutrition, integrated management.

INTRODUCTION

Peptic ulcer disease (PUD) is a chronic gastrointestinal disorder characterized by a break in the mucosal lining of the stomach or duodenum extending through the muscularis mucosae as a result of acid-peptic injury. Gastric ulcers commonly occur in the stomach, whereas duodenal ulcers are predominantly found in the proximal duodenum. Despite advancements in healthcare, PUD remains an important cause of morbidity worldwide, affecting millions of individuals annually and imposing a

considerable economic burden on healthcare systems. The epidemiology of PUD has changed significantly over the past few decades due to the declining prevalence of *Helicobacter pylori* infection in developed countries and the widespread use of acid-suppressive medications; however, increasing use of NSAIDs, antiplatelet agents, and an aging population continue to sustain disease prevalence and complications. The pathogenesis of PUD involves a complex interaction between aggressive factors and

mucosal defense mechanisms. Aggressive factors include gastric acid secretion, pepsin activity, *H. pylori* infection, NSAID use, smoking, alcohol consumption, and physiological stress, while protective factors comprise mucus secretion, bicarbonate production, mucosal blood flow, prostaglandins, and epithelial regeneration. Disruption of this balance results in mucosal injury, inflammation, and ulcer formation. Among the etiological factors, *H. pylori* infection and NSAID use account for the majority of PUD cases worldwide. *Helicobacter pylori*, a gram-negative spiral bacterium, colonizes the gastric mucosa and induces chronic inflammation through the release of virulence factors and stimulation of host immune responses. Persistent infection contributes to mucosal damage and increases the risk of gastric and duodenal ulcers as well as gastric malignancy. Likewise, NSAIDs impair mucosal defense by inhibiting cyclooxygenase enzymes and reducing prostaglandin synthesis, thereby compromising gastric mucosal integrity (6). Additional factors such as smoking, excessive alcohol intake, poor dietary habits, psychological stress, and irregular meal patterns have also been associated with delayed ulcer healing and increased recurrence rates. Pharmacotherapy remains the cornerstone of PUD management. Proton pump inhibitors are considered the most effective acid-suppressive agents and play a central role in ulcer healing and *H. pylori* eradication regimens. Other therapeutic options include histamine-2 receptor antagonists, antacids, sucralfate, bismuth compounds, and emerging agents such as potassium-competitive acid blockers. However, pharmacological treatment alone may not adequately address modifiable risk factors that contribute to disease persistence and recurrence. Recent evidence highlights the importance of integrating dietary and lifestyle interventions with pharmacotherapy to achieve optimal outcomes. A balanced diet rich in fruits, vegetables, fiber, flavonoids, and probiotics may support mucosal protection and reduce inflammation, whereas avoidance of smoking, excessive alcohol consumption, self-medication with NSAIDs, and stress can enhance treatment effectiveness. Patient education, adherence to treatment regimens, and preventive strategies are increasingly recognized as essential components of comprehensive PUD management. Therefore, the present review aims to provide a comprehensive

overview of peptic ulcer disease, focusing on the integration of dietary management, lifestyle modifications, and pharmacotherapy. By summarizing current evidence and recent advances, this review seeks to emphasize a patient-centered and multidisciplinary approach for improving ulcer healing, reducing recurrence, and enhancing quality of life. {3,4,6,7,8,9,10,}

2. Types of Peptic Ulcer Disease

Peptic ulcer disease (PUD) encompasses ulcerative lesions that develop in areas of the gastrointestinal tract exposed to gastric acid and pepsin. The most common types are gastric and duodenal ulcers, while esophageal and stress-related ulcers occur less frequently. Classification based on the anatomical location and underlying etiology is important because it influences clinical presentation, management, and prognosis.

2.1 Gastric Ulcer (GU)

Gastric ulcers are ulcers that develop in the lining of the stomach, most commonly along the lesser curvature of the antrum. They are primarily associated with *Helicobacter pylori* infection, prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs), smoking, and advanced age. Gastric ulcers are generally associated with normal or reduced gastric acid secretion but impaired mucosal defense mechanisms.

2.2 Duodenal Ulcer (DU)

Duodenal ulcers occur in the first part (bulb) of the duodenum and represent the most common type of peptic ulcer. They are strongly associated with *H. pylori* infection and increased gastric acid secretion.

2.3 Esophageal Ulcer

Esophageal ulcers occur in the lower esophagus and are commonly associated with gastroesophageal reflux disease (GERD). Other causes include medication-induced injury, infections (particularly in immunocompromised patients), and radiation therapy.

2.4 Stress-Related Mucosal Ulcer (Stress Ulcer)

Stress ulcers develop in critically ill patients due to severe physiological stress rather than psychological stress. They are commonly observed in patients with severe burns, major trauma, sepsis, head injury, or those requiring prolonged intensive care. {12,13,14,15,17}

3. Etiology And Risk Factors

Peptic ulcer disease (PUD) is a multifactorial disorder that develops when aggressive factors, such as gastric acid, pepsin, *Helicobacter pylori* (*H. pylori*), and non-steroidal anti-inflammatory drugs (NSAIDs), overwhelm the protective mechanisms of the gastric and duodenal mucosa [3,7]. Although the incidence of uncomplicated ulcers has declined in many regions due to improved eradication therapy and acid-suppressive medications, PUD remains a major cause of gastrointestinal morbidity worldwide.

Helicobacter pylori Infection

H. pylori is the most important infectious cause of PUD and is responsible for the majority of duodenal ulcers and a substantial proportion of gastric ulcers. The bacterium colonizes the gastric mucosa, producing urease and other virulence factors that induce chronic inflammation, disrupt epithelial integrity, and alter gastric acid secretion. Persistent infection increases the risk of recurrent ulcers, gastrointestinal bleeding, and gastric malignancy if left untreated.

Non-steroidal Anti-inflammatory Drugs (NSAIDs)

NSAIDs represent the second leading cause of PUD. These agents inhibit cyclooxygenase (COX)-1 and COX-2 enzymes, reducing prostaglandin synthesis. As prostaglandins are essential for maintaining mucosal blood flow, mucus secretion, and bicarbonate production, their inhibition weakens mucosal defense and predisposes patients to ulcer formation and complications. The risk increases with advanced age, prolonged NSAID therapy, concomitant corticosteroid or anticoagulant use, and previous ulcer history.

Gastric Acid Hypersecretion

Although acid alone rarely causes ulcers, excessive gastric acid secretion significantly contributes to mucosal injury in susceptible individuals. Conditions such as Zollinger–Ellison syndrome and increased vagal stimulation result in excessive acid production, overwhelming mucosal protective mechanisms and delaying ulcer healing.

Lifestyle and Behavioral Factors

Smoking impairs gastric mucosal blood flow, delays ulcer healing, reduces bicarbonate secretion, and increases the likelihood of ulcer recurrence. Excessive alcohol consumption damages the gastric mucosal barrier and may potentiate NSAID-induced injury. Chronic psychological stress, irregular eating habits, inadequate sleep, and poor dietary practices may aggravate symptoms and interfere with mucosal recovery, although they are generally considered modifying rather than primary causative factors.

Additional Risk Factors

Other important contributors include increasing age, family history of PUD, severe physiological stress (e.g., burns, trauma, sepsis), chronic systemic illnesses, and concurrent medications such as corticosteroids, antiplatelet agents, anticoagulants, selective serotonin reuptake inhibitors (SSRIs), and bisphosphonates. Genetic susceptibility and socioeconomic factors also influence disease prevalence and recurrence. {17,18,19,20}

4. Pathophysiology

The pathophysiology of peptic ulcer disease is characterized by an imbalance between aggressive luminal factors and the protective mechanisms of the gastroduodenal mucosa. Under normal physiological conditions, gastric acid and pepsin facilitate digestion while the mucosal barrier—comprising mucus, bicarbonate secretion, epithelial tight junctions, adequate mucosal blood flow, prostaglandins, and rapid epithelial regeneration—protects the gastrointestinal lining.

Normal Mucosal Defense

The gastric mucosa is protected by a mucus–bicarbonate layer that neutralizes hydrogen ions near

the epithelial surface. Prostaglandins enhance mucus and bicarbonate secretion, maintain mucosal perfusion, and stimulate epithelial repair. Tight junctions prevent back-diffusion of hydrogen ions, while rapid epithelial turnover promotes healing of superficial injury

Mechanism of Ulcer Formation

Ulcer formation occurs when aggressive factors exceed mucosal defenses. Increased exposure to gastric acid and pepsin results in epithelial injury, inflammation, and progressive tissue necrosis. Once the damage extends beyond the muscularis mucosae, a peptic ulcer develops.

Helicobacter pylori-Induced Injury

H. pylori survives in the acidic gastric environment by producing urease, which converts urea into ammonia and carbon dioxide, creating a localized alkaline microenvironment. The organism adheres to gastric epithelial cells and releases virulence factors such as CagA and VacA, inducing inflammatory cytokines, oxidative stress, epithelial apoptosis, and disruption of the mucosal barrier. Chronic inflammation alters gastric acid secretion and predisposes to both gastric and duodenal ulceration.

NSAID-Induced Mucosal Damage

NSAIDs reduce prostaglandin synthesis by inhibiting cyclooxygenase enzymes, resulting in decreased mucus and bicarbonate production, reduced mucosal blood flow, impaired epithelial regeneration, and increased susceptibility to acid-mediated injury. Direct topical irritation by NSAIDs further exacerbates epithelial damage.

Ulcer Healing

Successful ulcer healing requires eradication of *H. pylori* when present, suppression of gastric acid secretion, restoration of mucosal blood flow, epithelial regeneration, and elimination of precipitating risk factors. Persistent infection, continued NSAID exposure, smoking, poor medication adherence, and antimicrobial resistance contribute to delayed healing and recurrent ulcer disease.

Understanding these mechanisms forms the basis for current therapeutic strategies, which integrate eradication of *H. pylori*, potent acid suppression, mucosal protection, dietary optimization, and lifestyle modification to achieve sustained healing and reduce recurrence {21,22,23,25,26}

5. Diagnosis

The diagnostic approach includes clinical assessment, laboratory investigations, testing for *Helicobacter pylori* (*H. pylori*), upper gastrointestinal endoscopy, and selected imaging studies when indicated.

Clinical Evaluation

A detailed medical history should assess NSAID or aspirin use, previous ulcer disease, smoking, alcohol consumption, corticosteroid or anticoagulant therapy, and symptoms suggestive of *H. pylori* infection. Physical examination is often nonspecific but may reveal epigastric tenderness. Signs of anemia, hypotension, tachycardia, or peritoneal irritation should prompt evaluation for complications such as bleeding or perforation.

Laboratory Investigations

Routine laboratory tests support diagnosis and help evaluate complications. Complete blood count (CBC) may reveal iron-deficiency anemia or acute blood loss. Liver and renal function tests are useful before initiating therapy and in evaluating differential diagnoses. Stool occult blood testing can detect gastrointestinal bleeding, while serum electrolytes should be assessed in patients with persistent vomiting or suspected gastric outlet obstruction.

Detection of Helicobacter pylori

Identification of *H. pylori* is recommended in all patients with confirmed or suspected PUD because eradication significantly reduces ulcer recurrence. Diagnostic tests are classified as invasive and non-invasive.

Non-invasive tests include:

- Urea breath test (UBT), considered one of the most accurate methods for detecting active infection and confirming eradication.

- Stool antigen test, which has high sensitivity and specificity for active infection.
- Serological antibody testing, which is less useful for confirming eradication because antibodies may persist after successful treatment.

Invasive tests, performed during upper gastrointestinal endoscopy, include:

- Rapid urease test.
- Histopathological examination of gastric biopsy specimens.
- Culture and antimicrobial susceptibility testing in selected patients, particularly those with treatment failure or suspected antibiotic resistance.

Upper Gastrointestinal Endoscopy

Upper gastrointestinal endoscopy (esophagogastroduodenoscopy, EGD) is the gold standard for diagnosing PUD because it allows direct visualization of gastric and duodenal ulcers, assessment of ulcer size and location, identification of bleeding stigmata, and biopsy of suspicious gastric lesions to exclude malignancy. Endoscopy is strongly recommended in patients with alarm features such as gastrointestinal bleeding, anemia, unexplained weight loss, dysphagia, recurrent vomiting, or age above guideline-recommended thresholds.

Imaging Studies

Imaging is not routinely required for uncomplicated PUD but is valuable when complications are suspected. An upright chest or abdominal radiograph may demonstrate free subdiaphragmatic air in patients with perforated ulcers. Computed tomography (CT) provides excellent evaluation of perforation, penetration, abscess formation, or gastric outlet obstruction and is frequently used in emergency settings.

Differential Diagnosis

Several disorders may mimic PUD, including gastroesophageal reflux disease (GERD), functional dyspepsia, gastritis, gastric carcinoma, pancreatitis, biliary tract disease, inflammatory bowel disease, and cardiac ischemia. Appropriate clinical evaluation and

diagnostic testing are necessary to establish the correct diagnosis and avoid unnecessary treatment. {24,27,28,29}

6. Diet and Nutritional Management

Diet and nutritional management constitute an essential supportive component in the comprehensive management of peptic ulcer disease (PUD). Although dietary interventions alone cannot heal peptic ulcers, appropriate nutritional practices can alleviate symptoms, promote mucosal healing, improve the effectiveness of pharmacological therapy, and reduce the risk of ulcer recurrence. Contemporary clinical evidence no longer supports highly restrictive "ulcer diets"; instead, dietary recommendations should be individualized according to each patient's symptom tolerance, nutritional status, and underlying risk factors. A balanced diet rich in fruits, vegetables, whole grains, legumes, and lean protein sources provides essential vitamins, minerals, antioxidants, and dietary fiber that support tissue repair and maintain gastrointestinal health. Nutrients such as vitamins A, C, and E, together with zinc and selenium, contribute to epithelial regeneration and protection against oxidative stress, thereby facilitating gastric mucosal healing. Regular meal timing and healthy eating habits are equally important in minimizing gastric irritation. Patients are encouraged to consume small, frequent meals rather than large meals, which may increase gastric acid secretion and worsen discomfort. Eating slowly, chewing food thoroughly, and avoiding late-night heavy meals may further reduce dyspeptic symptoms and improve digestion. While no single food has been conclusively shown to cause peptic ulcers, certain dietary items—including highly spicy foods, fried or fatty meals, acidic foods, carbonated beverages, excessive caffeine, and chocolate—may aggravate symptoms in susceptible individuals. Therefore, these foods should be limited only if they consistently trigger discomfort, rather than being universally prohibited. Lifestyle-related dietary habits also influence ulcer healing. Excessive alcohol consumption damages the gastric mucosal barrier, delays ulcer healing, and increases the risk of gastrointestinal bleeding. Similarly, smoking impairs mucosal blood flow, reduces bicarbonate secretion, delays epithelial repair, and significantly increases the recurrence of peptic ulcers. Consequently, complete

smoking cessation and avoidance or substantial reduction of alcohol intake should be strongly recommended as integral components of ulcer management. Increasing evidence supports the use of probiotics as adjunctive therapy, particularly during *Helicobacter pylori* eradication treatment. Probiotic microorganisms, including *Lactobacillus*, *Bifidobacterium*, and *Saccharomyces boulardii*, help restore the intestinal microbiota, reduce antibiotic-associated adverse effects, improve treatment adherence, and may modestly enhance *H. pylori* eradication rates. Similarly, several functional foods and naturally occurring phytochemicals, such as honey, turmeric (curcumin), ginger, garlic, broccoli sprouts, berries, and green tea polyphenols, possess antioxidant, anti-inflammatory, and antimicrobial properties that may contribute to gastric mucosal protection and healing. Nevertheless, these dietary components should be considered complementary to standard pharmacotherapy rather than substitutes for evidence-based medical treatment. Adequate hydration should also be encouraged to maintain normal gastrointestinal function and overall health, while excessive intake of caffeinated or sugar-

sweetened beverages should be avoided if they aggravate symptoms. Patients receiving long-term proton pump inhibitor therapy may be at increased risk of deficiencies in vitamin B12, magnesium, calcium, and iron because of reduced gastric acidity. Therefore, periodic nutritional assessment and appropriate supplementation should be considered in individuals receiving prolonged acid-suppressive therapy, particularly older adults and patients with additional nutritional risk factors. Overall, dietary counselling should be integrated with pharmacotherapy and lifestyle modification as part of a multidisciplinary, patient-centered approach to PUD management. Individualized nutritional planning, patient education, and regular clinical follow-up not only improve symptom control and medication adherence but also contribute to ulcer healing, prevention of recurrence, and enhancement of long-term quality of life. Although dietary measures alone cannot replace pharmacological treatment, they remain an indispensable adjunct in the comprehensive management of peptic ulcer disease. {30,31,33,34,38,39}

Table: Summary of Diet and Nutritional Management of Different Types of Peptic Ulcers

Parameter	Gastric Ulcer	Duodenal Ulcer	Esophageal Ulcer	Stress-Related Mucosal Ulcer
Primary Nutritional Goal	Promote gastric mucosal healing and reduce gastric irritation	Promote duodenal healing, reduce acid exposure, and prevent recurrence	Promote esophageal healing, reduce reflux, and improve swallowing	Preserve gastrointestinal mucosal integrity and prevent stress-related bleeding
Meal Pattern	Small, frequent meals (5–6/day); avoid prolonged fasting	Small, frequent meals at regular intervals; avoid skipping meals	Small, frequent meals; eat slowly; avoid meals 2–3 hours before bedtime	Early enteral nutrition within 24–48 hours in critically ill patients; gradual oral feeding during recovery
Recommended Foods	Whole grains, cooked vegetables, bananas, apples, lean meat, fish, eggs, legumes, low-fat dairy	Whole grains, fruits, vegetables, lean proteins, yogurt, legumes, adequate fluids	Soft foods, porridge, mashed vegetables, bananas, melons, lean proteins, soups, low-fat dairy	Easily digestible foods, soft cereals, cooked vegetables, lean proteins, adequate fluids during recovery
Foods to Avoid	Spicy foods, fried foods, alcohol, excessive caffeine, carbonated drinks, smoking	Spicy foods, fatty meals, processed foods, alcohol, carbonated drinks, smoking	Citrus fruits, tomato products, spicy foods, chocolate, peppermint, caffeine, alcohol, very hot foods	Alcohol, tobacco, excessive caffeine, spicy and fried foods during recovery

Protein Requirement	Moderate-to-high protein intake to support mucosal repair	Adequate lean protein intake for tissue healing	High-quality protein to promote esophageal tissue repair	Increased protein (1.2–2.0 g/kg/day) to support healing during critical illness
Micronutrients	Vitamins A, C, E, zinc, selenium, antioxidants	Vitamins A, C, E, zinc, selenium, antioxidants	Vitamins A, C, E, zinc, selenium for epithelial repair	Vitamins A, C, E, zinc, selenium and other trace elements to support immune function
Role of Probiotics	May improve gut health and support <i>Helicobacter pylori</i> eradication	Beneficial as an adjunct to <i>H. pylori</i> eradication therapy	Not routinely recommended unless clinically indicated	May be considered based on ICU protocols and gastrointestinal tolerance
Hydration	Adequate water intake; avoid very hot beverages	Maintain adequate hydration throughout the day	Frequent water intake; avoid very hot or very cold drinks if symptomatic	Maintain fluid and electrolyte balance according to clinical condition
Lifestyle Advice	Stop smoking, avoid alcohol and NSAIDs, maintain healthy weight	Stop smoking, avoid alcohol, manage stress, avoid unnecessary NSAIDs	Remain upright after meals, elevate head during sleep, stop smoking and alcohol	Early nutritional support, avoid unnecessary NSAIDs, rehabilitation, adequate rest
Expected Benefits	Faster ulcer healing, symptom relief, reduced recurrence	Reduced acid-related injury, improved healing, lower recurrence	Reduced reflux symptoms, improved swallowing, enhanced mucosal healing	Reduced risk of stress ulcers, improved recovery, decreased gastrointestinal complications

7. Lifestyle Modifications for Different Types of Peptic Ulcers

Lifestyle modification is an essential component of the integrated management of peptic ulcer disease (PUD). In addition to pharmacotherapy and nutritional management, behavioral changes help reduce ulcer symptoms, promote mucosal healing, prevent recurrence, and improve patients' quality of life. Although lifestyle recommendations vary slightly depending on the type of ulcer, several measures such as smoking cessation, avoidance of alcohol and unnecessary NSAID use, stress reduction, and adherence to prescribed medications are universally recommended.

1. Lifestyle Modifications for Gastric Ulcer

Patients with gastric ulcer should adopt healthy lifestyle practices that minimize gastric mucosal injury and facilitate ulcer healing.

Recommended lifestyle modifications:

- Smoking cessation: Smoking delays ulcer healing, reduces gastric mucosal blood flow, and increases ulcer recurrence.
- Avoid alcohol consumption: Alcohol irritates the gastric mucosa and may delay tissue repair.
- Avoid unnecessary NSAIDs: NSAIDs should be discontinued whenever possible or used with gastroprotective therapy.
- Manage psychological stress: Yoga, meditation, deep breathing exercises, and relaxation techniques may improve symptom control.
- Maintain regular meal timings: Avoid skipping meals and overeating.
- Maintain a healthy body weight through regular physical activity.
- Ensure adequate sleep (7–8 hours/day) to support healing and overall health.
- Adhere strictly to prescribed medications, particularly proton pump inhibitors (PPIs) and *Helicobacter pylori* eradication therapy.

2. Lifestyle Modifications for Duodenal Ulcer

Lifestyle interventions aim to reduce gastric acid exposure and prevent recurrence.

Recommended lifestyle modifications:

- Stop smoking, as tobacco increases acid secretion and delays ulcer healing.
- Avoid alcohol, which may worsen duodenal inflammation.
- Avoid prolonged fasting and maintain regular meal schedules.
- Reduce emotional stress through mindfulness, yoga, meditation, or counselling.
- Avoid unnecessary NSAIDs and aspirin unless medically indicated.
- Exercise regularly (at least 150 minutes of moderate-intensity activity per week).
- Maintain healthy body weight.
- Complete *Helicobacter pylori* eradication therapy and attend follow-up testing to confirm eradication.

3. Lifestyle Modifications for Esophageal Ulcer

Most esophageal ulcers are associated with gastroesophageal reflux disease (GERD); therefore, lifestyle modifications focus on reducing acid reflux.

Recommended lifestyle modifications:

- Maintain an upright posture for at least 2–3 hours after meals.
- Avoid lying down immediately after eating.
- Elevate the head end of the bed by 15–20 cm to reduce nocturnal reflux.
- Maintain a healthy body weight, particularly in overweight or obese individuals.
- Avoid smoking and alcohol consumption, which weaken the lower esophageal sphincter.
- Wear loose-fitting clothing to reduce abdominal pressure.
- Avoid late-night meals and heavy dinners.
- Take medications with sufficient water and remain upright after swallowing pills to prevent medication-induced esophageal injury.
- Adhere to prescribed acid-suppressive therapy (PPIs or H₂ receptor antagonists).

4. Lifestyle Modifications for Stress-Related Mucosal Ulcer

Stress-related mucosal ulcers develop in critically ill patients due to physiological stress. Lifestyle modifications mainly apply during the recovery period.

Recommended lifestyle modifications:

- Early mobilization and rehabilitation following recovery from critical illness.
- Smoking cessation and avoidance of alcohol to support mucosal healing.
- Avoid unnecessary NSAIDs and corticosteroids, unless clinically indicated.
- Maintain adequate hydration and follow prescribed nutritional support.
- Ensure adequate sleep and rest to facilitate recovery.
- Manage underlying chronic diseases such as diabetes, kidney disease, or liver disease.
- Attend regular follow-up appointments after hospital discharge.
- Adhere to prescribed stress ulcer prophylaxis or acid-suppressive medications, if indicated. {32,33,36}

8. Pharmacotherapy

Pharmacotherapy is the cornerstone of peptic ulcer disease (PUD) management and aims to relieve symptoms, promote ulcer healing, eradicate *Helicobacter pylori* (*H. pylori*), prevent recurrence, and reduce complications. Proton pump inhibitors (PPIs), such as omeprazole, pantoprazole, esomeprazole, rabeprazole, and lansoprazole, are the first-line agents because they effectively suppress gastric acid secretion and promote rapid ulcer healing. Histamine-2 receptor antagonists (H₂RAs), including famotidine and cimetidine, may be used as alternatives in patients who cannot tolerate PPIs but are generally less effective [1–4]. Eradication of *H. pylori* is essential in infection-associated PUD. Current guidelines recommend bismuth-based quadruple therapy or clarithromycin-based triple therapy depending on local antibiotic resistance patterns. Mucosal protective agents such as sucralfate and bismuth compounds enhance ulcer healing, while

misoprostol is particularly effective for preventing NSAID-induced ulcers in high-risk patients. Antacids provide rapid symptomatic relief but do not promote ulcer healing and should be used only as adjunctive therapy [2–6]. Recently, potassium-competitive acid blockers (P-CABs), particularly vonoprazan, have emerged as effective alternatives to PPIs because they provide rapid and sustained acid suppression and

improve *H. pylori* eradication rates. Successful treatment also requires discontinuation of unnecessary NSAIDs, adherence to prescribed medications, and confirmation of *H. pylori* eradication after therapy. Combining pharmacotherapy with dietary modification, lifestyle changes, and patient education provides optimal healing and reduces ulcer recurrence [3–8].

Table: Pharmacotherapy for Different Types of Ulcers

Ulcer Type	First-Line Therapy	Additional/Adjunct Therapy	Treatment of Underlying Cause	Treatment Duration
Gastric Ulcer	Proton Pump Inhibitors (PPIs): Omeprazole, Pantoprazole, Esomeprazole, Rabeprazole, Lansoprazole	H ₂ receptor antagonists (Famotidine), Antacids (Magnesium hydroxide, Calcium carbonate), Sucralfate, Bismuth compounds, Misoprostol (NSAID-induced ulcers)	<i>Helicobacter pylori</i> eradication: Bismuth quadruple therapy (PPI + Bismuth + Tetracycline + Metronidazole) or Concomitant therapy (PPI + Amoxicillin + Clarithromycin + Metronidazole); discontinue NSAIDs whenever possible	PPIs: 6–8 weeks; <i>H. pylori</i> therapy: 10–14 days
Duodenal Ulcer	PPIs: Omeprazole, Pantoprazole, Esomeprazole, Rabeprazole	H ₂ receptor antagonists (Famotidine), Antacids, Sucralfate, Bismuth compounds	<i>Helicobacter pylori</i> eradication using guideline-recommended regimens; discontinue NSAIDs if present	PPIs: 4–8 weeks; <i>H. pylori</i> therapy: 10–14 days
Esophageal Ulcer	PPIs: Omeprazole, Pantoprazole, Esomeprazole, Rabeprazole	H ₂ receptor antagonists (Famotidine), Sucralfate suspension	Treat the underlying cause: discontinue offending drugs; Fluconazole for Candida esophagitis; Acyclovir for Herpes simplex esophagitis; Ganciclovir/Valganciclovir for CMV esophagitis	PPIs: 8–12 weeks; infection-specific therapy as indicated
Stress-Related Mucosal Ulcer	PPIs: Pantoprazole, Omeprazole, Esomeprazole	H ₂ receptor antagonists (Famotidine), Sucralfate (selected patients)	Early enteral nutrition, correction of hemodynamic instability, endoscopic hemostasis for active bleeding, blood transfusion if required	Continue prophylaxis while the patient remains at high risk or until recovery from critical illness

Abbreviations

- **PPI:** Proton Pump Inhibitor
- **H₂RA:** Histamine-2 Receptor Antagonist
- **NSAID:** Non-Steroidal Anti-Inflammatory Drug
- **CMV:** Cytomegalovirus

- ***H. pylori:*** *Helicobacter pylori*

CONCLUSION

Peptic ulcer disease (PUD) is a multifactorial gastrointestinal disorder primarily caused by *Helicobacter pylori* infection, NSAID use, and lifestyle-related risk factors. Effective management requires an integrated approach that combines pharmacotherapy, dietary management, and lifestyle

modification. Proton pump inhibitors, H. pylori eradication therapy, and mucosal protective agents remain the mainstay of treatment, while a balanced diet, adequate nutrition, smoking cessation, alcohol avoidance, stress management, regular physical activity, and adherence to medication support ulcer healing and reduce recurrence. A patient-centered, multidisciplinary approach not only improves clinical outcomes and quality of life but also minimizes complications and promotes long-term disease prevention. Future research should focus on personalized treatment strategies and nutritional interventions to further optimize the management of peptic ulcer disease. {38,37,40}.

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PubMed Search Links (each returns many research papers)

10. <https://pubmed.ncbi.nlm.nih.gov/?term=peptic+ulcer+disease>

11. <https://pubmed.ncbi.nlm.nih.gov/?term=peptic+ulcer+review>
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