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Review Article

Peptic Ulcer Disease: A Comprehensive Review of Pathophysiology and Allopathic vs Herbal Treatment Strategies

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ABSTRACT

Peptic ulcer disease (PUD) is a common acid-related gastrointestinal disorder characterized by mucosal damage in the stomach or duodenum. Its major etiological factors include Helicobacter pylori infection and non-steroidal anti-inflammatory drug (NSAID) use, along with lifestyle, genetic, and hypersecretory conditions. The present review provides a comprehensive overview of PUD, including its epidemiology, pathophysiology, clinical features, diagnostic approaches, and conventional allopathic management strategies such as proton pump inhibitors, H_2 receptor antagonists, cytoprotective agents, and H. pylori eradication regimens. In addition, it highlights the role of herbal medicines traditionally used in Ayurveda and other systems, supported by phytochemical and pharmacological evidence. The review also compares allopathic and herbal therapies, discusses current research trends such as nanotechnology and polyherbal formulations, and addresses limitations of herbal therapy. Overall, PUD management requires an integrated understanding of both evidence-based medicine and complementary approaches for improved therapeutic outcomes.

INTRODUCTION

Peptic ulcer disease is an acid-induced lesion of the gastrointestinal tract, most commonly occurring in the stomach or proximal duodenum, and is characterized by mucosal disruption that extends beyond the muscularis mucosa into the submucosa or even deeper layers such as the muscularis propria. [1] The global prevalence is estimated to be around 5–10%, although recent epidemiological data indicate a decline in incidence, hospital admissions, and mortality. This reduction is largely attributed to improved hygiene practices and the widespread use of effective therapies, particularly those targeting Helicobacter pylori infection. [2, 3] Traditionally, peptic ulcer formation was thought to result primarily from increased gastric acid secretion combined with

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dietary factors and stress; however, current understanding emphasizes a multifactorial pathogenesis involving an imbalance between aggressive factors and mucosal defense mechanisms.

The major risk factors for peptic ulcer disease include Helicobacter pylori infection, non-steroidal anti-inflammatory drug (NSAID) use, alcohol consumption, smoking, and conditions such as Zollinger–Ellison syndrome. [4] Among these, H. pylori infection and NSAID use are the most significant contributors to both gastric and duodenal ulcers. However, only a minority of individuals exposed to these risk factors develop ulcers, highlighting the role of genetic and individual susceptibility, including cytokine gene polymorphisms such as interleukin-1β (IL1B). [5] The risk of complications increases significantly with NSAID and aspirin use, especially when combined with anticoagulants, corticosteroids, or selective serotonin reuptake inhibitors, which further elevate the risk of upper gastrointestinal bleeding. [6] Interestingly, a proportion of cases—termed idiopathic peptic ulcers—occur in the absence of H. pylori infection, NSAID use, or aspirin exposure. These cases, which account for about one-fifth of patients, may be associated with mechanisms such as psychological stress, ischemia, drug-induced injury, radiotherapy, infections, eosinophilic infiltration, surgical alterations like gastric bypass, or metabolic disturbances, although their exact pathogenesis remains incompletely understood. [7, 8]

2. Epidemiology

Peptic ulcer disease (PUD) is a common global gastrointestinal disorder, with a lifetime risk of developing the condition estimated to be approximately 5% to 10%. It represents a significant public health concern due to its association with morbidity, complications, and healthcare utilization worldwide. However, in recent decades, there has been a noticeable decline in the incidence of PUD across many regions. This reduction is largely attributed to improved sanitation and hygiene practices, widespread eradication of Helicobacter pylori infection, and more cautious and rational use of non-steroidal anti-inflammatory drugs (NSAIDs), which are major contributing factors to ulcer formation. [5] Epidemiological patterns also show variation in ulcer types and demographic distribution. Duodenal ulcers are significantly more common than gastric ulcers, occurring approximately four times more frequently. In addition, duodenal ulcers show a higher prevalence in males compared to females, whereas gastric ulcers tend to have a more balanced gender distribution. [7]

3. Etiology

Peptic ulcer disease (PUD) has various causes; however, Helicobacter pylori-associated PUD and NSAID-associated PUD account for the majority of the disease etiology.[1]

Table 1: Cause of Peptic ulcer [6, 7]

Category	Cause	Mechanism/Details
Common Causes	Helicobacter pylori infection	Gram-negative bacillus colonizing gastric epithelium; responsible for ~90% of duodenal ulcers and 70–90% of gastric ulcers. Causes mucosal inflammation and damage.
	NSAIDs	Inhibit COX-1 enzyme, reducing prostaglandin synthesis, mucus production, bicarbonate secretion, and mucosal blood flow.
	Medications	Includes corticosteroids,bisphosphonates, potassium chloride, and fluorouracil.

Lifestyle Factors	Smoking	Increases risk of duodenal ulcers and impairs ulcer healing.
	Alcohol	Irritates gastric mucosa and increases gastric acid secretion.
Rare Causes	Zollinger–Ellison Syndrome	Gastrin-secreting tumor causing excessive gastric acid production.
	Malignancy	Gastric cancer, lung cancer, and lymphomas may present with ulceration.
	Stress-related ulcers	Associated with severe illness, burns, head injury, trauma, or intensive care admission.
	Viral infections	Certain viral infections can damage gastric mucosa, particularly in immunocompromised patients.
	Vascular insufficiency	Reduced blood supply to the gastric mucosa leading to ischemic injury.
	Radiation therapy	Causes direct mucosal injury and ulcer formation.
	Crohn Disease	Chronic inflammatory bowel disease that may involve the upper gastrointestinal tract.
	Chemotherapy	Cytotoxic agents damage rapidly dividing gastrointestinal mucosal cells.

Table 2: Virulence Factors of Helicobacter pylori [8-9]

Virulence Factor	Function
Urease	Converts urea into ammonia, neutralizing gastric acid and allowing bacterial survival.
CagA/VacA Toxins	Cause gastric mucosal inflammation, epithelial damage, and ulcer formation.
Flagella	Provide motility, enabling movement through gastric mucus toward epithelial cells.

Table 3: Hypersecretory Conditions Associated with PUD [10-12]

Condition	Mechanism
Zollinger–Ellison Syndrome	Gastrinoma causes excessive gastrin secretion and acid hypersecretion.
Systemic Mastocytosis	Increased histamine release stimulates gastric acid secretion.
Cystic Fibrosis	Associated with altered gastrointestinal secretions and increased ulcer risk.
Hyperparathyroidism	Hypercalcemia stimulates gastrin release, increasing acid production.
Antral G-cell Hyperplasia	Increased gastrin-producing cells lead to excessive gastric acid secretion.

Table 4: Comparison of Major Causes of Peptic Ulcer Disease [13-15]

Cause	Frequency	Primary Mechanism
H. pylori infection	Most common	Mucosal inflammation and epithelial damage
NSAID use	Second most common	Reduced prostaglandin-mediated mucosal protection
Hypersecretory states	Rare	Excess gastric acid secretion
Medications	Less common	Direct mucosal injury or impaired protection
Stress-related illness	Rare	Ischemia and impaired mucosal defense
Malignancy	Rare	Tumor-associated ulceration

4. Pathophysiology

The peptic ulcer disease (PUD) mechanism results from an imbalance between gastric mucosal protective and destructive factors.

Table 5: Risk factors predisposing to the development of PUD [16-21]

Risk Factor	How It Increases Risk of Peptic Ulcer Disease (PUD)
Helicobacter pylori infection	*H. pylori* colonizes the gastric mucosa and causes chronic inflammation (gastritis). It damages epithelial cells, decreases bicarbonate secretion, disrupts the mucus barrier, and may increase gastric acid secretion through increased gastrin release. These effects weaken mucosal defenses and promote ulcer formation, especially duodenal ulcers.
NSAID Use	NSAIDs inhibit cyclooxygenase (COX) enzymes, reducing prostaglandin synthesis. Prostaglandins normally stimulate mucus and bicarbonate secretion, maintain mucosal blood flow, and promote epithelial repair. Their loss makes the mucosa vulnerable to acid injury. NSAIDs may also directly damage the gastric epithelium.
First-Degree Relative with PUD	Individuals with a parent, sibling, or child who has PUD have a higher risk due to genetic susceptibility, inherited patterns of gastric acid secretion, increased likelihood of *H. pylori* infection within families, and shared environmental factors.
Emigrant from a Developing Nation	People who immigrate from regions with high *H. pylori* prevalence often acquire the infection during childhood because of crowded living conditions, poor sanitation, or limited access to clean water. Since *H. pylori* infection can persist for life, the risk of PUD remains elevated even after migration.
African American/ Hispanic Ethnicity	These populations have historically shown a higher prevalence of *H. pylori* infection and may experience socioeconomic and healthcare-access factors associated with increased ulcer risk. The association is primarily related to environmental and epidemiologic factors rather than ethnicity itself.

5. Symptoms

The symptoms of peptic ulcer disease (PUD) typically include epigastric pain, dyspepsia, nausea, vomiting, bloating, belching, early satiety, and loss of appetite. In complicated cases, patients may present with weight loss, hematemesis, melena, or signs of anemia due to gastrointestinal bleeding.

Table 6: Symptoms [22]

Symptom	Description
Epigastric pain (most common)	Burning, gnawing, or aching pain in the upper central abdomen (epigastrium).
Pain related to meals	Pattern depends on ulcer location (gastric vs. duodenal ulcer).
Nocturnal pain	Pain may wake the patient from sleep, especially with duodenal ulcers.
Bloating	Sensation of abdominal fullness or distension.
Belching	Frequent burping due to dyspepsia.
Nausea	Feeling of sickness in the stomach; may occur with or without vomiting.
Vomiting	Occurs in some patients, particularly if gastric outlet obstruction develops.
Early satiety	Feeling full after eating a small amount of food.
Loss of appetite	More common with gastric ulcers because eating may worsen pain.
Weight loss	Often seen in gastric ulcers due to reduced food intake.
Weight gain	May occur in duodenal ulcers because eating relieves pain, leading to increased food intake.

6. Diagnosis of Peptic Ulcer Disease

Diagnosis of peptic ulcer disease involves a combination of clinical evaluation, endoscopic assessment, laboratory investigations, and detection of Helicobacter pylori infection. The process begins with a detailed history focusing on epigastric pain, relation to meals, NSAID use, and associated alarm symptoms such as weight loss, vomiting, gastrointestinal bleeding, or anemia.

Table 7. Diagnosis of Peptic Ulcer Disease (PUD) [23-24]

Diagnostic Method	Purpose	Key Findings/ Advantages
Clinical Assessment	Initial evaluation of suspected PUD	Identifies symptoms, risk factors, and possible complications.
Upper Gastrointestinal Endoscopy (EGD)	Gold standard test for diagnosis	Direct visualization of ulcers, allows biopsy, assesses bleeding, and helps exclude malignancy.
H. pylori Testing	Detects *H. pylori* infection, a major cause of PUD	Can be performed using invasive or non-invasive methods.
Laboratory Investigations	Evaluates complications and associated conditions	Detects anemia, bleeding, and other abnormalities.

7. Allopathic Treatment

Allopathic treatment of peptic ulcer disease primarily focuses on reducing gastric acid secretion, eradicating underlying causes, and promoting mucosal healing. Conventional antiulcer therapy includes proton pump inhibitors, H2 receptor antagonists, antacids, potassium-competitive acid blockers, and cytoprotective agents. These drugs act by either suppressing acid production, neutralizing existing acid, or enhancing mucosal defense mechanisms. In cases associated with Helicobacter pylori infection, combination antibiotic regimens are used along with acid-suppressing agents to achieve eradication and prevent recurrence. Treatment selection depends on the severity of symptoms, underlying etiology, and presence of complications. [25] Overall, allopathic therapy remains the cornerstone of evidence-based peptic ulcer management.

Table 8. Mechanisms of action and adverse effects of the most commonly used antiulcer treatment options. [26-30]

Drug Class	Examples	Mechanism of Action	Common Adverse Effects
Proton Pump Inhibitors (PPIs)	Omeprazole, Lansoprazole, Rabeprazole, Esomeprazole, Pantoprazole	Irreversibly inhibit the gastric H^+/K^+-ATPase (proton pump) in parietal cells, producing profound suppression of gastric acid secretion.	Headache, abdominal pain, diarrhea, nausea, vomiting, constipation, flatulence, vitamin B12 deficiency, osteoporosis/fracture risk (long-term use)
H_2 Receptor Blockers	Cimetidine, Famotidine, Nizatidine, Ranitidine	Block histamine H_2 receptors on gastric parietal cells, reducing acid secretion.	Headache, dizziness, anxiety, depression, thrombocytopenia, rare cardiovascular effects
Antacids	Aluminum hydroxide, Magnesium hydroxide	Neutralize gastric acid and increase gastric pH (>4), reducing pepsin activity. Magnesium salts also retain water osmotically.	Nausea, vomiting, hypophosphatemia, chalky taste, constipation (Al), diarrhea (Mg), abdominal cramps, electrolyte imbalance
Potassium-Competitive Acid Blocker (P-CAB)	Vonoprazan	Reversibly inhibits H^+/K^+-ATPase by competing with potassium at the final step of acid secretion.	Nasopharyngitis, diarrhea, constipation, upper respiratory tract inflammation, eczema, back pain, contusion
Cytoprotective Agents	Misoprostol, Sucralfate	Protect the gastric mucosa. Misoprostol increases mucus and bicarbonate secretion and improves blood flow.	Diarrhea, abdominal pain, headache, constipation

		Sucralfate forms a protective barrier over ulcer sites.	

Table 9. Helicobacter pylori Eradication Treatment Regimens [31-34]

Treatment Line	Regimen	Duration	Eradication Rate
First-Line Therapy	Standard Triple Therapy: PPI + Clarithromycin + Amoxicillin *(or Metronidazole if penicillin allergy)*	7–14 days	70–85%
Second-Line Therapy	Bismuth Quadruple Therapy: PPI + Bismuth + Tetracycline + Metronidazole	14 days	77–93%
Second-Line Therapy	Non-Bismuth Concomitant Therapy: PPI + Clarithromycin + Amoxicillin + Metronidazole	14 days	75–90%
Second-Line Therapy	Levofloxacin Triple Therapy: PPI + Amoxicillin + Levofloxacin	14 days	74–81%
Salvage (Rescue) Therapy	Rifabutin-Based Triple Therapy: PPI + Rifabutin + Amoxicillin	10 days	66–70%

8. Herbal Treatment of Ulcers

Herbal medicines have been widely utilized for centuries in traditional medical systems such as Ayurveda, Traditional Chinese Medicine (TCM), and Unani for the management of gastrointestinal disorders, including peptic ulcer disease. Their continued use is largely attributed to their broad pharmacological activities, perceived safety profile, affordability, and strong cultural and community acceptance. [35] Unlike many synthetic drugs, medicinal plants often contain a complex mixture of bioactive compounds that act synergistically to produce therapeutic effects such as acid suppression, mucosal protection, antioxidant activity, and enhancement of gastric defense mechanisms. In recent years, advances in pharmacognosy, phytochemistry, and experimental pharmacology have validated the anti-ulcer potential of several herbal agents, supporting their traditional use with scientific evidence.

A wide range of medicinal plants has demonstrated significant efficacy in both the prevention and treatment of peptic ulcers. These plants contain diverse phytoconstituents such as flavonoids, tannins, alkaloids, saponins, and terpenoids, which contribute to their gastroprotective effects by inhibiting gastric acid secretion, enhancing mucus and bicarbonate production, and reducing oxidative stress and inflammation in the gastric mucosa. Experimental studies, including in vitro assays and in vivo animal models, as well as limited clinical trials, have shown promising results for many herbal formulations. [36, 37] This chapter aims to present a comprehensive review of important anti-ulcer medicinal plants, their active phytoconstituents, and the scientific evidence supporting their therapeutic role in peptic ulcer management, highlighting their potential as complementary or alternative approaches in modern gastroenterology.

Table 10: List of medicinal plants possess anti-ulcer activity [38-45]

Sr. No.	Common Name	Biological Source	Major Phytoconstituents	Mechanism of Antiulcer Action
1	Aloe Vera	Leaf gel of Aloe vera	Aloin, Aloe-emodin, Polysaccharides, Acemannan	Increases mucus secretion, antioxidant activity, promotes ulcer healing and tissue regeneration

2	Licorice	Roots of Glycyrrhiza glabra	Glycyrrhizin, Liquiritin, Flavonoids	Enhances mucus production, cytoprotective effect, inhibits gastric acid secretion
3	Turmeric	Rhizomes of Curcuma longa	Curcumin, Demethoxycurcumin, Volatile oils	Antioxidant, anti-inflammatory, inhibits gastric mucosal damage
4	Neem	Leaves and bark of Azadirachta indica	Nimbidin, Nimbin, Azadirachtin, Flavonoids	Reduces acid secretion, enhances mucosal defense, antioxidant activity
5	Indian Gooseberry (Amla)	Fruits of Phyllanthus emblica	Vitamin C, Gallic acid, Ellagic acid, Tannins	Antioxidant activity, increases mucosal protection, accelerates healing
6	Holy Basil (Tulsi)	Leaves of Ocimum sanctum	Eugenol, Ursolic acid, Rosmarinic acid	Reduces gastric acid secretion, antioxidant and anti-inflammatory effects
7	Bael	Fruits of Aegle marmelos	Marmelosin, Tannins, Coumarins	Cytoprotective action, reduces gastric acidity, enhances mucus production
8	Drumstick	Leaves of Moringa oleifera	Quercetin, Kaempferol, Vitamins, Alkaloids	Antioxidant activity, mucosal protection, inhibition of ulcer formation
9	Guava	Leaves of Psidium guajava	Quercetin, Tannins, Flavonoids	Anti-secretory, antioxidant, gastroprotective effects
10	Pomegranate	Peel and fruits of Punica granatum	Ellagitannins, Punicalagin, Flavonoids	Antioxidant activity, strengthens gastric mucosa, reduces ulcer index
11	Ginger	Rhizomes of Zingiber officinale	Gingerols, Shogaols, Zingerone	Anti-inflammatory, antioxidant, inhibits gastric acid secretion
12	Ashwagandha	Roots of Withania somnifera	Withanolides, Alkaloids, Sitoindosides	Stress-induced ulcer protection, antioxidant activity
13	Garlic	Bulbs of Allium sativum	Allicin, Sulfur compounds, Flavonoids	Antioxidant activity, anti-*H. pylori* activity, mucosal protection
14	Cabbage	Leaves of Brassica oleracea	Vitamin U (S-methylmethionine), Glucosinolates	Accelerates ulcer healing and promotes mucosal regeneration
15	Banana	Fruits of Musa paradisiaca	Leucocyanidin, Flavonoids, Pectin	Enhances mucus secretion, strengthens mucosal barrier, cytoprotection

Table 11: Comparison of Allopathic and Herbal Therapies [46]

Feature	Allopathic Treatment	Herbal Treatment
Scientific evidence	Extensive	Variable
Speed of healing	Rapid	Usually slower
H. pylori eradication	Effective	Limited evidence
Standardization	High (fixed doses, regulated formulations)	Often variable (depends on plant source, preparation, and dose)
Side effects	Well characterized and monitored	May be underreported or less well documented
Cost	Moderate to high	Often lower
Clinical guidelines	Established and evidence-based	Limited standardized clinical guidelines

9. Current Research Trends in Peptic Ulcer Disease

Current research in peptic ulcer disease is increasingly focused on developing safer and more effective therapeutic strategies beyond conventional acid-suppressing drugs. One major area of interest is the use of herbal formulations in combination with proton pump inhibitors (PPIs) to enhance ulcer healing and reduce side effects. Researchers are also exploring plant-derived compounds with anti-Helicobacter pylori activity, aiming to develop natural alternatives or adjuncts to antibiotic therapy, especially in the context of rising antibiotic resistance. Another promising field is nanotechnology-based drug delivery systems, which improve targeted delivery of anti-ulcer agents, enhance drug stability, and increase therapeutic efficacy at the gastric mucosal level. In addition, there is growing interest in polyherbal gastroprotective formulations, where multiple medicinal plants are combined to achieve synergistic effects such as acid reduction, mucosal protection, and anti-inflammatory activity. Finally, antioxidant-based mucosal healing strategies are being studied extensively, focusing on reducing oxidative stress in gastric tissues to promote faster and more complete ulcer healing. [47, 48]

10. Limitations of Herbal Therapy

Herbal therapy in peptic ulcer disease has several important limitations that restrict its use as a standalone treatment. One major concern is the lack of standardization, as herbal products often vary widely in preparation, dosage, and potency. This leads to inconsistent concentrations of active constituents, making therapeutic effects unpredictable. Additionally, there is a scarcity of large-scale, well-designed clinical trials, which limits strong scientific validation of their efficacy and safety. Herbal remedies may also pose risks of herb–drug interactions, especially when used alongside conventional medications such as proton pump inhibitors, antibiotics, or NSAIDs. Furthermore, quality control issues remain significant, including contamination, adulteration, and variability in manufacturing practices. Because of these limitations, herbal therapies are best used as complementary approaches rather than replacements for evidence-based treatments, particularly in clinically significant cases such as peptic ulcers associated with Helicobacter pylori infection or NSAID use. [49-52]

CONCLUSION

Peptic ulcer disease remains a significant gastrointestinal disorder despite a global decline in incidence due to improved hygiene, reduced H. pylori prevalence, and rational NSAID use. Allopathic therapy, including acid-suppressive agents and antibiotic-based eradication regimens, remains the cornerstone of effective and evidence-based management. However, increasing interest in herbal medicines highlights their potential as complementary therapies due to their gastroprotective, antioxidant, and anti-inflammatory properties. Although many medicinal plants show promising antiulcer activity, limitations such as lack of standardization, variable efficacy, and insufficient clinical trials restrict their standalone use. Future research focusing on integrated approaches, novel drug delivery systems, and validated herbal formulations may enhance treatment outcomes and provide safer, more effective management strategies for peptic ulcer disease.

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