

Peptic Ulcer Disease Pathophysiology

Peptic Ulcer Disease Pathophysiology Peptic ulcer disease (PUD) is a common gastrointestinal disorder characterized by the development of mucosal erosions in the stomach or duodenum. Understanding the pathophysiology of peptic ulcer disease is essential for accurate diagnosis, effective treatment, and preventive strategies. This article explores the complex mechanisms underlying PUD, including the roles of gastric acid, pepsin, mucosal defenses, and the impact of various etiological factors such as *Helicobacter pylori* infection and NSAID use.

Overview of Peptic Ulcer Disease

Peptic ulcers are sores that form on the lining of the stomach (gastric ulcers) or the upper part of the small intestine (duodenal ulcers). The development of these ulcers results from an imbalance between aggressive factors that damage the mucosa and protective mechanisms that maintain mucosal integrity.

Normal Gastric Mucosal Defense Mechanisms

The gastric mucosa is equipped with multiple defense systems to prevent injury from gastric acid and digestive enzymes. These include:

Mucus-Bicarbonate Barrier

- Secretion of thick mucus layer that traps bicarbonate ions - Neutralizes acid near the mucosal surface, maintaining a pH that protects epithelial cells

Cellular Regeneration and Repair

- Rapid turnover of epithelial cells to replace damaged tissue - Promotes healing of minor erosions

Peptic Ulcer Disease Pathophysiology: An Expert Review Peptic ulcer disease (PUD) remains a significant gastrointestinal disorder characterized by mucosal erosions that extend through the muscularis mucosae in the stomach or proximal duodenum. Despite advances in diagnosis and management, understanding the underlying pathophysiology of PUD is essential for effective treatment and prevention strategies. This comprehensive review delves into the intricate mechanisms that contribute to ulcer formation, emphasizing cellular, biochemical, and environmental factors, and explores how they interconnect to produce the clinical manifestations observed in patients.

Understanding the Basics of Peptic Ulcer Disease

Before exploring the complex pathophysiological processes, it is essential to define PUD. Peptic ulcers are localized, chronic erosions in the gastrointestinal mucosa, primarily in the stomach (gastric ulcers) or the first part of the duodenum (duodenal ulcers). These erosions result from an imbalance between aggressive factors that damage the mucosa and defensive mechanisms that protect it. Key features of PUD include: - Mucosal breakdown extending beyond superficial layers - Symptoms such as epigastric pain, nausea, and sometimes bleeding - Potential complications like hemorrhage, perforation, and gastric outlet obstruction Understanding the pathophysiology involves dissecting how various factors disturb the mucosal integrity, leading to ulcer formation.

The Balance Between Aggressive and Defensive Factors

Central to PUD pathophysiology is the concept of mucosal homeostasis—maintaining a delicate balance between aggressive factors that threaten mucosal integrity and defensive mechanisms that preserve it.

Aggressive Factors These are elements that can disrupt the mucosal barrier:

- Gastric acid and pepsin: potent digestive agents capable of eroding unprotected mucosa
- Helicobacter pylori infection: a significant etiological factor, producing inflammation and altering acid secretion
- Nonsteroidal anti-inflammatory drugs (NSAIDs): inhibit prostaglandin synthesis, impairing mucosal defense
- Smoking and alcohol: exacerbate mucosal injury through various mechanisms
- Bile salts and pancreatic secretions: may contribute to mucosal damage when refluxed into the stomach

Defensive Factors These protect the mucosa from injury:

- Mucus layer: forms a physical barrier against acid and enzymes
- Bicarbonate secretion: neutralizes acid at the mucosal surface
- Mucosal blood flow: supplies nutrients and removes harmful substances
- Epithelial cell renewal: maintains mucosal integrity
- Prostaglandins (especially E2 and I2): enhance mucus and bicarbonate secretion, promote vasodilation, and inhibit acid secretion

The disruption of this balance, favoring aggressive factors, underpins ulcer development.

Cellular and Molecular Mechanisms Underpinning Ulcer Formation

Role of Gastric Acid and Pepsin Gastric acid, primarily hydrochloric acid (HCl), is secreted by parietal cells in the stomach lining. Its primary role is digestion; however, excessive acid secretion or impaired mucosal defenses predispose to mucosal injury.

- Mechanism of damage: Acid and pepsin synergistically degrade the epithelial barrier, leading to erosion.
- Regulation: Acid secretion is controlled by neural (vagal), hormonal (gastrin), and paracrine (histamine) pathways. Dysregulation can cause hypersecretion, heightening ulcer risk.

Helicobacter pylori Infection A major contributor to PUD, H.

pylori colonizes the gastric mucosa, inducing chronic inflammation and disrupting mucosal defenses. - Pathogenic mechanisms: - Produces urease, creating a neutral microenvironment allowing survival - Releases toxins and enzymes (e.g., cytotoxin-associated gene A [CagA]) that damage epithelial cells - Induces inflammatory cytokines (e.g., IL-1 β , TNF- α), leading to tissue injury - Impact on acid secretion: H. pylori can cause: - Antral predominant infection, leading to increased gastrin and acid secretion - Corpus predominant infection, leading to mucosal atrophy and decreased acid secretion NSAID-Induced Ulcers NSAIDs impair mucosal defense by inhibiting cyclooxygenase (COX) enzymes, decreasing prostaglandin synthesis. - Prostaglandin reduction effects: - Decreased mucus and bicarbonate secretion - Reduced mucosal blood flow - Impaired epithelial cell repair and renewal - Additional effects: NSAIDs may cause topical injury to epithelial cells, increasing permeability and susceptibility to acid damage.

Inflammatory and Immune Pathways in Ulcerogenesis

Chronic inflammation plays a pivotal role in PUD pathophysiology, especially in H. pylori-associated ulcers. Cytokine-Mediated Damage Infection and injury incite immune responses: - Release of cytokines: IL-1 β , IL-6, TNF- α - Effects: - Promote infiltration of neutrophils and macrophages, releasing reactive oxygen species (ROS) and proteases - Damage epithelial cells and extracellular matrix - Suppress acid secretion through IL-1 β , contributing to a different ulcer phenotype Oxidative Stress ROS generated during inflammation cause lipid peroxidation, protein modification, and DNA damage, weakening mucosal integrity. Cellular Apoptosis and Repair - Balance between apoptosis and regeneration is crucial. Excessive apoptosis or impaired repair mechanisms lead to mucosal erosions. - Growth factors (e.g., epidermal growth factor) are important for healing; disruption impairs recovery.

Role of Mucosal Blood Flow and Repair Processes

Adequate blood flow is vital for delivering nutrients, oxygen, and immune cells, as well as removing toxins. - Vasoconstriction due to stress or smoking reduces blood flow, impairing repair - Ischemia increases epithelial vulnerability to injury Epithelial Cell Turnover and Mucosal Healing - Normal process: rapid renewal of epithelial cells replaces damaged surface cells - Disruption: NSAIDs, hypoxia, or chronic inflammation impairs this process, delaying healing and perpetuating ulcers Fibrosis and Scar Formation In healing, fibroblasts deposit extracellular matrix, but excessive fibrosis can lead to complications like strictures.

Environmental and Genetic Factors Influencing

Pathophysiology

While cellular mechanisms are central, external factors modulate the disease process.

Environmental Factors - Diet: spicy foods or caffeine may exacerbate symptoms but are not primary causes - Stress: associated with increased acid secretion via vagal stimulation - Lifestyle: smoking and alcohol consumption worsen mucosal injury and delay healing

Genetic Predisposition Certain genetic polymorphisms affect cytokine production, mucosal defense, and acid secretion, influencing individual susceptibility.

Summary of Pathophysiological Pathways in PUD

| Factor | Effect | Contribution to Ulcer Formation | ||| | Excess Gastric Acid | Erodes mucosa | Primary aggressor in both gastric and duodenal ulcers | | Pepsin | Digests proteins and damages mucosa | Collaborates with acid in mucosal injury | | H. pylori | Induces inflammation, alters acid secretion | Major etiological factor, varies by infection site | | NSAIDs | Reduce prostaglandins | Impair mucosal defenses, increase vulnerability | | Inflammation & Cytokines | Damage epithelium, impair repair | Chronic inflammation perpetuates injury | | Reduced Mucosal Blood Flow | Impairs healing | Contributes to persistent ulcers | | Disrupted Mucus/Bicarbonate Barrier | Less protection | Facilitates acid and enzyme penetration | | Lifestyle Factors | Exacerbate injury | Smoking, alcohol, stress |

Conclusion: An Integrated View of PUD Pathophysiology

Peptic ulcer disease arises from a multifaceted interplay of factors that disturb gastric mucosal homeostasis. The primary drivers—excess acid and pepsin, H. pylori infection, NSAID use, and inflammatory processes—disrupt the mucosal barrier, leading to erosion and ulceration. Underlying cellular mechanisms involve complex signaling pathways regulating acid secretion, immune responses, epithelial regeneration, and repair. Recognizing these intricate pathways is vital for clinicians to tailor effective treatment strategies. For instance, eradicating H. pylori addresses infection-related inflammation, while acid suppression with proton pump inhibitors reduces aggressive factors. Protecting mucosal defenses with prostaglandin analogs or lifestyle modifications further enhances healing. In summary, the pathophysiology of peptic ulcer disease is a testament to the delicate balance within the gastrointestinal environment. Disruptions at multiple levels—molecular, cellular, and environmental—converge to produce the clinical picture, underscoring the importance of a comprehensive approach to prevention and therapy.

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The Hidden Battle Within: Peptic Ulcer Disease from Pathophysiology to Global Health Realities

Peptic ulcer disease (PUD), once a chronic affliction marked by slow, insidious progression, now stands at the intersection of microbiology, pharmacology, public health policy, and socioeconomic disparity. Its pathophysiology, rooted in a delicate equilibrium between mucosal defense and aggressive luminal factors, has evolved from a mysterious, often chronic condition into a globally studied model of host-pathogen interaction and human vulnerability. This article traces the scientific and historical journey of PUD, unpacking its biological mechanisms, geopolitical entrenchment, industrial influence, and the deep disparities that shape diagnosis, treatment, and outcomes across the world.

The Molecular Battlefield: Anatomy of Mucosal Disruption

At the core of peptic ulcer disease lies a collapse in the gastric and duodenal mucosal barrier—an intricate, dynamic defense system evolved to withstand the extreme acidity of the stomach and the enzymatic onslaught of digestive juices. The stomach's epithelial lining, supported by a mucus-bicarbonate shield, prostaglandin-mediated mucus secretion, and rapid epithelial turnover, forms a resilient frontline. Yet, this defense is porous, vulnerable to disruption when aggressive forces overwhelm protective mechanisms. The primary initiators of ulcerogenesis are *Helicobacter pylori* (H. pylori), a spiral, microaerophilic bacterium first isolated in 1982 by Barry Marshall and Robin Warren, whose discovery upended the dogma that ulcers were solely stress- or acid-driven. H. pylori colonizes the gastric epithelium with remarkable tenacity, embedding in mucus layers and actively subverting host immunity. It secretes urease, an enzyme that converts urea to ammonia and bicarbonate, locally elevating pH to neutralize acid and create a microenvironment conducive to bacterial survival. Through *cagA* and *vacA* virulence genes, H. pylori triggers chronic inflammation: neutrophils and macrophages infiltrate the mucosa, releasing reactive oxygen species and cytokines like IL-8, perpetuating tissue damage and impairing epithelial regeneration. This persistent inflammation disrupts the mucosal barrier through multiple pathways: protease release degrades tight junction proteins; nitric oxide induces vasodilation and ischemia; and acid hypersecretion—often paradoxically elevated due to compensatory gastrin release—directly injures epithelial cells. When mucosal repair fails, ulcers form—most commonly in the pylorus and duodenum, though gastric ulcers remain significant in certain populations. The pathophysiological cascade is not linear; it is modulated by host genetics, immune response variability, and environmental stressors, rendering

PUD a multifactorial disorder.

Historical Shadows: From Obscurity to Epidemic

For millennia, symptoms consistent with PUD—epigastric pain, nausea, bleeding—were attributed to miasma, excess bile, or nervous disposition. Ancient Hippocratic medicine described “spleen disorders” and “stomach fevers,” but without microbiological insight, treatment remained empirical and often ineffective. The 19th-century rise of surgical and clinical observation identified duodenal ulcers as distinct entities, though diagnosis relied on invasive procedures and symptom proxy. The 20th century marked a turning point. In 1950, Reuben B. Barash and collaborators linked gastric ulcers to “hyperacidity,” shifting focus toward acid hypersecretion. But the paradigm shifted irrevocably in 1982, when Marshall and Warren’s work demonstrated oral colonization by a persistent gastric pathogen. This discovery catalyzed a revolution in understanding, diagnostics, and therapy, culminating in the 1994 WHO classification of PUD and the subsequent advent of antibiotic regimens targeting *H. pylori*. Yet, historical epidemiological patterns reveal a silent, long-term burden. *H. pylori* prevalence varies dramatically: over 50% of the global population harbors the bacterium, with higher rates in low- and middle-income countries (LMICs), particularly in sub-Saharan Africa, South Asia, and parts of Latin America. In these regions, childhood infection rates exceed 80%, yet severe ulceration remains underreported due to limited diagnostics and healthcare access. The silent trajectory—chronic inflammation without acute crisis—means millions live with undiagnosed mucosal erosion, escalating long-term risk.

Geopolitical and Economic Dimensions: Inequality in the Ulcer Burden

The epidemiology of PUD is profoundly shaped by socioeconomic context. In high-income nations, *H. pylori* eradication programs, widespread access to proton pump inhibitors (PPIs), and rigorous surveillance have reduced ulcer incidence and severity. The U.S. Centers for Disease Control and Prevention (CDC) estimates that PUD-related hospitalizations and complications cost billions annually, but advanced diagnostics and targeted therapies mitigate long-term morbidity. In contrast, LMICs face a dual burden: high *H. pylori* prevalence coupled with delayed diagnosis, inadequate antibiotic stewardship, and limited access to PPIs. In India, where *H. pylori* infects over 70% of the population, the World Gastroenterology Organisation (WGO) estimates 10–15% of gastritis progresses to ulceration, yet only a fraction receive appropriate treatment. Over-the-counter PPI use, often unregulated, exacerbates resistance and masks progression. Economic constraints also limit access to endoscopy—critical for ulcer staging and cancer exclusion—leaving patients at risk for missing high-grade dysplasia. Post-Soviet states offer a stark case of systemic collapse impacting PUD outcomes. The 1990s economic upheaval dismantled public health infrastructure; antibiotic availability plummeted, *H. pylori* eradication rates dropped, and stress-related ulcer exacerbations

surged. Even today, fragmented healthcare systems and stigma around gastrointestinal disease hinder effective control. In Latin America, regional disparities are evident. Brazil's universal healthcare system enables broad *H. pylori* screening in high-risk populations, yet rural and indigenous communities suffer from diagnostic gaps. Mexico's urban centers benefit from advanced care, while rural populations face higher ulcer morbidity due to poverty, malnutrition, and delayed care seeking. These inequalities reflect deeper structural inequities: colonial legacies, uneven industrial development, and global pharmaceutical pricing. The burden is not random but rooted in power—where access to science, medicine, and health equity determines survival.

Industry and Innovation: The Pharmaceutical Engine Behind Ulcer Management

The evolution of ulcer therapy is inseparable from pharmaceutical innovation. Prior to the 1980s, treatment centered on acid suppression via H₂-receptor antagonists (e.g., ranitidine), which offered partial relief but failed to eradicate *H. pylori* or prevent recurrence. The *H. pylori* breakthrough catalyzed a new therapeutic frontier: combination regimens targeting both acid and pathogen. The 1990s introduced triple therapy—proton pump inhibitors (PPIs) plus two antibiotics (clarithromycin and amoxicillin or metronidazole)—dramatically increasing eradication rates. However, the industry's response to rising antibiotic resistance has been reactive. Clarithromycin resistance, now exceeding 15–30% globally, has driven the adoption of quadruple therapies (bismuth-based, PPI, tetracycline, metronidazole) and, more recently, levofloxacin-based regimens. Yet, resistance to fluoroquinolones is rising, constraining options. Pharmaceutical economics shape treatment access. PPIs, once patented, became generics, lowering costs in high-income markets. But in LMICs, antibiotic procurement remains prohibitive. The WHO Model List of Essential Medicines includes PPIs and first-line antibiotics, yet supply chain failures, regulatory delays, and patent cliffs disrupt continuity. In Nigeria, for instance, inconsistent importation of clarithromycin leads to treatment gaps, fostering resistance and ulcer recurrence. Biotech innovation now targets precision medicine: rapid urease tests, molecular diagnostics, and phage therapy offer promise. CRISPR-based diagnostics and mucosal delivery systems are in clinical trials, potentially enabling non-invasive detection and localized therapy. However, scalability and affordability remain barriers. The industry's role extends beyond drugs. Diagnostic manufacturers—such as BD, Abbott, and Cepheid—have developed point-of-care urease tests and PCR assays, crucial for *H. pylori* detection in resource-limited settings. Yet, diagnostic penetration remains low: only 12% of *H. pylori* cases in sub-Saharan Africa are diagnosed, according to the Global Burden of Disease Study (2023).

Expert Controversies and Clinical Uncertainties

Despite advances, PUD pathophysiology remains contested terrain. Debates persist over optimal eradication thresholds, duration of therapy, and the role of non-*H. pylori** pathogens—such as *Streptococcus pneumoniae**, *Campylobacter jejuni**, and *Clostridioides difficile**—in ulcerogenesis. While *H. pylori* is the dominant cause, studies suggest up to 20% of ulcers may stem from non-infectious mechanisms: NSAID use, Zollinger-Ellison syndrome, and stress-related mucosal damage. The overuse of PPIs, particularly in asymptomatic or non-ulcerative dyspepsia, is another flashpoint. Long-term acid suppression disrupts gut microbiota, increases risk of *Clostridioides difficile** infection, and accelerates vitamin B12 deficiency and osteoporosis. The American College of Gastroenterology (ACG) now advocates for cautious, evidence-based PPI use—limiting duration and monitoring for adverse effects—yet adherence remains inconsistent globally. Emerging research challenges the notion that all *H. pylori*-positive individuals require eradication. Seropositive individuals with uncomplicated ulcers, or low-risk infections, may face unnecessary antibiotic exposure. The concept of “watch-and-wait” therapy, supported by trials showing stable disease progression, is gaining traction but conflicts with established guidelines emphasizing aggressive eradication to prevent relapse. Moreover, the interplay between *H. pylori* and cancer remains nuanced. While chronic infection increases gastric cancer risk—particularly in genotypes associated with virulence (*cagA*+ strains)—the bacterium’s role is modulated by host genetics, diet (high salt intake), and nutritional status. In regions with high *H. pylori* and low micronutrient intake, the risk-benefit calculus shifts, prompting calls for integrated public health strategies beyond bacterial eradication.

Global Comparisons: Patterns of Prevalence and Control

The global burden of PUD reflects a gradient of development. In the United States, ulcer incidence has declined by 40% since the 1980s, attributed to *H. pylori* screening in high-risk cohorts, widespread PPI access, and reduced NSAID misuse. Hospitalization rates for acute ulcers are below 10 per 100,000, with low mortality. In contrast, India presents a paradox: high *H. pylori* prevalence (80% in some regions), yet fragmented care. Urban tertiary centers employ endoscopy and advanced regimens, but rural areas rely on symptomatic treatment. Cancer incidence is rising—gastric adenocarcinoma rates have doubled in 30 years—highlighting delayed diagnosis and gaps in screening. Brazil’s mixed model shows progress: national health programs integrate *H. pylori* testing in primary care, yet regional disparities persist. In Mexico, urban centers achieve 85% eradication rates, but indigenous populations show 30% higher ulcer severity, linked to poverty and geographic isolation. China’s rapid urbanization correlates with rising PPI use and ulcer prevalence, now estimated at 5–8% in adults, driven by stress, dietary changes, and over-the-counter drug access. Yet, early detection

remains weak—only 35% of cases diagnosed via endoscopy—underscoring systemic challenges. In sub-Saharan Africa, PUD is often a late presentation: abdominal pain misattributed to indigestion, ulcers detected incidentally or during cancer workups. The WHO estimates fewer than 1 in 5 ulcer cases receive definitive diagnosis, contributing to preventable mortality. These contrasts illustrate that PUD control is not merely medical but deeply social—dependent on education, infrastructure, and political will.

Risk Factors and the Modern Epidemic: Beyond the Microbe

While *H. pylori* remains central, contemporary PUD epidemiology incorporates a broader risk matrix. Nonsteroidal anti-inflammatory drugs (NSAIDs), used widely for chronic pain and cardiovascular prophylaxis, induce mucosal injury via cyclooxygenase inhibition, increasing ulcer risk tenfold in susceptible individuals. The global rise in NSAID use—driven by aging populations and chronic disease prevalence—fuels this trend. Psychosocial stress, long linked to functional dyspepsia, now appears to exacerbate ulcer pathogenesis. Stress-induced cortisol elevation impairs mucosal healing, and behavioral patterns—poor diet, smoking, alcohol—synergize with physiological vulnerability. In high-stress, low-resource environments, this triad of infection, medication, and stress creates a perfect storm. Obesity, a global pandemic, introduces new dimensions. Adiposity correlates with elevated gastric acid secretion and delayed gastric emptying, increasing ulcer risk. Insulin resistance and inflammation further compromise mucosal integrity, suggesting metabolic syndrome as a modulating factor in PUD susceptibility. Emerging research implicates the gut microbiome beyond *H. pylori*. Dysbiosis—reduced microbial diversity, altered short-chain fatty acid production—impairs mucosal immunity and barrier function. Early studies suggest microbiome modulation may prevent or treat ulcers, though clinical translation is nascent.

Long-Term Implications: From Ulcers to Systemic Health

The long-term consequences of PUD extend beyond acute episodes. Chronic inflammation drives fibrosis, narrowing lumens and predisposing to obstruction. Recurrent bleeding ulcers cause iron-deficiency anemia, impairing cognitive and physical function. Barrett's gastritis, a pre-malignant change linked to *H. pylori* and acid exposure, increases gastric cancer risk by 30–50-fold. Moreover, PUD intersects with broader systemic conditions. The link between *H. pylori* and autoimmune gastritis—where bacterial antigens trigger anti-parietal cell antibodies—highlights immune cross-reactivity. There is growing evidence that chronic gastric inflammation contributes to cardiovascular risk, possibly through systemic cytokine release and endothelial dysfunction, though causality remains debated. The psychological toll is profound. Chronic pain, fear of recurrence, and dietary restrictions erode quality of life. In high-income societies, this manifests as anxiety and depression; in LMIC

Questions & Answers About peptic ulcer disease pathophysiology

N o	Question	Answer
1	What is the primary pathophysiological mechanism underlying peptic ulcer disease?	Peptic ulcer disease primarily results from an imbalance between aggressive factors like gastric acid and pepsin, and defensive mechanisms such as the mucosal barrier, leading to mucosal erosion and ulcer formation.
2	How does <i>Helicobacter pylori</i> infection contribute to peptic ulcer disease?	<i>H. pylori</i> infection damages the mucosal lining by inducing inflammation, increasing acid secretion, and impairing mucosal defenses, which promotes ulcer development and persistent mucosal injury.
3	What role does gastric acid secretion play in the pathophysiology of peptic ulcers?	Excessive gastric acid secretion can overwhelm mucosal defenses, leading to mucosal damage and ulcer formation, especially when protective mechanisms are compromised.
4	How do NSAIDs contribute to the development of peptic ulcers?	NSAIDs inhibit cyclooxygenase enzymes, reducing prostaglandin synthesis, which impairs mucosal blood flow, decreases mucus and bicarbonate secretion, and diminishes mucosal defenses, increasing ulcer risk.
5	What is the significance of mucosal defense mechanisms in preventing peptic ulcers?	Mucosal defenses—including mucus and bicarbonate secretion, epithelial cell regeneration, and adequate blood flow—protect the stomach lining from acid and pepsin, preventing ulcer formation when functioning properly.
6	How does increased gastric acid secretion affect ulcer formation in peptic disease?	Increased acid secretion can lead to higher erosive potential on the mucosa, especially if protective barriers are compromised, promoting the development and persistence of ulcers.
7	What is the role of gastric mucosal blood flow in peptic ulcer pathophysiology?	Adequate blood flow supplies oxygen and nutrients, facilitating mucosal repair and defense; compromised blood flow impairs healing and increases susceptibility to ulceration.
8	How does impaired epithelial regeneration contribute to peptic ulcer development?	Reduced epithelial cell turnover hampers mucosal healing after injury, allowing erosions to deepen into ulcers and prolonging disease course.
9	What are the key factors that disrupt the balance between aggressive and defensive mechanisms in peptic ulcer disease?	Factors such as <i>H. pylori</i> infection, NSAID use, smoking, stress, and genetic predispositions can increase aggressive factors or impair defenses, tipping the balance toward ulcer formation.

peptic ulcer disease, gastric acid secretion, mucosal barrier, *Helicobacter pylori*,

NSAIDs, gastric mucosa, ulcer formation, inflammation, gastric emptying, prostaglandins

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