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Overview of Peptic Ulcer Disease: Epidemiology, Causes, Pathophysiology, and Clinical Importance

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Keywords

Stomach ulcers, duodenal ulcers, gastric ulcers, Helicobacter pylori, epigastric pain, and abdominal pain.

Abstract

Peptic Ulcer Disease (PUD) is a prevalent gastrointestinal condition characterized by ulcerative lesions in the stomach, duodenum, or lower esophagus, resulting from an imbalance between aggressive factors such as gastric acid, pepsin, Helicobacter pylori infection, and NSAID use, and protective mechanisms including mucus and bicarbonate secretion. Affecting approximately 5–14% of the global population—primarily men aged 40 to 60 PUD presents with symptoms like epigastric pain and discomfort, with diagnosis often confirmed via upper gastrointestinal endoscopy. The pathophysiology of PUD involves complex interactions among genetic predisposition, environmental triggers, and lifestyle factors. Its chronic nature, characterized by alternating periods of flare-ups and remission, poses significant clinical and economic challenges. Advances in understanding gastric physiology and the identification of H. pylori have transformed the management and surgical approach to the disease, underscoring its continued relevance in modern gastroenterology.

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1. Introduction of Peptic Ulcer Disease

Peptic ulcer disease (PUD) is a persistent medical condition that involves the repeated development of ulcerative lesions in the stomach or duodenal lining, characterized by cycles of flare-ups and periods of relief. It occurs in 6% to 14% of the world's population, mostly men between 40 and 60. The paper's introduction provides an overview of the epidemiology, causes, and pathophysiology of gastric ulcers, emphasizing the complex interplay of genetic, environmental, and lifestyle factors contributing to the onset of peptic ulcer disease and its clinical importance [1]. PUD involves open sores in the stomach, duodenum, or esophagus, resulting from an imbalance between harmful elements like gastric acid and protective factors. Common causes include Helicobacter pylori infection, NSAID use, and alcohol intake. Peptic ulcer disease encompasses conditions with ulcers in the gastrointestinal tract due to a disparity between aggressive factors such as acid and pepsin, and defensive elements like gastric mucus and bicarbonate, leading to inflammation, tissue death, and ulcer formation [2]. Peptic ulcer disease is a widespread gastrointestinal condition characterized by epigastric pain and discomfort, primarily affecting the stomach and duodenum. Upper gastrointestinal endoscopy is the definitive method for diagnosing and assessing the disease. PUD refers to ulcers in the digestive tract lining, affecting 5–10% of the global population. It arises from an imbalance between aggressive and protective mucosal factors, resulting in gastric mucosal damage and imposing a significant economic burden on healthcare [3]. PUD involves a break in the stomach lining, small intestine, or lower esophagus. Gastric ulcers form in the stomach, while duodenal ulcers occur in the first part of the intestine, often causing abdominal pain and complications. Peptic ulcer disease is a chronic disruption of the stomach or duodenal mucosa, primarily due to Helicobacter pylori infection or nonsteroidal anti-inflammatory drug use, characterized by ulcers deeper than 5 mm penetrating through the muscularis mucosa [4]. Peptic ulcer disease has significantly influenced modern general surgery, evolving from surgical techniques like emptying and

patching operations to physiological procedures. Understanding gastric function and acid production has improved, especially with the identification of Helicobacter pylori as a causative factor [5]. PUD is a prevalent gastrointestinal illness involving the development of open sores or ulcers in the mucosal lining of the stomach (gastric ulcers) or the proximal small intestine, specifically the duodenum (duodenal ulcers) [6]. They develop when the mucosal lining is damaged by an imbalance between aggressive forces such as gastric acid and pepsin, and protective mechanisms like mucus and bicarbonate secretion. One of the greatest advances in gastroenterology was the discovery of Helicobacter pylori (H. pylori) as a primary cause of peptic ulcers [7]. This gram-negative rod colonizes the gastric mucosa and causes chronic inflammation, resulting in mucosal injury and ulceration. Apart from H. pylori infection, long-term use of NSAIDs like aspirin and ibuprofen is another significant etiological factor [8]. NSAIDs block prostaglandin synthesis, which weakens the gastric mucosal defense mechanism and makes it vulnerable to acid damage [9].

2. Pathophysiology

Peptic ulcer disease is composed of mucosal erosion within the duodenum or stomach and is primarily due to Helicobacter pylori infection, increased acid secretory function, and impaired mucosal defense mechanisms, leading to ulceration and its related symptoms [10]. Peptic ulcer disease is defined by mucosal erosion of the stomach or duodenum, which is primarily defined by Helicobacter pylori infection, increased acid secretion, and compromised mucosal defense mechanisms leading to ulcer formation and related symptoms. The lesions are referred to as peptic ulcers or erosions if they occur in mucosa exposed to acid-peptic secretions. The significant characteristic of pain in patients with peptic ulcer and this seems to be so whether the ulcer is in the stomach or the duodenum since the two cannot be differentiated on symptomatic grounds alone is that the initial discomfort occurs in the upper abdomen and not limited to the epigastrium, and the pain does actually tend to be intermittent and it often awakens the patient from sleep. In addition, it is characteristically relieved intermittently by taking antacids but not invariably by food intake. Bleeding, perforation, and retention of gastric content are the severe complications of peptic ulcer [11].

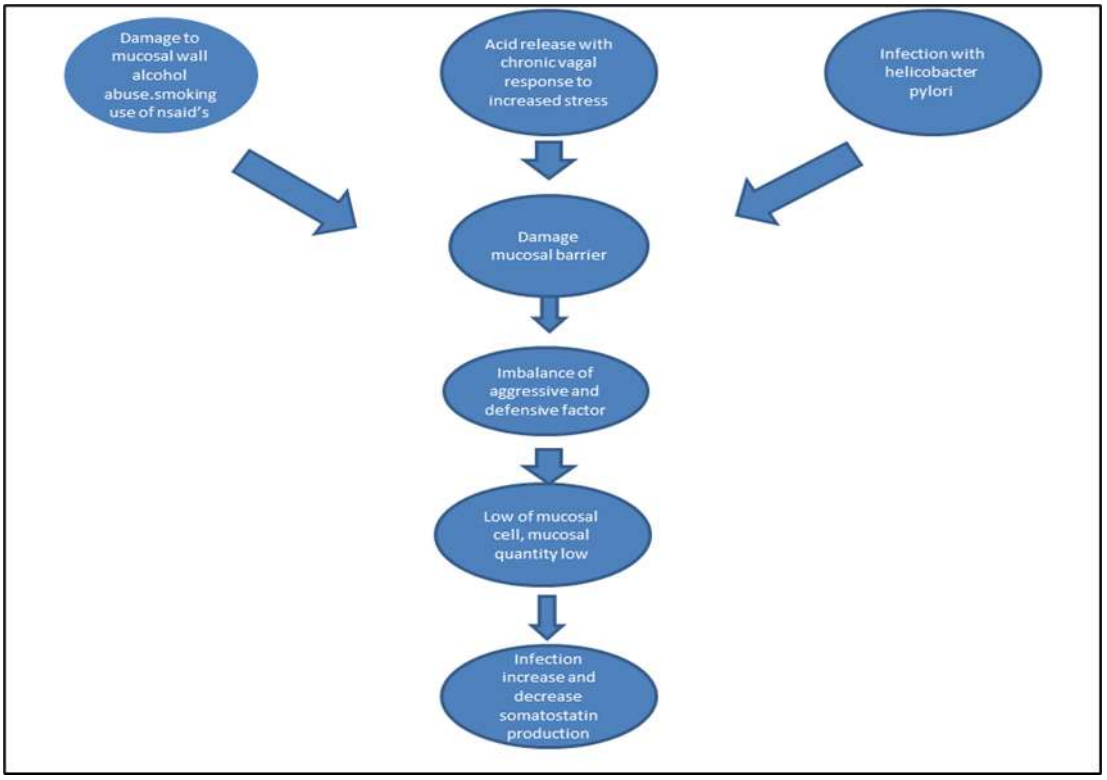

Figure 1: Pathophysiology of Mucosal Barrier Damage Leading to Gastrointestinal Infection.

3. Types of Peptic Ulcers

The two most common types of peptic ulcer disease are:

1. Gastric ulcers
2. Duodenal ulcers

They occur on the duodenum or stomach lining due to an imbalance between offensive and defensive factors [12].

2.1 Gastric Ulcers

Gastric ulcers are a type of peptic ulcer and involve mucosal erosion in the stomach. Factors such as Helicobacter pylori infection, use of NSAIDs, smoking, and alcohol consumption are of significance. Treatment includes proton pump inhibitors, H2 receptor antagonists, and surgery in case of complications. Gastric ulcers are open sores that develop in the stomach lining, typically resulting from causes like Helicobacter pylori infection, intake of NSAIDs, and excess alcohol consumption. Epigastric pain, bloating, and nausea are some of the symptoms, with acid reduction and H.pylori eradication being

treated [13]. Gastric ulcers are a subtype of peptic ulcer disease (PUD) that arise directly in the gastric mucosa. They are typified by focal erosion caused by an imbalance of protective mucosal factors versus destructive agents like pepsin and gastric acid. Clinically, gastric ulcers can be associated with epigastric pain after meals, nausea, bloating, or loss of weight. In severe forms, upper gastrointestinal bleeding or perforation can take place. Unlike duodenal ulcers, whose pain increases with food intake, gastric ulcer pain can worsen after consuming food, through direct acid exposure to the ulcerated mucosa [14].

3.2Duodenal Ulcers

Duodenal ulcers are a type of peptic ulcer disease involving mucosal damage in the duodenum, commonly with dyspepsia. Consumption of NSAIDs and Helicobacter pylori infection are frequent causes needing appropriate diagnosis and treatment based on the intensity of symptoms. Duodenal ulcers are a common type of peptic ulcer disease, seen in 5-10% of adults, predominantly men. They are more common than gastric ulcers, with over 60% of peptic ulcers occurring in the duodenum, necessitating proper nursing care and education. Duodenal ulcers refer to a form of peptic ulcers found in the initial part of the small intestine, called the duodenum [15]. Duodenal ulcers are caused by the erosion of the mucosa of the duodenum by the corrosive action of gastric acid and pepsin, which outweighs the defense of the mucosal lining. Duodenal ulcers are more prevalent than gastric ulcers and will usually involve younger people, generally between 30 and 50 years old. The most frequent etiology of duodenal ulcers is H. pylori infection, a gram-negative bacterium that inhabits the stomach and the proximal duodenum. The infection leads to chronic inflammation and augmentation of acid secretion, which results in mucosal damage. A second leading cause is the ingestion of NSAIDs, which compromise mucosal defense by interfering with prostaglandin production. Other causative variables are smoking, heavy alcohol drinking, and extreme physiological stress, particularly in hospitalized or seriously ill patients [16].

4. Causes and Risk Factors

The most common etiology of Cushing syndrome is the use of corticosteroid medications, with a frequency of 12 cases per 1 million people. The other etiologies sum up to 13 cases per 1 million. Women are more commonly affected by hypercortisolism secondary to pituitary tumors and adrenal tumors producing cortisol while hormone-secreting lung tumors are more common in men. Cushing syndrome is predominantly observed in adults, most of whom are affected in the range of 25–40 years of age, and is relatively rare in children, at an incidence rate of approximately 0.2 per million [17].

4.1 Helicobacter Pylori Infection

Helicobacter pylori is a Gram-negative intracellular bacterium that is associated with peptic ulcer disease and gastric cancer. It needs more than one method of diagnosis, and treatment involves more than one pharmacological modality to manage the infection and its resulting complications. Helicobacter pylori (H. pylori) is a gram-negative, spiral-shaped bacterium that infects the gastric mucosa and is identified as one of the most prevalent chronic bacterial infections globally. It has been estimated to infect more than 50% of the world's population, with increased prevalence rates in third-world countries attributed to poor hygiene and crowded housing conditions [18].

4.2 Non-Steroidal Anti-Inflammatory Drugs

Non-steroidal anti-inflammatory drugs (NSAIDs) are also widely applied in the management of inflammation and pain, particularly for chronic illnesses like arthritis. Their use, however, has serious side effects like gastrointestinal, cardiovascular, renal, and liver toxicities as well as hypersensitivity reactions. NSAIDs act through the inhibition of cyclooxygenase (COX) enzymes, namely COX-1 and COX-2, which are engaged in the production of prostaglandins—lipid molecules responsible for mediating pain, inflammation, and fever. COX-2 inhibition is the cause of the anti-inflammatory and analgesic actions, whereas COX-1 inhibition impacts protective prostaglandins in the gastrointestinal tract, kidneys, and platelets, thus tending to produce side effects [19].

4.3 Smoking and Alcohol Intake

Both smoking and alcohol consumption drastically lower the concentration of total protein and albumin, but raise CRP, LDL, triglycerides, and total cholesterol. Both of these habits trigger systemic inflammatory mechanisms and lipid derangements that cause arterial sclerosis as well as cardiovascular disease risk. Smoking and alcohol use are two of the most common modifiable lifestyle habits that contribute importantly to global health. Both are key causes of the burden of non-communicable diseases, such as cardiovascular disease, cancer, and gastrointestinal disorders. Cigarette smoking involves thousands of toxic chemicals that harm body tissues and interfere with normal physiological processes. It is linked with enhanced gastric acid secretion, decreased mucosal protection, and impaired ulcer healing, and thus it is a recognized risk factor for peptic ulcer disease and gastric cancer [20].

4.4 Anxiety and Stress

Stressful repeated or traumatic life events usually precede the development of anxiety disorders. The Stress Reactivity Index is pivotal in determining the magnitude of the "life event effect," and the index serves as a marker for vulnerability to stress. A vicious cycle may set in, perpetuating it even after the initiating stressors have abated, in anxious-vulnerable individuals. It emphasizes the interactive correlation of anxiety and stress and, therefore, necessitates consideration for understanding an individual's susceptibility to stressors. Stress can be described as a condition of mental or emotional tension caused by hostile or adverse situations. Chronic stress enhances one's vulnerability to disease. Gastro-oesophageal reflux disease, peptic ulceration, irritable bowel syndrome, Crohn's disease, and ulcerative colitis are diseases of the gastrointestinal tract that have high anxiety and stress levels. This paper aims to discuss

the research findings concerning possible connections between gastrointestinal disease and stress. Emotional stress (ES) has been suggested as one potential factor in the pathogenesis of duodenal ulcer (DU) disease. Recent, well-controlled research on the influence of ES on gastric acid secretion (GAS) in normal healthy subjects and in patients with inactive DU has not been done. Ten normal (N) men and 10 inactive DU men were studied on 2 different days. Randomly, subjects received either dichotomous listening (DL) to create stress or a control (non-DL) test. As well as assessing GAS in 15-min units, heart rate and blood pressure were assessed every 7.5 min, and visual analog scale assessments of emotion (relaxation, anxiety, anger, tension, and depress [21].

5. Symptoms and Diagnosis

Symptoms of peptic ulcer disease include epigastric pain, bloating, nausea, and life-threatening complications of bleeding or perforation. Endoscopy or Helicobacter pylori infection tests are used for diagnosis without invasive interventions [22].

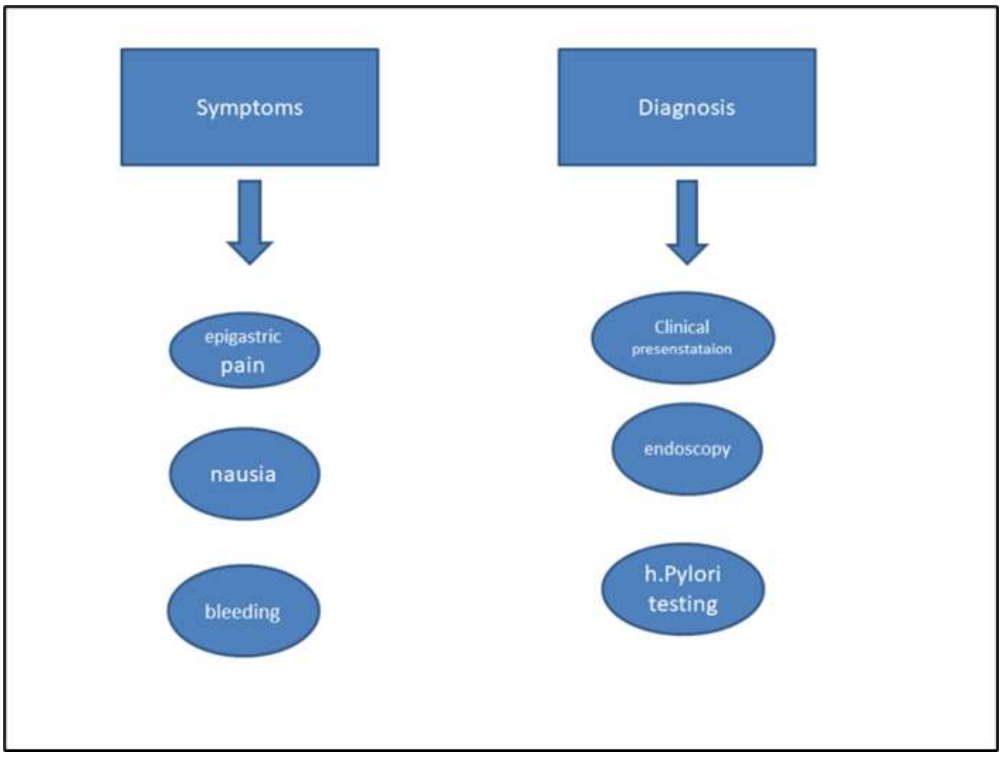

Figure 2: Symptoms and Diagnostic Approaches in Gastric Mucosal Damage.

5.1 Clinical Presentation

The presentation of peptic ulcers entails epigastric pain, bloating, nausea, and in severe cases, complications such as bleeding or perforation. The manifestations arise due to the inflammation of the ulcer's lining of the stomach or duodenum [23]. Most frequent presenting symptom of perforated peptic ulcer is acute onset of severe epigastric pain in 82.4% of patients. Delayed presentation after 48 hours is associated with higher postoperative complication and mortality. Clinical presentation of peptic ulcer disease is generally uncompleted. Treatment aims to eliminate Helicobacter pylori where found, utilizing acid-reducing therapy with such agents as proton pump inhibitors, and resolving complications such as bleeding through prompt endoscopy and high-dose PPI [24]. The presenting symptoms of peptic ulcers are a low symptom score, with key features being pain subsiding after meals, a history of peptic ulcer, and smoking. Postprandial pain and nausea are not characteristic of peptic ulcer disease. Peptic ulcer patients typically present with a variety of symptoms, mostly epigastric pain (85%). The symptoms may last for a period of up to 10 years, and food may exacerbate gastric ulcer pain but alleviate pain in duodenal ulcers. Silent ulcers present in 7% of the cases [25].

5.2 Endoscopy and Biopsy

The study reveals that intraoperative biopsy of perforated gastric ulcers is often redundant, as it does not alter clinical outcomes. Regardless of whether the biopsy is positive or not, upper gastrointestinal endoscopy is needed for further assessment and management of the condition [26]. Peptic ulcers, making direct visualization of the upper gastrointestinal tract possible. In the Koya City study, biopsies were obtained from antral and body mucosa of 267 patients with gastrointestinal complaints for histopathological examination. The results indicated that 91.76% of the cases had abnormal gastric mucosa, with gastritis being the most frequent diagnosis. This emphasizes the role of endoscopy and biopsy in diagnosis and peptic ulcer disease understanding [27]. Endoscopy and biopsy are fundamental diagnostic methods for peptic ulcers. Endoscopy facilitates direct observation of the gastric and duodenal mucosa, facilitating the detection of ulcers and an evaluation of their intensity. During the process, biopsy samples can be obtained to test for Helicobacter pylori infection and to exclude malignancy. These procedures increase

diagnostic accuracy and inform proper management options, enabling effective treatment of peptic ulcers and management of any complications that might develop [28]. Endoscopy and biopsy are essential in diagnosing peptic ulcers, especially in unusual instances of complication such as hepatic penetration. Through the process, direct visualization of the ulcer and obtaining tissue samples for histopathological examination is possible. In the case reported here, large ulcers with pseudo tumoral masses and liver tissue in biopsy samples were notable findings. Endoscopic biopsy will validate the diagnosis, which is frequently difficult because of the complexity of the symptoms and the requirement of precise histological evaluation [29]. Endoscopy is important for diagnosing gastric ulcers, but frequently cannot reliably distinguish between benign and malignant ulcers. Endoscopy diagnosed 65% of malignant gastric ulcers (MGUs) accurately in a study, but the accuracy increased significantly to 95% when combined with biopsy and cytology. It is vital that these methods be used in combination because they are complementary, and some diagnoses were made using a single modality only. Hence, biopsy and endoscopy should simultaneously be carried out to avoid accurate classification of peptic ulcers [30].

5.3 H. Pylori Testing

H. pylori testing is necessary to diagnose peptic ulcers since this bacterium is a leading cause of ulcer disease. The study compared two rapid stool antigen tests (Right Sign® and On-site®) for the detection of H. pylori with high sensitivity (96.6% and 96.9%, respectively) but reduced specificity (66.1% and 50%). While these tests can safely rule out H. pylori infection, positive results must be confirmed by more precise methods like HpSA-ELISA to ensure proper diagnosis and treatment [31]. H. pylori testing is needed in the treatment of peptic ulcers, particularly in those under 60 years with alarm-free dyspepsia. Noninvasive tests such as the urea breath test or stool antigen test are preferred to diagnose and later treat [32]. H. pylori diagnosis in peptic ulcers is vital, and according to the study, stool antigen tests were the best diagnostic test, superior to blood antibody tests and standard culture in detecting infections in patients in Lafia, Nigeria [33]. Post-treatment H. pylori testing for peptic ulcer disease is essential to validate eradication. Only 44% of patients were found to have been tested in the study, and significantly more in outpatients (57%) than inpatients (33%) and patients with GI follow-up (62%) [34]. H. pylori testing in peptic ulcers involves non-invasive tests such as stool antigen tests and breath tests, alongside invasive tests such as esophago-gastro-duodenoscopy biopsies. Proper testing is important for the proper treatment and management [35]. H. pylori diagnosis in peptic ulcers includes biopsy-based investigations (histology, culture, urease test) and serology. Bleeding decreases the sensitivity of culture and urease tests, but histology is reliable irrespective of bleeding status and hence is a method of choice [36]. H. pylori testing in peptic ulcers was done on 296 patients during acute bleeding, with a diagnostic yield of 12.5% after repeat testing on those with initial negative results. Use of proton-pump inhibitors and antibiotics decreased the diagnostic yield [37].

6. Treatment and Management

Today's peptic ulcer treatment includes pharmacotherapy like PPIs and H2 receptor antagonists, as well as surgery for complicated peptic ulcers. New modalities such as new drugs, probiotics, and minimally invasive therapies are also being explored [38]. Treatment of peptic ulcers includes the eradication of Helicobacter pylori if identified, the administration of acid-suppressing drugs such as PPIs, and the management of complications using early endoscopy. PPI prophylaxis is indicated for high-risk patients to avoid the recurrence of ulcers [39]. Existing management of peptic ulcers involves pharmacological interventions such as PPIs and H2 receptor antagonists, in addition to surgical procedures in complicated cases. New modalities in the form of new drugs, probiotics, and minimal procedures are also under investigation [40]. Modern management of peptic ulcers is covered in pharmacological interventions such as proton pump inhibitors (PPIs) and H2 receptor antagonists, as well as surgical procedures for complications. New modalities encompass new drugs, probiotics, and minimal invasive therapies, which are improving overall management techniques [41]. Peptic ulcer treatment has changed to include suppression of gastric acid with H2-receptor antagonists and proton pump inhibitors, as well as eradication of Helicobacter pylori infection with antibiotics, markedly decreasing complications and the necessity for surgery [42].

6.1 Drugs (Antibiotics, Acid Suppressants)

Peptic ulcer drugs include H2 blockers, anticholinergic, antacids, prostaglandin analogs, ulcer protectants, and PPIs. Potassium-competitive acid blockers (P-CABs) have recently been identified as superior substitutes for PPIs in the management of such conditions [43]. The first-line peptic ulcer disease drugs are proton pump inhibitors, amoxicillin, and clarithromycin as a routine triple therapy for the eradication of H. pylori. In resistant situations, anti-secretory drugs and selective COX-2 inhibitors are also used.[44]. Proton pump inhibitors are important in the treatment of peptic ulcers, with relief from symptoms and healing within 4 to 6 weeks. Furthermore, H. pylori eradication is generally achieved with a triple therapy of a proton pump inhibitor with two antibiotics for 7 to 14 days [45].

6.2 Lifestyle Modification

Lifestyle modifications for peptic ulcers include a balanced diet, exercise regularly, effective management of stress, avoiding smoking, alcohol in moderation, adequate sleep, and reduction in the use of analgesic drugs and glucocorticoids to improve healing and prevention [46]. Lifestyle changes for peptic ulcers involve dietary modifications, stress reduction strategies, and avoidance of smoking and alcohol use. These are all part of ulcer management, healing and preventing recurrence in addition to medical therapies such as proton pump inhibitors and antibiotics [47].

6.3 Surgical Management

Surgical management of peptic ulcers is necessary, especially in perforated ulcers. Despite a bias towards conservative management, an understanding of the impact of Helicobacter pylori eradication on surgical outcomes is crucial for maximizing treatment and reducing complications and recurrence rates [48]. Surgical treatment of peptic ulcers now is mainly for complications such as bleeding, perforation, and gastric outlet obstruction. Elective surgery is mostly forsaken, with endoscopic interventions being the preferred choice. Laparoscopic methods have no notable benefit over open surgery for these conditions [49].

6.4 Endoscopic Therapy

Endoscopic management of peptic ulcer disease includes injection therapies, thermal therapies, mechanical clips, hemostatic sprays, and endoscopic suturing. It is limited to Forrest 1a, 1b, and 2a lesions, whereas Forrest 2b lesions are managed with proton-pump inhibitors alone [50]. PUD is still one of the leading causes of upper gastrointestinal bleeding, which is an emergency condition with potential for life-threatening complications. While medical treatment including PPIs and eradicating Helicobacter pylori has revolutionized the long-term management of PUD, endoscopic therapy is still the mainstay for acute management of ulcer-related bleeding, particularly in patients presenting with high-risk stigmata such as active bleeding or visible vessels [51].

7. Complications and Follow-Up

Complications of peptic ulcer disease include dumping syndrome, diarrhea, stasis of the stomach, bile reflux, and Roux syndrome. These arise as a result of surgical management methods more common before the advent of effective medical management like PPIs and histamine-2 (H2) blockers. Follow-up care of PUD is typically observation of symptoms, monitoring of the effectiveness of medical therapy, and early detection of complications to prevent future health issues [52].

7.1 Bleeding and Perforation

Bleeding and perforation are the serious complications of peptic ulcers, most often presenting as acute abdominal pain. Diagnosis is confirmed by X-ray and CT scans, while minimally invasive surgery is increasingly preferred for treatment in suitable cases. [53]. Upper gastrointestinal bleeding due to peptic ulcers is the most common complication, and it occurs in about 50% of cases of non-variceal UGIB. It usually follows erosion into a submucosal artery and can present with hematemesis, melena, or hematochezia. Risk factors for bleeding from ulcers are Helicobacter pylori infection, NSAID use, anticoagulation, and corticosteroids [54]. Perforated peptic ulcers result when the ulcer perforates the entire thickness of the gastric or duodenal wall, resulting in gastric contents spilling into the peritoneal cavity. This causes chemical peritonitis and possible evolution to sepsis or septic shock. It is characterized clinically by acute, severe pain in the abdomen, frequently with peritonitis signs, and free air on imaging [55].

7.2 Obstruction and Stricture

Pyloric stricture and gastric outlet obstruction are peptic ulcer complications that might have to be managed, as treatment is challenging by conservative methods, with endoscopic procedures like balloon dilation and stenting emerging as modes of management [56]. Gastric outlet obstruction is a late and less frequent complication of PUD, representing 10–20% of all PUD complications in the pre-H. pylori treatment era, but still applicable in regions with limited access to healthcare. It is caused by edema, inflammation, or fibrotic stricture at the pylorus or duodenum that obstructs gastric emptying. Clinically, patients complain of nausea, vomiting of unaltered food, early satiety, weight loss, and abdominal distension [57].

7.3 Recurrence and Prevention

Peptic ulcer recurrence is influenced by the consumption of NSAIDs and Helicobacter pylori infection that disrupt mucosal resistance. Treatments like proton pump inhibitors (PPIs) and histamine-2 (H2) receptor antagonists have relapses and side effects. Prevention of peptic ulcers is advisable through the application of medicinal plants and their chemical structures, which have potential properties of treatment and prevention, and even reducing recurrence risks [58]. Recurrence of peptic ulcers is seen in as many as 10–20% of patients within a year, particularly in patients with untreated or chronic H. pylori infection, ongoing use of NSAIDs, or the presence of comorbidities. Despite apparent healing, failure to remove these pathogenic factors could result in re-ulceration, which is frequently followed by complications like bleeding or obstruction [59].

8. Special Consideration

Special caution in peptic ulcers includes the management of antiplatelet and antithrombotic drug-treated patients and the selection of appropriate primary and secondary prophylaxis. Most guidelines recommend proton pump inhibitors in patients at increased risk of developing complications or ulcers [60]. A specific note for peptic ulcers is NSAID-induced ulcer treatment by withdrawal of NSAIDs and the administration of anti-ulcer medications and prevention with PPI or VPZ in patients with a history of ulcers receiving NSAID or LDA therapy [61].

8.1 Peptic Ulcer Disease in Pregnancy

Peptic ulcer disease during pregnancy can occur with violent vomiting and epigastric pain. In one case report, a pregnant patient had duodenal ulceration, which responded well to treatment with omeprazole and H. pylori eradication, leading to symptom resolution and ulcer healing [62]. PUD can be described as a disruption in the duodenal or gastric mucosa brought about by a mismatch between the damaging factors like gastric acid and pepsin and the mucosal protective mechanisms. The two most prevalent etiologies are Helicobacter pylori infection and the long-term administration of NSAIDs. While PUD is fairly prevalent in the nonpregnant population, it is rare in pregnancy, with a projected incidence of 1 in 1,500 to 8,000 pregnancies [63].

8.2 Peptic Ulcer Disease in Children

Peptic ulcer disease in children is rare and was present in 0.98% of 6216 children in one study. They typically come with abdominal pain, and H. pylori was found in 20% of the cases without clinical variability [64]. PUD in children is defined as mucosal erosion of 0.5 cm or more in the stomach or duodenal lining due to an imbalance between aggressive factors like gastric acid and pepsin and mucosal defenses. Though less frequent than in adults, PUD can be found in children and is usually considered to be either primary or secondary. The primary ulcers are most commonly seen in Helicobacter pylori infection, whereas the secondary ulcers are most often connected with stress, serious illness, or intake of nonsteroidal anti-inflammatory drugs [65].

8.3 Peptic Ulcer Disease in the Elderly

Older patients with peptic ulcer disease may present with recurrent upper GI bleeding, like in an 87-year-old patient. Management complications are due to comorbidities, and multidisciplinary management is needed with early intervention and careful monitoring to prevent complications and mortality [66]. PUD as a mucosal break in the stomach or duodenum because of the corrosive action of gastric acid and pepsin is still a major health issue among the elderly. Despite the general decrease in incidence of PUD with the widespread application of PPIs and Helicobacter pylori eradication, disease burden among elderly people persists because of age-related physiological alterations and greater exposure to risk factors [67].

9. Future Directions and Emerging Trends

Avenues of future peptic ulcer research include finding novel therapeutic targets, enhancing diagnostic testing, and probing the function of gut microbiota in peptic ulcer pathology. Future tendencies are new medicines, probiotics, and reduced invasive procedures to manage them [68]. Current directions for treating peptic ulcers include future directions such as personalized medicine, new pharmacologic agents, and integrative strategies for treatment. Research should tackle challenges like antibiotic resistance and optimize individualized therapy to ensure enhanced patient outcomes and reduce gastric ulcer global burden [69]. As healthcare institutions struggle to contain ulcer-related morbidity and admissions, the combined power of precision medicine, digital health technologies, and worldwide antimicrobial stewardship will be integral in managing the ever-changing threat of PUD [70].

Conclusion

The conclusion for peptic ulcer disease is that healthy living, proper nutrition, and home remedies are effective treatments. Allopathic drugs, particularly antacids with proton pump inhibitors, also play an essential role in managing the condition. The study found that peptic ulcer disease negatively impacted patients' quality of life, moderated by environmental triggers and ulcer symptom-associated symptoms regardless of whether the majority reported insignificant effects on their physical activities as part of daily activities and merely a few reported poor overall health.

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Author Contributions

S.S. Conceptualized the study, **G.S.** Supervised the review, **T.S.** Prepared the manuscript draft**, S.H.M.** Data collection.

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Conflicts of Interest

No conflicts of interest are disclosed by the authors.

References

1. Zavhorodnia, A. (2023). Assessment And Examination of Information Concernin Individuals Affected by Ulcer Disease. Collection of scientific papers «ΛΌГОΣ», (December 22, 2023; Boston, USA), 346-347.
2. Pujari, N. M., Khushtar, M., Mishra, A., & Gupta, D. (2024). Comprehensive Insights into Gastric Ulcer Therapies: A Review of Current Approaches. Journal of Applied Pharmaceutical Sciences and Research, 7(1), 4-12.
3. Maisuria, P., Joshi, A., Saravaiya, S., Gajjar, S., Pateld, H., Pandyad, A., & Narkhede, S. Design, Development and Evaluation of a Polyherbal Floating Tablet for Gastric Ulcer.
4. Naik, A., Shankar, M., Sowjanya, R., Sireesha, B., & Yudharaj, P. (2016). Methods on employed in screening of antiulcer drugs-an overview. International Journal of Novel Trends in Pharmaceutical Sciences, 6(5), 111-119.
5. Sapkota, S., Oli, S. S., Karki, M., Rokaha, P. S., & Aryal, A. (2024). Peptic Ulcer Disease among Patients Undergoing Upper Gastrointestinal Endoscopy in a Tertiary Care Centre: A Descriptive Cross-sectional Study. JNMA: Journal of the Nepal Medical Association, 62(272), 226.
6. Srivastav, Y., Mansoori, M. F., Hameed, A., Hashmi, A., & Ahmad, M. I. Condensed Statement: Critical Framework of the Diagnosis along with Management of Peptic Ulcer Disease (PUD).
7. Nolan, D. J., & Traill, Z. C. (1997). The current role of the barium examination of the small intestine. Clinical radiology, 52(11), 809-820.
8. HANSEL, S. L. (2014). Peptic Ulcer Disease. Mayo Clinic Gastroenterology and Hepatology Board Review, 43.
9. Debas, H. T. (2004). Gastrointestinal surgery: pathophysiology and management. Springer Science & Business Media.
10. Mondal, P., Banerjee, D., Ghorai, P., Debnath, S., & Pal, N. Natural Remedies in the Treatment of Peptic Ulcer: A.

11. Agrawal, R. (2024). Pathophysiology, Risk Factors, and Advances in the Management of Peptic Ulcers: A Comprehensive Review.
12. Arushi, C. S., Chaudhary, S., Devi, M., Sharma, G., & Sharma, K. Gastro protective Mechanisms and its Augmentation by External Agents-A Review.
13. Agrawal, R. K. (2025). African Research Journal of Medical Sciences.
14. Maisuria, P., Joshi, A., Saravaiya, S., Gajjar, S., Pateld, H., Pandyad, A., & Narkhede, S. Design, Development and Evaluation of a Polyherbal Floating Tablet for Gastric.
15. GA, O. Q., Woolf, A., & Haddad, L. M. (2021). Duodenal ulcer (nursing).
16. Chinecherem, O. A., Ibiam, U. C., Chidiebere, U., & Chinedu, U. N. (2024). KNOWLEDGE, PREVALENCE AND RISK FACTORS OF EATING DISORDERS AND PEPTIC ULCER DISEASE AMONG CLINICAL STUDENTS AT THE UNIVERSITY OF NIGERIA TEACHING HOSPITAL, ITUKU-OZALLA, ENUGU. medRxiv, 2024-12.
17. Wirostko, B., & Christiansen, S. M. (2013). Causes and risk factors. Glaucoma: Basic and Clinical Perspectives, 19.
18. Bhattacharjee, A., Sahoo, O. S., Sarkar, A., Bhattacharya, S., Chowdhury, R., Kar, S., & Mukherjee, O. (2024). Infiltration to infection: key virulence players of Helicobacter pylori pathogenicity. Infection, 52(2), 345-384.
19. Arfijanto, M. V., Hadi, U., & Yoes Prijatna Dachlan, Y. (2020). Procalcitonin, IL-1ß, HSP10 and Resolvin D2 Mechanis as Sepsis Biomarkers in Sepsis Model. International Journal of Pharmaceutical Research, 12(4), 1388-1394.
20. Adunmo, G. O., Adeniyi, T. D., Olorunfemi, O. A., Adunmo, E. O., Stephen, D. A., & Ajayi, A. J. (2024). EFFECT OF ALCOHOL INTAKE AND CIGARETTE SMOKING ON LIPID PROFILE LEVELS AND SELECTED INFLAMMATORY BIOMARKERS. Era's Journal of Medical Research, 11(2).
21. y Revuelta, J. G. D. R. (1995). Stress and anxiety. Actas luso-espanolas de neurologia, psiquiatria y ciencias afines, 23(6), 310-313.
22. Maisuria, P., Joshi, A., Saravaiya, S., Gajjar, S., Pateld, H., Pandyad, A., & Narkhede, S. Design, Development and Evaluation of a Polyherbal Floating Tablet for Gastric Ulcer.
23. Maisuria, P., Joshi, A., Saravaiya, S., Gajjar, S., Pateld, H., Pandyad, A., & Narkhede, S. Design, Development and Evaluation of a Polyherbal Floating Tablet for Gastric Ulcer.
24. Trindade, M., Pinto, D. G., Carvalho, N., & Luz, C. (2025). Role of Intraoperative Biopsy and Postoperative Upper Gastrointestinal Endoscopy in Patients Undergoing Surgery for Perforated Gastric Ulcer: A Retrospective Study. Cureus, 17(1).
25. Metwally, M. A. E., Alfeky, H. M., Alegaily, H. S., Abbas, A. H., Abd Elmonem, A. M., & Gabal, H. M. R. (2023). Evaluation of two commercially available rapid stool antigen tests for the diagnosis of Helicobacter pylori infection. The Journal of Infection in Developing Countries, 17(05), 631-634.
26. Agrawal, R. K. (2025). African Research Journal of Medical Sciences.
27. Lakshmanan, M. (2021). Drugs used in acid peptic disorders. Introduction to Basics of Pharmacology and Toxicology: Volume 2: Essentials of Systemic Pharmacology: From Principles to Practice, 553-567.
28. Yegen, B. C. (2018). Lifestyle and peptic ulcer disease. Current pharmaceutical design, 24(18), 2034-2040.
29. Aljuhani, S. A., Sherwani, A. A., Alnamshah, F. O., Alaeq, R. A., Alrahma, H. A., Jarad, M. M., ... & Sahal, Y. (2024). Impact of Helicobacter pylori Eradication on Surgical Treatment of Peptic Ulcer Disease: Systematic Review. Cureus, 16(6).
30. Vakil, N. (2024). Endoscopic diagnosis, grading, and treatment of bleeding peptic ulcer disease. Gastrointestinal Endoscopy Clinics, 34(2), 217-229..
31. Dickler, C. J., & Spaniolas, K. (2019). Complications of Peptic Ulcer Disease. Clinical Algorithms in General Surgery: A Practical Guide, 157-160.
32. Danilo, C. O. C. O., & Leanza, S. (2022). A review on treatment of perforated peptic ulcer by minimally invasive techniques. Maedica, 17(3), 692.
33. Shim, K. N. (2019). Endoscopic Management for Pyloric Stricture and Gastric Outlet Obstruction: Dilation and Stenting. Therapeutic Gastrointestinal Endoscopy: A Comprehensive Atlas, 277-289.
34. Salman, A., Dhaneshwar, S., & Shafiulla, S. (2023). Post COVID-19: risk factors, prevention, and management of black fungus. Anti-Infective Agents, 21(1), 39-53.
35. Almadi, M. A., Lu, Y., Alali, A. A., & Barkun, A. N. (2024). Peptic ulcer disease. The Lancet, 404(10447), 68-81.
36. Satoh, K., Yoshino, J., Akamatsu, T., Itoh, T., Kato, M., Kamada, T., ... & Shimosegawa, T. (2016). Evidence-based clinical practice guidelines for peptic ulcer disease 2015. Journal of gastroenterology, 51, 177-194.
37. Frise, C. J., & Nelson-Piercy, C. (2012). Peptic ulcer disease in pregnancy. Journal of Obstetrics and Gynaecology, 32(8), 804-804
38. Güven, B., Sağ, E., Usta, D., İssi, F., & Çakır, M. (2020). Peptic ulcer disease in children. Mucosa, 3(1), 18-22.
39. Ahmad, H., Khan, U., Jannat, H., & Ahmad, N. (2023). Recurrent Gastrointestinal Bleeding in an Elderly Patient With Peptic Ulcer Disease: Successful Management Through Multidisciplinary Intervention and Close Monitoring. Cureus, 15(7).
40. Agrawal, R. (2024). Pathophysiology, Risk Factors, and Advances in the Management of Peptic Ulcers: A Comprehensive Review.
41. 41.. Pujari, N. M., Khushtar, M., Mishra, A., & Gupta, D. (2024). Comprehensive Insights into Gastric Ulcer Therapies: A Review of Current Approaches. Journal of Applied Pharmaceutical Sciences and Research, 7(1), 4-12.
42. Kulshreshtha, M., Srivastava, G., & Singh, M. P. (2017). Pathophysiological status and nutritional

therapy of peptic ulcer: An update. Environmental Disease, 2(3), 76-86.
43. Shanshal, S. A., Noori, A. S., Ghazi, J. A., Dahham, A. T., Saleh, A. S. M., & Al-Qazaz, H. K. (2022). Impact of peptic ulcer disease on the quality of life: A Cross-Sectional Study. Research Journal of Pharmacy and Technology, 15(7), 3267-3272.
44. Dworken, H. J. (1982). The Pathophysiology of Peptic Ulcer (pp. 129–172).
45. Chey, W. D., & Wong, B. C. Y. (2007). American College of Gastroenterology guideline on the management of Helicobacter pylori infection. The American Journal of Gastroenterology, 102(8), 1808–1825.
46. Chey, W. D., Leontiadis, G. I., Howden, C. W., & Moss, S. F. (2017). ACG Clinical Guideline: Treatment of Helicobacter pylori Infection. The American Journal of Gastroenterology, 112(2), 212–239.
47. Lanas, A., & Chan, F. K. L. (2017). Peptic ulcer disease. The Lancet, 390(10094), 613–624.
48. Chey, W. D., & Wong, B. C. Y. (2007). American College of Gastroenterology guideline on the management of Helicobacter pylori infection. The American Journal of Gastroenterology, 102(8), 1808–1825.
49. Lanas, A., & Chan, F. K. L. (2017). Peptic ulcer disease. The Lancet, 390(10094), 613–624.
50. Chey, W. D., & Wong, B. C. Y. (2007).
51. American College of Gastroenterology guideline on the management of Helicobacter pylori infection. The American Journal of Gastroenterology, 102(8), 1808–1825.
52. Chey, W. D., Leontiadis, G. I., Howden, C. W., & Moss, S. F. (2017). ACG Clinical Guideline: Treatment of Helicobacter pylori Infection. The American Journal of Gastroenterology, 112(2), 212–239.
53. Blaser, M. J., & Atherton, J. C. (2004). Helicobacter pylori persistence: biology and disease. The Journal of Clinical Investigation, 113(3), 321–333.
54. Vane, J. R., & Botting, R. M. (1998). Mechanism of action of nonsteroidal anti-inflammatory drugs. The American Journal of Medicine, 104(3A), 2S–8S.
55. World Health Organization (WHO). (2022). Tobacco. Retrieved from
56. Centers for Disease Control and Prevention (CDC). (2022). Health Effects of Cigarette Smoking. Retrieved from
57. Specialist, N. (2015). Examining the extent to which stress contributes to disease in the gastrointestinal tract: a literature review.
58. Bresnick, W. H., Rask-Madsen, C., Hogan, D. L., Koss, M. A., & Isenberg, J. I. (1993). The effect of acute emotional stress on gastric acid secretion in normal subjects and duodenal ulcer patients. Journal of Clinical Gastroenterology, 17(2), 117–122.
59. Ali, A. M., Mohamed, A. N., Mohamed, Y. G., & Keleşoğlu, S. İ. (2022). Clinical presentation and surgical management of perforated peptic ulcer in a tertiary hospital in Mogadishu, Somalia: a 5-year retrospective study. World Journal of Emergency Surgery, 17(1).
60. Almadi, M. A., Lu, Y., Alali, A., & Barkun, A. (2024). Peptic ulcer disease. The Lancet.
61. Werdmuller, B. F. M., van der Putten, A. B. M. M., & Loffeld, R. J. L. F. (1997). The clinical presentation of peptic ulcer disease. Netherlands Journal of Medicine, 50(3), 115–119>
62. Rehman, M., Khan, I. U., & Shah, M. (2011). Clinical Features and Endoscopic findings in Peptic Ulcer. Journal of Postgraduate Medical Institute, 12(1).
63. Salih, M. M. (2019). Histopathological Diagnosis of Peptic Ulcer Biopsies in Koya City, Kurdistan Region, Iraq. 9(2), 208–212.
64. Agrawal, R. (2024). Pathophysiology, Risk Factors, and Advances in the Management of Peptic Ulcers: A Comprehensive Review.
65. Mall, K. (1999). Duodenal ulcer with penetration into the liver. Endoscopic-biopsy diagnosis. Medizinische Klinik, 94(2), 101–104.
66. Moreno-Otero, R., Marrón, C., Cantero, J., Pajares, J. M., & Martinez-Raposo, A. (1989). Endoscopic biopsy and cytology in the diagnosis of malignant gastric ulcers. Diagnostic Cytopathology, 5(4), 366–370
67. Peptic Ulcer Disease and H. pylori Infection: Common Questions and Answers. (2023). 107(2), 165–172.
68. Nfongeh, J. F., Kabido, H. U., Pedro, A., Fadayomi, V. K., Ramalan, A. S., & Kpandem, Y. E. (2022). Comparative Assessment of Current Serological Methods against the Conventional in the Diagnosis of Helicobacter pylori Infections in Suspected Peptic Ulcer Patients Attending Health Facilities in Lafia, Nigeria. European Journal of Health Sciences, 7(3), 1–13.
69. Feder, R., Posner, S., Qin, Y., Zheng, J., Chow, S.-C., & Garman, K. S. (2018). Helicobacter pylori-associated peptic ulcer disease: A retrospective analysis of post-treatment testing practices. Helicobacter, 23(6).
70. Li Weiquan, J., & Jen Lock Khor, C. (2017). Testing And Treating Helicobacter Pylori Infection. The Singapore Family Physician, 43(2), 52–54.