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ORIGINAL ARTICLE

Clinical Impact of Helicobacter pylori Infection on Gastric Ulcer Severity and Symptom Burden: A Cross-Sectional Study

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ABSTRACT

Background: Gastric ulcer disease remains a significant cause of morbidity worldwide, particularly in regions where Helicobacter pylori infection is highly prevalent. The bacterium plays a central role in mucosal inflammation, ulcer formation, and progression to severe complications. Understanding how H. pylori influences ulcer severity and symptom burden is essential for optimizing diagnosis and clinical management.

Objective: To assess the clinical impact of H. pylori infection on the severity of gastric ulcers and the associated symptom burden among adults presenting with dyspepsia.

Methods: A cross-sectional study was conducted from June 2022 to July 2023 in the Medicine Department of Shaheed Mohtarma Benazir Bhutto General Hospital, Quetta, and Aziz Bhatti Shaheed Teaching Hospital, Gujrat, Pakistan. A total of 100 patients with endoscopically confirmed gastric ulcers were enrolled. H. pylori infection was identified using rapid urease testing, with histopathological confirmation in equivocal cases. Ulcer severity was classified using the Endoscopic Ulcer Severity Scale, and symptom burden was assessed through structured clinical evaluation. Statistical analyses were performed using SPSS version 26.

Results: Of the 100 patients, 64% were H. pylori positive. Severe ulcers were significantly more common among infected individuals (46.9%) compared with non-infected patients (19.4%) ($p = 0.003$). H. pylori-positive patients also exhibited a higher symptom burden, including intense epigastric pain, nocturnal pain, nausea, vomiting, early satiety, and upper gastrointestinal bleeding (all $p < 0.05$). The findings indicate a strong association between H. pylori infection and both advanced ulcer severity and worsened clinical presentation.

Conclusion: H. pylori infection is strongly associated with more severe gastric ulceration and a substantially higher symptom burden. Early diagnosis and prompt eradication therapy are critical for preventing complications, reducing symptom intensity, and improving overall patient outcomes in high-prevalence regions.

Keywords: Helicobacter pylori; gastric ulcer; ulcer severity; dyspepsia; gastrointestinal symptoms; symptom burden.

INTRODUCTION

Gastric ulcer disease continues to be a significant global health challenge, contributing to considerable morbidity, reduced quality of life, and recurrent healthcare utilization[1]. Although multiple etiological factors such as non-steroidal anti-inflammatory drug (NSAID) use, smoking, stress, and comorbid systemic illnesses have been implicated in ulcer development, the discovery of Helicobacter pylori (H. pylori) fundamentally reshaped the understanding of gastric ulcer pathophysiology. H. pylori, a spiral-shaped, microaerophilic Gram-negative bacterium, colonizes the gastric mucosa and induces chronic active gastritis, mucosal barrier disruption, epithelial injury, and ultimately ulcer formation[2,3].

Despite improvements in sanitation and antibiotic availability, H. pylori prevalence remains markedly high in many low- and middle-income countries, including Pakistan, where environmental, dietary, socioeconomic, and household transmission factors facilitate persistent spread[4]. In such populations, H. pylori infection serves as a major driver of gastric ulcer severity, increasing the risk of deeper mucosal penetration, recurrent ulceration, gastrointestinal bleeding, perforation, and other complications. Moreover, the inflammatory cascade triggered by the bacterium including cytokine release, oxidative stress, and altered gastric acid secretion amplifies the clinical symptoms experienced by patients, such as epigastric pain, nocturnal discomfort, nausea, vomiting, early satiety, and abdominal bloating[5,6].

Although international studies have consistently demonstrated strong links between H. pylori infection and ulcer disease, local evidence remains limited, particularly in the context of how infection status correlates with the depth of ulceration and the associated symptom burden in Pakistani adults[7]. Understanding these associations is essential for improving diagnostic strategies, guiding eradication therapy, reducing recurrence, and preventing severe complications. Early detection of H. pylori is especially important in high-prevalence regions, where delayed diagnosis can significantly worsen clinical outcomes[8].

Given these gaps, this study was designed to investigate the clinical impact of H. pylori infection on gastric ulcer severity and symptom burden among adults presenting with dyspeptic complaints. By analyzing endoscopic findings, symptom patterns, and infection status, the study aims to provide evidence-based insights that may support more targeted diagnostic and therapeutic approaches in routine clinical practice[9,10].

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MATERIALS AND METHODS

Study Design and Duration: This cross-sectional analytical study was conducted over a fourteen-month period from June 2022 to July 2023. The study aimed to evaluate the clinical impact of Helicobacter pylori infection on gastric ulcer severity and symptom burden among adult patients presenting with dyspepsia. The design allowed for the simultaneous assessment of exposure status (H. pylori positivity) and disease characteristics at the time of presentation.

Study Settings: The study was carried out in the Medicine Department of Shaheed Mohtarma Benazir Bhutto General Hospital in Quetta and Aziz Bhatti Shaheed Teaching Hospital in Gujrat, Pakistan. Both institutions are high-volume tertiary care centers serving diverse populations, with well-established diagnostic endoscopy facilities. These characteristics made them suitable for recruiting a representative sample of patients with suspected gastric ulcer disease.

Sample Size and Sampling Method: A total of 100 patients were included in the study. Participants were recruited through non-probability consecutive sampling, where every eligible patient presenting during the study period and diagnosed with a gastric ulcer on endoscopy was enrolled until the desired sample size was

achieved. This approach ensured practical feasibility while maintaining adequate sample representation.

Inclusion and Exclusion Criteria: Adults between 18 and 70 years of age presenting with upper gastrointestinal symptoms and subsequently confirmed to have a gastric ulcer on upper GI endoscopy were included. Only those patients who consented to participate and agreed to undergo H. pylori testing were enrolled. Patients were excluded if they had used non-steroidal anti-inflammatory drugs within the preceding four weeks, had received prior H. pylori eradication therapy, had undergone gastric surgery, were suspected of having gastric malignancy, or had severe systemic comorbidities that posed a contraindication to endoscopy. Pregnant and lactating women were also excluded.

Clinical Evaluation: Each patient underwent a detailed clinical assessment conducted by trained medical personnel. Information was recorded regarding the duration, character, and severity of epigastric pain, presence of nocturnal pain, early satiety, abdominal bloating, nausea, vomiting, hematemesis, melena, and other dyspeptic symptoms. Relevant clinical history including smoking, dietary habits, comorbidities, and previous gastrointestinal disorders was documented. A structured symptom assessment form was used to ensure consistency across both study sites.

Endoscopic Assessment: Upper gastrointestinal endoscopy was performed by senior gastroenterologists at both hospitals using standard protocols. During the procedure, the anatomical location of each gastric ulcer was noted, along with its size, depth, and the presence of stigmata of recent or active bleeding. Surrounding mucosal changes such as erythema, edema, erosions, and signs of chronic gastritis were documented. Ulcer severity was classified according to the Endoscopic Ulcer Severity Scale (EUSS), which categorizes lesions into mild, moderate, or severe based on morphological appearance, depth, and risk features.

Detection of H. pylori Infection: Determination of H. pylori infection was performed using the rapid urease test (RUT) as the primary diagnostic method. During endoscopy, biopsy samples were obtained from the antrum and body of the stomach and placed in urease reagent. A color change within fifteen minutes to two hours was considered indicative of a positive test. In cases where results were equivocal or inconsistent with clinical findings, additional biopsy samples were subjected to histopathological examination using hematoxylin–eosin and modified Giemsa staining to confirm the presence of H. pylori organisms.

Data Collection Procedure: All relevant demographic, clinical, endoscopic, and laboratory findings were recorded on a structured proforma developed specifically for this study. Data collection was supervised by consultant physicians to ensure accuracy and uniformity. The symptom burden, endoscopic findings, and H. pylori status were entered promptly after each patient encounter to minimize recall and transcription errors.

Data Analysis: Data were processed and analyzed using SPSS version 26. Continuous variables such as age were expressed as mean ± standard deviation and compared using the independent t-test. Categorical variables such as H. pylori status, ulcer severity categories, and symptom presence were analyzed using the Chi-square test. A p-value of less than 0.05 was considered statistically significant for all analyses, and results were presented using appropriate statistical measures.

Ethical Considerations: Ethical approval was obtained from the institutional review committees of both participating hospitals before initiation of the study. All participants provided written informed consent after a complete explanation of the study objectives and procedures. Confidentiality of personal and medical information was strictly maintained, and all procedures were conducted in accordance with the principles of the Declaration of Helsinki.

RESULTS

A total of 100 patients with endoscopically confirmed gastric ulcers were included in the study. Among these patients, 56% were male and 44% were female, with a mean age of 42.8 ± 10.2 years. The prevalence of Helicobacter pylori infection in the study population was 64%, while 36% tested negative. The baseline characteristics were similar across both groups, with no statistically significant difference in age or gender distribution, indicating comparability between infected and non-infected individuals.

Assessment of ulcer severity revealed a marked difference between the two groups. As shown in Table 1, severe gastric ulcers were significantly more common among H. pylori-positive patients. Nearly half of the infected individuals (46.9%) presented with severe ulcers compared to only 19.4% of the H. pylori-negative patients. Mild ulcers were substantially more frequent in the H. pylori-negative group. The difference in ulcer severity across groups was statistically significant (p = 0.003), establishing a strong association between H. pylori infection and more advanced ulceration.

Table 1. Distribution of Ulcer Severity by H. pylori Status

Ulcer Severity	H. pylori Positive (n=64)	H. pylori Negative (n=36)	p-value
Mild	12 (18.8%)	14 (38.8%)	0.003
Moderate	22 (34.4%)	15 (41.8%)	
Severe	30 (46.9%)	7 (19.4%)	

Table 2. Comparison of Symptom Burden Between H. pylori Positive and Negative Patients

Symptom	H. pylori Positive (n=64)	H. pylori Negative (n=36)	p-value
Intense epigastric pain	51 (79.7%)	18 (50.0%)	0.004
Nocturnal pain	39 (60.9%)	12 (33.3%)	0.01
Nausea/Vomiting	42 (65.6%)	14 (38.9%)	0.02
Early satiety	36 (56.2%)	12 (33.3%)	0.03
GI bleeding	19 (29.7%)	4 (11.1%)	0.01

Table 1 demonstrates the significant difference in ulcer severity between infected and non-infected patients, with severe ulcers predominantly associated with H. pylori positivity.

Regarding symptom burden, significant differences were observed across multiple clinical parameters. Patients with H. pylori infection experienced more intense epigastric pain, higher frequency of nocturnal pain, and increased occurrences of nausea, vomiting, and early satiety. Gastrointestinal bleeding events including hematemesis and melena were notably more common in the infected group. As illustrated in Table 2, the proportion of patients experiencing these symptoms was consistently higher among those who were H. pylori positive, with several differences reaching statistical significance.

Table 2 highlights the significantly higher symptom burden among H. pylori-positive patients, particularly regarding pain severity and gastrointestinal bleeding.

Overall, the results of this study indicate a clear and statistically meaningful relationship between H. pylori infection and both the severity of gastric ulceration and the intensity of clinical symptoms. The infected group consistently demonstrated more advanced endoscopic findings and heavier symptom burden compared to those without infection.

DISCUSSION

The findings of this cross-sectional study demonstrate a significant association between Helicobacter pylori infection and both the severity of gastric ulcers as well as the overall symptom burden among adults presenting with dyspepsia[11]. The prevalence of H. pylori infection in the study population was high, consistent with previous reports from Pakistan and other developing countries, where socioeconomic conditions, household crowding, and sanitation factors contribute to persistent transmission. This high prevalence reflects a continuing public health challenge, particularly in areas where screening and eradication programs remain limited[12].

A major observation of the study was the markedly greater severity of gastric ulcers in H. pylori-positive individuals[13]. Nearly

half of the infected patients presented with severe ulcers, compared to fewer than one-fifth of the non-infected group. This disparity reinforces the established understanding that H. pylori induces chronic mucosal inflammation, disrupts epithelial integrity, impairs bicarbonate secretion, and increases vulnerability to deeper ulceration[14]. The cytotoxin-associated gene A (CagA) and vacuolating cytotoxin A (VacA) virulence factors stimulate pro-inflammatory cascades and oxidative injury, contributing to the progression from superficial mucosal inflammation to ulcer formation and expansion. The present findings are consistent with regional and international studies that have identified H. pylori as a major driver of ulcer depth and complication risk[15].

The study also showed that the symptomatic presentation of gastric ulcer disease was substantially more severe among infected individuals. Higher rates of intense epigastric pain, nocturnal pain, nausea, vomiting, early satiety, and dyspepsia were recorded in the H. pylori-positive group[16]. These clinical manifestations are likely attributable to the persistent inflammatory response generated by the bacterium, which leads to impaired gastric motility, increased sensitivity of gastric nociceptors, and altered acid secretion. Moreover, infected patients demonstrated a significantly higher frequency of gastrointestinal bleeding, which represents a major clinical complication with potentially life-threatening consequences. This association aligns with previous literature highlighting that H. pylori-related ulcers exhibit friable mucosa, increased vascular fragility, and impaired mucosal healing capacity[17].

The results of this study have important clinical implications. In high-prevalence settings, early testing and treatment of H. pylori should be regarded as essential components of gastric ulcer management[18]. Failure to diagnose and eradicate the infection may lead to recurrent ulceration, chronic symptoms, and heightened risk of complications such as bleeding and perforation. Furthermore, prompt identification of H. pylori status may reduce unnecessary long-term use of acid-suppressive therapy and allow clinicians to adopt curative rather than palliative treatment strategies. Endoscopic evaluation remains crucial in distinguishing ulcer severity and confirming mucosal pathology, but integration of non-invasive testing and primary-care–based screening may help expand early detection efforts in resource-limited environments[19].

While the present study provides valuable insight into the relationship between H. pylori status and clinical severity of gastric ulcers, certain limitations should be considered. The cross-sectional design limits the ability to infer causality, although the biological plausibility strongly supports the observed associations[20]. The study was conducted in two hospitals, which may not fully reflect national prevalence patterns. Additionally, the diagnosis of H. pylori relied primarily on rapid urease testing, although histopathology was used in equivocal cases to strengthen diagnostic accuracy. Despite these limitations, the study adds important evidence to the regional literature and highlights the need for broader community-based screening programs and more robust eradication strategies[17-20].

CONCLUSION

This study demonstrates that Helicobacter pylori infection significantly influences both the severity of gastric ulcers and the intensity of clinical symptoms experienced by affected individuals. Infected patients were more likely to present with severe ulceration, heightened pain, increased nocturnal discomfort, more frequent gastrointestinal symptoms, and a substantially greater risk of bleeding. These findings underscore the critical importance of early detection and eradication of H. pylori as an integral part of gastric ulcer management, particularly in regions with high infection prevalence. Timely diagnosis, appropriate antibiotic therapy, and close clinical follow-up can substantially improve patient outcomes, prevent complications, and reduce the overall burden of gastric ulcer disease. Future research should focus on larger multicenter studies, long-term follow-up of eradication outcomes, and evaluation of population-level screening strategies to optimize ulcer prevention and management in Pakistan and similar high-prevalence regions.

Authors' Contributions: W.B. conceived the study idea, supervised data collection, and contributed to manuscript drafting. S.A. performed clinical evaluations, assisted in data interpretation, and reviewed the final document. R.A. contributed to patient recruitment, clinical assessment, and manuscript editing. S.K. assisted with endoscopic evaluation, data accuracy, and critical revision of the manuscript. J.H. performed diagnostic assessments, contributed to analysis, and reviewed the discussion section. A.A.K. contributed to data acquisition, literature review, and manuscript formatting. All authors approved the final version of the manuscript.

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