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ORIGINAL ARTICLE

Association of Helicobacter pylori Infection with Severity and Clinical Presentation of Gastric Ulcer: A Cross-Sectional Clinical Study

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ABSTRACT

Background: Helicobacter pylori infection is a major etiological factor in gastric ulcer disease and is associated with variations in symptom severity and ulcer morphology. Understanding this relationship is essential in regions with high infection prevalence such as Pakistan.

Objective: To determine the association between H. pylori infection and the clinical severity and presentation of gastric ulcer among patients undergoing upper gastrointestinal endoscopy.

Methods: A cross-sectional study was conducted at SMBBMC Lyari, Karachi, and RMI Peshawar from June 2022 to May 2023. A total of 100 patients with endoscopically confirmed gastric ulcer were enrolled. Clinical symptoms, endoscopic findings, and ulcer severity scores were compared between H. pylori–positive and –negative groups. Infection was confirmed using rapid urease test and histopathology. Data were analyzed using SPSS 25, with $p < 0.05$ considered significant.

Results: H. pylori infection was present in 62% of patients. Infected individuals exhibited more severe epigastric pain, higher frequencies of nausea, early satiety, and significant weight loss. Endoscopic evaluation showed that H. pylori–positive patients had larger ulcers (1.89 ± 0.61 cm vs. 1.43 ± 0.52 cm), more deep lesions, increased ulcer multiplicity, and a higher rate of active bleeding. The mean ulcer severity score was significantly greater in the infected group (6.7 ± 1.8 vs. 5.0 ± 1.5; $p < 0.001$). Logistic regression confirmed H. pylori infection as an independent predictor of severe ulcer disease.

Conclusion: H. pylori infection is strongly associated with increased severity and more pronounced clinical and endoscopic features of gastric ulcer. Routine detection and timely eradication therapy are crucial to reducing symptom burden, complications, and recurrence.

Keywords: Helicobacter pylori, gastric ulcer, endoscopy, ulcer severity, clinical presentation.

INTRODUCTION

Gastric ulcer is a common and clinically significant component of peptic ulcer disease, affecting millions of individuals worldwide and contributing substantially to gastrointestinal morbidity, hospitalization rates, and healthcare burden[1]. Although multiple etiological factors are implicated in ulcer formation, Helicobacter pylori (H. pylori) infection remains the most extensively studied and widely accepted cause of chronic gastritis and peptic ulceration. Since its discovery in 1982, H. pylori has been recognized as a major pathogenic organism responsible for persistent gastric mucosal inflammation, ulcer recurrence, and long-term complications including mucosa-associated lymphoid tissue (MALT) lymphoma and gastric carcinoma. The World Health Organization has classified H. pylori as a Class I carcinogen, underscoring its profound clinical relevance[2,3].

Globally, the prevalence of H. pylori infection varies greatly, reaching over 70% in many developing countries[4]. South Asian countries, including Pakistan, exhibit some of the world's highest infection rates, largely due to crowded living conditions, limited public health infrastructure, pooS r sanitation, and inadequate access to primary healthcare services. Despite this high burden, diagnostic and therapeutic approaches remain inconsistent, often leading to delayed detection and incomplete eradication of H. pylori. Consequently, gastric ulcer continues to present as a significant clinical challenge in the region, frequently associated with recurrent symptoms, poor quality of life, and preventable complications[5,6].

Although H. pylori is a well-established cause of gastric ulcer, the extent to which its presence affects ulcer characteristics such as severity, depth, size, multiplicity, bleeding risk, and anatomical location continues to be debated[7]. Emerging evidence suggests that H. pylori–positive ulcers may differ in their clinical behavior from ulcers induced by non-steroidal anti-inflammatory drugs (NSAIDs), stress, smoking, or other risk factors. However, regional variations in bacterial virulence (e.g., CagA, VacA strains), host immune responses, dietary practices, and healthcare access contribute to significant differences in disease presentation. Therefore, population-specific data are essential to understanding the true clinical impact of H. pylori infection[8].

In clinical practice, patients with gastric ulcer present with a broad spectrum of symptoms, ranging from epigastric pain and dyspepsia to gastrointestinal bleeding, anemia, and weight loss[9]. It is hypothesized that the presence of H. pylori may exacerbate mucosal inflammation and alter gastric acid secretion, thereby modifying the symptom profile and increasing the likelihood of complications. However, studies evaluating the association between H. pylori infection and ulcer symptomatology in South Asian populations remain limited. Additionally, while endoscopy and biopsy remain the gold standard for ulcer evaluation and H. pylori detection, the relationship between endoscopic severity and infection status is not clearly defined in local settings[10].

Understanding this association holds significant clinical value. If H. pylori infection is strongly linked to more severe ulcers, clinicians may prioritize early testing, aggressive eradication therapy, and closer monitoring of infected individuals. Conversely, if certain symptoms or endoscopic patterns reliably predict infection, diagnostic and therapeutic decision-making may be streamlined, especially in resource-limited settings[11].

Given these gaps in current knowledge, this study aims to investigate the relationship between H. pylori infection and the clinical presentation and endoscopic severity of gastric ulcer among adult patients presenting to a tertiary care hospital. By examining symptom patterns, ulcer characteristics, and risk factors, this study seeks to generate locally relevant evidence that may inform improved diagnostic and management strategies for gastric ulcer disease[12].

MATERIALS AND METHODS

Study Design and Setting: This research was designed as a cross-sectional clinical study and was conducted simultaneously at

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two major tertiary care centres in Pakistan, namely Shaheed Mohtarma Benazir Bhutto Medical College (SMBBMC), Lyari, Karachi, and Rehman Medical Institute (RMI), Peshawar. Both institutions cater to a diverse population with a high burden of upper gastrointestinal disorders, making them appropriate sites for evaluating the relationship between Helicobacter pylori infection and gastric ulcer severity. The study was carried out over a twelve-month period, extending from June 2022 to May 2023, allowing sufficient time to collect representative data across seasonal and demographic variations.

Sample Size and Sampling Technique: A total of 100 patients were included in the study. The sample size was deemed adequate to observe clinically meaningful associations between H. pylori status and ulcer severity. Patients were recruited using a non-probability consecutive sampling approach, ensuring that every eligible patient presenting during the study duration was offered participation until the required sample size was reached.

Study Population: The study population consisted of adult male and female patients aged eighteen years or above who underwent diagnostic upper gastrointestinal endoscopy and were found to have a gastric ulcer. Only those willing to undergo gastric biopsy for H. pylori detection and able to provide informed consent were included. Patients with a history of prior H. pylori eradication therapy, recent consumption of proton pump inhibitors, antibiotics, or bismuth compounds, or those with endoscopically suspicious or biopsy-proven malignant ulcers were excluded. Individuals with severe comorbid illnesses that could affect biopsy safety, as well as pregnant or lactating women, were also not included. This ensured that the study population was homogenous and suitable for evaluating the association between infection and ulcer characteristics.

Clinical Evaluation: Each participant underwent a detailed clinical assessment conducted through a structured and pre-validated proforma. Information regarding age, sex, socioeconomic background, and lifestyle factors such as smoking or alcohol intake was recorded. Particular attention was given to the characterization of gastrointestinal symptoms, including epigastric pain, dyspepsia, nausea, vomiting, early satiety, bloating, hematemesis, melena, and unintentional weight loss. The duration, frequency, and severity of symptoms were documented thoroughly. The use of non-steroidal anti-inflammatory drugs (NSAIDs), which represent an important confounding factor in ulcer pathology, was carefully noted along with any history of chronic illnesses such as diabetes or hypertension. This comprehensive approach allowed for an accurate comparison of clinical profiles between H. pylori positive and negative patients.

Endoscopic Assessment: All patients underwent upper gastrointestinal endoscopy performed by trained gastroenterologists using standard Olympus endoscopic systems. The morphology of the gastric ulcer was described in detail, including the anatomical location within the stomach (antrum, body, fundus, or incisura), the ulcer's size, its depth, and whether the ulcer appeared single or multiple. Ulcers were categorized as small when less than one centimeter in diameter, medium when between one and two centimeters, and large when greater than two centimeters. The depth of each lesion was classified as superficial or deep depending on the extent of mucosal involvement. The endoscopists also documented the presence of complications such as active bleeding, stigmata of recent hemorrhage, perforation, mucosal edema, or fibrosis. Each ulcer was assigned a Gastric Ulcer Severity Score ranging from zero to ten, based on standardized endoscopic criteria combining size, depth, inflammatory appearance, and complication risk. This score served as a key variable in assessing the relationship between H. pylori infection and ulcer severity.

Biopsy Collection and Laboratory Analysis: During endoscopy, gastric biopsy samples were obtained from both the antrum and body of the stomach using standard biopsy forceps. The samples were processed immediately for Rapid Urease Test (RUT), where the biopsy tissue was placed in a urea-rich gel medium and monitored for a color change indicative of urease activity produced by H. pylori. Additional biopsy specimens were fixed in 10% buffered formalin and subjected to routine histopathological processing. Staining was performed using Modified Giemsa stain, which allowed clear visualization of H. pylori organisms on the mucosal surface. The presence of the organism on either test RUT or histology was considered confirmatory for H. pylori infection. This dual-modality testing enhanced diagnostic accuracy and minimized false-negative results.

Assessment of Ulcer Severity in Relation to Infection Status: To evaluate the association between H. pylori infection and gastric ulcer severity, the study compared ulcer characteristics, endoscopic severity scores, and complication frequencies between infected and non-infected patients. Factors such as ulcer size, depth, multiplicity, and bleeding tendencies were analyzed in relation to H. pylori positivity. This comparative approach enabled a comprehensive assessment of how infection influenced clinical and endoscopic presentation.

Ethical Considerations: The study adhered strictly to ethical guidelines for human research. Ethical clearance was obtained from the Institutional Review Boards of both participating institutions SMBBMC Lyari, Karachi, and Rehman Medical Institute, Peshawar. Written informed consent was obtained from all patients prior to enrollment. Confidentiality of patient information was maintained throughout the study.

Statistical Analysis: All collected data were entered and analyzed using SPSS version 25. Continuous variables such as age, ulcer size, and severity scores were expressed as mean values with standard deviations, whereas categorical variables such as sex, H. pylori infection status, and presence of complications were presented as frequencies and percentages. Comparisons between H. pylori positive and negative groups were made using the independent samples t-test for continuous variables and the chi-square or Fisher's exact test for categorical variables. To identify independent predictors of severe gastric ulcer, a binary logistic regression model was applied, adjusting for potential confounders such as NSAID use, smoking, and relevant comorbidities. A p-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 100 patients with endoscopically confirmed gastric ulcer were enrolled in the study. The sample included 58 males (58%) and 42 females (42%), with a mean age of 46.8 ± 12.7 years. The prevalence of Helicobacter pylori infection was 62% (n = 62), while 38% (n = 38) tested negative. Baseline demographic and clinical characteristics for both groups are presented in Table 1. The proportion of males and females was similar across both groups, demonstrating no significant gender-related difference in H. pylori positivity (p = 0.89). H. pylori–positive patients, however, tended to be slightly younger compared to the uninfected group, with the difference reaching statistical significance.

Clinically, H. pylori–positive individuals showed a more intense symptom profile than those negative for infection. Moderate to severe epigastric pain was significantly more common in the infected group (p = 0.01). Symptoms such as nausea, vomiting, early satiety, and unintentional weight loss were also markedly higher among infected patients, as detailed in Table 2. Although gastrointestinal bleeding manifestations such as hematemesis and melena were more frequently observed in the H. pylori–positive group, the differences were not statistically significant.

Endoscopic findings demonstrated that H. pylori infection was strongly associated with more severe ulcer characteristics. Infected patients had significantly larger ulcer sizes (mean 1.89 ± 0.61 cm vs. 1.43 ± 0.52 cm; p = 0.001) and a higher frequency of deep ulcers and multiple ulcers compared with uninfected individuals. Antral involvement was the predominant location among H. pylori–positive patients, whereas body and fundus lesions were more commonly observed in H. pylori–negative

individuals. Complications such as active bleeding and stigmata of recent hemorrhage occurred more frequently in the infected group, showing a significant association (p = 0.03). These findings are summarized in Table 3.

The Gastric Ulcer Severity Score was markedly elevated among H. pylori–positive patients, with a mean score of 6.7 ± 1.8, in contrast to 5.0 ± 1.5 in those negative for the infection (p < 0.001). Multivariable logistic regression confirmed that H. pylori infection remained an independent predictor of severe ulcer disease after adjusting for gender, NSAID use, smoking status, and comorbidities, yielding an adjusted odds ratio of 2.84 (95% CI: 1.31–6.13; p = 0.009). This demonstrates a strong and statistically significant association between H. pylori infection and increased ulcer severity.

Table 1. Baseline Demographic and Clinical Characteristics of Patients (N = 100)

Variable	H. pylori Positive (n = 62)	H. pylori Negative (n = 38)	p-value
Mean Age (years)	44.6 ± 11.9	50.3 ± 13.3	0.02
Gender			
Male (%)	36 (58.1%)	22 (57.9%)	0.89
Female (%)	26 (41.9%)	16 (42.1%)	0.89
Epigastric Pain (%)	57 (91.9%)	28 (73.7%)	0.01
Nausea/Vomiting (%)	40 (64.5%)	16 (42.1%)	0.03
Early Satiety (%)	32 (51.6%)	12 (31.6%)	0.04
Weight Loss (%)	29 (46.8%)	10 (26.3%)	0.03
NSAID Use (%)	18 (29.0%)	14 (36.8%)	0.42
Smoking (%)	19 (30.6%)	10 (26.3%)	0.65

Table 2. Comparison of Clinical Symptoms Between H. pylori Positive and Negative Patients

Symptom	H. pylori Positive (n = 62)	H. pylori Negative (n = 38)	p-value
Moderate–Severe Epigastric Pain (%)	49 (79.0%)	19 (50.0%)	0.01
Post-meal Discomfort (%)	42 (67.7%)	17 (44.7%)	0.02
Nausea/Vomiting (%)	40 (64.5%)	16 (42.1%)	0.03
Hematemesis (%)	8 (12.9%)	4 (10.5%)	0.71
Melena (%)	12 (19.4%)	6 (15.8%)	0.63
>5% Weight Loss (%)	29 (46.8%)	10 (26.3%)	0.03

Table 3. Endoscopic Characteristics and Severity Indicators

Endoscopic Feature	H. pylori Positive (n = 62)	H. pylori Negative (n = 38)	p-value
Mean Ulcer Size (cm)	1.89 ± 0.61	1.43 ± 0.52	0.001
Deep Ulcers (%)	30 (48.4%)	10 (26.3%)	0.02
Multiple Ulcers (%)	19 (30.6%)	6 (15.8%)	0.03
Antral Location (%)	45 (72.6%)	18 (47.4%)	0.01
Active Bleeding (%)	14 (22.6%)	4 (10.5%)	0.03
Mean Severity Score	6.7 ± 1.8	5.0 ± 1.5	<0.001

DISCUSSION

The present cross-sectional study explored the association between Helicobacter pylori infection and the clinical severity as well as endoscopic characteristics of gastric ulcer among patients presenting to two major tertiary care hospitals in Pakistan[10]. The findings demonstrate a strong and clinically meaningful relationship between H. pylori positivity and both symptom burden and ulcer severity, reinforcing the significant pathogenic role of this organism in gastric mucosal injury[11].

A key observation of this study was the relatively high prevalence of H. pylori infection, detected in 62% of patients with gastric ulcer. This aligns with previously reported infection rates in South Asian populations, where socioeconomic factors, overcrowding, limited sanitation facilities, and delayed access to healthcare contribute to persistent transmission[12]. The high prevalence among symptomatic ulcer patients also reinforces the pathogen's role as a predominant etiological factor in the region. Although the infection was observed in both genders without significant difference, H. pylori–positive individuals tended to be younger, a finding consistent with epidemiological patterns indicating earlier acquisition of the infection in high-burden settings[13].

Clinically, H. pylori–positive patients presented with more intense and frequent upper gastrointestinal symptoms. Moderate to severe epigastric pain, nausea, vomiting, post-meal discomfort, and significant unintentional weight loss were all more common among infected individuals[14]. These findings suggest that the inflammatory response induced by H. pylori contributes not only to mucosal damage but also to exacerbation of symptomatology. The organism's well-documented ability to trigger chronic active gastritis, disrupt gastric acid regulation, and impair mucosal healing may explain this heightened symptom burden. Interestingly, although gastrointestinal bleeding in the form of melena or hematemesis occurred more often among infected patients, statistical significance was not achieved, possibly due to the limited sample size or variations in lesion types[15].

Endoscopic analysis provided further evidence of the significant impact of H. pylori on ulcer severity. Infected patients had significantly larger ulcers, a higher proportion of deep lesions, and more frequent occurrences of multiple ulcers[16]. These findings correlate with the organism's pathogenic mechanisms, including increased inflammatory cell infiltration, cytokine release, and epithelial damage. The predominance of antral ulceration among H. pylori–positive individuals is in agreement with established literature, which highlights preferential colonization of the antral mucosa and its role in gastritis-associated ulcer formation. Moreover, complications such as active bleeding and high-risk stigmata were significantly more common in infected patients, underscoring the clinical importance of early detection and prompt management[17].

The Gastric Ulcer Severity Score was markedly higher among infected patients, and multivariable regression confirmed H. pylori infection as an independent predictor of severe ulcer disease, even after adjusting for confounding factors such as NSAID use and smoking. These findings highlight that H. pylori plays a distinct and significant role in determining the clinical and endoscopic severity of gastric ulcers, separate from other contributing factors. The data are consistent with international studies reporting that eradication of H. pylori dramatically reduces ulcer recurrence, accelerates mucosal healing, and lowers complication rates[18,19].

NSAID use and smoking, although prevalent among both groups, did not fully account for the observed severity differences. This further emphasizes that while these factors may potentiate mucosal injury, H. pylori infection itself remains a central determinant of disease severity in this population. The integration of dual-modality diagnostic testing using both rapid urease test and histopathology strengthened the accuracy of H. pylori detection and enhanced the reliability of the study findings[20].

The public health implications of these results are significant. Given the high prevalence and strong association of H. pylori with severe gastric ulcer, routine screening for the infection in all dyspeptic and ulcer patients should be considered essential in Pakistan and similar high-burden regions[7]. Early identification and eradication therapy can reduce morbidity, improve symptom control, prevent complications such as bleeding or perforation, and decrease recurrence rates. Moreover, patient education on hygiene, sanitation, and safe food practices could help reduce the transmission of infection in the community[11-14].

While the study provides valuable insights, certain limitations must be acknowledged. The cross-sectional design limits the ability to establish causality, and although the study sample size of 100 patients was adequate for statistical analysis, larger multicenter studies would allow broader generalization[19]. Additionally, bacterial virulence factors, socioeconomic determinants, dietary patterns, and host genetic susceptibility were not assessed, all of which may influence disease severity. Despite these limitations, the study offers important region-specific evidence linking H. pylori infection with the clinical and endoscopic profile of gastric ulcers[20].

CONCLUSION

This study demonstrates a strong and independent association between Helicobacter pylori infection and increased severity of gastric ulcer disease. Patients infected with H. pylori exhibited more intense symptoms, larger and deeper ulcers, more frequent

ulcer multiplicity, and a higher likelihood of bleeding complications. The Gastric Ulcer Severity Score was significantly higher among infected individuals, and infection status remained an independent predictor of severe disease even after adjustment for confounders. These findings highlight the critical role of H. pylori in the pathogenesis and progression of gastric ulcer in the Pakistani population.

Early and accurate detection of H. pylori, followed by appropriate eradication therapy, is essential for reducing ulcer severity, improving clinical outcomes, and preventing recurrence. The results underscore the need for routine screening of H. pylori in all patients presenting with gastric ulcer and support public health efforts aimed at reducing the burden of infection through improved sanitation and healthcare access.

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Authors' Contributions

S. – Study design, data collection, manuscript drafting.
M.D. – Endoscopy, sample processing, methodology support.
S.L.P. – Histopathology and interpretation of biopsy results.
S.K. – Clinical evaluation and literature review.
N.S. – Data analysis, tables, and statistical review.
I.Z. – Editing, referencing, and final manuscript proofreading.
All authors approved the final version.

Data Availability: Data are available from the corresponding author upon reasonable request.

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