



ASSOCIATING *H. PYLORI* BACTERIAL DENSITY WITH UPPER GI SYMPTOMS AND ENDOSCOPIC FINDINGS: A CROSS-SECTIONAL STUDY

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Abstract

Objective: To examine the association between *Helicobacter pylori* density of gastric mucosal biopsy and the severity of gastric mucosal inflammation and evaluate the relationship with endoscopic findings and upper gastrointestinal symptoms in endoscopic patients.

Study Design: This was a cross-sectional study.

Place and Duration: This study was conducted at Al Tibri Medical College and Hospital ISRA, University Karachi Campus Pakistan from August 2024 to August 2025

Methods: Ninety-two patients with upper gastrointestinal symptoms underwent endoscopy with gastric antral biopsies. Biopsies were stained with hematoxylin and eosin and Giemsa and were graded for gastritis and *H. pylori* density by one histopathologist using the Sydney System (0-3 and 0-6 scales, respectively). Patients on anti-*H. pylori* therapy were excluded. Data was analyzed using SPSS 20.0, using Chi-Square tests and Pearson correlation with a $p < 0.05$.

Results: Among 92 patients (59.8% female, mean age 39.21 ± 13.48 years), 65.2% (n=60) had no *H. pylori*, 10.9% (n=10) mild, 16.3% (n=15) moderate, and 7.6% (n=7) severe density. Antral gastritis and duodenitis each affected 65.2% (n=60), esophagitis 65.2% (n=60), gastric and duodenal ulcers 5.4% (n=5), mild gastritis 5.4% (n=5), moderate gastritis 37.8% (n=35), and severe gastritis 21.7% (n=20). Epigastric pain occurred in 76.1% (n=70) patients, nausea in 54.3% (n=50), retrosternal burning in 49.0% (n=45), and hematemesis in 2.2% (n=2). Esophagitis was significantly correlated with increased *H. pylori* density ($p < 0.001$). Moderate gastritis ($p = 0.077$),

duodenitis ($p=0.326$), and vomiting ($p=0.206$) were non-significant trends, and all other symptoms and findings were non-associated ($p=0.221-0.966$).

Conclusion: Higher *H. pylori* density is significantly associated with the severity of esophagitis but not with most gastrointestinal symptoms or other endoscopic findings. Future multicenter studies may elucidate these relationships.

Keywords: *Helicobacter pylori*, bacterial density, gastric inflammation, gastritis, esophagitis, gastrointestinal symptoms.

Introduction

Helicobacter pylori, often called *H. pylori*, is a Gram-negative bacterium that primarily settles in the stomach lining. Discovered in the early 1980s, this Gram-negative, spiral-shaped bacterium thrives in the acidic environment of the stomach by producing urease, which neutralizes gastric acid and facilitates adherence to epithelial cells [1, 2]. It is one of the major contributors to chronic gastritis, peptic ulcers, gastric adenocarcinoma, and mucosa-associated lymphoid tissue lymphoma [3]. Globally, the prevalence of this infection is approximately 43.9% in adults, compared to 52.6% prior to 1990, and in children and adolescents, it is approximately 35% with no significant decrease in recent years [4]. In Pakistan, prevalence rates vary among different groups and geographical regions, with a study reporting a range of 22.41% to 50.89% in individuals with stomach complaints, and a high burden in certain areas, such as Lahore and Dera Ghazi Khan [5, 6]. This infection typically begins in childhood, and its health effects can be devastating in some cases where treatment is not sought [3].

H. pylori produces gastric damage by a complex interplay of bacterial factors such as cytotoxin-associated gene A (CagA) and vacuolating cytotoxin A (VacA), and host immune responses [1, 7]. Neutrophil and mononuclear cell infiltration are observed when *H. pylori* is colonized, causing active and chronic gastritis, as categorized by the new Sydney system [8]. Bacterial density is a crucial factor. Studies have shown that higher densities (e.g., massive clusters covering at least half of the mucosal lining) correlate with increased levels of inflammatory activity, as indicated by elevated polymorphonuclear infiltrates and cytokine secretion, such as IL-8 [9, 10]. Multiple studies demonstrate specific causal links between bacterial density and tissue-level consequences. Likewise, moderate to severe chronic gastritis is characterized by a higher density in more than 90% of high-load cases, where the level of inflammation is more active and may lead to tissue atrophy or cellular changes [3, 9].

H. pylori infection leads to upper gastrointestinal (GI) symptoms such as dyspepsia, epigastric pain, bloating, nausea, vomiting, and acid reflux. However, they are not always present and can overlap with functional disorders [11]. Acute infections may lead to temporary symptoms, whereas chronic colonization may be subclinical and only 10-20% may result in overt manifestation [12]. Density thresholds are found in quantitative tests. Bacterial counts of approximately 10^4 CFU are linked with vomiting and acid reflux. In contrast, higher levels (10^5 - 10^8) correspond to more advanced symptoms of pain and early satiety, which may be indicative of atrophy transitions [11].

Endoscopy demonstrates nodular lesions, gastritis, and ulcers, frequently worse in the antrum with patterns that vary according to infection stage and area of stomach [13]. Typical symptoms are an inflamed stomach lining in approximately forty percent of cases, and more than half are positive for the bacteria [14]. Some tools are used to predict infection, such as the Kyoto classification, which depends on the nature of visible blood vessels or mucus accumulation, and more recent techniques rely on imaging to estimate bacterial load [14]. The relationships between bacterial levels, symptoms such as stomach aching or bloating, and findings on endoscopy are not necessarily direct, which points to research gaps. Certain studies indicate associations with continuous abdominal pain, yet density does not powerfully influence the majority of signs across all groups [10]. Endoscopy correlations are also confounded, with no obvious association to most of the results observed except conditions such as esophagitis [14], and some studies report greater bacterial loads in ulcers or inflamed regions [15].

To fill these gaps, the current study examines the relationship between the density of *H. pylori* in gastric biopsies and the severity of mucosal inflammation, expanding the analysis to all endoscopic results as well as prevalent upper GI symptoms in patients undergoing endoscopy.

Methodology

In this study 92 patients with upper gastrointestinal symptoms were studied who needed an endoscopy and biopsy.

The reasons behind the endoscopy, such as epigastric pain, nausea, or retrosternal burning, were reported. All procedures were performed by the same endoscopist using a Fujifilm Endoscope ELUXEO 7000 series. To minimize variation in observations, only 92 gastric antral biopsies from patients with chronic gastritis were included, without any exclusions. Patient-form information, including age, gender, symptoms, medical history, medications (particularly antibiotics), and endoscopic results, was all recorded.

A single histopathologist carried out biopsies. General staining was performed using hematoxylin and eosin on tissues, with Giemsa stain to aid in the identification of *H. pylori*. To determine the features of chronic gastritis, the Sydney System was used. This 0-3 graded gastritis: 0 = none, 1=mild, 2=moderate, and 3=severe. Histopathology provided a comparable review of *H. pylori* density. Density grading was classified as follows: 0, no bacteria; 1, *H. pylori* at one location; 2, scattered bacteria at scattered locations; 3, scattered bacteria at scattered locations; 4, scattered bacteria at scattered locations; 5, scattered bacteria at scattered locations; 6, scattered bacteria at scattered locations; 6, thick, unbroken layer across surface. They grouped these to analyze them: none (0), mild (1-2), moderate (3-4), and severe (5-6).

SPSS version 20.0 on Windows was used to analyze the data. Means and standard deviations were used to summarize continuous variables like age. Categorical variables, including the severity of gastritis, were expressed in percentages. The Chi-Square test was used to compare categorical data. Pearson correlation analysis was used to test the relationships among variables, including relationships between density and inflammation. The results were assessed at a 95% confidence interval, with $p < 0.05$ being significant.

Results

The sample size for this study consisted of 92 patients, comprising 55 females ($n=55$, 59.8%) and 37 males ($n=37$, 40.2%). The age range of participants was 18-83 years, with a mean of 39.21 ± 13.48 years. The average age of female patients was 37.45 ± 13.22 years, and for males it was 41.15 ± 13.76 years (Table 1).

Table 1: Demographic Characteristics of Patients (n=92)

Parameter	n (%)
Total Sample Size	92 (100)
Gender Distribution	
Female	55 (59.8)
Male	37 (40.2)
Mean Age	39.21 ± 13.48
Age Range	18 - 83 years
Mean Age by Gender	
Female	37.45 ± 13.22
Male	41.15 ± 13.76

Endoscopic findings consisted of antral gastritis and duodenitis in 60 ($n=60$, 65.2%), and gastric and duodenal ulcers in 5 ($n=5$, 5.4%). Severe gastritis was observed in 20 patients ($n=20$, 21.7%), and esophagitis in 60 patients ($n=60$, 65.2%). In terms of gastrointestinal symptoms, 70 patients ($n=70$, 76.1%) reported epigastric pain, and 50 patients ($n=50$, 54.3%) reported nausea. Only two

patients (n=2, 2.2%) had hematemesis. The presence of *H. pylori* was positive in 32 (n=60, 34.8%) and negative in 60 (n=32, 65.2%) patients (Table 2).

Table 2: Distribution of Patients According to Endoscopic Findings and Gastrointestinal Symptoms

Parameters	Yes (%)	n (%)
Endoscopic Findings		
Duodenal Ulcer	5 (5.4)	87 (94.6)
Gastric Ulcer	5 (5.4)	87 (94.6)
Antral Gastritis	60 (65.2)	32 (34.8)
Duodenitis	60 (65.2)	32 (34.8)
Gastritis Mild	5 (5.4)	87 (94.6)
Gastritis Moderate	35 (37.8)	57 (62.2)
Gastritis Severe	20 (21.7)	72 (78.3)
Esophagitis	60 (65.2)	32 (34.8)
Gastrointestinal Symptoms		
Hematemesis	2 (2.2)	90 (97.8)
Malena	3 (3.3)	89 (96.7)
Retrosternal Burning	45 (49.0)	47 (51.0)
Belching	5 (5.4)	87 (94.6)
Burping	4 (4.3)	88 (95.7)
Nausea	50 (54.3)	42 (45.7)
Vomiting	28 (30.4)	64 (69.6)
Epigastric Pain	70 (76.1)	22 (23.9)
Evaluation of <i>H. pylori</i>		
Positive	60 (65.2)	
Negative	32 (34.8)	

Most patients (n=60, 65.2%) had no detectable *H. pylori* presence. A smaller portion (n=10, 10.9%) had mild density, and 15 patients (n=15, 16.3%) had moderate *H. pylori* density. Only seven patients (n=7, 7.6%) showed severe *H. pylori* density. These findings suggest that most patients have either no infection or a mild to moderate degree of *H. pylori* colonization (Figure 1).

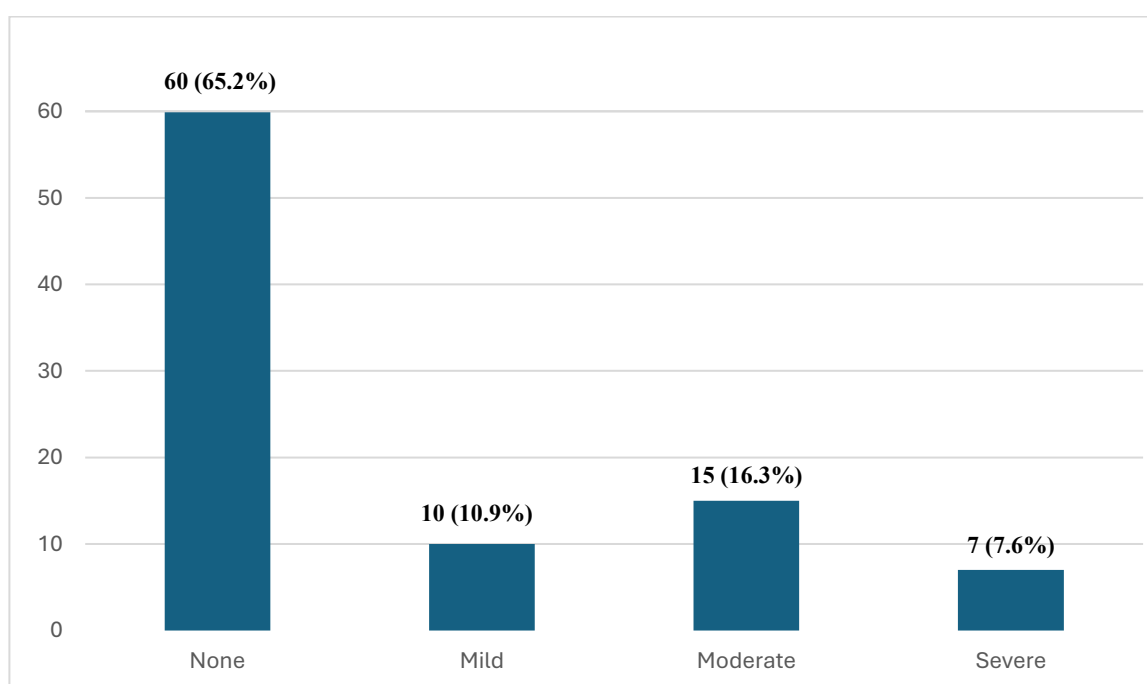


Figure 1: Distribution of *H. pylori* Density Among Patients

The analysis of *H. pylori* density and its association with gastrointestinal symptoms and endoscopic findings shows that most gastrointestinal symptoms, such as epigastric pain (n=22, 23.9%), nausea (n=42, 45.7%), and recurrent retrosternal burning (n=28, 30.4%), do not show a significant association with *H. pylori* density. Vomiting (n=64, 69.6%) is more common in patients with no *H. pylori* density, though this trend is not statistically significant (p=0.206). Notably, esophagitis (n=31, 33.7%) is significantly linked to higher *H. pylori* densities (p<0.001), with a notable increase in severity. Other findings, such as moderate gastritis (n=35, 37.8%) and duodenitis (n=28, 30.4%), show trends toward association with *H. pylori* density but are not statistically significant (p-values ranging from 0.077 to 0.911). (Table 3).

Table 3: Association Between Endoscopic Findings, Gastrointestinal Symptoms, and *H. pylori* Density

Parameters	None (%)	Mild (%)	Moderate (%)	Severe (%)	p-value
Endoscopic Findings					
Duodenal Ulcer	55 (59.8)	2 (2.2)	5 (5.4)	30 (32.6)	.468
Gastric Ulcer	60 (65.2)	2 (2.2)	5 (5.4)	25 (27.2)	.911
Antral Gastritis	60 (65.2)	6 (6.5)	6 (6.5)	20 (21.7)	.442
Duodenitis	56 (60.9)	6 (6.5)	7 (7.6)	23 (25.0)	.326
Gastritis Mild	59 (64.1)	2 (2.2)	13 (14.1)	18 (19.6)	.646
Gastritis Moderate	57 (62)	9 (9.8)	10 (10.9)	16 (17.4)	.077
Gastritis Severe	60 (65.2)	2 (2.2)	7 (7.6)	23 (25.0)	.657
Esophagitis	31 (33.7)	9 (9.8)	13 (14.1)	39 (42.4)	<.001
Gastrointestinal Symptoms					
Hematemesis	90 (97.8)	0 (0)	1 (1.1)	1 (1.1)	.911
Malena	89 (96.7)	0 (0)	1 (1.1)	2 (2.2)	.911
Retrosternal Burning	28 (30.4)	5 (5.4)	9 (9.8)	25 (27.2)	.270
Burping	47 (51.1)	3 (3.3)	7 (7.6)	35 (38.0)	.800
Belching	46 (50.0)	2 (2.2)	8 (8.7)	36 (39.1)	.800
Nausea	42 (45.7)	5 (5.4)	11 (12.0)	34 (36.9)	.947
Vomiting	64 (69.6)	3 (3.3)	11 (12.0)	14 (15.2)	.206
Epigastric Pain	22 (23.9)	7 (7.6)	11 (12.0)	52 (56.5)	.966

*P = <.001, Statistically significant

Hematemesis and malena showed no significant association, with 1 (100%) positive case for each and 30 (34.9%) negative cases for both symptoms (p=1.000). Retrosternal burning had a higher percentage of positive cases (16, 42.1%) compared to negative cases (22, 57.9%), but the association was not significant (p=0.221). Both burping and belching showed no significant association, with similar percentages in positive and negative groups (p=1.000). Nausea and vomiting showed no significant correlation with *H. pylori*, with 17 (38.6%) and 10 (47.6%) positive cases, respectively. Finally, there was no significant difference for epigastric pain (p=0.903) (Table 4).

Table 4: Association between *H. pylori* & Gastrointestinal Symptoms

Parameters	Positive	Negative	p-value
Hematemesis			1.000
Yes	1 (100%)	0 (0%)	
No	30 (34.9%)	56 (65.1%)	
Malena			1.000
Yes	1 (100%)	0 (0%)	
No	30 (34.9%)	56 (65.1%)	
Retrosternal Burning			.221

Yes	16 (42.1%)	22 (57.9%)	
No	14 (29.8%)	33 (70.2%)	
Burping			1.000
Yes	2 (40.0%)	3 (60.0%)	
No	23 (34.2%)	44 (65.8%)	
Belching			1.000
Yes	2 (40.0%)	3 (60.0%)	
No	23 (34.2%)	44 (65.8%)	
Nausea			.673
Yes	17 (38.6%)	27 (61.4%)	
No	13 (37.1%)	22 (62.9%)	
Vomiting			.220
Yes	10 (47.6%)	11 (52.4%)	
No	18 (31.6%)	39 (68.4%)	
Epigastric Pain			.903
Yes	21 (36.2%)	37 (63.8%)	
No	14 (33.3%)	28 (66.7%)	

Discussion

This cross-sectional study investigated the relationship between *H. pylori* levels in gastric biopsies and the degree of mucosal inflammation in patients, while evaluating the associations with various endoscopic findings and common gastrointestinal symptoms in patients undergoing upper endoscopy. Our positive percentage stood at 65.2% on patients with *H. pylori*, like other reports where gastritis is reported to be a common phenomenon in *H. pylori* infection, with an average positive percentage of over 60 percent in infected individuals. In Pakistan, studies show certain variations: Sardar et al. [16] discovered 73.3% in dyspeptic patients, meaning it is strongly correlated with such symptoms as epigastric pain, 86.4%. Maqsood et al. reported a reduced rate (22.41%), which could be attributed to differences in detection methods or local determinants [6]. This is compared to a lower level in developed settings, with 13.2% in Ireland [17]. This highlights the geographic discrepancies, and socioeconomic problems can compound the burdens in our scenario.

The rates of antral gastritis and duodenitis were 65.2% each, esophagitis was 65.2%, and the ulcer (gastric and duodenal) rate was 5.4% in our cohort. These trends are similar to those reported by Sharma et al., where the highest incidence was gastritis (79.32%) in the *H. pylori*-positive cohort and duodenal ulcers (8.38%) [18]. Nevertheless, our rates of ulcers were lower than those in a different study, 12.3% of cases represented duodenal ulcers, and 85.7% of these ulcers were *H. pylori*-positive, which indicates we may be experiencing milder disease or earlier treatment in our population [19]. The severity trends of gastritis in our study are similar to those of Sayin (2019), in which more severe inflammation was positively related to the higher *H. pylori* density ($p < 0.001$) [8]. Symptomatically, 76.1% of the patients complained of epigastric pain, 54.3% nausea, and 49.0% retrosternal burning. This observation is consistent with Sardar et al., who attributed *H. pylori* to nausea/vomiting (60.6%) and high odds ratios to epigastric pain [16]. However, this contrasts with that of Zheng et al., who found no significant relationships between symptoms and children. They did, however, correlate well with nodular lesions and active gastritis [3].

We found that *H. pylori* density did not significantly contribute to most gastrointestinal symptoms, such as epigastric pain, nausea, and retrosternal burning, with p-values of 0.270-0.966. Patients without *H. pylori* were more likely to vomit ($p=0.206$). Such symptom findings are in line with several studies. In a pediatric cohort, no significant differences were observed in abdominal pain or vomiting between the infected and uninfected groups. However, the positive group had more inflammation [3]. This observation supports our previous observations that symptoms must be caused by factors beyond bacterial load alone, including host response. However, this is in contrast

with a study by Sayin (2019), which finds correlations between higher densities and the severity of dyspepsia in adults; higher colonization levels and the severity of dyspepsia symptoms in cases of chronic gastritis [8].

Our endoscopic findings are contrary to those found in the literature, which generally show an inverse correlation. A single cross-sectional study by Niknam et al. showed that there is no relationship between erosive GERD and *H. pylori* infection, and that the prevalence of positivity showed no significant difference between mild and severe disease ($p=0.214$), indicating a protective effect of this bacterium against reflux [20]. Meta-analyses also exhibit this protective pattern, showing decreased odds of GERD with infection [21]. Our gastritis and duodenitis trends also correlate with others positively. Moderate-to-severe chronic gastritis and biopsy activity were linked to higher densities ($p < 0.05$), with dose-response associations [10]. Our observations are also associated with the association of duodenitis in Lebanese patients, which connects infection to inflammation [13].

In terms of bleeding, we do not find any correlation with hematemesis or melena as opposed to evidence that *H. pylori* cause upper GI bleeding in the form of ulcers. In a study of pediatric bleeding, *H. pylori* had a positivity rate of 37% and the older age and crowding were identified as factors [22]. The results may inform guided testing, reduce unnecessary endoscopies in symptom-driven cases, and encourage the study of non-bacterial symptom drivers to develop more personalized therapies.

There are a few limitations of this study. Being a cross-sectional design, it cannot establish causality between *H. pylori* density and symptoms or endoscopic findings but instead illustrates associations. Its generalizability could be constrained by the small sample size of 92 patients across one center in Pakistan, especially to different populations or regions with varying infection rates.

Conclusion

The findings of this study showed that the prevalence of *H. pylori* was not seen in many patients; moderate density was predominant in positive patients, and only a significant relationship was found with esophagitis, and not with most symptoms. Overall, these findings underscore the intricate relationship between bacterial load and gastric health. Future directions could be strengthened by generalizability through larger multicenter trials and longitudinal studies to determine subsequent alterations in density over time or after treatment. Exploring molecular mechanisms or more sophisticated imaging modalities is also helpful in understanding these relations in more depth.

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