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HISTOPATHOLOGICAL AND BIOCHEMICAL CORRELATION OF HELICOBACTER PYLORI INFECTION IN CHRONIC GASTRITIS

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ABSTRACTS

Background: Helicobacter pylori (H. pylori) is a major cause of chronic gastritis and persistent gastric mucosal inflammation. Histopathological assessment provides direct evidence of mucosal injury, while the relationship between bacterial density and different histological changes helps characterize disease severity.
Objective: To evaluate the histopathological and biochemical correlation of H. pylori infection in patients with chronic gastritis.
Study Design & Setting: This was a descriptive analytical cross-sectional study conducted in the Department of Pathology Bahria University Health Sciences, Karachi from August 2025 to January 2026.
Methodology: A total of 150 adult patients with chronic gastritis were included through consecutive sampling. Gastric biopsy specimens were processed and examined using hematoxylin and eosin and modified Giemsa staining. Histopathological changes, including chronic inflammation, neutrophilic activity, glandular atrophy, intestinal metaplasia, and H. pylori density, were graded according to the Updated Sydney System. Blood samples were also analyzed for selected biochemical parameters. Associations between H. pylori status and histopathological findings were assessed using appropriate statistical tests, while correlations were evaluated using correlation analysis.
Results: H. pylori infection was identified in 78 (52.0%) patients. Among positive cases, mild, moderate, and severe bacterial density was observed in 39 (50.0%), 29 (37.2%), and 10 (12.8%), respectively. Chronic inflammation was significantly associated with H. pylori status ($p=0.003$), as was neutrophilic activity ($p<0.001$). Glandular atrophy and intestinal metaplasia were not significantly associated with infection ($p=0.089$ and $p=0.126$, respectively). H. pylori density showed significant positive correlations with chronic inflammation ($r=0.521$, $p<0.001$), neutrophilic activity ($r=0.596$, $p<0.001$), and glandular atrophy ($r=0.228$, $p=0.045$).
Conclusion: H. pylori infection was common among patients with chronic gastritis and was significantly associated with inflammatory histopathological changes. Increasing bacterial density was correlated with greater chronic inflammation and neutrophilic activity.

KEYWORDS: Contraceptive prevalence; Depot medroxyprogesterone acetate; DMPA-SC; Perception; Reproductive autonomy; Self-administration

INTRODUCTION

Chronic gastritis is a persistent inflammatory condition of the gastric mucosa characterized histologically by infiltration of inflammatory cells, with variable epithelial injury, glandular atrophy, intestinal metaplasia, and other mucosal changes.[1,2] Among its recognized causes, Helicobacter pylori (H. pylori) is the predominant infectious etiological factor and is particularly associated with chronic active gastritis.[3] H. pylori is a Gram-negative, microaerophilic, spiral-shaped bacterium capable of colonizing the gastric mucosa and persisting for prolonged periods. Transmission is thought to occur mainly through oral–oral or fecal–oral routes and is influenced by socioeconomic and environmental conditions.[4]

H. pylori infection remains one of the most prevalent chronic bacterial infections worldwide. A systematic review and meta-analysis covering studies from 1980 to 2022 estimated that global prevalence declined from 58.2% (95% CI: 50.7–65.8%) during 1980–1990 to 43.1% (95% CI: 40.3–45.9%) during 2011–2022, with substantial geographic variation and higher prevalence generally reported in lower-income settings.[5] Following colonization, bacterial urease activity facilitates survival in the acidic gastric environment, while adhesins,

cytotoxins, and inflammatory mediators contribute to epithelial injury and sustained mucosal inflammation.[6] Persistent infection may consequently produce neutrophilic activity, lymphocytic infiltration, glandular atrophy, intestinal metaplasia, and, in advanced cases, dysplastic or neoplastic changes.[7]

Histopathological assessment therefore provides a direct means of characterizing the gastric mucosal response to H. pylori. Gastric biopsy specimens can be evaluated using hematoxylin and eosin staining for chronic inflammation, neutrophilic activity, atrophy, and intestinal metaplasia, while special stains such as Giemsa can facilitate identification of H. pylori.[8] The Updated Sydney System provides a standardized framework for grading chronic inflammation, activity, atrophy, intestinal metaplasia, and bacterial density. Studies using this system have demonstrated associations between H. pylori positivity and the severity of chronic inflammation and other histological alterations.[9,10]

Beyond bacterial detection, biochemical assessment can provide complementary information about the inflammatory response associated with H. pylori infection. Persistent colonization stimulates epithelial and immune cells to release inflammatory mediators and may alter circulating biochemical markers reflecting systemic or mucosal inflammation. When these biochemical findings are interpreted alongside the degree of chronic inflammation, neutrophilic activity, glandular atrophy, intestinal metaplasia, and bacterial density observed on gastric biopsy, they may provide a broader characterization of the host response to infection.[11,12]

Chronic H. pylori infection produces variable degrees of gastric inflammation, and histopathological severity may differ considerably among affected patients. However, Pakistani literature remains relatively limited in simultaneously correlating histopathological severity with biochemical parameters in patients with chronic gastritis. This study will therefore generate integrated histopathological and biochemical data, allowing assessment of whether biochemical alterations correspond with the degree of mucosal inflammation and H. pylori involvement. The findings will add locally derived evidence to the existing literature and help clarify the variability of H. pylori-associated chronic gastritis patterns reported across Pakistani populations.

MATERIALS AND METHODS

Study design and setting

This was a descriptive analytical, cross-sectional study that was conducted in the Department of Pathology Bahria University Health Sciences, Karachi from August 2025 to January 2026. The study population consisted of adult patients who underwent upper gastrointestinal endoscopy and gastric biopsy for evaluation of symptoms suggestive of chronic gastritis.

Sample Size

A total of 150 patients were included in the study. The sample size was taken as 150 based on the expected prevalence of H. pylori infection and to provide adequate precision for estimation of histopathological and biochemical associations. The final sample was increased/maintained at 150 patients to allow reliable subgroup and correlation analysis.

Sampling Technique and Patient Selection

Patients were selected through consecutive non-probability sampling according to predefined inclusion and exclusion criteria. Adult patients of either gender who underwent upper gastrointestinal endoscopy and had gastric mucosal biopsy specimens showing features of chronic gastritis were included. Patients with a previous history of H. pylori eradication therapy, previous gastric surgery, gastric malignancy, active gastrointestinal bleeding, severe systemic illness, or inadequate/poorly preserved biopsy material were excluded. Patients receiving medications that could substantially alter the biochemical parameters under assessment were also excluded where applicable.

Data Collection

After obtaining informed written consent, demographic and relevant clinical information was recorded on a structured proforma. Age, gender, presenting symptoms, relevant medical history, medication history, and endoscopic findings were documented. Gastric biopsy specimens were obtained during routine upper gastrointestinal endoscopy. Biopsy specimens were appropriately labeled and transported to the pathology laboratory for processing. A venous blood sample was also collected from each participant for assessment of the selected biochemical parameters. The biochemical investigations were performed using standard laboratory procedures and the results were recorded for subsequent analysis.

Histopathological Examination

Gastric mucosal biopsy specimens were fixed in 10% neutral buffered formalin, processed routinely, embedded in paraffin wax, and sectioned at approximately 4–5 μm thickness. Tissue sections were stained with hematoxylin

and eosin (H&E) for assessment of gastric mucosal architecture and inflammatory changes. A modified Giemsa stain was performed for detection of H. pylori, as this stain facilitates visualization of the organisms along the gastric epithelial surface. Histopathological evaluation was performed using light microscopy.
The histological features were assessed according to the Updated Sydney System, which evaluates chronic inflammation, neutrophilic activity, glandular atrophy, intestinal metaplasia, and H. pylori density. Each parameter was graded as 0 (absent), 1 (mild), 2 (moderate), or 3 (severe) according to the degree of involvement observed microscopically. The presence and density of H. pylori organisms were recorded separately. The histopathological findings were subsequently correlated with the biochemical parameters. The Updated Sydney System has been widely used for standardized assessment and grading of chronic gastritis and H. pylori-associated mucosal changes.

Biochemical Assessment
Blood samples were obtained from the enrolled patients and were analyzed for the predefined biochemical parameters relevant to the study. The samples were processed according to standard laboratory protocols, and biochemical results were recorded in the study proforma. The biochemical findings were categorized according to the laboratory reference ranges and were compared with H. pylori status and the severity of histopathological changes. Continuous biochemical values were also retained for correlation analysis with the individual Updated Sydney System grades. Previous evidence has demonstrated associations between histological severity of gastritis and systemic laboratory parameters, supporting assessment of biochemical changes alongside gastric histopathology.

Assessment of H. pylori Infection
H. pylori infection was determined histologically by identification of characteristic curved or spiral-shaped organisms along the gastric foveolar epithelium and within the surface mucus, particularly on Giemsa-stained sections. Patients were categorized according to the presence or absence of H. pylori. In positive cases, bacterial density was additionally graded according to the Updated Sydney System. Histopathological evidence of H. pylori was then compared with the degree of chronic inflammation, neutrophilic activity, glandular atrophy, and intestinal metaplasia.

Data Analysis
The collected data were entered and analyzed using IBM SPSS Statistics. Quantitative variables such as age and biochemical parameters were expressed as mean ± standard deviation or median with interquartile range, depending on the distribution of the data. Qualitative variables such as gender, H. pylori status, and histopathological grades were presented as frequencies and percentages. The association between categorical variables was assessed using the Chi-square test or Fisher's exact test, where appropriate. Differences in biochemical parameters between H. pylori-positive and H. pylori-negative groups were assessed using the independent-samples t test or Mann–Whitney U test according to data distribution. Correlation between biochemical parameters and histopathological grades was assessed using Pearson or Spearman correlation analysis, as appropriate. A p-value of ≤ 0.05 was considered statistically significant.

Ethical Considerations
Ethical approval was obtained from the relevant institutional ethical committee before commencement of the study. Written informed consent was obtained from all participants after explaining the purpose and procedures of the study. Patient confidentiality was maintained throughout data collection, laboratory processing, analysis, and reporting. Participation was voluntary, and patients were allowed to withdraw from the study at any stage without affecting their clinical care.

RESULTS
Among the 150 patients, the largest proportion belonged to the 31–40 years age group, comprising 42 (28.0%) patients, followed by the 41–50 years group with 39 (26.0%). Patients aged 18–30 and 51–60 years each accounted for 27 (18.0%), while 15 (10.0%) patients were aged >60 years. Males were predominant, with 86 (57.3%) patients compared with 64 (42.7%) females. Most patients were from urban areas, accounting for 91 (60.7%), whereas 59 (39.3%) were from rural areas, as shown in Table 1.

Table 1. Distribution of patients according to demographic characteristics (n=150)

Variable	Category	Frequency (%)
Age (years)	18–30	27 (18.0)
	31–40	42 (28.0)

	41–50	39 (26.0)
	51–60	27 (18.0)
	>60	15 (10.0)
Gender	Male	86 (57.3)
	Female	64 (42.7)
Residence	Urban	91 (60.7)
	Rural	59 (39.3)

Regarding clinical presentation, epigastric pain was the most frequently reported symptom, occurring in 104 (69.3%) patients, followed by dyspepsia in 97 (64.7%) and bloating in 76 (50.7%). Heartburn was reported by 63 (42.0%) patients, while nausea/vomiting was present in 51 (34.0%). Early satiety and anorexia were reported in 38 (25.3%) and 29 (19.3%) patients, respectively, as given in Table 2.

Table 2. Distribution of patients according to clinical presentation (n=150)

Clinical feature	Frequency (%)
Epigastric pain	104 (69.3)
Dyspepsia	97 (64.7)
Nausea/vomiting	51 (34.0)
Heartburn	63 (42.0)
Early satiety	38 (25.3)
Bloating	76 (50.7)
Anorexia	29 (19.3)

Histopathological examination showed that chronic inflammation was predominantly moderate, observed in 68 (45.3%) patients, followed by mild inflammation in 54 (36.0%) and severe inflammation in 24 (16.0%). Neutrophilic activity was absent in 52 (34.7%) patients, while mild, moderate, and severe activity was observed in 48 (32.0%), 37 (24.7%), and 13 (8.7%), respectively. Glandular atrophy was absent in 118 (78.7%) patients and was mild, moderate, and severe in 20 (13.3%), 9 (6.0%), and 3 (2.0%), respectively. Intestinal metaplasia was absent in 126 (84.0%) patients, whereas mild, moderate, and severe changes were observed in 15 (10.0%), 7 (4.7%), and 2 (1.3%), respectively. H. pylori density was absent in 72 (48.0%) patients, while mild, moderate, and severe bacterial density was recorded in 39 (26.0%), 29 (19.3%), and 10 (6.7%), respectively, as presented in Table 3.

Table 3. Histopathological findings of gastric biopsies according to the Updated Sydney System (n=150)

Histopathological parameter	Grade 0 n (%)	Grade 1 n (%)	Grade 2 n (%)	Grade 3 n (%)
Chronic inflammation	4 (2.7)	54 (36.0)	68 (45.3)	24 (16.0)
Neutrophilic activity	52 (34.7)	48 (32.0)	37 (24.7)	13 (8.7)
Glandular atrophy	118 (78.7)	20 (13.3)	9 (6.0)	3 (2.0)
Intestinal metaplasia	126 (84.0)	15 (10.0)	7 (4.7)	2 (1.3)
H. pylori density	72 (48.0)	39 (26.0)	29 (19.3)	10 (6.7)

Histopathological examination identified H. pylori infection in 78 (52.0%) of the 150 patients, whereas 72 (48.0%) patients were negative for H. pylori, as shown in figure 1.

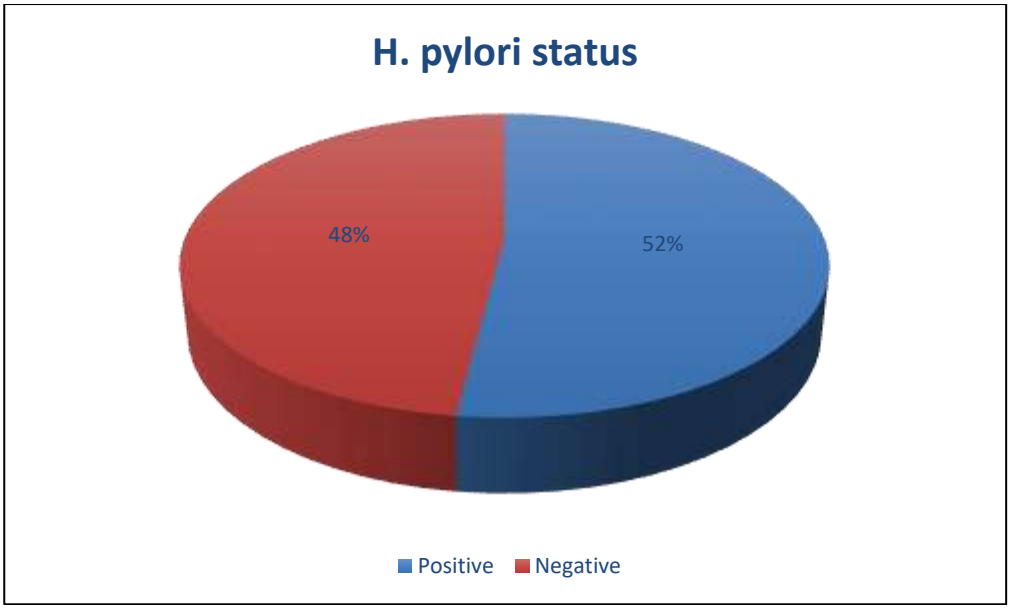

Figure 1. Frequency of *Helicobacter pylori* infection among study participants (n=150)

Among the 78 H. pylori-positive patients, mild bacterial density was the most common finding, occurring in 39 (50.0%) patients, followed by moderate density in 29 (37.2%). Severe H. pylori density was observed in 10 (12.8%) patients, as given in figure 2.

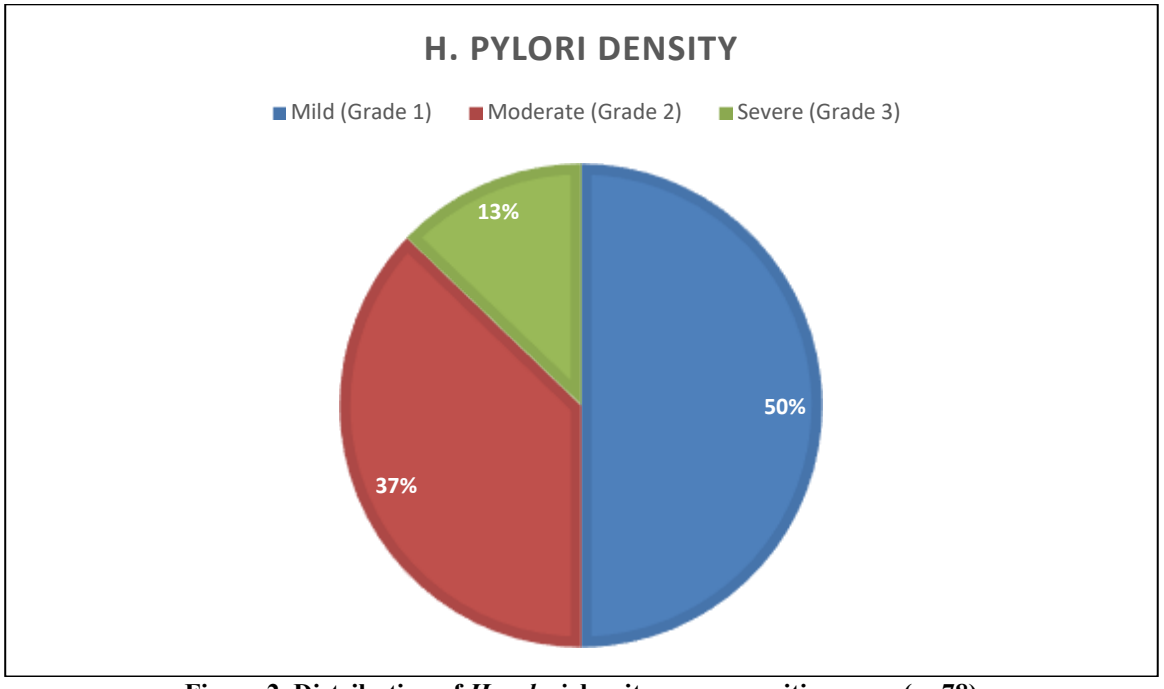

Figure 2. Distribution of *H. pylori* density among positive cases (n=78)

The association between *H. pylori* status and histopathological features showed that chronic inflammation was significantly different between *H. pylori*-positive and negative patients (p=0.003). Among *H. pylori*-positive patients, moderate chronic inflammation was most frequent, observed in 42 (53.8%), followed by severe inflammation in 17 (21.8%) and mild inflammation in 19 (24.4%). Similarly, neutrophilic activity was significantly associated with *H. pylori* status (p<0.001), with moderate and mild activity observed in 28 (35.9%) and 27 (34.6%) *H. pylori*-positive patients, respectively. In contrast, glandular atrophy was not significantly associated with *H. pylori* status (p=0.089), and intestinal metaplasia also showed no statistically significant association (p=0.126). As given in Table 4**.**

Table 4. Association of *H. pylori* status with histopathological features of chronic gastritis (n=150)

Histopathological feature	Grade	*H. pylori* positive n(%)	*H. pylori* negative n (%)	p-value
Chronic inflammation	Grade 0	0 (0.0)	4 (5.6)	0.003
	Grade 1	19 (24.4)	35 (48.6)	
	Grade 2	42 (53.8)	26 (36.1)	
	Grade 3	17 (21.8)	7 (9.7)	
Neutrophilic activity	Grade 0	13 (16.7)	39 (54.2)	<0.001
	Grade 1	27 (34.6)	21 (29.2)	
	Grade 2	28 (35.9)	9 (12.5)	
	Grade 3	10 (12.8)	3 (4.2)	
Glandular atrophy	Grade 0	56 (71.8)	62 (86.1)	0.089
	Grade 1	14 (17.9)	6 (8.3)	
	Grade 2	6 (7.7)	3 (4.2)	
	Grade 3	2 (2.6)	1 (1.4)	
Intestinal metaplasia	Grade 0	61 (78.2)	65 (90.3)	0.126
	Grade 1	11 (14.1)	4 (5.6)	
	Grade 2	5 (6.4)	2 (2.8)	
	Grade 3	1 (1.3)	1 (1.4)	

Grade 0 = absent, Grade 1 = mild, Grade 2 = moderate, Grade 3 = severe

Among the 78 *H. pylori*-positive patients, *H. pylori* density showed a significant positive correlation with chronic inflammation ($r=0.521$, $p<0.001$) and neutrophilic activity ($r=0.596$, $p<0.001$). A weaker but statistically significant positive correlation was also observed with glandular atrophy ($r=0.228$, $p=0.045$). In contrast, the correlation between *H. pylori* density and intestinal metaplasia was weak and statistically non-significant ($r=0.194$, $p=0.089$), as given in Table 5**.**

Table 5. Correlation between *H. pylori* density and histopathological parameters among *H. pylori*-positive patients (n=78)

Histopathological parameter	Correlation coefficient (r)	p-value
Chronic inflammation	0.521	<0.001
Neutrophilic activity	0.596	<0.001
Glandular atrophy	0.228	0.045
Intestinal metaplasia	0.194	0.089

DISCUSSION

Helicobacter pylori is a common gastric pathogen and an important cause of chronic gastritis. Persistent infection produces chronic mucosal inflammation through bacterial virulence factors and host immune responses.[13] Histopathological examination allows assessment of chronic inflammation, neutrophilic activity, glandular atrophy, and intestinal metaplasia. The Updated Sydney System provides a standardized approach for grading these histopathological changes and H. pylori density. Biochemical abnormalities may accompany the inflammatory response and can provide additional information about disease activity. Correlation of biochemical findings with histopathological severity can therefore provide a comprehensive assessment of H. pylori-associated chronic gastritis.[14,15]

The present study demonstrated that H. pylori infection was present in 52.0% of patients with chronic gastritis, with moderate chronic inflammation being the predominant histopathological finding. The infection was significantly associated with both chronic inflammation and neutrophilic activity, while no significant association was observed with glandular atrophy or intestinal metaplasia. Moreover, increasing H. pylori density was positively correlated with chronic inflammation, neutrophilic activity, and glandular atrophy. These findings collectively indicate that the inflammatory component of chronic gastritis was more closely related to active H. pylori infection and bacterial burden than the more advanced mucosal alterations assessed in the present study.

The H. pylori prevalence of 52.0% observed in our population was within the range reported in previous studies, although some variation was evident. Yakoob et al. (2010) reported a higher prevalence of 62.5%,[16] whereas the lower frequency of 38.0% reported by Anjana and Yevoor (2021) and 46.78% by Shukla et al. (2024) illustrates the variability of H. pylori detection across different study populations.[18,20] In contrast, the prevalence reported by Ullah et al. (2025), 55.0%, was very close to our finding. Such variation may reflect differences in geographic population, patient characteristics, disease severity, biopsy site, and diagnostic methodology rather than a

fundamental inconsistency in the epidemiology of infection.[21] The relatively close prevalence in our study and the recent Pakistani study by Ullah et al. also suggests that the 52.0% frequency observed in our patients was compatible with contemporary local findings.[21]

The association between infection and inflammatory activity was one of the most consistent findings across the available literature and was strongly supported by our results. In our study, chronic inflammation differed significantly according to H. pylori status (p=0.003), with moderate inflammation occurring in 42 (53.8%) infected patients compared with 26 (36.1%) uninfected patients, while severe inflammation was present in 17 (21.8%) and 7 (9.7%), respectively. This pattern agrees with the earlier observation of Yakoob et al. (2010), who found a significant relationship between H. pylori infection and gastric inflammatory activity (p=0.002).[16] It was also consistent with the findings of Ullah et al. (2025), who demonstrated a significant relationship between infection and moderate-to-severe inflammation (p<0.00001). Rather than simply indicating the presence of infection, these findings suggest that bacterial colonization was accompanied by a greater inflammatory burden within the gastric mucosa. The higher proportion of moderate and severe inflammation among our infected patients supports the recognized role of persistent H. pylori colonization in maintaining chronic mucosal inflammation.[21]

A similar and even stronger relationship was observed for neutrophilic activity. In our cohort, neutrophilic activity was significantly associated with H. pylori status (p<0.001); among infected patients, 27 (34.6%) had mild, 28 (35.9%) had moderate, and 10 (12.8%) had severe activity, whereas 39 (54.2%) of the uninfected patients had no neutrophilic activity. This finding closely agrees with Anjana and Yevoor (2021), who also reported a significant association between H. pylori infection and neutrophilic activity (p<0.001). The findings of Ullah et al. (2025) similarly showed a significant relationship between infection and neutrophilic activity (p<0.00001). The consistency across these studies is biologically plausible because neutrophilic infiltration represents an active inflammatory response to bacterial colonization. Thus, in our study, neutrophilic activity appeared to be a more responsive histological indicator of current H. pylori-associated inflammation than the chronic structural changes of the gastric mucosa.[21]

The relationship between bacterial density and inflammatory severity further strengthened this observation. Among the 78 infected patients, increasing H. pylori density correlated positively with chronic inflammation (r=0.521, p<0.001) and neutrophilic activity (r=0.596, p<0.001). The stronger correlation with neutrophilic activity suggests that bacterial burden may be particularly related to the active component of gastritis. This finding was in agreement with Khalid et al. (2018), who reported a positive correlation between H. pylori density and neutrophilic activity (r=0.416). The stronger correlation observed in our study (r=0.596) may reflect differences in the distribution of bacterial density or histopathological severity between the two study populations. Importantly, the direction of association was the same, supporting the concept that greater bacterial colonization accompanies greater inflammatory activity.[17]

Our findings concerning glandular atrophy and intestinal metaplasia were less consistent with some previous evidence. H. pylori density showed a weak but statistically significant correlation with glandular atrophy in our study (r=0.228, p=0.045), although overall glandular atrophy was not significantly associated with H. pylori status (p=0.089). Khalid et al. (2018) reported a stronger correlation between bacterial density and atrophy (r=0.306), suggesting a similar direction of relationship. In contrast, the relatively weak correlation in our study indicates that bacterial density alone may not adequately explain the development of glandular atrophy, which may also depend on duration of infection, host response, age, and other environmental factors.[17] The absence of a significant association between overall infection status and atrophy in our study is also compatible with Ullah et al. (2025), who reported no significant relationship between H. pylori infection and atrophy (p=0.430).[21]

For intestinal metaplasia, our findings differed from Khalid et al. (2018) and Shukla et al. (2024). Although H. pylori density showed a positive correlation with intestinal metaplasia in our patients, the relationship was weak and did not reach statistical significance (r=0.194, p=0.089), and the association between H. pylori status and intestinal metaplasia was also non-significant (p=0.126).[17,20] Khalid et al. reported a significant positive correlation between bacterial density and intestinal metaplasia (r=0.287),[17] while Shukla et al. found a statistically significant association between H. pylori and intestinal metaplasia.[20] The discrepancy may partly be related to the relatively limited number of patients with metaplastic changes in our study: intestinal metaplasia was absent in 126 (84.0%) patients and present in only 24 (16.0%). Conversely, the absence of a significant association in our study was consistent with the findings of Ullah et al. (2025), who reported intestinal metaplasia in only 0.6% of cases and found no significant association with H. pylori (p=0.149). These differences suggest that the relationship between infection and metaplastic transformation may be influenced by the duration and stage of gastric mucosal injury and may not be adequately represented by bacterial presence alone.[21]

The biochemical findings provided additional support for the inflammatory nature of H. pylori-associated gastritis. In our study, CRP was significantly higher in H. pylori-positive patients than in negative patients (8.64±4.21 vs. 5.17±2.96 mg/L, p<0.001), and CRP demonstrated positive correlations with chronic

inflammation (r=0.472, p<0.001), neutrophilic activity (r=0.513, p<0.001), and H. pylori density (r=0.438, p<0.001). These findings were consistent with the observations of Altun et al. (2019), who found higher hs-CRP levels in H. pylori-positive patients than in negative patients (2.5±1.9 vs. 2.0±1.7 mg/dL, p=0.001) and reported significant relationships between hs-CRP, severe chronic inflammation (p=0.015), and increasing H. pylori severity (p=0.001). Although the absolute CRP values cannot be directly compared because of differences in measurement units and assay characteristics, the consistent positive direction of the association supports a link between H. pylori-related gastric inflammation and systemic inflammatory activity.[19]

Overall, the present findings were most strongly aligned with previous evidence regarding the inflammatory component of H. pylori-associated gastritis. The significant associations with chronic inflammation and neutrophilic activity, together with the positive correlations between bacterial density and these parameters, indicate that bacterial burden was closely related to active gastric inflammation. In contrast, the weaker and largely non-significant relationships with atrophy and intestinal metaplasia suggest that these structural changes may develop through a more complex and prolonged process. The biochemical findings, particularly the relationship of CRP with both histopathological severity and bacterial density, further supported the inflammatory pattern observed histologically.

STUDY LIMITATIONS

The inclusion of biochemical parameters provided an additional dimension for assessing disease-associated changes. However, the cross-sectional design limited assessment of temporal or causal relationships. The single-center setting and relatively modest sample size may limit generalizability of the findings

CONCLUSION

H. pylori infection was frequently observed among patients with chronic gastritis and was significantly associated with chronic inflammation and neutrophilic activity. Increasing H. pylori density showed positive correlations with chronic inflammation, neutrophilic activity, and glandular atrophy. The combined assessment of histopathological and biochemical findings provided a broader characterization of H. pylori-associated chronic gastritis.

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REFERENCES:

1. Ma XZ, Zhou N, Luo X, Guo SQ, Mai P. Update understanding on diagnosis and histopathological examination of atrophic gastritis: A review. World journal of gastrointestinal oncology. 2024 Oct 15;16(10):4080.
2. Chinese Society of Gastroenterology, Cancer Collaboration Group of Chinese Society of Gastroenterology, Chinese Medical Association. Guidelines for diagnosis and treatment of chronic gastritis in China (2022, Shanghai). Journal of Digestive Diseases. 2023 Mar;24(3):150-80.
3. Ali A, AlHussaini KI. Helicobacter pylori: a contemporary perspective on pathogenesis, diagnosis and treatment strategies. Microorganisms. 2024 Jan 22;12(1):222.
4. Ali A, AlHussaini KI. Helicobacter pylori: a contemporary perspective on pathogenesis, diagnosis and treatment strategies. Microorganisms. 2024 Jan 22;12(1):222.
5. Li Y, Choi H, Leung K, Jiang F, Graham DY, Leung WK. Global prevalence of Helicobacter pylori infection between 1980 and 2022: a systematic review and meta-analysis. The lancet Gastroenterology & hepatology. 2023 Jun 1;8(6):553-64.
6. Almarmouri C, El-Gamal MI, Haider M, Hamad M, Qumar S, Sebastian M, Ghemrawi R, Muhammad JS, Burucoa C, Khoder G. Anti-urease therapy: a targeted approach to mitigating antibiotic resistance in Helicobacter pylori while preserving the gut microflora. Gut Pathogens. 2025 May 28;17(1):37.
7. Sharndama HC, Mba IE. Helicobacter pylori: an up-to-date overview on the virulence and pathogenesis mechanisms. Brazilian Journal of Microbiology. 2022 Mar;53(1):33-50.
8. Wang YK, Li C, Zhou YM, Zeng L, Li YY, Huang SL, Zhu CY, Wang Y, Wang SN, Chen XD. Histopathological features of Helicobacter pylori infection in gastric mucosa. Journal of Inflammation Research. 2022 Jan 1:6231-43.

9. Lee HS. Histopathologic diagnosis of H. pylori infection and associated gastric diseases. InHelicobacter pylori 2024 Mar 1 (pp. 143-152). Singapore: Springer Nature Singapore.
10. Ansari S, Yamaoka Y. Helicobacter pylori infection, its laboratory diagnosis, and antimicrobial resistance: a perspective of clinical relevance. Clinical microbiology reviews. 2022 Sep 21;35(3):e00258-21.
11. Săsăran MO, Meliț LE, Dobru ED. MicroRNA modulation of host immune response and inflammation triggered by Helicobacter pylori. International journal of molecular sciences. 2021 Jan 30;22(3):1406.
12. Shatila M, Thomas AS. Current and future perspectives in the diagnosis and management of Helicobacter pylori infection. Journal of Clinical Medicine. 2022 Aug 30;11(17):5086.
13. Clyne M, Ó Cróinín T. Pathogenicity and virulence of Helicobacter pylori: A paradigm of chronic infection. Virulence. 2025 Dec 31;16(1):2438735.
14. Santacroce L, Topi S, Cafiero C, Palmirotta R, Jirillo E. The role of the immune response to Helicobacter pylori antigens and its relevance in gastric disorders. Gastrointestinal Disorders. 2025 Jan 14;7(1):6.
15. Fei X, Li N, Xu X, Zhu Y. Macrophage biology in the pathogenesis of Helicobacter pylori infection. CritiCal reviews in MiCrobiology. 2025 May 4;51(3):399-416.
16. Yakoob MY, Hussainy AS. Chronic gastritis and Helicobacter pylori: a histopathological study of gastric mucosal biopsies. J Coll Physicians Surg Pak. 2010;20(11):773-5.
17. Khalid H, Zubair A, Kiran N, Aiza. Histopathological evaluation of H. pylori density and its correlation with activity, atrophy and intestinal metaplasia. J Islam Int Med Coll. 2018;13(1):26-31.
18. Anjana ML, Yevoor K. Evaluation of chronic gastritis with Helicobacter pylori using updated Sydney system. Int J Res Med Sci. 2021;9(9):2728-32.
19. Altun E, Dişibeyaz S, Köksal AS, et al. The role of high sensitive C-reactive protein and histopathological evaluation in chronic gastritis patients with or without Helicobacter pylori infection. Acta Cir Bras. 2019;34(3):e201900301.
20. Shukla GT, Yadav S, Shukla A, Yadav KK, Varma AV, Nandedekar S, Senger M, Gupta S. Histopathological features of chronic gastritis and its association with Helicobacter pylori infection. The Korean Journal of Gastroenterology. 2024 Oct 25;84(4):153-9.
21. Ullah O, Rahman H, Ijaz S. Helicobacter pylori-Associated Infection: A Comprehensive Histopathological Analysis of Gastric Biopsies from Patients of Pakistan. Microbiology Research. 2025 Nov 2;16(11):232.