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Research Article

Histological Footprint of Helicobacter Pylori: Insights from the Updated Sydney Grading System

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ABSTRACT
Background: Helicobacter pylori is a common chronic bacterial infection associated with chronic gastritis, peptic ulcer disease, gastric adenocarcinoma, and mucosa-associated lymphoid tissue lymphoma. Histopathological examination of gastroduodenal biopsies remains the gold standard for its detection and assessment of mucosal changes.
Aim: To determine the prevalence of H. pylori in gastroduodenal biopsies, evaluate associated histopathological changes using the Updated Sydney Classification, and correlate bacterial density with histological findings.
Materials and Methods: This prospective observational study included 122 gastroduodenal biopsy specimens collected over two years. Sections were stained with Hematoxylin and Eosin and modified Giemsa stain. Histopathological parameters were graded according to the Updated Sydney Classification.
Results: H. pylori was detected in 68 (55.7%) cases, with higher prevalence in gastric (58.4%) than duodenal (48.5%) biopsies. Chronic gastritis was the most frequent lesion. Moderate bacterial density was the commonest finding (41.9%), followed by severe (35.5%) and mild (22.6%) density. Active chronic gastritis was present in 51.6% of cases, and H. pylori density showed a significant association with neutrophilic activity (p=0.0094).
Conclusion: H. pylori infection was strongly associated with chronic gastritis and active inflammation. Routine histopathological evaluation using H&E, modified Giemsa staining, and the Updated Sydney Classification provides reliable detection and standardized grading, supporting its routine use in gastric biopsy reporting to guide clinical management and improve patient outcomes.

Keywords: Helicobacter Pylori; Chronic Gastritis; Gastroduodenal Biopsy; Histopathology; Updated Sydney Classification; Modified Giemsa Stain; Gastric Mucosa.

INTRODUCTION

Helicobacter pylori (H. pylori) is a spiral-shaped, Gram-negative, microaerophilic bacterium that selectively colonizes the gastric mucosa and remains one of the most common chronic bacterial infections affecting humans worldwide. Despite significant advances in sanitation and healthcare, nearly half of the global population continues to harbor this organism, with a substantially higher prevalence in developing countries due to overcrowding, poor hygiene, and lower socioeconomic conditions. H. pylori infection is recognized as a major public health concern because of its close association with chronic gastritis, peptic ulcer disease, gastric mucosal atrophy, intestinal metaplasia, mucosa-associated lymphoid tissue (MALT) lymphoma, and gastric adenocarcinoma. The World Health Organization has classified H. pylori as a Group I carcinogen owing to its established role in gastric carcinogenesis.[1,2]

The pathogenesis of H. pylori infection is complex and involves multiple bacterial virulence factors, including cytotoxin-associated gene A (CagA), vacuolating cytotoxin A (VacA), urease production, adhesins, and flagella-mediated motility. These factors enable the organism to survive in the acidic gastric environment, adhere to epithelial cells, evade host immune responses, and induce chronic inflammation. Persistent infection results in progressive mucosal injury, ultimately leading to structural and functional alterations in the gastric epithelium. However, the clinical manifestations vary considerably among individuals, ranging from asymptomatic colonization to severe gastroduodenal diseases depending on bacterial virulence, host genetic

susceptibility, immune response, and environmental influences.[3,4]

Histopathological examination of gastroduodenal biopsy specimens remains the cornerstone for evaluating H. pylori-associated gastric pathology. Endoscopic biopsy not only permits direct visualization of mucosal architecture but also allows simultaneous identification of the organism and assessment of associated inflammatory changes. Routine hematoxylin and eosin (H&E) staining often demonstrates chronic active gastritis characterized by lymphoplasmacytic infiltration, neutrophilic activity, glandular atrophy, and intestinal metaplasia. Special stains such as Giemsa, Warthin–Starry silver stain, and immunohistochemistry further enhance the detection of H. pylori, particularly in cases with low bacterial density or after partial treatment.[5,6]

The updated Sydney System continues to provide a standardized framework for the histopathological assessment of gastritis by grading parameters such as chronic inflammation, neutrophilic activity, glandular atrophy, intestinal metaplasia, and H. pylori density. This systematic evaluation assists pathologists in accurately correlating microscopic findings with disease severity and predicting the risk of progression to premalignant lesions. Recent international consensus guidelines also emphasize that every diagnosed H. pylori infection should be considered an infectious disease warranting eradication therapy, highlighting the importance of precise histopathological diagnosis in routine clinical practice.[2,7]

Gastroduodenal biopsies play a pivotal role in differentiating inflammatory, ulcerative, premalignant, and malignant lesions of the upper gastrointestinal tract. Although several non-invasive diagnostic modalities, including the urea breath test, stool antigen test, and serology, are available, histopathology remains indispensable because it simultaneously provides information regarding bacterial presence, mucosal morphology, disease activity, and associated complications. In addition, histopathological examination facilitates the detection of incidental lesions such as dysplasia, early gastric carcinoma, and MALT lymphoma, thereby influencing therapeutic decisions and long-term patient management.[5,8]

In countries with a high prevalence of H. pylori infection, understanding its histopathological spectrum is particularly important for early diagnosis and prevention of gastric cancer. Regional variations in bacterial prevalence, host response, and environmental factors further necessitate local studies to determine disease patterns. Evaluating gastroduodenal biopsies for H. pylori infection and associated morphological changes provides valuable epidemiological data while strengthening the role of pathology in guiding appropriate clinical intervention.[1,9,10]

The present study aims to determine the prevalence of Helicobacter pylori infection in gastroduodenal endoscopic biopsy specimens and evaluate the associated histopathological changes. The objectives are to assess the morphological patterns of chronic gastritis using the Updated Sydney grading system, grade the density of H. pylori colonization, correlate bacterial density with histological findings, and compare the study results with those reported in recent national and international literature.

MATERIALS AND METHODS

Study Design: Prospective observational, hospital-based histopathological study.

Study Population: Patients presenting with dyspeptic symptoms who underwent upper gastrointestinal endoscopy, with gastric (antrum and corpus) and/or duodenal biopsies submitted for histopathological examination.

Sample Size: A total of 122 endoscopic biopsy specimens were included in the study, comprising 89 gastric biopsies and 33 duodenal biopsies.

Study Duration: Two years (August 2015 to August 2017).

Study Place: Department of Pathology, Narayana Medical College and Hospital, Nellore, Andhra Pradesh, India.

Inclusion Criteria

- Patients of all age groups and both sexes presenting with symptoms of dyspepsia who underwent upper gastrointestinal endoscopy.
- Gastric (antrum and/or corpus) and duodenal biopsy specimens received for histopathological evaluation.
- Adequately preserved biopsy specimens suitable for microscopic examination.
- Patients who provided informed consent for endoscopy and biopsy.

Exclusion Criteria

- Inadequate, autolyzed, or poorly preserved biopsy specimens.
- Repeat biopsy specimens from the same patient during the study period.

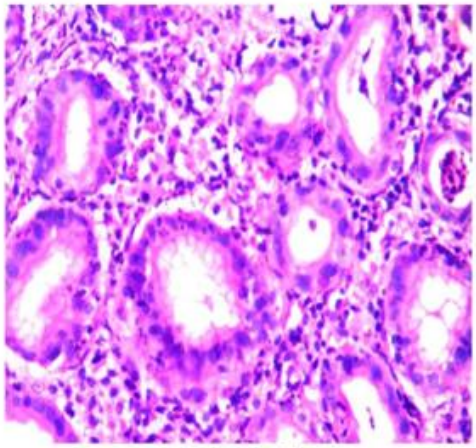
- Patients with incomplete clinical or endoscopic information.
- Biopsies showing extensive tissue artifacts that interfered with histopathological interpretation.
- Patients who declined participation or whose consent was unavailable.

Methodology

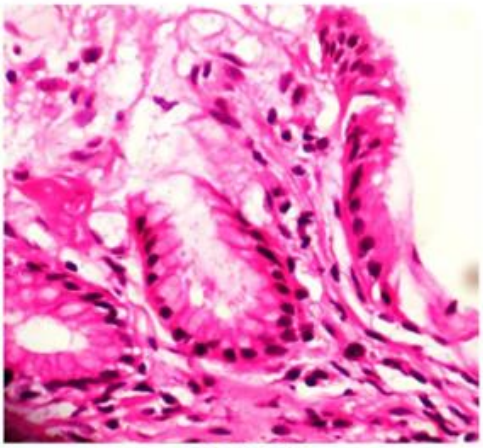
- Endoscopic biopsy specimens were immediately fixed in 10% neutral buffered formalin, processed routinely, and embedded in paraffin wax.
- Sections of 4 µm thickness were prepared using a rotary microtome.
- Tissue sections were stained with Hematoxylin and Eosin (H&E) for routine histopathological evaluation.
- Modified Giemsa stain was used for the identification of Helicobacter pylori organisms.
- Histopathological diagnosis was established based on microscopic examination of all biopsy specimens.
- Gastritis was graded according to the Updated Sydney Classification System using a semi-quantitative scoring method.
- The following parameters were graded as mild, moderate, or severe:
  - Helicobacter pylori density
  - Neutrophilic (activity) infiltration
  - Mononuclear inflammatory infiltrate
  - Glandular atrophy
  - Intestinal metaplasia
- Minor histopathological features were recorded as either present or absent.
- H. pylori colonization was assessed by evaluating the extent of bacterial involvement over the gastric surface epithelium.
- Inflammatory activity was determined by the density of neutrophilic infiltration within the gastric crypts.
- Chronic inflammation was assessed by the density of mononuclear inflammatory cell infiltrate.
- The extent of intestinal metaplasia and glandular atrophy was evaluated according to the proportion of affected gastric glands.
- The presence or absence of epithelial dysplasia was also documented as a potential premalignant histological finding.

Statistical Analysis: All statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) software, version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean ± standard deviation (SD), whereas categorical variables were expressed as frequency and percentage. The prevalence of Helicobacter pylori infection was compared across different demographic, clinical, endoscopic, and histopathological variables. Associations between categorical variables were assessed using the Pearson's Chi-square test, while Fisher's exact test was applied whenever the expected cell count was less than five. Histopathological parameters graded according to the Updated Sydney Classification (including Helicobacter pylori density, neutrophilic activity, chronic inflammation, intestinal metaplasia, and glandular atrophy) were analyzed using Chi-square or Fisher's exact test, as appropriate. A two-tailed p-value <0.05 was considered statistically significant. The results were presented in tables and graphs to facilitate comparison and interpretation of the findings.

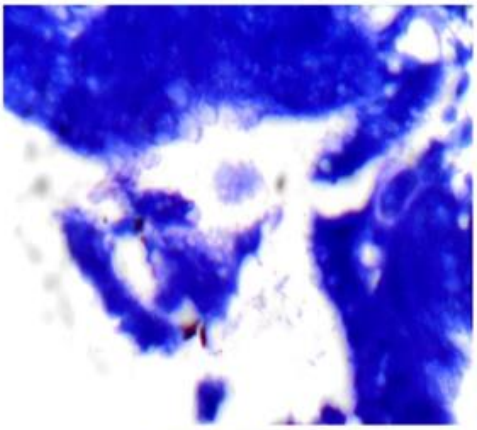
Gastroduodenal Biopsies



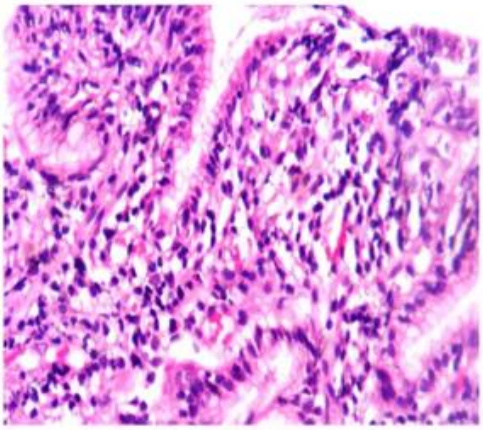
(H&E, X 400)
Chronic Gastritis with Acute Inflammation



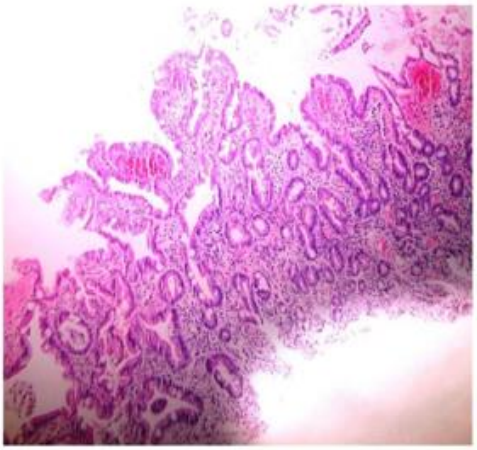
(H&E, X 1000)
Chronic Gastritis with Acute Inflammation showing Helicobacter Pylori



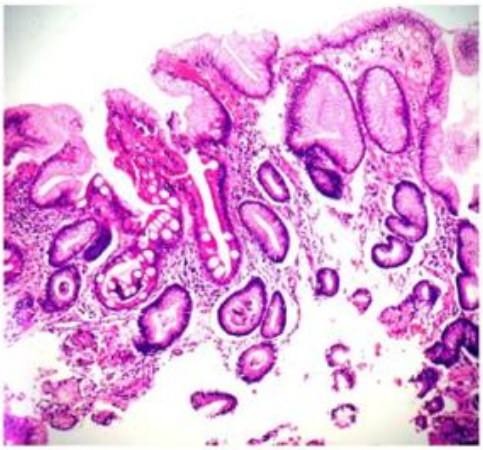
(GIEMSA X 1000)
Chronic Gastritis with Acute Inflammation showing Helicobacter Pylori



(H&E, X 400)
Chronic Gastritis with Chronic Inflammation

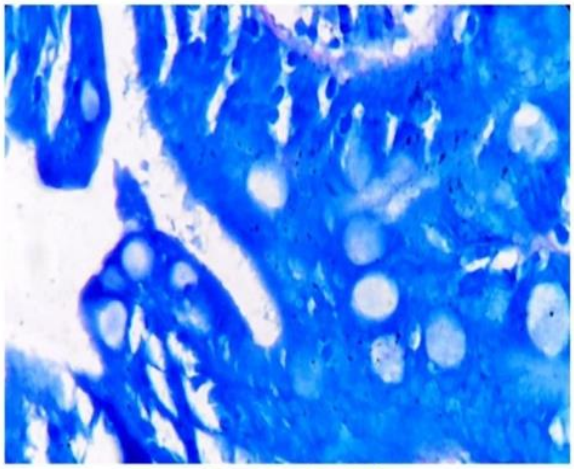


(H&E, X 100)
Chronic Gastritis with Intestinal Metaplasia

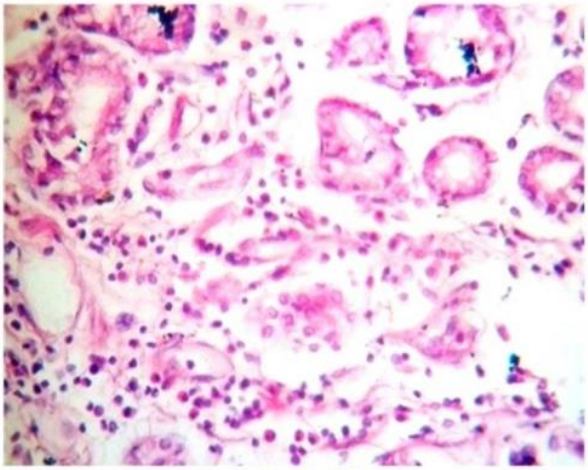


(H&E, X 400)
Chronic Gastritis with Intestinal Metaplasia

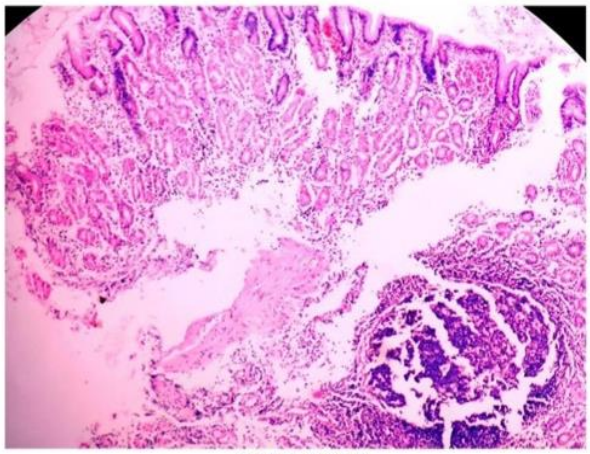
Gastroduodenal Biopsies



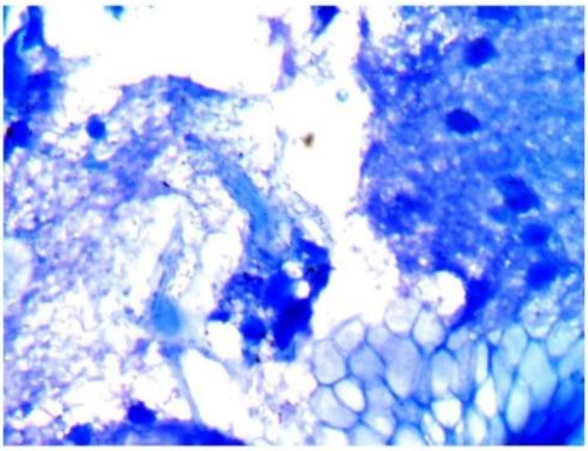
(GIEMSA X 1000)
Chronic Gastritis with Intestinal Metaplasia showing Helicobacter Pylori



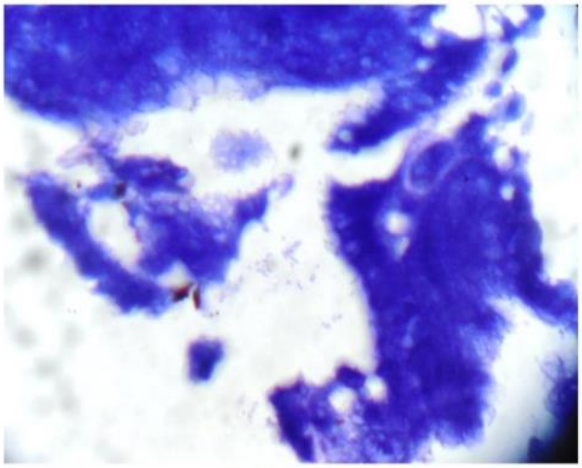
(H&E, X 400)
Chronic Gastritis with Glandular Atrophy



(H&E X 100)
Chronic Gastritis with Lymphoid Follicle



(GIEMSA, X 1000)
Helicobacter Pylori density 3+



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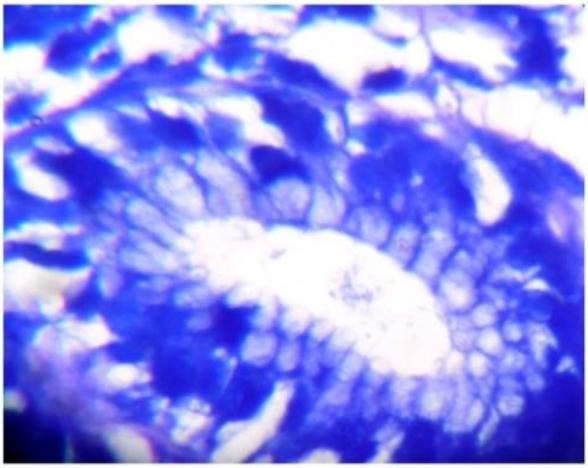
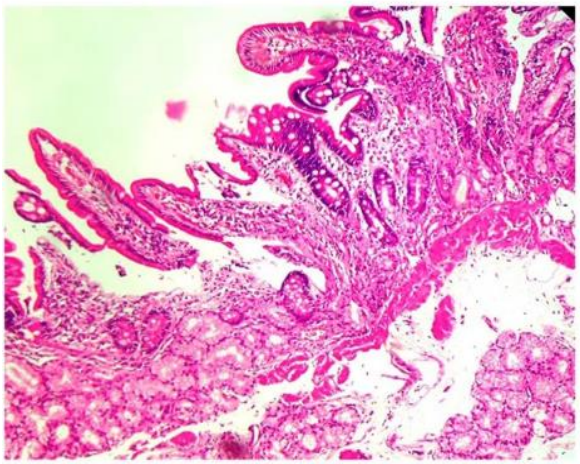
Helicobacter Pylori density 2+

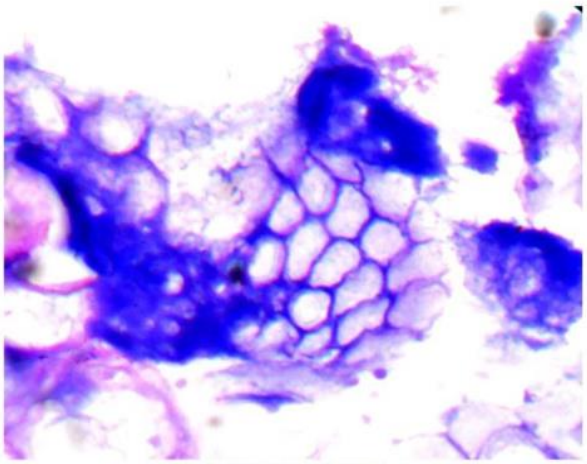


(GIEMSA, X 1000)
Helicobacter Pylori density 1+

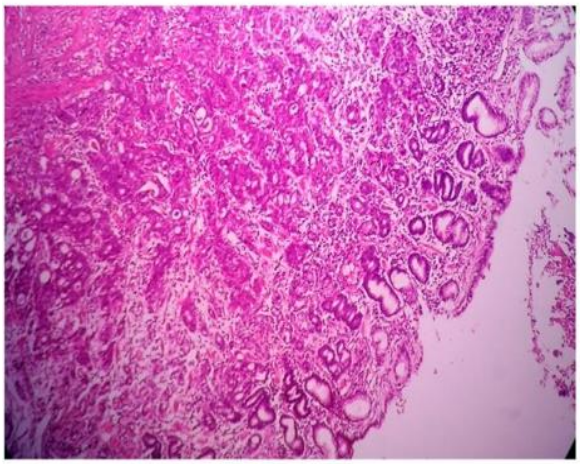
Gastroduodenal Biopsies



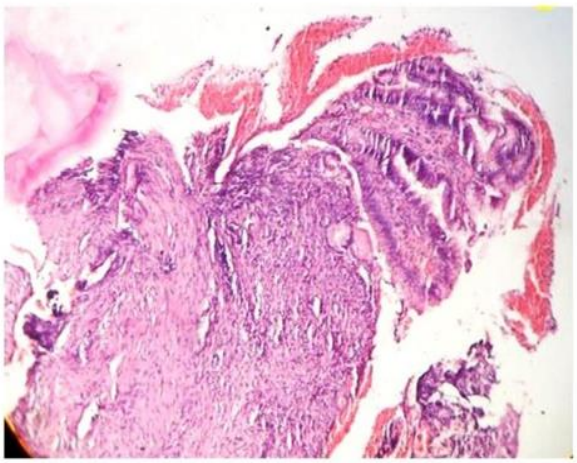
(H&E, X 100)
Chronic duodenitis



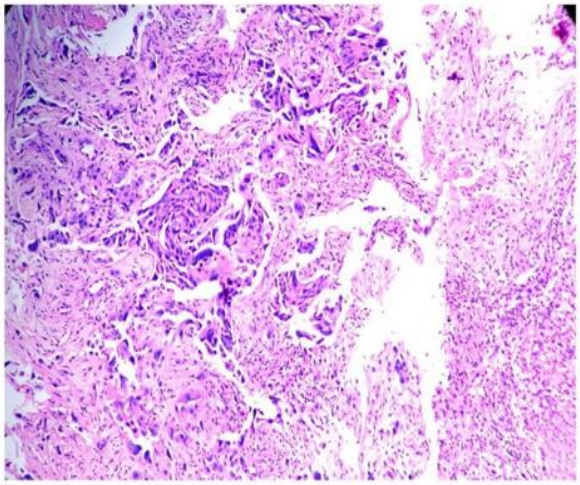
(GIEMSA X 1000)
Chronic Duodenitis with Helicobacter Pylori



(H&E, X 100)
Gastric Adenocarcinoma



(H&E, X 100)
Duodenal Ulcer



(H&E, X 100)
Duodenal Adenocarcinoma

RESULT

Table 1: Baseline Demographic and Clinicopathological Characteristics (N=122)

Variable	Category	H. Pylori Positive N (%)	H. Pylori Negative N (%)	Total N (%)	P-Value
Age Group	10–19	4	3	7	0.9462
	20–29	4	3	7	
	30–39	7	6	13	
	40–49	10	11	21	
	50–59	13	7	20	
	60–69	20	14	34	
	>70	10	10	20	
Gender	Male	47	34	81	0.4747
	Female	21	20	41	
Lesion Type	Benign	51	34	85	0.1508
	Malignant	17	20	37	

Table 2: Distribution of H. Pylori According To Site and Histopathological Lesions

Variable	Category	Positive	Negative	Total	P- Value
Site	Gastric	52	37	89	0.326
	Duodenal	16	17	33	
Gastric Lesions	Gastritis	31	19	50	0.4987
	Ulcer	6	3	9	
	Carcinoma	15	15	30	
Duodenal Lesions	Duodenitis	11	10	21	0.4713
	Ulcer	3	2	5	
	Carcinoma	2	5	7	

Table 3: Correlation of H. Pylori Density with Sydney System Histopathological Variables

Histological Variable	Absent	Mild	Moderate	Severe	P- Value
Neutrophilic Activity	15	3	9	4	0.0094
Chronic Inflammation	16	3	5	7	
Intestinal Metaplasia	27	1	2	1	
Glandular Atrophy	28	0	2	1	

Table 4: Association of H. Pylori with Other Histopathological Findings

Variable	Positive	Negative	P- Value
Lymphoid Follicles	7	5	0.0001
Active Chronic Gastritis	16	15	
Dysplasia	2	29	

Table 5: Updated Sydney Classification in H. Pylori Positive Chronic Gastritis (N=31)

Sydney Classification	Mild	Moderate	Severe	Total	P- Value
Inflammation (Active)	3	9	4	16	0.9077
Inflammation (Chronic)	3	5	7	15	
Atrophy	0	2	1	3	
Metaplasia	1	2	1	4	
H. Pylori Density	7	13	11	31	
Dysplasia	1	1	0	2	

Table 6: Summary of Histopathological Findings in H. Pylori Positive Cases (N=31)

Finding	Number (%)
Mild H. Pylori Density	7 (22.6)
Moderate H. Pylori Density	13 (41.9)
Severe H. Pylori Density	11 (35.5)

Active Chronic Gastritis	16 (51.6)
Chronic Gastritis Without Activity	15 (48.4)
Intestinal Metaplasia	4 (12.9)
Glandular Atrophy	3 (9.7)
Lymphoid Follicles	7 (22.6)
Dysplasia	2 (6.5)

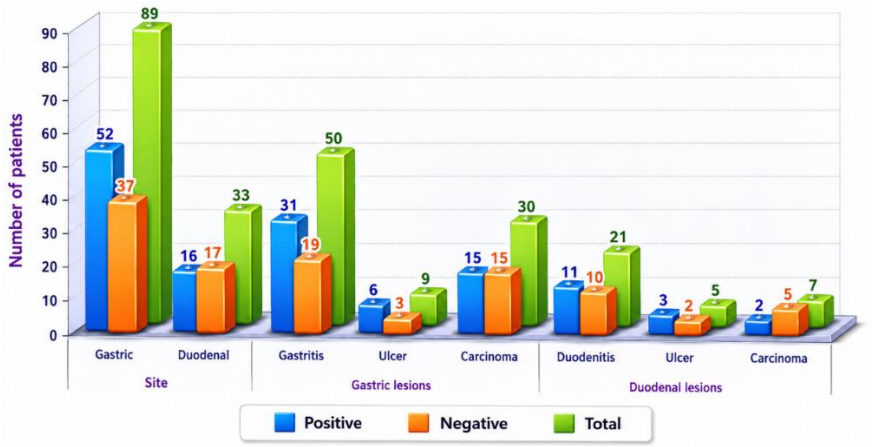


Figure: 1. Distribution of Helicobacter pylori Infection According to Anatomical Site and Histopathological Lesions

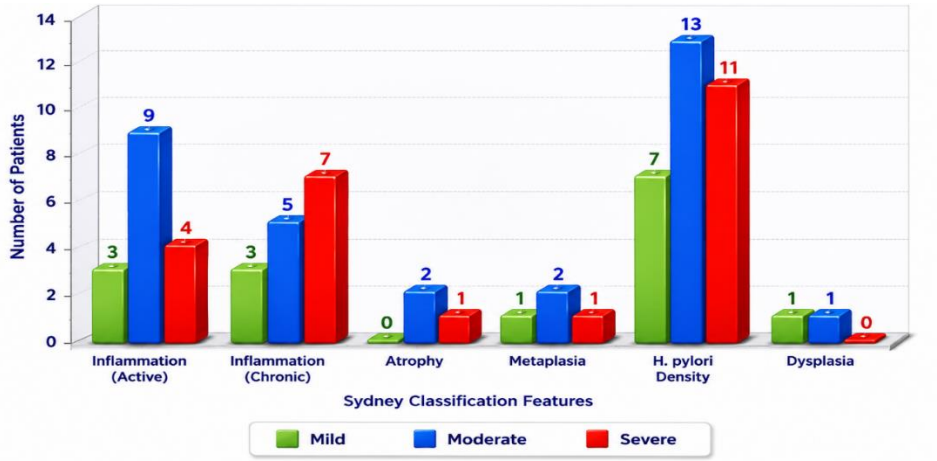


Figure: 2. Severity Distribution of Histopathological Changes According to the Updated Sydney Classification

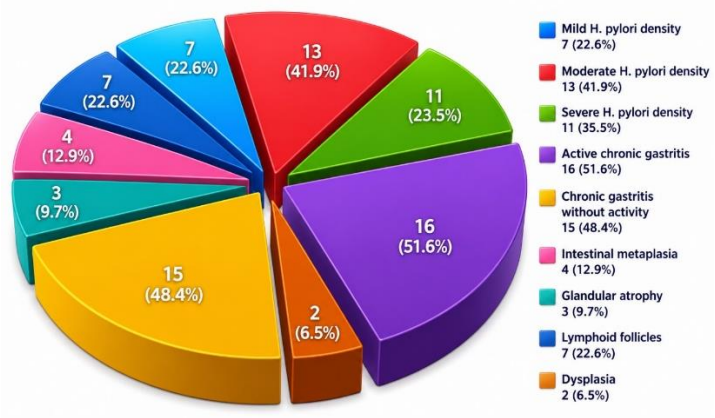


Figure: 3. Frequency Distribution of Histopathological Findings in Helicobacter pylori-Positive Gastric Biopsies

A total of 122 gastroduodenal biopsy specimens were included in the study, of which 68 (55.7%) were positive and 54 (44.3%) were negative for Helicobacter pylori. The highest number of patients belonged to the 60–69 years age group (34/122; 27.9%), followed by the 40–49 years (21/122; 17.2%), 50–59 years (20/122; 16.4%), and >70 years (20/122; 16.4%) age groups. Among the H. pylori-positive cases, the largest proportion was observed in patients aged 60–69 years (20 cases), followed by 50–59 years (13 cases) and 40–49 years (10 cases). However, the distribution of H. pylori positivity across different age groups was not statistically significant (p = 0.9462).

There was a clear male predominance, with 81 (66.4%) male patients and 41 (33.6%) female patients. Among males, 47 (58.0%) were positive for H. pylori, whereas 21 (51.2%) females were positive. Nevertheless, no statistically significant association was found between gender and H. pylori infection (p = 0.4747).

Regarding lesion type, 85 (69.7%) biopsy specimens represented benign lesions, while 37 (30.3%) were malignant. H. pylori positivity was observed in 51 (60.0%) benign lesions and 17 (45.9%) malignant lesions. Although infection was relatively more frequent in benign lesions, the association between lesion type and H. pylori positivity was not statistically significant (p = 0.1508).

Of the 122 biopsy specimens, 89 (73.0%) were obtained from the stomach and 33 (27.0%) from the duodenum. H. pylori positivity was identified in 52 (58.4%) gastric biopsies compared with 16 (48.5%) duodenal biopsies. Although gastric biopsies demonstrated a relatively higher prevalence of infection, the difference between gastric and duodenal sites was not statistically significant (p = 0.326).

Among gastric lesions, gastritis was the most frequent diagnosis (50 cases), with 31 (62.0%) cases demonstrating H. pylori positivity. Gastric ulcers accounted for 9 cases, of which 6 (66.7%) were positive, while 15 (50.0%) of 30 gastric carcinoma cases were positive for H. pylori. The association between different gastric lesions and H. pylori infection was not statistically significant (p = 0.4987).

Within the duodenum, duodenitis was the commonest lesion (21 cases), showing 11 (52.4%) positive cases. Duodenal ulcer constituted 5 cases, with 3 (60.0%) positive, whereas 2 (28.6%) of 7 duodenal carcinoma cases were positive for H. pylori. No statistically significant association was observed between the type of duodenal lesion and H. pylori infection (p = 0.4713).

The histopathological grading according to the Updated Sydney System demonstrated a significant relationship between H. pylori density and inflammatory changes. Regarding neutrophilic activity, 15 cases showed no activity, whereas 3, 9, and 4 cases demonstrated mild, moderate, and severe activity, respectively. This distribution was found to be statistically significant (p = 0.0094), indicating a close association between bacterial density and active inflammation.

For chronic mononuclear inflammation, 16 cases showed no significant inflammation, while 3, 5, and 7 cases exhibited mild, moderate, and severe chronic inflammation, respectively, reflecting increasing inflammatory infiltrates with higher bacterial colonization.

Intestinal metaplasia was absent in 27 cases, while only 1, 2, and 1 cases demonstrated mild, moderate, and severe metaplasia, respectively, indicating that metaplastic changes were relatively infrequent.

Similarly, glandular atrophy was absent in 28 cases, with only 2 moderate and 1 severe cases observed, suggesting that glandular atrophy occurred in a relatively small proportion of H. pylori-positive biopsies.

Among the 31 chronic gastritis cases positive for H. pylori, lymphoid follicles were identified in 7 cases, whereas 5 cases among the negative group also demonstrated lymphoid follicles. This association was found to be highly statistically significant (p = 0.0001), highlighting the strong relationship between H. pylori infection and lymphoid follicle formation.

Active chronic gastritis was observed in 16 positive cases compared with 15 negative cases. Although active inflammation was more common among infected patients, the difference was not statistically significant.

Dysplasia was detected in only 2 H. pylori-positive cases, while 29 cases showed no dysplastic changes, indicating that epithelial dysplasia was an uncommon finding in the present study.

Among the 31 cases of H. pylori-positive chronic gastritis, active inflammation was graded as mild in 3 (18.8%), moderate in 9 (56.2%), and severe in 4 (25.0%) of the 16 active gastritis cases. Chronic inflammation without active neutrophilic infiltration was observed in 15 cases, including 3 mild (20.0%), 5 moderate (33.3%), and 7 severe (46.7%) cases. The overall distribution of inflammatory grades did

not demonstrate statistical significance (p = 0.9077).

Glandular atrophy was identified in only 3 cases, including 2 moderate and 1 severe case, whereas intestinal metaplasia was present in 4 cases, comprising 1 mild, 2 moderate, and 1 severe lesion.

The density of H. pylori colonization showed that 7 (22.6%) cases had mild, 13 (41.9%) had moderate, and 11 (35.5%) had severe bacterial density, indicating that moderate colonization was the most frequently encountered pattern.

Dysplasia was uncommon and identified in only 2 cases, including 1 mild and 1 moderate lesion, with no severe dysplasia recorded.

Among the 31 H. pylori-positive chronic gastritis cases, moderate bacterial density was the predominant finding, observed in 13 (41.9%) patients, followed by severe density in 11 (35.5%) and mild density in 7 (22.6%) patients. Regarding inflammatory activity, 16 (51.6%) patients demonstrated active chronic gastritis, while 15 (48.4%) had chronic gastritis without active neutrophilic inflammation. Intestinal metaplasia was identified in 4 (12.9%) cases, and glandular atrophy was present in 3 (9.7%) cases. Lymphoid follicles, a characteristic histological feature associated with H. pylori infection, were observed in 7 (22.6%) patients. Dysplasia was the least frequent finding and was identified in only 2 (6.5%) cases. Overall, these findings indicate that moderate-to-severe H. pylori colonization was commonly associated with chronic inflammatory changes, whereas premalignant lesions such as intestinal metaplasia, glandular atrophy, and dysplasia were comparatively infrequent in the study population.

DISCUSSION

In the present study, the highest proportion of Helicobacter pylori infection was observed in patients aged 60–69 years, with a male predominance (66.4%). Although H. pylori positivity was more frequent among males and in older individuals, neither age nor gender showed a statistically significant association with infection. Similar observations were reported by Kamboj et al.[11], who found that H. pylori infection occurred predominantly in middle-aged and elderly adults with a slight male preponderance but without significant age- or sex-related differences. Likewise, Rana et al.[12] demonstrated that demographic variables were poor predictors of H. pylori infection, emphasizing that environmental and socioeconomic factors play a greater role than age or gender alone.

Gastric biopsies demonstrated a higher prevalence of H. pylori positivity than duodenal biopsies, and gastritis represented the commonest lesion associated with infection. However, no statistically significant association was observed between biopsy site or lesion type and H. pylori positivity. Comparable findings were reported by Al-Humayed et al.[13], who observed that H. pylori was detected predominantly in gastric mucosal biopsies, especially in chronic gastritis, while its prevalence in duodenal lesions and malignant conditions was comparatively lower. Similarly, Ahmed et al.[14] found chronic gastritis to be the most frequent histopathological diagnosis among H. pylori-infected patients, supporting the present findings.

The present study demonstrated a significant association between H. pylori density and neutrophilic activity, whereas intestinal metaplasia and glandular atrophy were observed only in a minority of cases. This finding supports the well-established concept that increasing bacterial colonization is associated with active mucosal inflammation. Similar results were reported by Shrestha et al.[15], who found a strong positive correlation between H. pylori density and the severity of chronic inflammatory infiltrates based on the Updated Sydney System. Likewise, Abdullah et al.[16] reported that higher bacterial density significantly correlated with active gastritis but showed a weaker association with intestinal metaplasia and glandular atrophy.

Lymphoid follicles were significantly associated with H. pylori infection in the present study, whereas dysplasia remained an uncommon finding. Similar observations were made by Khan et al.[17], who identified lymphoid follicle formation as one of the characteristic histological markers of chronic H. pylori infection. Furthermore, Lee et al.[18] demonstrated that although dysplasia may occur in longstanding infection, it is relatively uncommon in routine biopsy specimens and usually develops after prolonged progression through atrophy and intestinal metaplasia.

Moderate H. pylori density was the predominant histological pattern observed in the present study, followed by severe colonization. Chronic inflammation and active gastritis were the principal microscopic findings, whereas atrophy and intestinal metaplasia were relatively infrequent. Comparable results were reported by Singh et al.[19], who found that moderate

bacterial density was the most frequent grade and closely correlated with chronic inflammatory changes. Similarly, Molina-Infante et al.[20] observed that most biopsy-proven H. pylori gastritis cases exhibited mild-to-moderate colonization with variable inflammatory activity, while advanced precancerous lesions were comparatively uncommon.

Overall, the present study highlights that chronic active gastritis remains the most common histopathological manifestation of H. pylori infection, with moderate-to-severe bacterial density being associated with greater inflammatory activity. Premalignant lesions such as intestinal metaplasia, glandular atrophy, and dysplasia were encountered in relatively few patients. These findings are in agreement with recent literature, which emphasizes that histopathological examination using the Updated Sydney System remains a reliable method for evaluating H. pylori infection, grading mucosal inflammation, and identifying patients at risk for disease progression.[15,19]

CONCLUSION

The present study demonstrated that Helicobacter pylori infection remains a common finding in gastroduodenal biopsy specimens, with a higher prevalence in gastric lesions, particularly chronic gastritis. Histopathological examination using routine hematoxylin and eosin (H&E) and modified Giemsa staining, in conjunction with the Updated Sydney Classification, provides a reliable and standardized approach for detecting H. pylori infection and grading the associated mucosal changes. The predominance of moderate bacterial density and its strong association with active chronic inflammation further support the diagnostic value of systematic histopathological assessment. Although intestinal metaplasia, glandular atrophy, and dysplasia were observed in a smaller proportion of cases, their identification highlights the importance of early diagnosis and timely eradication therapy to prevent progression to premalignant and malignant gastric lesions. The findings of this study strongly support the routine incorporation of the Updated Sydney Classification into histopathology reporting, as it enables uniform assessment of gastritis severity, facilitates effective clinicopathological correlation, improves communication between pathologists and clinicians, and assists in guiding appropriate patient management and follow-up. Standardized Sydney grading should therefore be considered an integral component of routine gastric biopsy reporting to enhance diagnostic accuracy, optimize therapeutic decision-making, and improve long-term patient outcomes..

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