RESEARCH PAPER

Prevalence of Helicobacter pylori in Endoscopic Gastric Biopsies among Chronic Gastritis Patients: A Retrospective Study at a Tertiary Care Centre

Dr. Reddyvari Ganga Siva Subbaramireddy[1], Dr. Dinesh Kumar T[2*], Dr. Senthil Kumar[3], Dr. P Akshaya Poorani[4]

[1]Post Graduate, Department of General Surgery, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Kelambakkam - 603103, Tamil Nadu, India. Email: rgsreddi180@gmail.com

[2*]Professor of General Surgery, Department of General Surgery, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Kelambakkam - 603103, Tamil Nadu, India. Email: dineshkumar.t1986@gmail.com (Corresponding Author)

[3]Professor of General Surgery, Department of General Surgery, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Kelambakkam - 603103, Tamil Nadu, India. Email: drsenthilchri@gmail.com

[4]Assistant Professor, Department of General Surgery, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Kelambakkam - 603103, Tamil Nadu, India. Email: akshayapandurangan@gmail.com

ABSTRACT

Background: Helicobacter pylori is a highly prevalent gastric pathogen implicated in chronic gastritis and its complications, including peptic ulcer disease and gastric malignancy. Despite its significant burden, institutional data on prevalence and histopathological predictors remain limited in tertiary care settings in India.

Objectives: To determine the prevalence of H. pylori infection in endoscopic gastric biopsy specimens among patients with chronic gastritis, to evaluate associated histopathological findings, and to identify independent predictors of H. pylori positivity.

Methods: A retrospective observational study was conducted at a tertiary care teaching hospital over 12 months (August 2024–July 2025). A total of 100 patients diagnosed with chronic gastritis who underwent upper gastrointestinal endoscopy with biopsy were included using consecutive sampling. Data were collected from medical records, endoscopy registers, and histopathology reports. Variables included demographic characteristics, biopsy site, and histopathological features such as lymphoid follicles, foveolar hyperplasia, glandular atrophy, and intestinal metaplasia. Statistical analysis was performed using descriptive statistics, and associations were assessed using odds ratios with 95% confidence intervals. Logistic regression analysis was used to identify independent predictors.

Results: The prevalence of H. pylori infection was 35%. Among positive cases, mild colonization was most common (46%), followed by moderate (40%) and severe (14%). Lymphoid follicles (OR = 4.50, 95% CI: 1.80–11.20, $p < 0.001$) and foveolar hyperplasia (OR = 2.40, $p = 0.01$) showed significant association with infection. On multivariate analysis, lymphoid follicles (AOR = 3.90, $p = 0.002$) and antral involvement (AOR = 3.20, $p = 0.01$) were identified as independent predictors. No significant association was observed with gender or residence.

Conclusion: The study demonstrates a 35% prevalence of H. pylori among chronic gastritis patients in a tertiary care setting. Lymphoid follicle formation and antral biopsy site involvement are strong independent predictors of infection. These findings support routine H. pylori testing and targeted biopsy strategies to improve diagnostic accuracy and guide early treatment.

Keywords: Helicobacter pylori, chronic gastritis, gastric biopsy, prevalence, lymphoid follicles, histopathology

How to cite this article: Subbaramireddy RGS, Dinesh Kumar T, Senthil Kumar, Poorani PA. Prevalence of Helicobacter pylori in Endoscopic Gastric Biopsies among Chronic Gastritis Patients: A Retrospective Study at a Tertiary Care Centre. Int J Drug Deliv Technol. 2026;16(4): 90-98. DOI: 10.25258/ijddt.16.4.11

Source of support: Nil.

Conflict of interest: None

Introduction

Helicobacter pylori (H. pylori) is a gram-negative, spiral-shaped, microaerophilic bacterium that

colonises the human gastric mucosa and is recognised as one of the most prevalent chronic bacterial infections worldwide.[1] It is estimated to infect approximately 44% of the global population, with considerably higher prevalence rates observed in low- and middle-income countries, where rates may exceed 70-80% due to socioeconomic determinants such as poor sanitation, overcrowding, and limited access to clean drinking water.[2] In India, epidemiological data suggest a prevalence ranging from 40% to 80% depending on the region, age group, and socioeconomic status of the population studied.[3]

H. pylori infection plays a central and well-established role in the pathogenesis of a spectrum of upper gastrointestinal diseases. Following colonisation of the gastric antrum and body, the organism triggers a persistent inflammatory response characterised by infiltration of neutrophils and mononuclear cells into the lamina propria, culminating in the histopathological picture of chronic active gastritis.[4] Left untreated, this chronic inflammatory milieu progressively disrupts the normal gastric mucosal architecture, potentially leading to gastric ulceration, glandular atrophy, intestinal metaplasia, and in a subset of patients, gastric adenocarcinoma or mucosa-associated lymphoid tissue (MALT) lymphoma.[5] The bacterium was formally classified as a Group I carcinogen by the International Agency for Research on Cancer (IARC) in 1994, underscoring its oncogenic potential and the public health imperative of early detection.[6]

Chronic gastritis represents one of the most common indications for upper gastrointestinal endoscopy in clinical practice. Patients typically present with dyspepsia, epigastric pain, nausea, bloating, and early satiety. The Sydney System, updated in 1996, provides a standardised framework for the histopathological classification of gastritis, incorporating grading of H. pylori density, neutrophilic activity, chronic inflammation, glandular atrophy, and intestinal metaplasia at defined biopsy sites including the antrum, body, and incisura angularis.[7] Accurate histopathological reporting of gastric biopsies remains the gold standard for confirming H. pylori infection in routine clinical settings, particularly where non-invasive tests are unavailable or inconclusive.[8]

The antrum has long been recognised as the preferential site of H. pylori colonisation, particularly in patients with duodenal ulcer disease. Histopathological features strongly associated with H. pylori infection include the formation of lymphoid follicles in the lamina propria, foveolar hyperplasia, and neutrophilic infiltration indicative of active gastritis. Lymphoid follicle formation, in particular, is regarded as a highly specific histological marker for H. pylori-related gastritis and its presence on biopsy should prompt systematic evaluation for the organism.[9] Understanding the distribution of these histopathological findings across different biopsy sites carries important diagnostic and prognostic implications, especially for identifying patients at risk of advanced mucosal disease.[10]

Despite the well-documented global burden of H. pylori infection, its prevalence varies considerably across geographic regions, clinical settings, and patient populations, and robust institutional data from tertiary care centres in India remain essential to inform local clinical decision-making. The present retrospective study was therefore undertaken to determine the prevalence of H. pylori infection in endoscopic gastric biopsy specimens among patients with chronic gastritis presenting to a tertiary care teaching hospital, to characterise the associated histopathological findings, and to identify independent predictors of H. pylori positivity using logistic regression analysis. The findings are intended to contribute to the evidence base supporting systematic H. pylori testing and treatment protocols in comparable clinical settings.

Methodology

This study was a retrospective observational study, conducted by reviewing previously recorded patient data. It aimed to assess the prevalence of Helicobacter pylori infection in gastric biopsy specimens of chronic gastritis patients. The study was carried out in the Department of General Surgery at a tertiary care teaching hospital

This centre caters to a large patient population, making it suitable for obtaining adequate biopsy samples and clinical records. The study was conducted over a period of 12 months (August 2024 to July 2025). All eligible cases within this time frame were included for analysis.

Study Population

The study population consisted of patients:

- Diagnosed clinically with chronic gastritis
- Undergoing upper gastrointestinal (UGI) endoscopy
- Who had gastric biopsy specimens collected

Sample Size

A total of 100 cases were included in the study.

The sample size was determined based on:

- Availability of eligible case records during the study period
- Feasibility within the retrospective design

Sampling Technique

A consecutive sampling method was used:

- All patients meeting the inclusion criteria during the study period were included
- This minimizes selection bias and ensures representativeness of routine clinical practice

Inclusion Criteria

- Presented with symptoms suggestive of chronic gastritis
- Underwent UGI endoscopy with biopsy
- Had complete demographic details (age, sex, residence)
- Had histopathological reports of gastric biopsy (antrum, body, fundus, etc.)

Exclusion Criteria

- Had taken proton pump inhibitors (PPIs) or antibiotics within 4 weeks
- Had a history of previous gastric surgery
- Had inadequate or insufficient biopsy specimens

Data Collection Procedure

Data were collected retrospectively from hospital medical records, endoscopy registers, and histopathology reports of patients diagnosed with chronic gastritis who underwent upper gastrointestinal endoscopy during the study period. Relevant variables including demographic details such as age, sex, residence, and socioeconomic status were recorded. Information regarding biopsy sites (antrum, body, and fundus) and endoscopic findings were also documented. Histopathological examination reports were reviewed to extract details on the presence of *Helicobacter pylori* and associated features such as chronic inflammation, inflammatory activity, glandular atrophy, intestinal metaplasia, lymphoid follicles, foveolar hyperplasia, and ulceration. All data were systematically entered into a structured data collection sheet for further analysis.

Data Analysis

The collected data were compiled and entered into a computerized database using statistical software such as Microsoft Excel or SPSS. Descriptive statistics were applied to summarize the data. Categorical variables such as gender distribution, biopsy site involvement, histopathological findings, and *H. pylori* positivity were expressed as frequencies and percentages. Continuous variables like age were presented as mean and standard deviation. The prevalence of *H. pylori* infection was calculated as a proportion of total cases. The results were presented using tables, charts, and graphs for better interpretation and comparison.

Ethical Considerations

Ethical approval for the study was obtained from the Institutional Ethics Committee prior to data collection. As this was a retrospective study, informed consent from patients was waived. Confidentiality of patient information was strictly maintained by anonymizing all data and avoiding the use of personal identifiers. The data were used solely for academic and research purposes, and all procedures were conducted in accordance with ethical principles and institutional guidelines.

Results

Table 1: Demographic Characteristics of study participants (n = 100)

Variable	Category	n (%)
Age (years)	Mean ± SD	42 ± 14
	21–60 years	82 (82%)
Gender	Male	38 (38%)
	Female	62 (62%)
Residence	Urban	71 (71%)
	Rural	29 (29%)

Table 1 shows the demographic profile of the study participants (n = 100). The mean age of the participants was 42 ± 14 years, with the majority (82%) belonging to the 21–60 years age group. Females constituted a higher proportion (62%) compared to males (38%). Most of the participants were from urban areas (71%), while 29% were from rural areas.

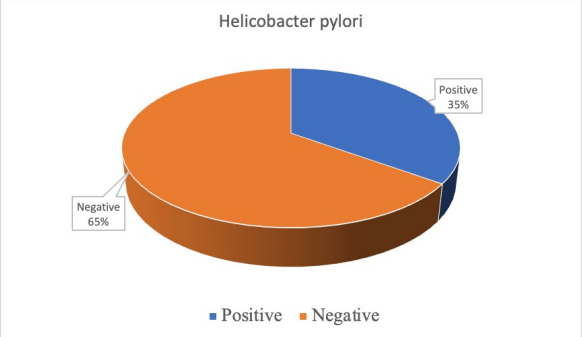

Figure 1: Prevalence of Helicobacter pylori (n = 100)

Figure 1 illustrates the prevalence of Helicobacter pylori infection among the study population. Out of 100 participants, 35% were positive for H. pylori, while 65% were negative.

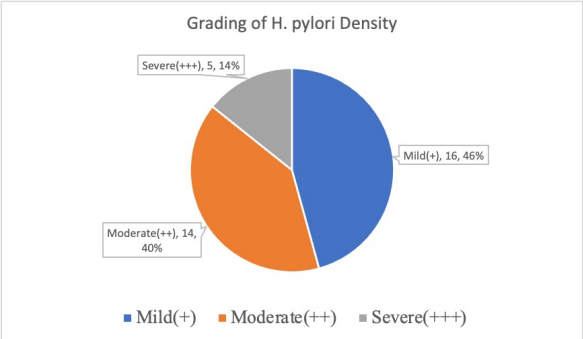

Figure2: Grading of *H. pylori* Density (n = 35)

Figure 2 shows the distribution of *H. pylori* density among positive cases (n = 35). Mild colonization was the most common (46%), followed by moderate (40%) and severe (14%) grades.

Table 2: Association of Demographic Variables with H. pylori

Variable	Category	Positive n (%)	Negative n (%)	p-value
Gender	Male	15 (42.9%)	23 (35.4%)	0.48
	Female	20 (57.1%)	42 (64.6%)	
Residence	Urban	26 (74.3%)	45 (69.2%)	0.62
	Rural	9 (25.7%)	20 (30.8%)	

Table 2 presents the association between demographic variables and *Helicobacter pylori* infection. Among the positive cases, 57.1% were females and 42.9% were males. Urban residents accounted for 74.3% of positive cases. However, no statistically significant association was observed between *H. pylori* infection and gender (p = 0.48) or residence (p = 0.62).

Table 3: Association of Histopathological Findings with H. pylori

Finding	Category	n (%)	OR	95% CI	p-value
Lymphoid follicles	Present	22 (62.9%)	4.50	1.80 – 11.20	**<0.001***
	Absent (Reference)	13 (37.1%)	1.00		
Foveolar hyperplasia	Present	10 (28.6%)	2.40	1.05 – 5.80	**0.01***
	Absent (Reference)	25 (71.4%)	1.00		
Glandular atrophy	Present	3 (8.6%)	1.80	0.45 – 7.20	0.08
	Absent (Reference)	32 (91.4%)	1.00		
Intestinal metaplasia	Present	2 (5.7%)	1.50	0.30 – 6.80	0.12
	Absent (Reference)	33 (94.3%)	1.00		
Ulcerated lesions	Present	4 (11.4%)	1.30	0.40 – 4.10	0.30
	Absent (Reference)	31 (88.6%)	1.00		

Table 3 demonstrates the association between histopathological findings and *H. pylori* infection. Lymphoid follicles were significantly associated with *H. pylori* infection (OR = 4.50, 95% CI: 1.80–11.20, p < 0.001). Foveolar hyperplasia also showed a significant association (OR = 2.40, 95% CI: 1.05–5.80, p = 0.01). In contrast, glandular atrophy, intestinal metaplasia, and ulcerated lesions did not show statistically significant associations.

Table 4: Association of Biopsy Site (Antrum) with Histopathological Findings

Finding	Category	Antrum n (%)	Other Sites n (%)	OR	95% CI	p-value
Lymphoid follicles	Present	25 (71.4%)	10 (28.6%)	3.80	1.50 – 9.50	**<0.001***
	Absent (Reference)	10 (28.6%)	25 (71.4%)	1.00		

Foveolar hyperplasia	Present	18 (51.4%)	5 (14.3%)	5.20	1.70 – 15.80	**<0.001***
	Absent (Reference)	17 (48.6%)	30 (85.7%)	1.00		
Glandular atrophy	Present	3 (8.6%)	1 (2.9%)	2.10	0.20 – 20.0	0.05
	Absent (Reference)	32 (91.4%)	34 (97.1%)	1.00		
Intestinal metaplasia	Present	2 (5.7%)	1 (2.9%)	1.90	0.15 – 22.0	0.10
	Absent (Reference)	33 (94.3%)	34 (97.1%)	1.00		

Table 4 shows the association between biopsy site and histopathological findings. The antral region demonstrated a significantly higher prevalence of lymphoid follicles (OR = 3.80, p < 0.001) and foveolar hyperplasia (OR = 5.20, p < 0.001) compared to other sites. Glandular atrophy showed borderline significance (p = 0.05), while intestinal metaplasia did not show a significant association.

Table 5: Logistic Regression Analysis for Predictors of H. pylori Infection

Variable	**Category**	**OR**	**95% CI**	**p-value**	**AOR**	**95% CI**	**p-value**
Lymphoid follicles	**Present**	4.50	1.80 – 11.20	<0.001	3.90	1.50 – 10.10	**0.002***
	Absent (Reference)	1.00			1.00		
Foveolar hyperplasia	**Present**	2.40	1.05 – 5.80	0.01	2.10	0.90 – 4.80	0.08
	Absent (Reference)	1.00			1.00		
Antral involvement	**Yes**	3.80	1.50 – 9.50	<0.001	3.20	1.20 – 8.50	**0.01***
	No (Reference)	1.00			1.00		

Table 5 presents the results of logistic regression analysis for predictors of *H. pylori* infection. On multivariate analysis, lymphoid follicles (AOR = 3.90, 95% CI: 1.50–10.10, p = 0.002) and antral involvement (AOR = 3.20, 95% CI: 1.20–8.50, p = 0.01) were identified as independent predictors. Foveolar hyperplasia did not retain statistical significance after adjustment (AOR = 2.10, p = 0.08).

Discussion

The present retrospective study examined the prevalence of *Helicobacter pylori* infection in endoscopic gastric biopsy specimens among 100 patients with chronic gastritis at a tertiary care teaching hospital in India. An overall prevalence of 35% was recorded, with the majority of positive cases demonstrating mild to moderate colonisation density. The study further characterised the histopathological correlates of infection and identified lymphoid follicle formation and antral biopsy site involvement as independent predictors of *H. pylori* positivity on multivariate logistic regression analysis. The following sections interpret these findings in the context of existing literature and discuss their clinical implications.

Prevalence of H. pylori infection

The overall prevalence of *H. pylori* infection in this series was 35%, a figure that is broadly consistent with the reported prevalence range in tertiary care hospital-based studies across India, which spans 30% to 60% depending on the geographic region, study design, and detection method employed.[11] The lower end of this range, which includes the present study, likely reflects the patient profile at a tertiary centre — where prior antibiotic or proton pump inhibitor (PPI) exposure, exclusion of cases with recent antibiotic use, and a higher proportion of urban patients may collectively reduce the observed prevalence relative to community-based estimates.

Globally, *H. pylori* prevalence in symptomatic patients undergoing endoscopy has been reported between 30% and 70% across different settings, with the highest rates documented in sub-Saharan Africa and South Asia.[2] A systematic review and meta-analysis by Hooi et al. estimated a global prevalence of approximately 44% in the general population, with South Asia contributing rates of 35-45% in endoscopy-based series.[2] The 35% prevalence observed in the current study is therefore in close agreement with the existing literature and reflects the genuine epidemiological burden of infection at this institutional level.

Grading of H. pylori colonisation density

Among the 35 positive cases, mild colonisation was the most frequent grade (46%), followed by moderate (40%) and severe (14%). This distribution — where mild and moderate grades together account for 86% of positive cases — is consistent with published data from Indian tertiary hospitals, where heavy bacterial loads are less frequently encountered than in primary care or community settings.[8] The predominance of mild colonisation in hospital-based series has been attributed to several factors, including partial suppression of bacterial density by prior antisecretory therapy, dilution of heavily colonised antral mucosa by sampling from multiple biopsy sites, and a relative enrichment of urban, health-seeking patients in tertiary referral populations.

The clinical significance of colonisation density merits attention. Studies have demonstrated a dose-response relationship between *H. pylori* density and the severity of neutrophilic infiltration, mucosal inflammatory activity, and the degree of glandular injury.[12] Higher bacterial loads have also been associated with a greater likelihood of virulent strains expressing CagA and VacA virulence factors, which are key determinants of ulcerogenicity and carcinogenic risk. The low proportion of severe colonisation in the present series may therefore partly explain the relatively low rates of glandular atrophy and intestinal metaplasia observed.

Demographic characteristics and their association with H. pylori

The study population had a mean age of 42 ± 14 years, with 82% of participants in the 21-60 year age group. This age distribution is typical of symptomatic chronic gastritis cohorts presenting for endoscopy, as this working-age group bears the greatest burden of dyspeptic symptoms and is most likely to seek endoscopic evaluation.[13] Females constituted the majority of the study population (62%), which may reflect the referral patterns and gender composition of the surgical outpatient department at this institution rather than a true epidemiological gender preponderance.

No statistically significant association was observed between *H. pylori* infection and gender ($p = 0.48$) or residential status ($p = 0.62$) in this study. This is consistent with the established epidemiological view that while socioeconomic factors such as sanitation and crowding are major determinants of *H. pylori* acquisition at the population level, gender and urban versus rural residence do not independently predict infection status within a hospital-based symptomatic cohort of comparable socioeconomic background.[13] Several Indian studies have similarly reported no significant gender difference in *H. pylori* prevalence among endoscopy patients, supporting the observation that both sexes are equally susceptible to infection once exposed to the organism.

Lymphoid follicles as a histopathological marker of H. pylori infection

The most striking histopathological finding in this study was the strong and statistically significant association between lymphoid follicle formation and *H. pylori* positivity (OR = 4.50, 95% CI: 1.80-11.20, $p < 0.001$), which remained robust on multivariate analysis (AOR = 3.90, 95% CI: 1.50-10.10, $p = 0.002$). Lymphoid follicles were identified in 62.9% of *H. pylori*-positive cases, compared to 37.1% of negative cases.

This finding is strongly corroborated by the existing literature. Genta et al. first established in a landmark study that lymphoid follicle formation in the gastric lamina propria is a highly specific and sensitive histological indicator of *H. pylori* infection, occurring in response to the organism's potent B-cell stimulatory activity mediated through its outer membrane proteins.[5] The formation of organised lymphoid aggregates with germinal centres represents a characteristic tissue response to persistent *H. pylori* colonisation and is the same immunological substrate that underlies the pathogenesis of gastric MALT lymphoma, the development of which is strongly dependent on sustained antigenic stimulation by *H. pylori.*[14] The high odds ratio observed in the present study reinforces the diagnostic value of lymphoid follicles as a surrogate histological marker for *H. pylori* infection, particularly in settings where Giemsa or immunohistochemical staining for the organism may not routinely be performed.

Foveolar hyperplasia and its significance

Foveolar hyperplasia was identified in 28.6% of *H. pylori*-positive cases and showed a significant univariate association with infection (OR = 2.40, 95%

CI: 1.05-5.80, p = 0.01). However, this association did not retain statistical significance on multivariate analysis after adjustment for lymphoid follicles and antral involvement (AOR = 2.10, p = 0.08), suggesting that foveolar hyperplasia is a dependent rather than independent marker of *H. pylori* infection in this cohort.

Foveolar hyperplasia is a reactive mucosal change characterised by elongation and tortuosity of the foveolae, occurring in response to chronic mucosal injury and the sustained elevation of gastrin levels triggered by *H. pylori*-induced antral inflammation.[15] It is well recognised as a non-specific reactive change that accompanies chronic gastritis of varying aetiology, including chemical or reactive gastropathy due to bile reflux or non-steroidal anti-inflammatory drug use, which likely accounts for its loss of independent significance after multivariate adjustment. Nonetheless, its presence on biopsy should heighten clinical suspicion for *H. pylori* infection and prompt specific organism identification, particularly when lymphoid follicles are co-present.

Glandular atrophy, intestinal metaplasia, and ulcerated lesions

Glandular atrophy, intestinal metaplasia, and ulcerated lesions did not show statistically significant associations with *H. pylori* positivity in this study (p = 0.08, p = 0.12, and p = 0.30, respectively). Atrophy was present in only 8.6% and intestinal metaplasia in 5.7% of positive cases. These relatively low rates deserve consideration in the context of study design and patient demographics.

The Correa cascade describes a well-established multistep histopathological progression from chronic non-atrophic gastritis through atrophic gastritis, intestinal metaplasia, dysplasia, and ultimately gastric adenocarcinoma.[5] This cascade typically unfolds over decades, and its advanced stages — atrophy and metaplasia — are therefore more prevalent in older cohorts with longer infection duration. The mean age of 42 years in the present study population, combined with the retrospective design and the 12-month study window, may have been insufficient to capture a substantial burden of these advanced pre-malignant changes. Additionally, the exclusion of patients with recent PPI use would have removed a subset of patients with severe mucosal disease requiring antisecretory therapy, potentially underestimating the true prevalence of atrophic changes in the endoscopy population.

Importance of antral biopsy site

Antral biopsy site involvement was confirmed as an independent predictor of *H. pylori* infection on multivariate analysis (AOR = 3.20, 95% CI: 1.20-8.50, p = 0.01). The antrum demonstrated a significantly higher prevalence of both lymphoid follicles (OR = 3.80, p < 0.001) and foveolar hyperplasia (OR = 5.20, p < 0.001) compared to body and fundal biopsy sites.

This finding is entirely consistent with the well-established tropism of *H. pylori* for the antral mucosa, particularly in patients without concomitant acid hypersecretion or atrophy that might shift the distribution of colonisation proximally. El-Zimaity and Graham demonstrated that antral biopsies alone provide the highest diagnostic yield for *H. pylori* detection in most clinical scenarios, and that the incorporation of additional corpus biopsies according to the Updated Sydney System significantly improves diagnostic sensitivity only in selected circumstances such as PPI use or advanced atrophic disease.[10] The practical implication for endoscopists in a resource-limited setting is that targeted antral sampling — at minimum from the greater and lesser curvatures of the antrum — should be prioritised for *H. pylori* detection in patients with chronic gastritis symptoms.

Multivariate predictors and clinical implications

Logistic regression analysis identified two independent predictors of *H. pylori* positivity: lymphoid follicle formation (AOR = 3.90) and antral biopsy site involvement (AOR = 3.20). These two variables together define a practical histopathological profile that, in the absence of organism identification by Giemsa or immunohistochemical staining, should be treated as a strong surrogate indicator for *H. pylori* infection by the reporting pathologist and the treating clinician.

From a clinical standpoint, the findings of this study support the adoption of a systematic *H. pylori* testing and treatment protocol in all patients undergoing endoscopy for chronic gastritis at this institution. The Maastricht V/Florence Consensus guidelines recommend a test-and-treat strategy for all patients with *H. pylori* infection, irrespective of the severity of presenting symptoms, given the significant reduction in peptic ulcer disease recurrence, regression of low-grade MALT lymphoma, and potential reduction in gastric cancer risk that eradication therapy confers.[8] The 35% institutional prevalence documented here provides a robust evidence base for justifying this investment at the departmental and hospital policy level.

Limitations of the study

This study has several limitations that must be acknowledged. The retrospective design introduces the inherent risk of incomplete data and selection bias, as only patients with complete histopathological and demographic records were eligible for inclusion. The detection of *H. pylori* was based exclusively on histopathological examination of haematoxylin and eosin (H&E) stained sections, without systematic use of Giemsa staining, Warthin-Starry silver stain, or immunohistochemistry, which may have resulted in some degree of underestimation of true infection prevalence, particularly in cases of sparse colonisation. Non-invasive confirmatory tests such as the urea breath test or stool antigen test were not employed. The relatively modest sample size of 100 cases, while adequate for a preliminary institutional audit, limits the statistical power to detect modest associations, particularly for low-frequency histological outcomes such as glandular atrophy and intestinal metaplasia. Finally, the absence of virulence factor analysis (CagA, VacA status) prevents further stratification of infection risk. A prospective study with standardised multi-site biopsy protocols, confirmatory staining, and virulence typing would substantially strengthen these findings.

Conclusion

This retrospective study demonstrates an *H. pylori* prevalence of 35% among chronic gastritis patients undergoing endoscopic biopsy at a tertiary care centre, consistent with regional and national data. Histopathologically, lymphoid follicle formation and antral biopsy site involvement emerged as independent predictors of *H. pylori* positivity, reinforcing their diagnostic utility in routine pathological reporting. The predominance of mild to moderate colonisation density and the low rates of glandular atrophy and intestinal metaplasia in this cohort suggest that a significant proportion of patients are being identified at an early, reversible stage of mucosal disease. These findings have direct implications for institutional policy: systematic *H. pylori* testing of all patients undergoing endoscopy for chronic gastritis, coupled with prompt eradication therapy in positive cases and endoscopic surveillance for those with atrophic changes, is warranted. Prospective studies with larger sample sizes, standardised biopsy protocols, and supplementary staining techniques are recommended to further validate and expand upon these observations.

References

1. Suerbaum S, Michetti P. Helicobacter pylori infection. N Engl J Med. 2002;347(15):1175-86.
2. Hooi JKY, Lai WY, Ng WK, Suen MMY, Underwood FE, Tanyingoh D, et al. Global prevalence of Helicobacter pylori infection: systematic review and meta-analysis. Gastroenterology. 2017;153(2):420-9.
3. Ghoshal UC, Chaturvedi R, Correa P. The enigma of Helicobacter pylori infection and gastric cancer. Indian J Gastroenterol. 2010;29(3):95-100.
4. Wroblewski LE, Peek RM Jr, Wilson KT. Helicobacter pylori and gastric cancer: factors that modulate disease risk. Clin Microbiol Rev. 2010;23(4):713-39.
5. Correa P. Human gastric carcinogenesis: a multistep and multifactorial process. Cancer Res. 1992;52(24):6735-40.
6. International Agency for Research on Cancer. Schistosomes, Liver Flukes and Helicobacter pylori. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol 61. Lyon: IARC; 1994.
7. Dixon MF, Genta RM, Yardley JH, Correa P; International Workshop on the Histopathology of Gastritis. Classification and grading of gastritis: the updated Sydney System. Am J Surg Pathol. 1996;20(10):1161-81.
8. Malfertheiner P, Megraud F, O’Morain CA, Gisbert JP, Kuipers EJ, Axon AT, et al. Management of Helicobacter pylori infection: the Maastricht V/Florence Consensus Report. Gut. 2017;66(1):6-30.
9. Genta RM, Hamner HW, Graham DY. Gastric lymphoid follicles in Helicobacter pylori infection: frequency, distribution and response to triple therapy. Hum Pathol. 1993;24(6):577-83.
10. El-Zimaity HMT, Graham DY. Evaluation of gastric mucosal biopsy site and number for identification of Helicobacter pylori or intestinal metaplasia: role of the Sydney System. Hum Pathol. 1999;30(1):72-7.
11. Thyagarajan SP, Ray P, Das BK, Ayyagari A, Khan AA, Dharmalingam S, et al. Geographical difference in antimicrobial resistance pattern of Helicobacter pylori clinical isolates from Indian patients: multicentric study. J Gastroenterol Hepatol. 2003;18(12):1373-8.

12. Rugge M, Genta RM; OLGA Group. Staging and grading of chronic gastritis. Hum Pathol. 2005;36(3):228-33.
13. Sonnenberg A, Everhart JE. The prevalence of self-reported peptic ulcer in the United States. Am J Public Health. 1996;86(2):200-5.
14. Wotherspoon AC, Ortiz-Hidalgo C, Falzon MR, Isaacson PG. Helicobacter pylori-associated gastritis and primary B-cell gastric lymphoma. Lancet. 1991;338(8776):1175-6
15. Sobala GM, King RF, Axon AT, Dixon MF. Reflux gastritis in the intact stomach. J Clin Pathol. 1990;43(4):303-6.