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Histopathological spectrum of gastric biopsies in patients with chronic dyspepsia: A cross-sectional observational study.

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Abstract

Background:
Chronic dyspepsia remains one of the most common gastrointestinal complaints, with etiologies ranging from benign inflammatory changes to premalignant and malignant conditions.

Objectives:
To determine the histopathological spectrum of gastric mucosal lesions in patients presenting with chronic dyspepsia and to evaluate the correlation of *H. pylori* infection with histopathological patterns.

Methods:
This cross-sectional observational study was conducted on 50 patients with chronic dyspepsia who underwent upper gastrointestinal endoscopy at a tertiary care hospital. Gastric biopsies were obtained primarily from the antrum and processed for histopathological examination using hematoxylin and eosin staining. Modified Giemsa stain was used for the detection of *H. pylori*. Data were analyzed using descriptive statistics and chi-square test, with $p < 0.05$ considered significant.

Results:
The mean age of the study population was 44.6 ± 12.4 years, with a male-to-female ratio of 1.3:1. The antrum was the predominant site of biopsy (68%). Chronic non-specific gastritis (44%) was the most common histological finding, followed by chronic active gastritis (22%), reactive (chemical) gastropathy (6%), atrophic gastritis (4%), intestinal metaplasia (4%), and gastric adenocarcinoma (4%). *Helicobacter pylori* was detected in 28% of cases, showing a statistically significant association with chronic active and atrophic gastritis ($\chi^2 = 9.84$; $p = 0.021$). Overall, inflammatory lesions constituted 78%, premalignant lesions accounted for 8%, and malignant lesions comprised 6% of the total biopsies.

Conclusion:
Chronic gastritis, predominantly involving the antrum, was the leading cause of dyspepsia. The significant association of *H. pylori* with chronic active and atrophic gastritis highlights its pivotal role in gastric mucosal injury and carcinogenic transformation.

Recommendations:
Routine gastric biopsies with *H. pylori* detection should be incorporated in all patients with persistent dyspepsia. Early eradication therapy, histological surveillance for atrophic or metaplastic changes, and lifestyle modifications may reduce progression to malignancy.

Keywords: *Chronic dyspepsia, Gastric biopsy, Histopathology, Helicobacter pylori, Gastritis.*
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Introduction

Dyspepsia is one of the most frequent upper gastrointestinal complaints encountered in clinical practice, accounting for nearly **30–40%** of gastroenterology consultations worldwide [1]. It encompasses a constellation of symptoms such as epigastric discomfort, bloating, nausea, and postprandial fullness that significantly impair quality of life. Chronic or persistent dyspepsia, defined as symptoms lasting for more than three months, may result from a wide spectrum of structural, infectious, inflammatory, or neoplastic disorders of the stomach [2].

Histopathological examination of gastric biopsies provides a definitive diagnosis in such patients by revealing the entire spectrum of mucosal alterations, ranging from chronic gastritis to premalignant and malignant lesions. Among the various etiological factors, *Helicobacter pylori* (*H. pylori*) infection plays a pivotal role in the pathogenesis of chronic gastritis, peptic ulcer disease, and gastric carcinoma [3]. The bacterium induces both inflammatory and degenerative changes in the gastric mucosa through complex host–pathogen interactions, leading to progressive glandular atrophy, intestinal metaplasia, and dysplasia.

The prevalence and histopathological pattern of gastric lesions in chronic dyspepsia exhibit significant geographic and ethnic variability, reflecting differences in dietary habits, socioeconomic status, *H. pylori* prevalence, and host genetic susceptibility [4]. Although non-invasive diagnostic tests such as the urea breath test and stool antigen detection are widely used for *H. pylori* screening, histopathological examination remains the gold standard for identifying both *H. pylori* infection and the associated mucosal pathology [5]. Given the high burden of dyspepsia and its potential to mask serious underlying disease, early histological evaluation is crucial for accurate diagnosis, appropriate therapy, and prevention of complications. Hence, the present study was designed to analyze the histopathological spectrum of gastric biopsies in patients with chronic dyspepsia and to assess the association between *H. pylori* infection and histopathological changes, thereby contributing to improved diagnostic and therapeutic strategies in clinical practice.

Methodology

Study Design and Setting

The study was conducted in the Department of Pathology at Neelima Institute of Medical Sciences, Hyderabad, Telangana, India. The institute is a large tertiary-care teaching hospital serving both urban and semi-urban communities and receives a steady referral load for upper gastrointestinal endoscopy from the Departments of Gastroenterology and General Medicine. This environment made it an appropriate setting for evaluating gastric biopsy specimens from patients with persistent dyspepsia.

This hospital-based cross-sectional observational study was carried out over 12 months, from June 2024 to May 2025. The primary objective was to examine the histopathological spectrum of gastric lesions in adults presenting with chronic dyspepsia and to determine the association between Helicobacter pylori infection and specific histopathological patterns.

Study Population

The study included 50 adult patients presenting with symptoms of chronic dyspepsia who underwent upper gastrointestinal endoscopy during the study period. Chronic dyspepsia was defined as upper abdominal discomfort, epigastric pain, bloating, or nausea persisting for more than three months. All eligible patients who met the inclusion criteria were consecutively enrolled to ensure a uniform and unbiased selection process.

Sample Size Determination

The sample size was calculated using the standard formula for estimating a proportion in a cross-sectional study:

$n = Z^2 \times P \times Q / d^2$

Where:

Z = 1.96 at 95% confidence level

P = expected prevalence of chronic gastritis in dyspeptic patients (assumed as 50% based on previous regional studies)

$Q = 1 - P$

d = allowable error (14%)

Substituting these values:

$n = (1.96)^2 \times 0.5 \times 0.5 / (0.14)^2$

$n \approx 49$

Thus, the minimum required sample size was 49. The study finally included 50 gastric biopsy specimens, which met and slightly exceeded the calculated requirement.

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Inclusion Criteria

Patients aged 18 years and above presenting with chronic dyspepsia.
Patients undergoing endoscopic gastric biopsy for diagnostic evaluation.
Patients who provided written informed consent for participation.

Exclusion Criteria

Patients with a history of gastric surgery or malignancy.
Individuals on proton pump inhibitors, H2 blockers, or antibiotics within two weeks before biopsy.
Patients with coagulopathies or contraindications for endoscopic biopsy.
Biopsies with inadequate or autolyzed tissue for histological interpretation.

Data Collection

Detailed clinical history, demographic data, and presenting symptoms were recorded using a structured proforma. Endoscopic findings, including site and gross appearance of mucosa, were documented by the gastroenterologist.

Biopsy Procedure and Processing

Endoscopic biopsies were obtained predominantly from the gastric antrum, and in selected cases from the body or both sites, depending on mucosal pathology. Tissues were immediately fixed in 10% neutral buffered formalin, processed routinely, and embedded in paraffin blocks. Sections of 4–5 µm thickness were stained with Hematoxylin and Eosin (H&E) for general histopathological assessment.
Detection of *H. pylori* was performed using Modified Giemsa stain, and in doubtful cases, Warthin–Starry silver stain was applied. Lesions were classified according to the Updated Sydney System for grading gastritis, considering parameters such as chronic inflammation, activity, atrophy, intestinal metaplasia, and bacterial density.

Bias and Measures to Minimize It

Several steps were taken to reduce potential sources of bias. Selection bias was minimized by including all consecutive eligible patients undergoing endoscopic gastric biopsy during the study period. Information bias was reduced by using a structured proforma to record clinical and endoscopic details uniformly. Detection bias was limited by having all biopsy specimens processed using standardized protocols and examined by two independent pathologists who were blinded to the patient's clinical information. Any discrepancies in interpretation were resolved through joint review and consensus. These measures helped maintain consistency and reliability in histopathological assessment.

Statistical Analysis

All data were entered into Microsoft Excel and analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics such as frequency and percentage were used for categorical variables, while mean and standard deviation were calculated for continuous variables. The Chi-square (χ^2) test was applied to assess the association between H. pylori infection and histopathological findings. A p-value < 0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of Neelima Institute of Medical Sciences, Hyderabad. Written informed consent was obtained from all participants before inclusion in the study. Confidentiality and ethical principles were maintained throughout the research process.

RESULTS

Participant Flow

During the study period, 56 adults presenting with symptoms of chronic dyspepsia underwent preliminary screening for eligibility. Of these, 4 individuals were excluded because they had taken proton pump inhibitors or antibiotics within two weeks before endoscopy, and 2 were excluded due to inadequate or autolyzed biopsy tissue. A total of 50 participants met the inclusion criteria and were enrolled consecutively. All enrolled participants completed the endoscopic biopsy procedure, and none were lost to follow-up or excluded after recruitment.

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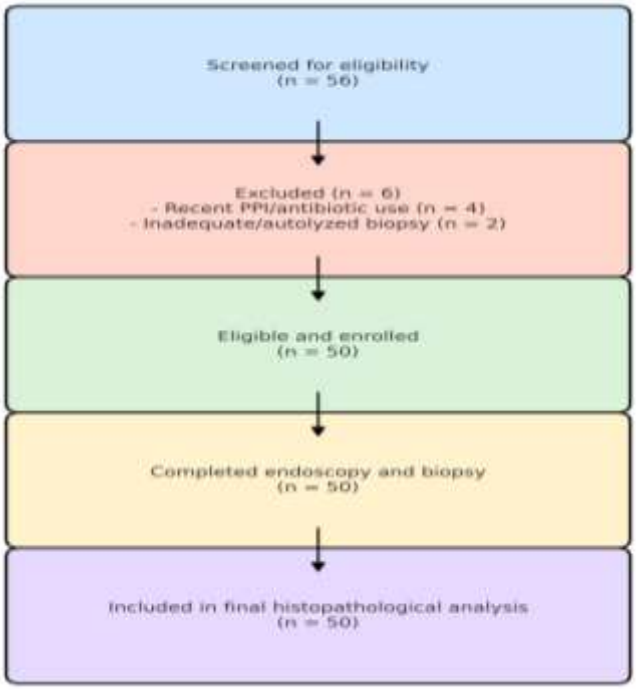

Figure1. Participant Flow Diagram

A total of 50 gastric biopsy specimens from patients with chronic dyspepsia were evaluated. The study cohort had a mean age of 44.6 ± 12.4 years, with ages ranging from 19 to 73 years. The largest age group was 41–50 years (28%), followed by 31–40 years (24%) and 51–60 years (18%). Males constituted 56% of the sample, giving a male-to-female ratio of 1.3:1.

Clinical Presentation:

Epigastric pain was the most frequently reported symptom, present in 74% of patients. Bloating was noted in 52%, while nausea and vomiting were observed in 32% and 26% of individuals, respectively (Table 1).

Table 1. Demographic and Clinical Profile of Patients (n = 50)

Variable	Category	No. of Cases	Percentage (%)
Age group (years)	18 – 30	9	18.0
	31 – 40	12	24.0
	41 – 50	14	28.0
	51 – 60	9	18.0
	> 60	6	12.0
Sex	Male	28	56.0
	Female	22	44.0
Predominant symptoms	Epigastric pain	37	74.0
	Bloating	26	52.0
	Nausea	16	32.0

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	Vomiting	13	26.0

Endoscopic and Biopsy Site Findings

As shown in Table 2, the antrum was the most frequent site of biopsy (68%), followed by the body (22%) and both sites (10%). Gross mucosal changes were observed in 61% of specimens, most commonly erythematous mucosa (36%), while 39% of biopsies appeared macroscopically normal. Erosions or ulcerations were noted in 17%, and nodularity or mucosal thickening in 8% of cases.

Table 2. Site and Gross Findings of Gastric Biopsies (n = 50)

Parameter	Observation	No. of Cases	Percentage (%)
Site of biopsy	Antrum	34	68.0
	Body	11	22.0
	Both sites	5	10.0
Gross mucosal changes	Normal mucosa	19	38.0
	Erythematous mucosa	18	36.0
	Erosions/ulcerations	9	18.0
	Nodularity or thickening	4	8.0

Histopathological Spectrum

Microscopic examination revealed that chronic non-specific gastritis was the most prevalent lesion, observed in 44% of cases, followed by chronic active gastritis (22%) and H. pylori–associated gastritis (6%) (Table 3). Reactive (chemical) gastropathy accounted for 6%, atrophic gastritis for 4%, and intestinal metaplasia for 4%. Among neoplastic lesions, gastric adenocarcinoma and MALT-type lymphoma were identified in 4% and 2%, respectively.

Overall, inflammatory lesions constituted 78% of all biopsies, premalignant lesions accounted for 8%, and malignant lesions comprised 6%.

Table 3. Histopathological Spectrum of Gastric Lesions

Histopathological Diagnosis	No. of Cases	Percentage (%)
Chronic non-specific gastritis	22	44.0
Chronic active gastritis	11	22.0
Reactive (chemical) gastropathy	3	6.0
Atrophic gastritis	2	4.0
Intestinal metaplasia	2	4.0
H. pylori–associated gastritis	3	6.0
Lymphoid hyperplasia	1	2.0
Gastric adenocarcinoma	2	4.0
MALT-type lymphoma	1	2.0
Total	**50**	**100.0**

Correlation with *Helicobacter pylori* Infection

Helicobacter pylori organisms were detected in 28% of cases (Table 4). The highest positivity was observed among patients with atrophic gastritis (50%) and chronic active gastritis (54.5%), whereas lower rates were noted in chronic gastritis (27.3%) and intestinal metaplasia (50%). A statistically significant association was found between H. pylori infection and chronic active/atrophic gastritis (χ^2 = 9.84; p = 0.021).

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Table 4. Association Between H. pylori Positivity and Histopathological Patterns (n = 50)

Histological Diagnosis	No. of Cases	H. pylori Positive (n)	Percentage (%)
Chronic gastritis	22	6	27.3
Chronic active gastritis	11	6	54.5
Atrophic gastritis	2	1	50.0
Reactive gastropathy	3	0	0.0
Intestinal metaplasia	2	1	50.0
Neoplastic lesions	3	0	0.0
Total	**50**	**14**	**28.0**

$\chi^2 = 9.84$, $p = 0.021$ (significant correlation between **H. pylori** *infection and chronic active/atrophic gastritis).*

Discussion

The present cross-sectional study evaluated the histopathological spectrum of gastric biopsies in 50 patients with chronic dyspepsia at Neelima Institute of Medical Sciences, Hyderabad. Inflammatory lesions formed the major proportion of gastric mucosal pathology, and a clear association was observed between Helicobacter pylori (H. pylori) infection and the severity of gastritis.

Chronic non-specific gastritis (44%) was the most frequent lesion, followed by chronic active gastritis (22%) and H. pylori–associated gastritis (6%). Antral involvement (68%) was predominant, which is in keeping with the usual pattern of H. pylori colonization, where infection commonly begins in the antrum and may later extend to the corpus.

The overall H. pylori positivity rate of 28% was lower than the 40–60% reported in earlier Indian series [8]. This difference may be related to improving sanitation, changing dietary habits, widespread use of proton pump inhibitors, and incidental antibiotic exposure in the community. Despite the relatively lower prevalence, H. pylori infection showed a statistically significant association with chronic active and atrophic gastritis ($p = 0.021$), supporting its central role in mucosal inflammation and in the Correa cascade from chronic gastritis to glandular atrophy, intestinal metaplasia, dysplasia, and carcinoma [6].

Premalignant lesions, namely atrophic gastritis and intestinal metaplasia, were present in 8% of biopsies, while malignant lesions (adenocarcinoma and MALT lymphoma) accounted for 6%. Similar ranges for atrophic gastritis (5–12%) and intestinal metaplasia (4–10%) have been reported in other studies [9,10]. Persistent H. pylori–induced inflammation appears to drive this transition, and meta-analyses indicate that eradication therapy alone may not completely abolish the risk of gastric cancer once metaplastic or dysplastic changes have developed [6,7]. Reversal of established atrophic gastritis or intestinal metaplasia after eradication also remains uncertain and often incomplete [7].

Lower H. pylori detection rates in neoplastic biopsies in this study are compatible with the so-called "vanishing bacterium" phenomenon, in which progressive glandular atrophy and intestinalization of the gastric mucosa create an unfavorable environment for bacterial survival [11]. Along with cumulative genetic and epigenetic alterations induced by long-standing inflammation, this mechanism underlies H. pylori–related gastric carcinogenesis [11].

The present findings are broadly in agreement with previous work that described a comparable distribution of intestinal metaplasia, dysplasia, and H. pylori infection in gastric biopsies [10]. Ultrastructural observations from earlier studies have also outlined a stepwise evolution of chronic gastritis, starting with epithelial degeneration and inflammatory infiltration, followed by glandular distortion, and finally progression to atrophy and metaplasia [12]. The pattern observed in the current series fits within this morphological sequence.

Clinical Implications

This study reinforces that gastric biopsy remains indispensable in evaluating chronic dyspepsia, as significant mucosal pathology, including early neoplasia, can be identified in symptomatic individuals. Routine *H. pylori* detection should be integrated into diagnostic protocols, and patients with atrophic or metaplastic changes should undergo regular histological surveillance. Strengthening early eradication programs and lifestyle modifications can further reduce gastric cancer risk in high-burden regions like India.

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Generalizability

The findings of this study can be generalized to patients presenting with chronic dyspepsia in similar tertiary-care settings across India, where *H. pylori* prevalence and dietary factors are comparable, though regional variations may influence the exact histopathological distribution.

Conclusion

The present study highlights that chronic gastritis is the predominant histopathological finding among patients with chronic dyspepsia, most frequently involving the gastric antrum. *Helicobacter pylori* infection was significantly associated with chronic active and atrophic gastritis, reinforcing its central role in gastric mucosal inflammation and progression toward premalignant changes. Although malignant lesions were relatively uncommon, the detection of atrophy and intestinal metaplasia in a subset of biopsies underscores the importance of early endoscopic and histological evaluation. Routine gastric biopsy with *H. pylori* detection should be advocated in all persistent dyspeptic patients to ensure timely eradication, surveillance, and prevention of advanced gastric pathology.

Strengths and Limitations

The strength of this study lies in its systematic approach using both routine and special stains (H&E and modified Giemsa), ensuring reliable detection of *H. pylori* and mucosal changes. However, limitations include a single-center design with a modest sample size and the absence of molecular or immunohistochemical confirmation of *H. pylori*. The cross-sectional nature precludes longitudinal assessment of histological progression following eradication therapy.

Recommendations

Routine endoscopic gastric biopsy with *Helicobacter pylori* detection should be performed in all patients with chronic or recurrent dyspepsia to identify underlying mucosal pathology at an early stage. *H. pylori*–positive individuals should receive appropriate eradication therapy and follow-up evaluation to prevent recurrence or progression to atrophic or metaplastic changes. Patients with atrophic gastritis or intestinal metaplasia should undergo periodic endoscopic surveillance. Clinicians should promote awareness regarding dietary habits, hygiene, and the avoidance of unnecessary proton pump inhibitor use. Multicenter studies with larger sample sizes and inclusion of molecular diagnostic methods are recommended to enhance diagnostic accuracy and population applicability.

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List of Abbreviations

H&E – Hematoxylin and Eosin
H. pylori – *Helicobacter pylori*
MALT – Mucosa-Associated Lymphoid Tissue
GI – Gastrointestinal
IEC – Institutional Ethics Committee
SD – Standard Deviation
SPSS – Statistical Package for the Social Sciences
PPI – Proton Pump Inhibitor
DNA – Deoxyribonucleic Acid
PCR – Polymerase Chain Reaction

Source of funding

The study had no funding.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

Concept and design of the study, results interpretation, review of literature, and preparation of the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript. -Concept and design of the study, results interpretation, review of literature, preparing the first draft of the manuscript, and revision of the manuscript. -Review of literature and preparing the first draft of the manuscript. Statistical analysis and interpretation. -Concept and design of the study, results interpretation, review of literature, and preparation of the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript**.**

Data availability

Data available on request

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