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Histopathological Analysis Of Chronic Gastritis Using The Updated Sydney System.

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ABSTRACT

Helicobacter pylori (H. pylori) is a gram-negative bacterium implicated in various gastric pathologies, including chronic gastritis, peptic ulcers, and gastric carcinoma. The updated Sydney Classification provides a standardized framework for assessing gastritis, incorporating anatomical, morphological, and etiological features to enhance diagnostic accuracy. This study aims to evaluate the histopathological characteristics of chronic gastritis in gastric antral biopsies using the updated Sydney Classification and to assess the association between these findings and H. pylori infection. A retrospective analysis was conducted on 200 gastric antral biopsy specimens obtained from patients presenting with upper gastrointestinal symptoms over a period of two years. Tissue samples were stained with hematoxylin and eosin (H&E) for morphological evaluation and Giemsa stain for *H. pylori* detection. Histological parameters were graded from 0 to 3+ (mild, moderate, severe) according to the updated Sydney Classification. Data were analyzed using SPSS version 22.0. Among the 200 biopsies, 175 (87.5%) exhibited features of chronic gastritis. H. pylori was identified in 70 samples (40%). Neutrophilic infiltration was present in 89 cases (50.8%), with 70 (78.6%) being H. pylori-positive ($p<0.001$). Chronic inflammatory changes were noted in 162 samples (92.6%). Intestinal metaplasia was observed in 27 cases (15.34%) and glandular atrophy in 13 cases (7.4%), though no significant association with H. pylori was found for either. Utilizing the updated Sydney Classification for histological examination of gastric biopsies proves effective in diagnosing chronic gastritis and detecting H. pylori infection. The strong correlation between neutrophilic activity and H. pylori underscores the importance of early detection to avoid further complications.

Keywords: Chronic gastritis, Helicobacter pylori, Updated Sydney System, Gastric biopsy, Histopathology.

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INTRODUCTION

Gastritis is defined as inflammation of the gastric mucosa, represents a spectrum of histopathological changes that can be acute or chronic in nature. While acute gastritis is usually self-limiting and associated with direct mucosal injury from nonsteroidal anti-inflammatory drugs (NSAIDs), alcohol, or stress-related factors. The chronic gastritis is a more persistent condition frequently associated with infectious or autoimmune etiologies, the most common being infection with Helicobacter pylori (H. pylori) [1].

Discovered in the early 1980s, H. pylori is a spiral-shaped Gram-negative bacterium that colonizes surface of the gastric epithelium [2]. It has since been implicated as a major etiological agent in chronic gastritis, peptic ulcer disease, gastric atrophy, intestinal metaplasia, and gastric adenocarcinoma [3,4]. The presence of H. pylori induces a cascade of inflammatory changes, beginning with superficial gastritis and, in some cases, progressing to multifocal atrophic gastritis and intestinal metaplasia—lesions that are considered precancerous. The chronicity and extent of mucosal inflammation directly influence the risk of progression to malignancy, making early detection and classification of gastritis essential for effective patient management [5].

To provide a standardized and reproducible framework for the histopathological assessment of gastritis, the Updated Sydney System was introduced in the early 1990s and later revised [6]. This system integrates topographical distribution, morphological features, and etiological associations to enable a comprehensive evaluation of gastric biopsies. It recommends obtaining multiple biopsy samples from defined regions of the stomach—typically the antrum, corpus, and incisura angularis—to assess the extent and severity of disease [7].

The Updated Sydney System evaluates five key histological parameters:

- Chronic inflammation (characterised by the presence of lymphocytes and plasma cells in the lamina propria)
- Neutrophil activity (active inflammation)
- Glandular atrophy (loss of native gastric glands)
- Intestinal metaplasia (presence of goblet cells)
- Presence and density of H. pylori.

Each of these features is semi-quantitatively graded on a scale from 0 (absent) to 3 (marked/severe). This grading helps stratify the severity of disease, guide clinical decisions, and monitor therapeutic outcomes [6].

MATERIALS AND METHODS

The present study was retrospective conducted in the Department of Pathology at Government Medical College, Patiala, over a duration of two-year from 2020 to 2022. The study was approved by the Institutional Ethics Committee prior to initiation. A total of 200 gastric antral biopsies were included in the study. Patients aged 14 years and above presenting with upper gastrointestinal symptoms such as dyspepsia, epigastric pain, bloating, nausea, vomiting, or heartburn were included in the study. Patients who had received treatment with antibiotics or proton pump inhibitors within the preceding four weeks were excluded from the study in order to avoid confounding effects on Helicobacter pylori (H. pylori) detection.

The biopsies were obtained from patients undergoing upper gastrointestinal endoscopy. Tissue specimens were fixed in 10% neutral buffered formalin, processed using routine histological techniques, and embedded in paraffin blocks. Sections of 4–5 µm thickness were cut and stained with Hematoxylin and Eosin (H&E) for routine morphological evaluation and with Giemsa stain specifically for the detection of H. pylori.

Histological parameters were assessed and graded according to the Updated Sydney System. The following features were evaluated:

Chronic Inflammation: Graded as mild (1+), moderate (2+), or severe (3+), based on the density of mononuclear inflammatory infiltrates in the lamina propria.

Neutrophilic Activity: Defined by the presence of polymorphonuclear leukocytes in the lamina propria, gastric pits, or surface epithelium; graded as mild (1+), moderate (2+), or severe (3+).

Glandular Atrophy: Graded based on the extent of loss of gastric glands.

Intestinal Metaplasia: Identified by the presence of goblet cells and columnar epithelial cells resembling intestinal mucosa.

H. pylori Presence: Graded as mild (1+), moderate (2+), or severe (3+) based on bacterial load observed in the superficial gastric mucosa.

All data were entered and analyzed using SPSS software version 22.0. Descriptive statistics were used to summarize demographic and histopathological findings. The chi-square test was employed to determine associations between the presence of H. pylori and various histopathological parameters. A p-value of <0.05 was considered statistically significant.

RESULTS

The present study analyzed 200 gastric antral biopsies obtained from patients with upper gastrointestinal symptoms. The age of the patients ranged from 14 to 78 years, with a mean age of 46.8 years. A slight male predominance was observed, with 116 males (58.2%) and 84 females (41.8%), yielding a male-to-female ratio of 1.4:1.

Out of 200 cases, chronic gastritis was seen in 175 cases. Among 175 cases of chronic gastritis, chronic inflammation was the most frequent finding, seen in 92.6% (n = 162) of cases.(Figure 1) Among these, moderate chronic inflammation (2+) was the predominant grade. Neutrophilic infiltration, indicative of active gastritis, was seen in 89 cases (50.8%).(Figure 2) Glandular atrophy was present in 13 cases (7.4%), all of which were graded as mild (1+).(Figure 3) Intestinal metaplasia was observed in 27 cases (15.34%), most being of mild (1+) severity.(Figure 4) The histopathological grading of chronic gastritis using Updated Sydney system is shown in Table 1.

Helicobacter pylori was detected in 70 out of 175 cases (40%) using Giemsa staining.(Figure 5) Grading of H. pylori Colonization Among Positive Cases shown in Table 2.

A statistically significant association was found between H. pylori positivity and neutrophilic infiltration ($p < 0.001$). However, no statistically significant correlation was observed between H. pylori infection and either glandular atrophy ($p = 0.312$) or intestinal metaplasia ($p = 0.174$). The association Between H. pylori and Histopathological Features is shown in Table 3.

Table 1: Histopathological grading of chronic gastritis (n=175) using Updated Sydney system

Histopathological features	Mild (1+)	Moderate (2+)	Severe (3+)	Total
Chronic inflammation	36	95	31	162
Neutrophilic activity	51	29	9	89
Intestinal metaplasia	23	4	0	27
Glandular atrophy	13	0	0	13
H. Pylori positive	19	40	11	70

Table 2: Grading of H. pylori Colonization Among Positive Cases (n = 70)

Grading of Colonization	Number of Cases	Percentage (%)
Mild (1+)	19	27.15%
Moderate (2+)	40	57.14%
Severe (3+)	11	15.71%

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Table 3: Association Between H. pylori and Histopathological Features

Histopathological Feature	Present (n)	H. pylori Positive (n)	H. pylori Negative (n)	p-value
Neutrophilic Infiltration	89	70	19	<0.001
Glandular Atrophy	13	5	8	0.312
Intestinal Metaplasia	27	8	19	0.174

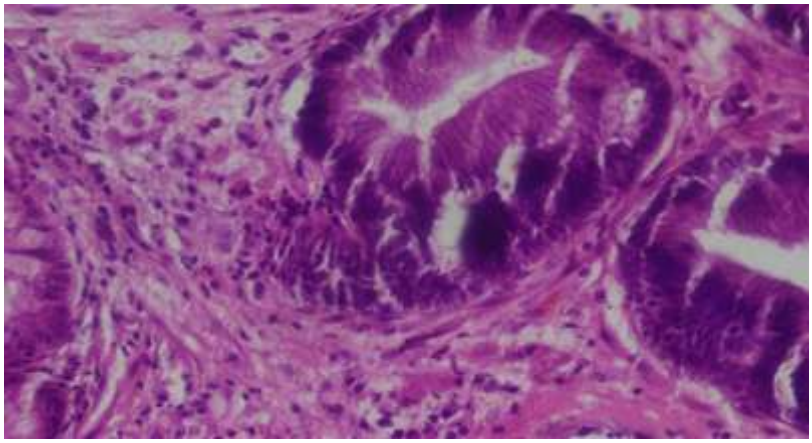

Figure 1: Dense inflammatory infiltrate : Chronic inflammation (H & E; X400)

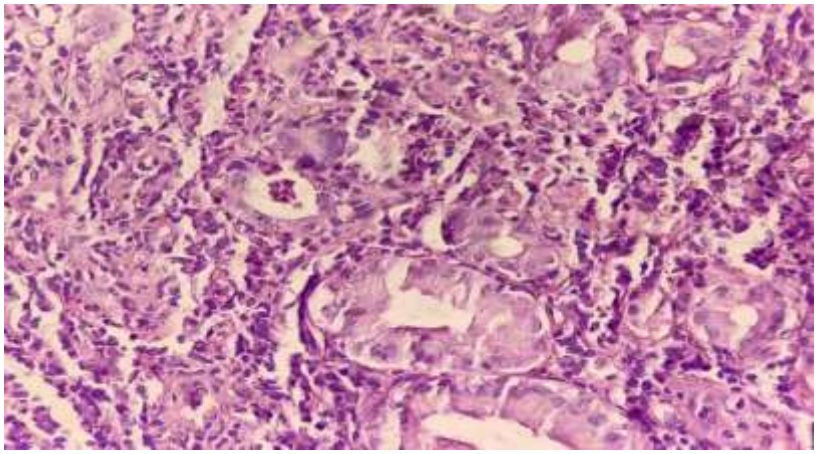

Figure 2: Presence of neutrophils in the lumen of glands : Activity (H & E; X400)

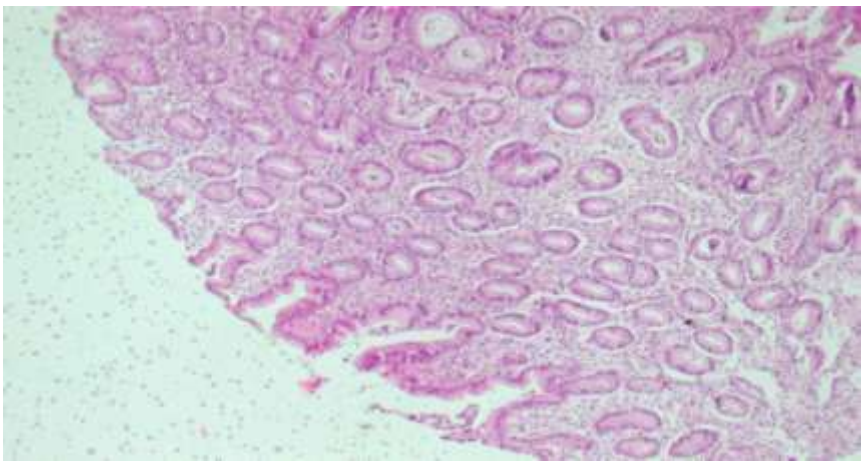

Figure 3: Loss of gastric pits : Atrophy (H&E; X40)

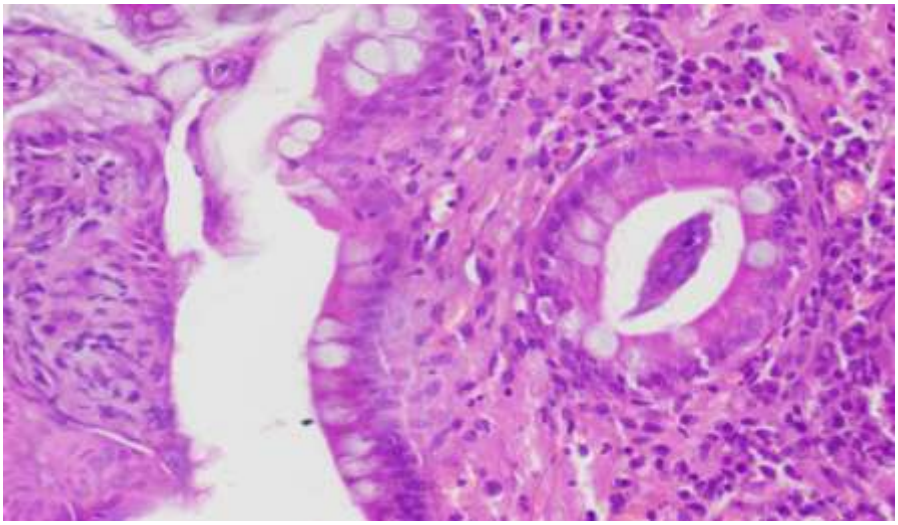

Figure 4: Presence of goblet cells : Intestinal metaplasia (H & E; X400)

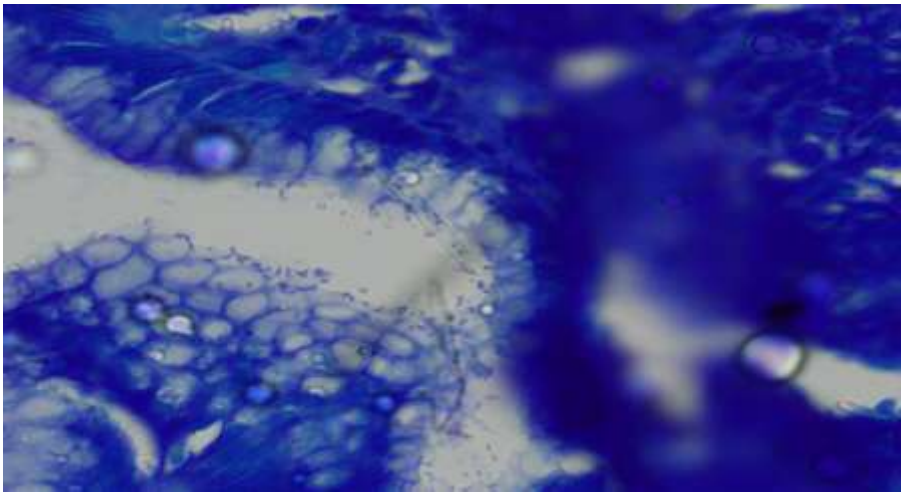

Figure 5: H. Pylori colonisation (Giemsa; X1000)

DISCUSSION

Chronic gastritis is a widespread pathological condition often linked with Helicobacter pylori infection, especially in developing countries like India. The Updated Sydney Classification provides a systematic and internationally accepted method for grading histological changes in gastric mucosa. This study aimed to analyze the histopathological features of chronic gastritis in gastric antral biopsies and evaluate their correlation with H. pylori infection. The findings of the present study reaffirm the significant etiopathogenic role of H. pylori in chronic gastritis and underscore the utility of histological examination in routine diagnosis.

In the current study, H. pylori was detected in 35% of all biopsy samples and in 40% of those with chronic gastritis. These findings are consistent with several studies across India and other parts of the Indian subcontinent. For instance, Khan et al. reported a H. pylori prevalence of 46.5% in Northern Maharashtra [8], while Sharma et al. observed a rate of 60% in a cohort from Nepal [9]. Similarly, Girish et al. from Karnataka found a prevalence of 38% [10], and Sahu et al. in Berhampur reported 53.6% [11]. This variation in prevalence may be attributed to geographic, socioeconomic, and dietary factors, as well as the diagnostic methodologies employed—ranging from histology and urease testing to serology and PCR-based assays.

Despite methodological differences, these studies consistently report that a substantial proportion of patients with gastritis are infected with H. pylori. The moderate prevalence observed in our study is

likely reflective of our population's demographic profile and the single-modality diagnostic approach (Giemsa staining), which, while specific, can underestimate true prevalence compared to PCR or multiple test strategies [12].

The Sydney system recommends the assessment of five key histological parameters: chronic inflammation, neutrophil activity (active inflammation), glandular atrophy, intestinal metaplasia, and the presence of H. pylori. In our study, chronic inflammation was noted in 92.6% of cases, with most graded as moderate (2+), highlighting the high burden of long-standing mucosal injury in this population.

Neutrophilic infiltration was present in 50.8% of biopsies and showed a strong and statistically significant association with H. pylori infection ($p < 0.001$), revealing that the neutrophil infiltration is a reliable marker of active H. pylori-associated gastritis. These results align closely with those reported by Abbas et al. and Sinha et al., who also noted a statistically significant association between neutrophilic activity and H. pylori presence [12,13]. In the Karnataka study by Girish et al., similar findings were reported, emphasizing that active inflammation is one of the earliest and most prominent histological changes in H. pylori-infected mucosa [10]).

Glandular atrophy was identified in 7.4% of biopsies, and intestinal metaplasia in 15.34%, with no statistically significant association with H. pylori positivity ($p = 0.312$ and $p = 0.174$, respectively). These findings suggest that while H. pylori initiates mucosal injury, the progression to atrophy and metaplasia likely depends on the chronicity and duration of infection, host immune response, and possibly environmental factors such as dietary nitrates and smoking. A longer follow-up or larger cross-sectional sample could potentially reveal stronger associations [9].

Contrastingly, studies like those by Sharma et al. and Sahu et al. reported higher frequencies of atrophic changes and metaplasia, particularly in older individuals, suggesting that these premalignant changes may be underrepresented in short-duration or retrospective studies without longitudinal follow-up [9,11].

Globally, the prevalence and histopathological impact of H. pylori vary by region. A study in Turkey by Uygun et al. reported H. pylori prevalence of over 70%, with strong histological associations [14], while research in Nigeria showed prevalence ranging from 55–65% in symptomatic individuals [15]. Western studies, such as those from the United States, report lower prevalence (20–30%) due to improved hygiene and widespread use of antibiotics [16], but still confirm the critical role of H. pylori in cases of non-autoimmune gastritis.

When comparing the present study with international cohorts, it is evident that while H. pylori prevalence is influenced by regional endemicity, its histological manifestations—particularly chronic inflammation and neutrophilic infiltration—remain consistent across diverse populations. This reaffirms the pathogen's universal role in the pathogenesis of gastritis and related gastroduodenal disorders.

The present study has certain limitations. Being retrospective in nature, the study is inherently limited by potential selection and information biases. Patient follow-up and treatment outcomes could not be assessed. H. pylori was detected solely through Giemsa staining. The addition of rapid urease tests, immunohistochemistry, or PCR could have improved diagnostic sensitivity and specificity [17]. The absence of follow-up biopsies limits our understanding of the natural history and progression of gastric mucosal lesions. Correlating histological findings with clinical symptoms, endoscopic impressions, or treatment response would have enhanced the translational relevance of the study.

The findings of this study highlight the utility of routine histopathological examination and H. pylori detection in the management of patients with dyspeptic symptoms. Given the significant association of H. pylori with active inflammation, timely diagnosis and eradication therapy may prevent disease progression to atrophy, metaplasia, and eventually gastric malignancy [18].

Future studies should consider incorporating a prospective design, multimodal diagnostic tools, and long-term follow-up, along with clinical correlation, to better understand the progression of gastritis and the role of H. pylori in gastric carcinogenesis.

CONCLUSION

In conclusion, the updated Sydney Classification proves to be an effective tool for the histopathological assessment of chronic gastritis and the detection of H. pylori infection. The significant association between neutrophilic activity and H. pylori underscores the importance of early detection and treatment to prevent the progression of gastritis to more severe conditions such as peptic ulcers and gastric carcinoma. Given the high prevalence of H. pylori infection in the Indian subcontinent, routine application of the Updated Sydney Classification in histopathological assessments is recommended to enhance diagnostic accuracy and guide appropriate therapeutic interventions.

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