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Original Research Article

Chronic Gastritis Histopathological Spectrum and Association between Pathological Features and *H. Pylori*

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Abstract

Background: A Helicobacter pylori (*H. pylori*) infection is the most common cause of chronic gastritis (CG), with approximately 50% of the world's population infected. Long-term infection increases the risk of progression to gastric cancer. This study evaluated the histopathological changes in CG using the Updated Sydney System (USS) to estimate the prevalence and correlation of *H. pylori* gastritis with other histological variables.

Methods: This prospective observational study was conducted in the Department of Pathology of JLNMCH, Bhagalpur, Bihar from March 2025 to August 2025. 62 gastric endoscopic mucosal biopsies taken from patients presenting with dyspepsia were included in this study. Slides were stained with routine H and E and Giemsa for *H. pylori* detection in chronic gastritis cases. Grading of the variables were done with reference to Sydney system of classification.

Results: Out of 62 gastric biopsy specimens, 55 cases (88.7%) were histopathological diagnosed as chronic gastritis. Among chronic gastritis, 21 (38%) cases showed *H. pylori* and majority of these being moderately (2+) positive. 27 (49%) cases showed neutrophilic activity with most of them showed mild (1+) activity. Chronic inflammation was seen 52 (94.5%) with majority of these graded as moderate (2+). Intestinal metaplasia was seen in 8 (14.5%) of cases with majority being mild (1+). Atrophy was seen only in 3 (5.4%) of cases with majority being mild (1+). Significant statistical association was found between *H. pylori* and neutrophilic activity ($p<0.001$).

Conclusion: Histological evaluation of chronic gastritis using updated Sydney system of classification helps in detection of *H. pylori* infection and prevents further progression of the disease.

Keywords: Chronic Gastritis; Helicobacter pylori; Updated Sydney system.

Introduction

Chronic gastritis (CG) is the chronic inflammation of the gastric mucosa associated with varying degrees of superficial and glandular epithelial damage.[1] Helicobacter pylori (H. pylori) is a gram-negative bacterium and the most common cause of CG. Approximately 50% of the world's population is infected with H. pylori.[2] The percentage is higher in developing countries and more in men than women.[3] Long-term infection increases the risk of progression to gastric cancer. Gastric carcinoma is the second most common malignancy worldwide.[4,5]

An accurate diagnosis of H. pylori-associated gastritis is crucial in clinical practice because H. pylori-associated gastritis indicates the early phase of gastric carcinogenesis.[6-8] H. pylori infection is a treatable condition. Therefore, early detection and eradication improve the quality of life with a decreased severity of dyspeptic symptoms and a decrease in the risk of gastric malignancy. The Updated Sydney System (USS) for grading and classifying CG was conceived to provide a unified and standardized application to interpret antral biopsies.[9]

In the USS, the following five histological parameters are graded as mild, moderate, or severe: chronic inflammation, activity (neutrophilic infiltration), atrophy, intestinal metaplasia, and H. pylori density.[9]

Material and Methods

This study was both retrospective and prospective conducted in the department of pathology, Jawaharlal Nehru Medical College, Bhagalpur, Bihar from March 2025 to August 2025. All biopsies obtained for various symptoms of

dyspepsia like abdominal pain, nausea, vomiting, bloating, heartburn, were included in the study. Exclusion criteria included absolute/relative contraindication to upper GI endoscopy and patients put on H. pylori eradication or acid suppressive therapy in the last 2 weeks. Written informed consent was taken from all the patients for the study. Upper gastrointestinal endoscopic biopsy samples received in 10% formalin were taken for the study. While embedding care was taken to see that the mucosal surface was placed 90 degree to the cutting surface. 4-5 µm thick sections were cut on a Leica microtome. Routine haematoxylin and eosin staining was done on all biopsies.

Special stain like Giemsa was done for the detection of H. pylori in chronic gastritis cases. The slides were evaluated, and morphological variables of chronic gastritis were graded according to updated Sydney system. The data obtained was analysed using SPSS 21.0 software. Microsoft word and excel was used to assimilate the data and prepare the article.

Results

A total of 62 gastric biopsy samples of dyspepsia were included in the study. The endoscopic mucosal biopsies were taken for histopathological evaluation. Out of 62 patients, chronic gastritis was observed in 55 (88.7%) cases, dysplasia in 2 (3.2%), gastric ulcer in 2 (3.2%), polyp in 1 (1.6%), adenocarcinoma in 1 (1.6%) and no significant pathology in 1 (1.6%) case. 55 chronic gastritis cases were further evaluated.

Among chronic gastritis cases, majority of cases were seen in the 41-50 years age group followed by 51-60 years age group (Table 1). There were 36 males and 19 female patients with an M: F ratio of 1.9: 1. Most frequent site sampled for biopsy was from gastric antrum constituting 46cases (83.6%).

Table 1: Age distribution in chronic gastritis

Age groups (years)	No. of cases	Percentage
11-20	3	5.45%
21-30	6	10.9%
31-40	9	16.36%
41-50	18	32.72%
51-60	12	21.8%
61-70	4	7.27%
>70	3	5.45%

Histological grading of chronic gastritis was done by updated Sydney system (Table 2).

Chronic inflammation was present in 52 (94.5%) cases of chronic gastritis out of which, 8 (15.3%) had mild, 34 (65.3%) had moderate and 12 (23%) had severe chronic inflammation. Neutrophilic activity was seen in 27 (49%) cases of which 20 (74%) had mild, 5 (18.5%) had moderate and 2 (7.4%) had severe neutrophilic activity.

Intestinal metaplasia was seen in 8 (14.5%) cases with 7 cases (87.5%) having mild and 1 case (12.5%) having moderate intestinal metaplasia. Glandular atrophy was seen in 3 (5.4%) cases which were of mild grade.

Table 2: Histological grading of chronic gastritis by updated Sydney system

Variables in chronic gastritis	Mild/1+	Moderate/2+	Severe/3+	Total	Percentage
H. pylori	8	11	2	21	38%
Neutrophils	20	5	2	27	49%
Chronic inflammation	8	34	12	52	94.5%
Intestinal metaplasia	7	1	-	8	14.5%
Atrophy	3	-	-	3	5.4%

H. pylori was identified in 21 (38%) cases of chronic gastritis on gastric mucosal biopsies. Out of these 8(38%) had mild, 11 (52.3%) had moderate and 2 (9.5%) had severe H. pylori colonization. Further, association of H. pylori with neutrophils, lymphoid aggregates, intestinal metaplasia and atrophy in chronic gastritis was also evaluated (Table 3). 27 cases of chronic gastritis showed neutrophilic activity. 24 of these 27 cases (88.8%) were positive for H. pylori. The association was clinically significant ($p<0.001$). 52 cases of chronic gastritis showed lymphoid aggregates. 18 of these 52 cases (34.6%) were positive for H. pylori. 8 cases of chronic gastritis showed intestinal metaplasia and 2 out of these 8 cases (25%) were positive for H. pylori. 3 cases of chronic gastritis showed atrophy. Out of these 3 cases none were positive for H. pylori.

Table 3: Association of H. pylori with neutrophilic activity, lymphoid aggregates, intestinal metaplasia and atrophy in chronic gastritis

Histological variables	No. of cases	H. pylori positive	Percentage
Neutrophilic activity	27	24	88.88%
Lymphoid aggregates	52	18	34.61%
Intestinal metaplasia	8	2	25%
Atrophy	3	-	-

Discussion

In the present study, chronic gastritis was noticed in a wide age group ranging from 12-80 years with a mean age of 49.6 years. This is similar to other studies in which mean age was 47 years and 48 years in another study.[10-12] The M: F ratio in our study is 1.9: 1 which is similar to studies done by Chen et al, Pruthi et al and where they have reported a M: F ratio of 1.8: 1, 2.3: 1 respectively.[13,14] The most common site of gastric biopsies was antrum in the present study, which is in line with studies conducted by Garg et al and Park et al.[11,15] H. pylori was positive in 21 (38%) cases of total chronic gastritis cases. This is almost consistent with studies done by Pruthi et al, and Dhakhwa et al, in which H. pylori was positive in 47% and 44% of cases respectively.[14,16] However H. pylori positivity was higher in study done by Qureshi et al and lesser in study done by Hassan et al which were 15.5% and 93.7% respectively.[10,17] These variations in the result can be attributed to biopsy sampling, where multiple biopsies are required to improve the results.14 The use of immunostains are also helpful for better detection of H. pylori.[19]

Neutrophilic infiltration was seen in 27 (49%) cases with majority of them showing mild activity. 24 of these 27 cases (88.8%) were positive for H. pylori. The association was clinically significant ($p<0.001$) which was proved in other studies as well.[11,13,16] Stolte et al stated that neutrophilic activity is an almost universal phenomenon in H. pylori gastritis.

Neutrophils are a very sensitive indicator for the presence of H. pylori and disappear within few days of cure of infection.[20] 52 (94.5%) cases of chronic gastritis showed moderate to marked chronic inflammation. However, a statistically significant relationship was not demonstrated between H. pylori infection and grade of chronic inflammation. This contrasts with the study done by Genta et al in which 91.8% patients with H. pylori gastritis showed lymphoid aggregates and was concluded as statistically significant.[21]

8 (14.5%) cases of chronic gastritis showed intestinal metaplasia. This is similar to the study done by Hassawi et al in which 15% of cases showed this change.[22] However 5% of cases showed intestinal metaplasia in the study done by Dhakhwa et al and in contrary 23% of cases showed intestinal metaplasia in study done by Nuaimy et al.[16,23]

The latter may be attributed to the fact that the diagnostic rate of intestinal metaplasia could be improved by the use of special stain for mucin. In addition, biopsy taken from the area of incisura angularis also increases the rate of detection of intestinal metaplasia as it initially develops in this region.[24] There was no significant statistical association between H. pylori and intestinal metaplasia which is in line with other studies.[16,18]

Only 3 (5.4%) cases of chronic gastritis showed atrophy. None of them were positive for H. pylori. Likewise, it was concluded the association was insignificant in studies conducted by Garg et al and Maharjan et al.[11,18]

Conclusion

Histopathological evaluation of endoscopic gastric biopsy using updated Sydney system of classification is of value in detection of H. pylori and various histological changes of chronic gastritis. In our study neutrophilic activity was significantly associated with H. pylori infection. Thus, search for H. pylori should be initiated if neutrophils are seen in the antral biopsies. The detection of H. pylori can help in early treatment and further advancement of disease.

References

1. Cheli R, Testino G, Giacosa A, Cornaggia M. Chronic gastritis: its clinical and physiopathological meaning. J Clin Gastroenterol 1995; 21:193-197.
2. Feldman M, Lee EL. Gastritis. In: Feldman M, ed. Sleisenger and Fordtran's gastrointestinal and liver disease. 10th ed. Elsevier Saunders publishers, 2016:868.
3. Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. Am J SurgPathol 1996; 20:1161-1181.
4. Satoskar A, Vora IM. Incidence of Helicobacter pylori associated gastritis in the urban population from India. Trop Geogr Med 1994; 46:167-168.
5. Maitra TN, Ghosh S. Gastritis and Helicobacter (Campylobacter) pylori--merely

one more piece in the jigsaw puzzle or the final answer? Indian J Pathol Microbiol 1991; 34:67-79.
6. Yakoob MY, Hussainy AS. Chronic gastritis and Helicobacter pylori: a histopathological study of gastric mucosal biopsies. J Coll Physicians Surg Pak 2010; 20:773-775.
7. Abu-Ahmad NM, Odeh A, Sallal AKJ. Prevalence of Helicobacter pylori gastritis at the north of Jordan. Jordan J BiolSci 2011; 4:71-76.
8. Nai GA, Parizi AC, Barbosa RL. Association between Helicobacter pylori concentration and the combining frequency of histopathological findings in gastric biopsies specimens. Arq Gastroenterol 2007; 44:240-243.
9. Warren JR, Marshall B. Unidentified curved bacilli on gastric epithelium in active chronic gastritis. Lancet 1983; 1:1273-1275.
10. Hassan TMM, Najjar SI, Zahrani IH, Alanazi FIB, Alotibi MG. Helicobacter pylori chronic gastritis updated Sydney grading in relation to endoscopic findings and H. pylori IgG antibody: diagnostic methods. J MicroscUltrastruct. 2016;4(4):167-74.
11. Garg B, Sandhu V, Sood N, Sood A, Malhotra V. Histopathological analysis of chronic gastritis and correlation of pathological features with each other and with endoscopic findings. Pol J Pathol. 2012;63(3):172-8.
12. Udoh MO, Obaseki DE. Histopathological Evaluation of H. Pylori Associated Gastric Lesions In Benin City, Nigeria. East Afr Med J. 2012;89(12):408-13.
13. Chen XY, Liu WZ, Shi Y, Zhang DZ, Xiao SD, Tytgat GN. Helicobacter pylori associated gastric diseases and lymphoid tissue hyperplasia in gastric antral mucosa. J Clin Pathol. 2002;55(2):133-7.
14. Pruthi S, Nirupama M, chakraborti S. Evaluation of gastric biopsies in chronic gastritis: Grading of inflammation by Visual Analogue Scale. Med J DY Patil Univ. 2014; 7:463-7.
15. Park J, Kim MK, Park SM. Influence of Helicobacter pylori colonization on histological grading of chronic gastritis in Korean patients with peptic ulcer. Korean J Intern Med. 1995;10(2):125-9.
16. Dhakhwa R, Acharya IL, Shrestha HG, Joshi DM, Lama S, Lakhey M. Histopathologic study of chronic antral gastritis. J Nepal Health Res Counc. 2012;10(1):57-60.
17. Qureshi NA, Hallissey MT, Fielding JW. Outcome of index upper gastrointestinal endoscopy in patients presenting with dysphagia in a tertiary care hospital-A 10 years review. BMC Gastroenterol. 2007; 7:43.
18. Maharjan S, Ranabhat S, Tiwari M. Helicobacter pylori associated chronic gastritis and application of visual analogue scale for the grading of the histopathological parameters in Nepal. Biomed J Sci Tech Res. 2017; 1:28-34.
19. Cartun RW, Kryzmowski GA, Pedersen CA, Morin SG, Kruiningen HJ, Berman MM. Immunocytochemical identification of Helicobacter pylori in formalin-fixed gastric biopsies. Mod Pathol. 1991;4(4):498-502.
20. Stolte M, Eidt S. Chronic erosions of the antral mucosa: a sequela of Helicobacter pylori-induced gastritis. Z Gastroenterol. 1992;30(12):846-50.
21. Genta RM, Hamner HW. The significance of lymphoid follicles in the interpretation of gastric biopsy specimens. Arch Pathol Lab Med. 1994;118(7):740-3.
22. Hassawi BA, Khalil KH, Sharafadin ZA. Prevalence of intestinal metaplasia and dysplasia in infectious and non-infectious chronic gastritis. Int J Res Med Sci. 2015; 3:2228-31.
23. Nuaimy WMT, Faisal HM. Endoscopical and histopathological interpretation of gastritis in Nineveh Province. Ann Coll Med Mosul. 2019; 41:28-35.
24. Uedo N, Kanzaki H, Ishihara R. Endoscopic diagnosis of gastric intestinal metaplasia. Gastroenterol Endosc. 2014; 56:1941-52.