International Journal of Clinical and Diagnostic Pathology

ISSN (P): 2617-7226
ISSN (E): 2617-7234
www.patholjournal.com
2019; 2(2): 352-355
Received: 16-05-2019
Accepted: 20-06-2019

Dr. Ganga H
Assistant Professor, Department of Pathology, Subbaiah Institute of Medical Sciences, Shimoga, Karnataka, India

Dr. Indudhara PB
Associate Professor, Department of Pathology, Subbaiah Institute of Medical Sciences, Shimoga, Karnataka, India

Corresponding Author:
Dr. Indudhara PB
Associate Professor, Department of Pathology, Subbaiah Institute of Medical Sciences, Shimoga, Karnataka, India

Histopathological study of endoscopic gastric biopsies in dyspeptic patients with special correlation to helicobacter pylori

Dr. Ganga H and Dr. Indudhara PB

DOI: https://doi.org/10.33545/pathol.2019.v2.i2f.127

Abstract
Aims: 1. To identify various pathological lesions in gastric biopsies in patients with dyspeptic symptoms 2. To determine that *H. pylori* is a major etiologic organism in the pathogenesis of chronic gastritis and to assess the graded variables of Helicobacter associated gastritis.
Materials and methods: A total of 93 cases with complaint of dyspepsia were studied during the period July 2016 to July 2017. Endoscopic examinations as well as biopsy specimens from gastric mucosa were studied in all the cases for the various mucosal lesions.
Results: Out of 93 cases studied, dyspepsia was most common clinical symptom seen in 80 patients. On endoscopy 18 patients showed normal appearance and erythema was the most common finding in 40 patients. On histopathology, 6 were normal, 80 cases showed various forms of chronic gastritis and 7 cases were malignant. Out of 80 cases of chronic gastritis, 43 showed *H. pylori* positivity. Most of the *H. pylori* positive cases were associated with activity and mononuclear inflammatory infiltrate.
Conclusion: Prevalence of *H. pylori* in the present study was 46.2% in patients undergoing upper gastro-intestinal endoscopic biopsies. *H. pylori* associated gastritis is the predominant type of gastritis. Accurate endoscopic and histopathological examination of gastritis according to the Sydney grading system is a valuable indicator of *H. pylori* infection.

Keywords: Dyspepsia, *H pylori*, endoscopic gastric biopsy

Introduction
Gastro-duodenal diseases are the commonest diseases in adult population worldwide. Dyspepsia, which includes upper abdominal discomfort with or without nausea, vomiting or bloating, is one of the most common presentations in any Gastroenterology Department. Endoscopic gastric mucosal biopsies are frequently done for such patients mainly to rule out various forms of gastritis including *H pylori* induced gastritis. *H. pylori* have been implicated in several gastrointestinal diseases, including gastritis, peptic ulcer, duodenal ulcer, and gastric adenocarcinoma. The prevalence of *Helicobacter pylori* infection varies from country to country with large differences between developed and developing countries [1, 2, 3, 4]. Studies reported that its prevalence ranged from <15% in some populations to virtually 100%, depending on socioeconomic status and country development [5, 6]. Epidemiological evidence suggests that many people acquire infection in childhood. The prevalence of the infection increases with age, although this may be a large cohort effect. The mode of transmission is unknown but proposed route include oral-oral, faecal –oral [7].
Helicobacter pylori, the spiral-shaped gram-negative bacterium is found to colonize in gastric mucosa or adherent to the epithelial lining of the stomach [8]. The organism was first discovered and reported in 1983 by Warren and Marshall. International agency for research on cancer has categorized *H pylori* as a group 1 carcinogen [9]. The recognition that *Helicobacter pylori* play a pivotal role in the pathogenesis of several gastroduodenal pathologies makes its diagnosis necessary in many different circumstances [10, 11].
A number of tests may be used to confirm the presence of *Helicobacter pylori*. These fall into 2 categories; those that rely on non-invasive methods to detect the infection, such as serology, urea breath test, fecal antigen test and invasive requiring endoscope evaluation includes bacteriologic culture, histopathologic studies, cytological examination of smear, rapid urease test or CLO test and molecular studies. Several studies have shown high sensitivity and specificity of rapid urease test,

histology and culture for detection of *Helicobacter pylori* in gastric biopsy. Mergaud and Fallone consider histological methods as the "Gold Standard" for the demonstration of *H. pylori* in endoscopic biopsies, taken from patients with the gastritis- peptic ulcer syndrome. Factors other than the direct demonstration of the organism, can support the histological diagnosis of *H. pylori*. The incidence of gastric carcinoma has declined worldwide; it still remains the second most common cause of cancer death. *H pylori* and carcinoma stomach coexistence is high in India. In view of the above it was decided to take up a study to find out various histopathological gastric mucosal lesions and its correlation with *H pylori* infection.

AIMS and objectives

To identify various pathological lesions in gastric biopsies in patients with dyspeptic symptoms.

To determine that *H. pylori* is a major etiologic organism in the pathogenesis of chronic gastritis and to assess the graded variables of Helicobacter associated gastritis.

Materials and Methods

This study was conducted in the Department of pathology, Subbaiah institute of medical sciences, Shivamogga. A total of 93 endoscopic gastric biopsies were studied from July 2016 to July 2017 prospectively. The patient's age, sex, detailed clinical history, upper gastrointestinal endoscopy findings and other relevant laboratory investigations were documented.

Inclusion Criteria: (i) Cases of all ages and both sexes with upper gastrointestinal symptomatology (ii) Full mucosal thickness endoscopic biopsies incorporating muscularis mucosae. Exclusion criteria: (i) Patients who had taken Hp eradication treatment such as antibiotics, proton pump inhibitors, or H2 antagonists in the 4 weeks before endoscopy. (ii) Superficial gastric biopsy specimens.

The endoscopic biopsies studied were taken from the antrum, corpus, and edge of ulcer or any areas of mucosal abnormalities. Serial sections of 4-5µ thickness were obtained from formalin fixed paraffin embedded tissue and stained with hematoxylin and eosin (H and E). Giemsa stain was done for each of the biopsy specimen to demonstrate *H. pylori. H pylori* were identified as curved rods or coccoid forms. Lymphocytes and plasmocytes infiltration indicate chronic inflammation of *H pylori* and polymorphonuclear (PMN) cell infiltration stand for activity of *H pylori* infection. The histological glandular atrophy was identified when the gastric glands were correspondingly decreased in amount and/or widely separated.

Cases of chronic gastritis were graded according to the grading system provided by Houston-updated Sydney system which was depended on the intensity of mononuclear inflammatory cellular infiltrates within the lamina propria into four scales as

Grade 0: Absent inflammation
Grade 1: Mild inflammation
Grade 2: Moderate inflammation
Grade 3: Severe inflammation

Other Morphological changes such as reparative atypia, dysplasia, metaplasia, & malignant change were noted.

Results

A total of 93 cases were studied with most common symptom being dyspepsia. The age range of the patients was from 18 to 75 yrs with a mean age of 55 yrs. Maximum numbers of cases, belonged to age group of 51-60 years (32%). There were 58 male patients and 35 female patients with M: F ratio of 1.65: 1.

Clinical profile

Dyspepsia was the main indication for endoscopic biopsy in our study. It was present in 80 out of 93 patients. In dyspeptic symptoms following were the common presentations either alone or in combinations. Vague upper abdominal pain, nausea, vomiting and burning sensation in the epigastrium after meal. Other major indications for gastric biopsies were anemia, weight loss and upper GI bleed.

Table 1: Clinical profile

Symptoms	No of Cases
Dyspepsia	80 (86.02%)
Anemia	18 (19.3%)
Weight loss	10 (10.7%)
GI bleed	5 (5.37%)

Endoscopic findings

Out of 93 cases, 86 were benign lesions and 7 malignant lesions. Among 86 benign lesions, 18(20.9%) patients had normal appearance, 40 (46.5%) cases appeared erythematous, 14(16.2%) ulcerative, 7(8.13%) cases of thickened gastric fold, and 4(3.44%) patient showed erosion and healed scar, in 3(3.48%) case showed nodularity. Out of 7 malignant lesions, 4(57.1%) appeared polypoid, 2(28.5%) were ulcerative and 1(14.2%) was showing flattening of mucosa. Regarding the anatomical location of endoscopic gastritis, antral gastritis was found in 48 patients, and antrum predominant pangastritis and corpus predominant pangastritis in 10 patients each.

Histopathological findings

Out of 93 cases, 6 were normal in histopathology. 80 cases showed various forms of chronic gastritis and 7 cases were malignant.

Out of 80 cases of chronic gastritis, 56 cases showed neutrophilic activity of varying grades with mild in 27 (48.2%) cases, moderate in 18(32.14%) and severe activity/ crypt abscess in 11(19.6%) cases. *H. pylori* were seen in 43 cases with mild density in 10(23.25%) cases, moderate in 15(34.8%) cases and severe in 18(41.8%) cases. Mononuclear infiltration of varying grades was seen in 42 cases, with G1 (Grade 1) in 22(52.3%) cases, G2 (Grade 2) in 18(42.8%) cases and G3 (Grade 3) in 2(4.7%) cases. Lymphoid follicle formation was noted in 4 cases. Intestinal metaplasia of varying grades was seen in 24 cases with mild in 12 (50%) cases, moderate in 10(41.6%) cases and severe in 2(8.3%) cases. Dysplastic changes were associated with metaplasia was found in 4 cases. 18 cases showed presence of atrophy among which 10 cases were associated with intestinal metaplasia.

Table 2: Histopathological grading in chronic gastritis (updated Sydney system).

	Neutrophilic Activity	Hp Colonization	Atrophy	Intestinal Metaplasia	Mononuclear Infiltrate
Mild	27	10	11	12	22
Moderate	18	15	07	10	18
Severe	11	18	00	02	02

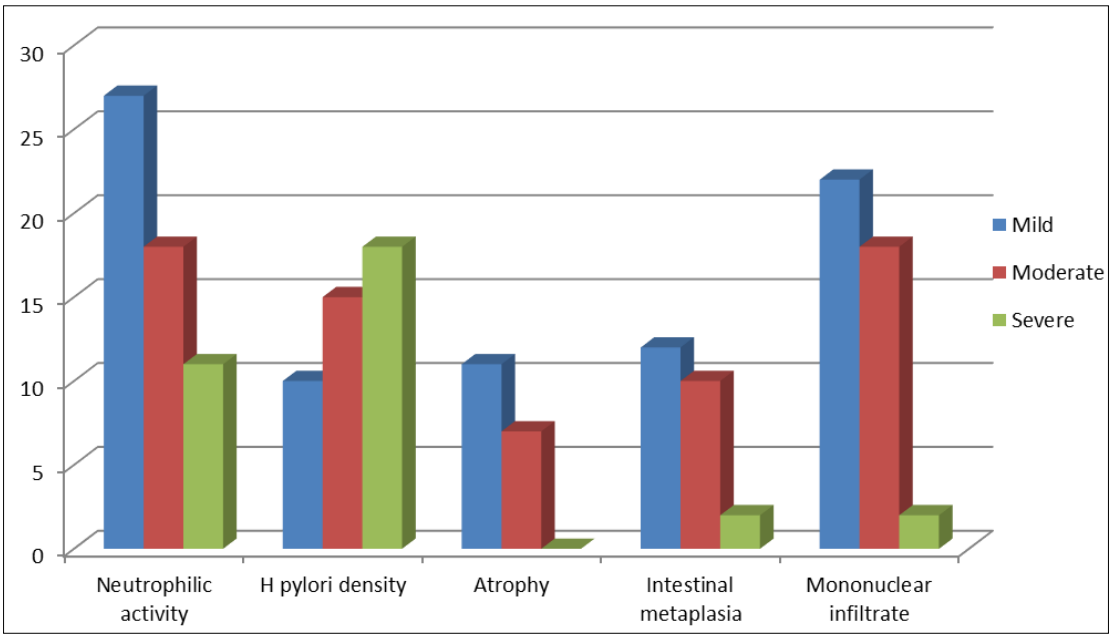

Fig 1: Histopathological grading in chronic gastritis (Updated Sydney system)

There were 7 malignant lesions of stomach. Out of which 5 (71.4%) were intestinal type of adenocarcinoma and 2 (28.5%) were diffuse adenocarcinoma. Site wise distribution showed 5 (71.4%) cases from antrum and 2 (28.5%) cases from body of stomach.

Discussion

Biopsy sampling of the gastric mucosa at diagnostic endoscopy provides information that cannot be obtained otherwise. Microscopic examination of gastric biopsy specimens gives, in addition to *H. pylori* status, information about the grade, extent, and topography of gastritis and atrophy-related alterations in the gastric mucosa. This information provides further possibilities for the assessment of risk and likelihood of various gastric disorders.

Endoscopic appearance

In this study, the endoscopic findings of gastritis revealed a significant relation between the following variables: erythema, ulceration and mucosal nodularity. Antral type of gastritis that is major forms of gastritis. Our endoscopic findings are in agreement with Khakoo *et al*. and Calabrese *et al*.

Histologic Appearance

Chronic gastritis was seen in 80 of 93 cases (86.02%).Chronic inflammation was located in the antrum in the 58 (72.5%) out of 80 cases similar to Singh R *et al*. study. The main reason for antral predominant gastritis could be due to the predominantly antral location of *H. pylori*. According to Mysorekar *et al*. 52% of Indians with dyspepsia and 44% of control subjects have active *H. pylori* infection by second decade of life.

In the present study histological gastritis was seen in 33.3% of endoscopically normal patients. Whereas in a study by Prabhu SR *et al*. showed histological gastritis in 66% of asymptomatic and endoscopically normal individuals. Endoscopic mucosal breaks (erosions) have a stronger association with histological gastritis. In our study *H. pylori* was identified in 43(53.7%) of 80 cases of chronic gastritis. In a study by Singh *et al*. 96% of chonic gastritis showed *H. pylori* positivity. In the present study 56 (70%) cases showed presence of activity similar to the study by Zhang *et al*.

The normal gastric mucosa contains very few lymphocytes in the lamina propria. Lymphoid follicles and aggregates are characteristic of *H pylori* associated gastritis. Lymphoid follicle prevalence between 27.4% and 100% has been reported in gastric mucosa from patients with *H. pylori* associated gastritis.

Intestinal metaplasia was present in 24(30%) of the 80 cases showing evidence of gastritis. This figure is similar to 20% reported in the study by Satarkar *et al*. In western literature intestinal metaplasia has been found in 12-51% of cases.

No correlation was seen between the presence of *H.pylori* colonization and intestinal metaplasia. This is similar to the findings of Satarkar *et al*. who also did not find significant relationship between the two. 18(22.55%) of the 80 cases showed atrophy and 10 were associated with intestinal metapalsia. Atrophy in the fundal and body mucosa is closely linked to loss of acids secretion and to the development of intestinal metaplasia, which in turn is linked to an increased risk of gastric cancer.

Seven (7.5%) out of 93 gastric endoscopic biopsies were diagnosed as gastric carcinoma. Chronic inflammation plays an important role in development of various cancers, including *H. pylori* associated gastric cancer. Gastric carcinoma is an important cause of cancer related death, so

understanding the pathogenesis of *H. pylori* induced gastric carcinoma may improve the risk stratifcation for prevention and therapy. This is the only cancer which can be prevented by antibiotics by eradication of *H. Pylori.*

Conclusion

Chronic gastritis was commonest histological diagnosis in this study. *H. Pylori* was the commonest cause for gastritis (53%), mostly in the antral region. Symptomatic cases normal on endoscopy may show chronic gastritis histologically. Histopathology is the most sensitive test for diagnosing *H. pylori* on endoscopic biopsies. *H. pylori* gastritis usually shows increased neutrophilic activity but can also present with increased mononuclear inflammatory infiltrate and lymphoid follicles in chronic gastritis. Intestinal metaplasia and atrophy indicates the chronicity of the disease.

References

1. Rotimi O, Cairns A, Gray S, Mooyyedi P, Dixon MF. Histological identification of helicobacter pylori: comparison of staining methods. J Clin Pathol. 2000; 53:756-9.
2. Rugge M, Pennelli G, Pilozzi E, Fassan M, Ingravallo G, Russo VM *et al*. Gastritis: The histology report. Dig Liver Dis. 2011; 43:S73-384.
3. Sipponen P, Kosunen TU, Valle J, Riihela M, Seppala K. Helicobacter pylori infection and chronic gastritis in gastric cancer. J Clin Pathol. 1992; 45:319-23.
4. Sokwala A, Shah MV, Devoni S, Youga G. Helicobacter pylori: A randomized comparative trial of 7-day versus 14-day triple therapy. S Afr. Med J. 2012; 102(6-2):368-71.
5. Malaty HM. Epidemiology of Helicobacter pylori infection. Best Pract Res Clin Gastroenterol. 2007; 21(2):205-14.
6. Carrasco G, Corvalan AH. Helicobacter pylori-induced chronic gastritis and assessing risks for gastric cancer. Gastroenterol Res Pract, 2013, 393015.
7. Fuccio L, Laterza L, Zagari RM, Cennamo V, Grilli D, Bazzoli F. Treatment of Helicobacter pylori infection. BMJ. 2008; 337:a1454.
8. Dooley CP, Cohen H, Fitzgibbons PL, Bauer M, Appleman MD, Perez-Perez GI *et al*. Prevalence of Helicobacter pylori infection and histologic gastritis in asymptomic persons. N Eng J Med. 1989; 321(23):1562-6.
9. Wong BC, Lam SK, Wong WM, Chen JS, Zheng TT, Feng RE *et al*. Helicobacter pylori eradication to prevent gastric cancer in a high- risk region of China-a randomized controlled trial. JAMA. 2004; 291(2):187-94.
10. Graham DY, Malati HM, Evans DG, Evans DJ *et al.* Epidemiology of Helicobacter pylori in an asymptomic population in the United Satets: effect of age, race and socioeconomic status. Gastroenterology. 1991; 100(6):1495-501.
11. Glupczynski Y. Microbiological and serological tests for H.pylori: An overview. Br Med Bull. 1998; 54(1):175-86.
12. Versalovic J. Helicobacter pylori: pathology and diagnostic strategies. Am J Clin Pathol. 2003; 119(3):403-12.
13. Anim JT, Al-Sobkie N, Prasad A, John B *et al*. Assesment of different methods for staining *H. pylori* in endoscopic gastric biopsies. Acta histochem. 2000; 102(2):129-37.
14. Prabhu SR, Amrapurkar AD, Amrapurkar DN. Role of Hlicobacter pylori in gastric carcinoma. Natl. Med J India. 1995; 8(2):58-60.
15. 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 Surg Path. 1996; 20(10):1161-81.
16. Khakooet SI, Lobo AJ, Shepherd NA, Wilkinson SP. Histological assessment of the Sydney classification of endoscopic gastritis. Gut. 1994; 35(9):1172-5.
17. Calabrese C, Di Febo G, Brandi G, Morselli-Labate AM, Areni A, Scialpi C *et al*. Correlation between endoscopic features of gastric antrum, histology and Helicobacter pylori infection in adults. Ital. J Gastroenterol Hepatol. 1999; 31(5):359-65.
18. Singh R, Laxmi TV, Verma KS, Dung R. Chronic Gastritis: Helicobacter pylori Infection: A Clinico-Endoscopic and Histological evaluation. Global Journal for research analysis. 2017; 6:2.
19. Mysorekar VV, Chitralekha, Dandekar P, Prakash BS. Antral histopathological changes in acid peptic disease associated with helicobacter pylori. Indian J Pathol Microbiol. 1999; 42(4):427-34.
20. Prabhu SR, Ranganath S, Amarapurkar DN. Helicobacter pylori in normal gastric mucosa. JAPI. 1994; 42:863-4.
21. Elta GH, Appleman HD, Behler EB, Wilson JA, Nostrant TJ. A study of the correlation between endoscopic and histological diagnosis in gastroduodenitis. Am J Gastroenterol. 1987; 82(8):749-53.
22. Zhang JK. Evaluation of a new classification of gastritis-Sydney system.International conference of Gastroenterology-1991. J Gastroenterol Hepatol. 1992; 6:200-1.
23. Genta RM, Hammer HW, Graham DY. Gastric lymphoid follicles in Helicobacter pylori infection: frequency, distribution, and response to triple therapy. Hum Pathol. 1993; 24:577-83.
24. Satarkar SP, Sawant P, Nanovadekar S, Shroff C. Helicobacter pylori and intestinal metaplasia of gastric mucosa. Indian J Gastroenterol. 1997; 16:16-7.
25. Zaninotto G, Avellini C, Barbazza R. Prevalance of intestinal metaplasia in the distal oesophagus Oesophagogastric Jn. & gastric cardia in symptomatic patient in north east Italy, A prospective descriptive survey.The Italian ulcer study group. 2001; 33:316-21.
26. Guarner J, Herrera Goepfort R, Mohar A. Atrophy and extend of intestinal metaplasia in a cohort of H pylori infected population. Human pathol. 2001; 32:31-5.
27. Uemura N, Okamoto S, Yamamoto S, Matsumura N, Yamaguchi S, Yamakido M. Helicobacter pylori infection and the development of gastric cancer. N. Engl. J Med. 2001; 345(11):784-9.