

Histopathological Spectrum of Gastric Biopsies in a Tertiary Care Hospital

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ABSTRACT

Objective: This study was carried out to determine the histopathological spectrum of gastric lesions at a tertiary care hospital

Study Design: A retrospective study

Descriptive Study: This study was conducted at Dr. Tahir Laboratory from Gastroenterology unit of Hamdard University Hospital Karachi from July 2009 to August 2012.

Patients and Methods: The gastric mucosal biopsies of 280 patients received at Dr. Tahir Laboratory from Gastroenterology unit of Hamdard University Hospital Karachi.

Results: A slight higher frequency of gastric disease seen in females with age range of 17 years to 78 years was observed. The clinical presentations mostly seen were abdominal pain, dyspepsia, vomiting, diarrhea, decreased appetite and weight loss. The histopathology revealed chronic active gastritis (H Pylori positive and negative) followed by malignant gastric ulcer. A number of biopsies were unremarkable histologically.

Conclusion: The more prevalent lesions in this series were chronic active gastritis. H. pylori associated gastritis was seen in majority of the patients. Thus gastric biopsy is an essential tool for diagnosis and confirmation of clinically suspected cases.

Key Words: Histopathological Spectrum, Gastric Biopsies, atrophy, intestinal metaplasia, dysplasia

INTRODUCTION

The upper gastrointestinal flexible fibreoptic endoscope was first used in late 60s. It demonstrated success in the early diagnosis of upper gastrointestinal lesions¹. The endoscopic biopsy is helpful in the diagnosis of specific entity, offers H. pylori status and plans for specific medical or surgical treatment^{2,3}. Endoscopic screening may detect gastric mucosal lesions at an early stage like atrophy, intestinal metaplasia and dysplasia so as to prevent progress of these lesions to invasive cancer⁴.

Diagnostic endoscopy is an invasive technique but has proved to be a simple and safe procedure⁵. In routine clinical practice, histology is often considered as the "gold standard" against which other tests are compared. Biopsy provides an excellent opportunity for the clinician and histopathologist to correlate the clinical data, endoscopic findings and pathological lesions. Helicobacter pylori (H. pylori) was identified by Warren and Marshall⁶ in 1983 in gastric antral mucosa, which is associated with chronic gastritis, peptic ulcer⁷, non-ulcer dyspepsia, gastric carcinoma and B-cell mucosa associated lymphoid tissue (MALT) lymphoma^{8,9}. This has gained even more importance since H. Pylori has also been classified as group 1 carcinogen by the World Health Organization (WHO) due to its role in the etiology of gastrointestinal cancers^{10,11}.

Helicobacter pylori (H. pylori) infection is chronic and common throughout the world, with a higher prevalence in developing than in developed countries¹². H. pylori infection causes gastritis and is the most important risk factor for peptic ulcer disease (gastric and duodenal)¹³ and it also contributes to the onset of gastric cancer and primary gastric B-cell lymphoma.¹⁴ H. pylori is present in a significant number of dyspeptic patients with endoscopically normal stomach¹⁵. A strong relationship is also documented between H. pylori and peptic ulcer disease. However, it is more common in duodenal ulcer than gastric ulcer and its frequency increases with the increasing age¹⁶. The incidence of H. pylori infection in the patients with gastroesophageal reflux disease varies widely in literature from 3% to 90% and with approximately a consensus of 35% in most series¹⁷. 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% have been reported in gastric mucosa from patients with H pylori associated gastritis¹⁸, but this association becomes weaker in adults with chronic non-active, particularly atrophic gastritis¹⁹. In the light of existing literature it is seen that H. pylori infection is associated with the severity of gastritis followed by gastric malignancies and primary gastric B cell lymphoma²⁰.

MATERIALS AND METHODS

Two hundred eighty consecutive endoscopic gastric biopsies received in Dr. Tahir Laboratory, Hamdard University Hospital Karachi during July 2009 to August 2012. This is a descriptive study. The specimens were mostly received from the Department of Gastroenterology Hamdard University Hospital. The relevant clinical information and demographic data was obtained from the histopathology request form. The gastrointestinal symptoms included abdominal pain, dyspepsia, vomiting, diarrhea and weight loss. Patients of all ages and both sexes having undergone gastric biopsy were included in the study.

The tissue was fixed in 10% formaldehyde, processed four to five sections of 4 micron thickness were cut on rotary microtome and stained with Haematoxylin and Eosin (H&E), Periodic-acid Schiff (PAS) and Giemsa staining was also performed.

All these biopsies were graded morphologically according to updated Sydney system emphasizing the importance of combining topographical, morphological and etiological aspects together for making clinical diagnosis of chronic gastritis. Two pathologists examined the sections independently and final histological diagnosis was made in the light of clinical and endoscopic findings.

Statistical Analysis: SPSS version 10.0 was used to analyze the data. Percentages were calculated to describe the data.

RESULTS

A total of 280 gastric biopsies were examined in the three year period from July 2009 to August 2012 at Department of Histopathology Dr. Tahir Laboratory at Hamdard University Hospital Karachi.

The age of patients varied from 17 to an age of 78 years. Out of 280 gastric biopsies, 124(44.28%) were from male patients and 151(53.28%) from female. The majority of the patients were biopsied for either gastritis, dyspepsia, gastric / duodenal ulcer or chronic diarrhoea. The specimens varied from 1 to 4 tiny fragments of antral / body parts of the stomach tissue (average 2-3).

Out of 280 biopsies 141 (50.35%) cases were diagnosed as Helicobacter positive chronic active gastritis, 41 (14.64%) were found to be H. Pylori negative active gastritis, 70 (25%) cases were labeled as Chronic non specific gastritis. 3 (1.07%) patients revealed dysplasia, follicular gastritis was found in 03 (1.07%) cases. 1 (.35%) case was positive for chronic granulomatous inflammation consistent with tuberculosis. 04 (1.42%) show signet cell type carcinoma whereas intestinal type gastric carcinoma found in 02 (071%) cases. 09 (3.12%) cases were unremarkable for any active pathology.

Table No.1: Total number of cases in three years with gender distribution.

No. of cases	Male (%)	Female (%)
280	124 (44.28 %)	151 (53.92 %)

Table No.2: Spectrum of gastric lesions from 2009-2012

Sr. No.	Gastric Lesion	No. of cases	%
1	H. Pylori associated chronic active gastritis	141	50.35 %
2	Chronic Active gastritis (H. pylori -ve)	41	14.64 %
3	Dysplasia	03	1.07 %
4	Chronic non specific gastritis	70	25 %
5	Follicular gastritis	03	1.07 %
6	Chronic caseating granulomatous inflammation	01	0.35 %
7	Unremarkable pathology	15	5.35 %
8	Signet cell gastric carcinoma	04	1.42 %
9	Intestinal type gastric carcinoma	02	0.71 %

DISCUSSION

Demographic data related to age, sex and the clinical presentation in our cases showed trends similar to other reported studies^{21,22}. However a local study by Siddiqui S et al is in consistent with our data as H. pylori infection is more common in middle aged adults²³.

International and national literature shows a wide variation of H pylori associated gastritis from 30-90%.²⁴ In another study the H pylori positive gastritis was seen in 37.3% (391/1046) and H pylori negative gastritis in 53.2% (567/1046)²⁵. A local study conducted by Mohsin A. et al in hospital setting showed 43.6% frequency of H. pylori in gastritis²⁶.

According to our observations Helicobacter pylori associated gastritis was seen in 50.35% cases, it is comparable to international and national studies. Waleed MA et al found the overall prevalence of H. pylori in 362 dyspeptic patients was 49.7% with some variation in age, race and sex. They suggested the use of noninvasive test in their community for example urea breath test or stool antigen test.²⁷

However in a local study which was carried out in Northern Punjab and Khyber Pakhtunkhwa region histologically 85.89% cases revealed chronic active gastritis and chronic inactive gastritis and H. pylori positivity was seen in 70%, involving the adult population²⁸.

Comparing the frequency of malignant primary gastric tumors our experience was 2.6% cases of gastric adenocarcinoma (Signet ring type 1.42% and intestinal type 0.71%. however the frequency of gastric adenocarcinoma was slightly higher in study by Mubarak et al found 5.71% cases which were clinically

suspected of gastric tumors and stated a diagnostic yield of over 95% for endoscopic gastric biopsy²⁸

CONCLUSION

We conclude that chronic active gastritis was the commonest lesion noted in the endoscopic antral biopsies. Majority of the cases were associated with *H. pylori* infection, which reveals its high prevalence in Pakistan. Malignant tumors as a group were next frequently seen pathological entity.

REFERENCES

1. Blackstone MO. Endoscopic Interpretation. Normal and pathologic appearances of the Gastrointestinal tract. Raven Press New York 1984; 1: 13-15.
2. Sipponen P, Stolte M. Clinical impact of routine biopsies of the gastric antrum and body. *Endoscopy* 1997; 29: 671-8.
3. Sipponen P. Update on the pathologic approach to the diagnosis of gastritis, gastric atrophy and *Helicobacter pylori* and its sequelae. *J Clin Gastroenterol* 2001; 32: 196-202.
4. Suvakovic Z, Bramble MG, Jones R, Wilson G, Idle N, Ryott J, et al. Improving the detection rate of early gastric cancer requires more than open access gastroscopy: a five year study. *Gut* 1997; 41 (3): 308 – 313.
5. Pasricha PJ. Gastrointestinal Endoscopy. In: Lee Goldman J, Bennett C, editors. *Cecil Textbook of Medicine*. WB Saunders; 2000.p.649-650.
6. Warren JR, Marshall BJ. Unidentified curved bacilli on gastric epithelium in active chronic gastritis. *Lancet* 1983; 1: 1273 – 1275.
7. Kazi JI, Jafarey NA, Alam SM, et al. Association of *Helicobacter pylori* with acid peptic disease in Karachi. *J Pak Med Assoc* 1990; 40: 240.
8. Forman D, Webb P, Parsonnet J. *Helicobacter pylori* and gastric cancer. *Lancet* 1995;345:1591-4.
9. Uemura N, Okamoto S, Yamamoto S, Matsumura N, Yamagudi S, Yamakido M, et al. *Helicobacter pylori* infection and the development of gastric cancer. *N Engl J Med* 2001; 345: 784 – 9.
10. Versalovic J. *Helicobacter pylori*. Pathology and diagnostic strategies. *Am J Clin Pathol* 2003;119: 403 – 412.
11. Madan E, Kemp J, Westblom TU, Subik M, Sexton S, Cook J, et al. Evaluations of staining methods for identifying *Campylobacter pylori*. *Am J Clin Path* 1988; 90: 450.
12. Santos IS, Boccio J, Santos AS, Valle NC, Halal CS, Bachilli MC, et al. Prevalence of *Helicobacter pylori* infection and associated factors among adults in Southern Brazil: a population-based cross-sectional study. *BMC Public Health* 2005; 5:118.
13. Rosenstock S, Jørgensen T, Bonnevie O, Andersen L. Risk factors for peptic ulcer disease: a population based prospective cohort study comprising 2416 Danish adults. *Gut* 2003;52: 186-93.
14. Hsu PI, Lai KH, Hsu PN, Lo GH, Yu HC, Chen WC, et al. *Helicobacter pylori* infection and the risk of gastric malignancy. *Am J Gastroenterol* 2007; 102: 725-30.
15. Hassan SR, Abbas Z. Presence of *helicobacter pylori* in dyspeptic patients with endoscopically normal stomach. *Pak J Med Sci* 2007; 23: 335-9.
16. Ghani MH, Ghouri AA, Hanif R, Bux H, Ahmed S, Humaira M. Frequency of *helicobacter pylori* in patients with peptic ulcer disease. *Med Channel* 2007; 13: 60-5.
17. Grande M, Cadeddu F, Villa M, Attina GM, Muzi MG, Nigro C, et al. *Helicobacter pylori* and gastroesophageal reflux disease. *World J Surg Oncol* 2008; 6: 74.
18. 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; 55:133-7.
19. Skiewicz K, Kobierska G. Lymphoid aggregates in gastric biopsies: relationship to other mucosal lesions. *Arch Immunol Ther Exp (Warsz)* 2000; 48: 201-4.
20. Engel LS, Chow WH, Vaughan TL, Gammon MD, Risch HA, Stanford JL, et al. Population attributable risks of esophageal and gastric cancers. *J Natl Cancer Inst* 2003; 95: 1404-13.
21. Khaar HB, Umar M, Khurram M et al. Endoscopic and Histopathological Evaluation of 306 Dyspeptic patients. *Pak J Gastroenterol* 2003; 17: 4 – 7.
22. Malekzadeh R, Sotoudeh M, Derakhshan MH, Mikaeli J, Yazdanbod A, Merat S, et al. Prevalence of gastric precancerous lesions in Ardabil, a high incidence province for gastric adenocarcinoma in the northwest of Iran. *J Clin Pathol* 2004; 57:37-42.
23. Siddiqui ST, Naz E, Danish F, Mirza T, Aziz S, Ali A, Frequency of *Helicobacter pylori* in biopsy proven gastritis and its association with lymphoid follicle formation. *JPMA* 2011;61(2), JPMA 61:138; 138-141
24. Shaheen B, Shaikh N, Zahir N, Akhter S, Vashwani A, Manzoor H. Histopathologic spectrum of upper gastrointestinal endoscopy. *Medical Channel* 2009;15(4):11-14
25. Wabinga H. *Helicobacter pylori* and histopathological changes of gastric mucosa in Uganda population with varying prevalence of stomach cancer. *Afr Health Sci* 2005; 5: 234-7.
26. Mohsin A, Qayyum A, Hussain I, Mirza A, Shah AA, Zaidi SNR. *Helicobacter Pylori* Prevalence and Eradication Study: *Helicobacter Pylori* Prevalence: An experience with patients presenting to Jinnah Hospital, Lahore. *Ann King Edward Med Col* 1999; 5: 95-6.
27. Waleed M A, Siddique I, Alateeqi N, Nakib B. Prevalence of *Helicobacter pylori* infection among new outpatients with dyspepsia in Kuwait. *BMC Gastroenterol* 2010;10:14
28. Afzal S, Ahmad M, Mubarik A, Saeed F, Rafi S, Saleem N, Hussain A, morphological spectrum of gastric lesion- endoscopic biopsy findings. *PAFMJ* 2006;56(2):143-9.