

The Histopathological Study on *Helicobacter pylori* Associated Gastroduodenal Diseases in a Tertiary Care Hospital, Mysore

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Abstract: *Gastroduodenal diseases are common disorders which are known to be caused by Helicobacter.pylori. Histopathological study is the mainstay in the diagnosis of H.pylori. The study sets out to evaluate histopathological diagnosis of gastric biopsy and to correlate histopathological and endoscopic findings. A total of 48 biopsy samples were studied retrospectively using revised Sydney system. Antral biopsies were stained with Haematoxylin and eosin for light microscopy. Giemsa was done wherever necessary. Among 48 patients 36 were males and 12 were females (3:1). Histopathology revealed varying degrees of chronic gastritis, with or without H.pylori, activity, intestinal metaplasia and atrophy. Out of 48 patients, 35(72%) were histopathologically positive for H.pylori. Endoscopic findings revealed 13(27%) duodenal ulcer cases, 9(18.7%) gastric ulcer cases, 4(8.3%) gastroduodenal ulcer cases and 16(33.3%) erosions. Endoscopic findings of 6 (12.5%) patients revealed normal picture. In conclusion, Endoscopic and histopathological examination go hand in hand in the diagnosis of gastro duodenal diseases. This study emphasizes the necessity of correlating histopathological findings with the endoscopic impression.*

Keywords: *Helicobacter pylori*, Gastroduodenal diseases, Histopathology, Endoscopy, Antral biopsy.

1. Introduction

In 1982 Warren and Marshal described *Helicobacter pylori* as a helical shaped Gram negative bacterium, it is found to be associated with gastritis, gastric ulcer, duodenal ulcer, gastric cancer and other gastroduodenal diseases [1]-[4]. Half of the world population is known to be infected with *Helicobacter pylori* and existence of diseases are higher in developing countries compared to developed countries [5]. Overcrowding and poor hygiene conditions are the known reasons for the infection. Contaminated food and water, direct person to person contact are the possible mode of transmission of infection [6]. The bacterium infects about 80% of the population in India and most of them acquire the infection since 10 years of age [7]. In India the prevalence of dyspepsia and peptic ulcer are common but information available in this regard is little [8]. In 1990 Sydney system was introduced to grade the gastritis and also to reduce the diagnostic confusion. In 1994, Sydney system was updated to get brief description of chronic gastritis[9].Nowadays many methods are available for detecting *H.pylori*, the most sensitive and commonest being the histological method [10]. In 2/3rd cases of gastroduodenal diseases, abnormalities are revealed from the gastric biopsy specimen by the histopathological study [3]. Types of gastritis is best revealed by histopathological study, so the results would be better if endoscopic examination is followed by histopathological study [11].

2. Materials and Methods

The subjects were patients who underwent upper GI endoscopy in a tertiary care hospital in Mysore, for various gastroduodenal diseases. The selected subjects were residing in and around Mysore, Karnataka, South India. Patients below 20 years of age were excluded from this study. From May 2014 – December 2014, a total of 48 patients were selected for this study. All patients had physical examination, their demographic data were collected along with pre-approved consent. Ethical clearance was obtained from the institute.

Endoscopic Procedure:

Endoscopic examination was performed by a single gastroenterologist who was aware of clinical symptom status of each subject. Subjects underwent an upper GI endoscopy with Olympus GIF H190/ GIF H150 endoscope under topical or local pharyngeal anaesthetic i.e., 4% xylocaine. The endoscope and biopsy forceps were sterilized with 2% glutaraldehyde under the brand name korsolex. All patients had an evaluation of the esophagus, stomach and duodenum and abnormalities were documented. Past ulceration was inferred if there was definitive evidence of scarring or deformed pylorus/ D1.

Histopathological procedure:

Antral biopsy specimens were taken in 10% formalin for overnight fixation. After processing paraffin blocks, 5µm sections were cut from it. The sections were stained with Haematoxylin and eosin along with Giemsa for light

microscopy. The severity and activity of gastritis, atrophy, and intestinal metaplasia, in addition to *H pylori* density, were evaluated according to the updated Sydney system (Refer fig 1,2,3). The infiltration of gastric mucosa by mononuclear cells and neutrophils, atrophy and intestinal metaplasia were graded as follows: 0, none; 1, mild; 2, moderate; 3, severe. Atrophy was defined as the loss of inherent glandular tissue, with or without replacement by intestinal-type epithelium.

3. Results

A total of 48 gastric biopsies were studied retrospectively over a period of 7 months, from May 2014 to December 2014. Out of these 36 were males and 12 were females, resulting in male to female ratio 3:1 with the age ranging from 21 years to 90 years and the mean age was 46. Most common age group affected was 40-60years (Table 1).

Table 1: Age wise distribution of gastroduodenal diseases

Age group	Total No of cases	Percentage (%)
20 – 30	5	10.4
31 – 40	11	22.9
41 – 50	12	25
51 – 60	12	25
61 – 70	5	10.4
71 – 80	1	2
81 – 90	2	4.1

Endoscopic Findings:

Out of 48, 13 patients had duodenal ulcer, 9 had gastric ulcer, 4 had both gastric and duodenal ulcer, 16 had erosions, and 6 were normal (Table 2).

Table 2: Endoscopic findings in all the patients

Endoscopic findings	No of cases	Percentage (%)
Gastric erosions	16	33.3
Duodenal ulcer	13	27
Gastric ulcer	9	18.5
Gastroduodenal ulcer	4	8.3
Normal	6	12.5

Histopathological findings

Inflammatory infiltrate was seen maximally in duodenal ulcer (76.9%), followed by gastric ulcer(66.6%), gastric erosions(37.5%) and both gastroduodenal ulcer(50%).In 12.5% of cases having a normal looking mucosa on endoscopy, inflammatory infiltrate was seen in 66.6%. Histologically *H. pylori* density and lymphoid follicles were maximum in duodenal ulcer. Table 3 and 4 shows endoscopic and histopathological correlation of grading and non-grading variables respectively.

Table 3: Endoscopic and histopathological correlation of grading variables

		D	N	MN
E (n=16) 33.3%	Mi	2(12.5%)	2(12.5%)	3(18.75%)
	Mo	6(37.5%)	0	3(18.75%)
	Se	2(12.5%)	0	0
DU (n=13) 27%	Mi	3(38.4%)	5(38.4%)	1(7.6%)
	Mo	3(23%)	0	7(53.8%)
	Se	5(38.4%)	1(7.69%)	2(15.3%)

GU (n= 9) 18.5%	Mi	4(44.4%)	3(33.3%)	3(33.3%)
	Mo	3(33.3%)	0	3(33.3%)
	Se	1(11.1%)	1(11.1%)	0
GDU (n=4) 8.3%	Mi	2(50%)	1(25%)	2(50%)
	Mo	0	1(25%)	0
	Se	1(25%)	1(25%)	0
N (n=6) 12.5%	Mi	2(12.5%)	0	1(6.25%)
	Mo	0	1(6.25%)	0
	Se	1(6.25%)	0	1(6.25%)

E-Erosions, DU- Duodenal ulcer, GU-Gastric ulcer, GDU-Gastroduodenal ulcer, N-Normal, n-Total Number, Mi-Mild, Mo-Moderate, Se-Severe, D- *H.pylori* density, N-Neutrophils (Activity), MN-Mononuclear infiltrate (Inflammatory infiltrate)

Table 4: Endoscopic and histopathological correlation of grading variables

		A	IM	D
E (n=16) 33.3%	Mi	0	0	0
	Mo	0	0	0
	Se	0	0	0
DU (n=13) 27%	Mi	1(7.6%)	1(7.6%)	0
	Mo	0	0	0
	Se	1(7.6%)	0	0
GU (n= 9) 18.5%	Mi	0	2(22.2%)	0
	Mo	1(11.1%)	0	0
	Se	0	0	0
GDU (n=4) 8.3%	Mi	0	0	0
	Mo	0	0	0
	Se	0	0	0
N (n=6) 12.5%	Mi	0	0	0
	Mo	0	0	0
	Se	0	0	0

E-Erosions, DU- Duodenal ulcer, GU-Gastric ulcer, GDU-Gastroduodenal ulcer, N-Normal, n-Total Number, Mi-Mild, Mo-Moderate,Se-Severe,A-Atrophy, IM-Intestinal Metaplasia, D-Dysplasia

Table 5: Endoscopic and histopathological correlation of non-grading variables

F	E TC=16	DU TC=13	GU TC=9	GDU TC=4	N TC=6
G	7(43.7%)	9(69.2%)	3(33.3%)	2(50%)	3(50%)
LF	0	2(15.4%)	1(11.1%)	1(25%)	1(16.6%)
HE	2(12.5%)	0	0	0	0

F-Features, E-Antral Erosions, DU-Duodenal ulcers, GU-Gastric ulcers,GDU-Gastric and Duodenal ulcers, N-Normal, TC-Total Cases, G-Gastritis, LF-Lymphoid follicle, HE-Hyperplastic epithelium

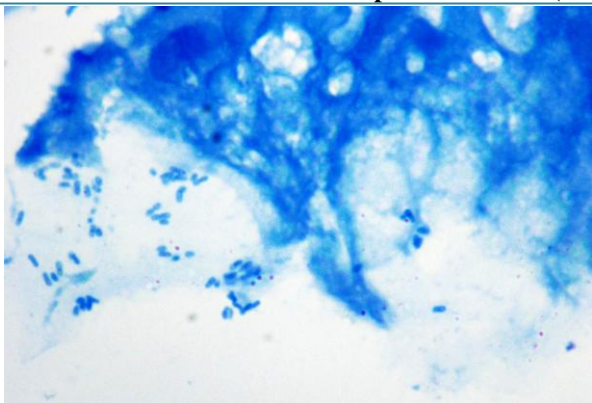


Figure 1: GIMESA X 1000 spiral *H.pylori* are seen

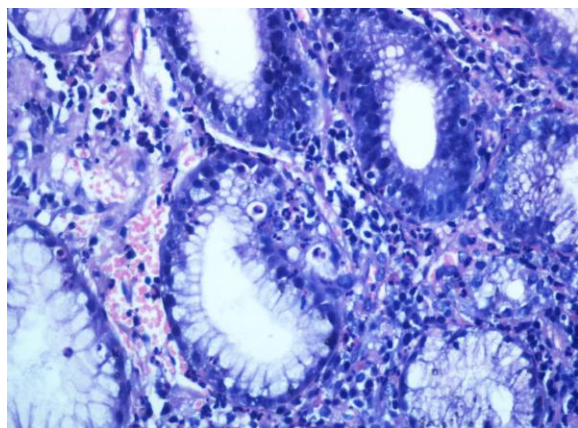


Figure 2: H&E x 400, Chronic gastritis with activity (intraepithelial neutrophils)

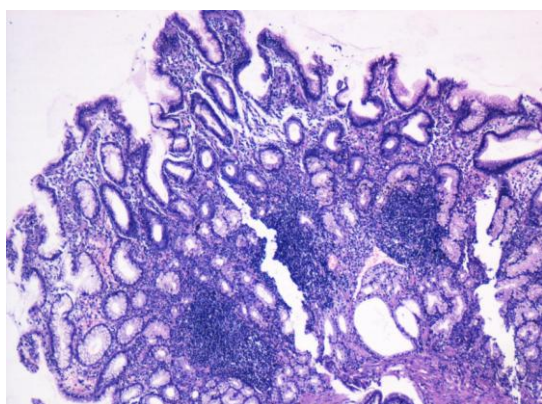


Figure 3: H&E X 20 sections shows *H.pylori* associated gastritis with hyperplastic foveolae and lymphoid follicle

4. Discussion

In our present study, the most common age group was 40 – 60 years with the mean age range of 46 years, this is consistent with studies done by Adyin et al [12]. Mustapa et al [13] who reported a mean age of 47.2 and 47 years. The Male:Female ratio during this study was 3 :1. Kumar et al. also found similar M:F of 2.7:1 [14].

In this study we have concentrated only on antral biopsies since most of the studies have reported antrum as the most likely site of histopathological findings in gastritis [15]. The criteria laid down by Aydin et al. [12] are extremely useful in analysing the gastric biopsies for suspected gastritis.

The endoscopic findings in our study revealed the presence of erosions in 33%, duodenal ulcer in 27%, gastric ulcer in 18%, gastroduodenal ulcer in 8% and normal gastric mucosa in 12.5% of cases. This is in contradiction with Khakoo et al.[16] in which they found erythema in the majority of the cases (45.8%). In 20% of cases who were endoscopically normal, inflammation was revealed on histology; which was also observed by Khan et al. [17] who had 32% of patients with chronic gastritis histologically but their endoscopic findings were normal, thereby emphasizing the role of biopsy even in endoscopically normal individuals. In patients with duodenal ulcer, colonization by *H. pylori* was seen in 84.6% of cases; inflammatory infiltrate was present in all the cases with the majority (50%) having only moderate inflammation while 36.3% had mild inflammation. A study done by Witteman et al. [18] observed chronic infiltrate in all biopsies but the majority had moderate inflammation which is similar to our study. In this study, the majority of patients with duodenal ulcer had more severe gastritis on histology as compared to subjects having erosions. In 33.3% of the cases, activity was seen which is in accordance with a study published by Misra et al.[19]. The occurrence of atrophy (6.25%) and intestinal metaplasia (6.25%) was infrequent, which was also observed in studies by Atisook et al. [20] and Nawfal et al. [21] *Helicobacter pylori* was observed in 66.6% of all the cases, which is in accordance with the studies done by Kumar et al.[14] and Gill et al.[22] which showed positivity in 78% and 65% of cases respectively.

5. Conclusion

H.pylori infection is more prevalent in developing countries like India. Early diagnosis helps in early initiation of treatment and thereby eradication of *H.pylori*. Endoscopic examination and biopsy is a convenient procedure for accurate diagnosis. Endoscopy is incomplete without biopsy and histopathology is the gold standard for the diagnosis of endoscopically detected lesions. Endoscopic biopsy correlation is important in understanding the biology and pathophysiology of the disease. Awareness of the histomorphological features that are typical of *H.pylori* gastritis would be helpful to the clinicians to identify other conditions like atrophic gastritis and intestinal metaplasia and thereby prevent the progression to carcinoma. This correlation provides new information about the prevalence of the disease and thereby assists in improving patient management.

6. Future Scope

Further studies are required with more number of subjects to establish the use of endoscopy and histopathology in the diagnosis of *H.pylori* associated gastroduodenal diseases.

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Conflict of Interest: None

References

- [1] M Hemalata et al, prevalence of *Helicobacter pylori* infection and histomorphologic spectrum in endoscopic biopsies. *Ijbr*2013; 4(11): 608-615.
- [2] Adisa JO et al, *Helicobacter pylori* associated gastritis in North – eastern Nigeria A histomorphological study. *E-International scientific research journal* 2011; 3: 1-4
- [3] Owen DA Gastritis and carditis. *Mod pathol* 2003;16(4):325-341
- [4] Owen DA Gastritis and duodenitis. In *applenac HD ed Pathology of the esophagus, stomach and duodenum Churchill Living stone*. 1986: 43-49.
- [5] Dorer MS, Talarico S, Salama NR. *Helicobacter pylori's* unconventional role in health and disease. *PLoS Pathog* 2009; 5:e1000544. Epub 2009 Oct 26.
- [6] Salih BA. *Helicobacter pylori* infection in developing countries: theburden for how long? *Saudi J Gastroenterol* 2009; 15:201-7.
- [7] Poddar U, Yachha SK. *Helicobacter pylori* in children: an Indian perspective. *Indian Pediatr* 2007; 44:761-70.
- [8] Tovey FI Peptic ulcer In India and Bangladesh. *Gut*, 1979;20:329-47
- [9] Dixon MF Genta RM, Yardely JH Classification and grading of gastritis. The updated Sydney system. International workshop on the histopathology of gastritis Houston. 1994, *Am J Surg.Pathol.*1996; 20:1161-81
- [10] Zhang C, Yamda N. Wu YL, Wen M Matsukura N. *Helicobacter pylori* infection, glandular atrophy and intestinal metaplasia in superficial gastritis, gastric erosions, gastric ulcer and early gastric cancer. *World Gastro enteral.*2005;11(6);791-796.
- [11] Sharma P, Topalovski M, Mayo M S, Sampliner RE. *Helicobacter pylori* eradication dramatically improves inflammation in the Gastric cardia. *Am J Gastroenterol.* 2009;95(11);3107-3111.
- [12] Adyin O, Egilmez R karabacak T, Kanika A. Interobserver variation in histopathological assessment of *Helicobacter pylori* gastritis, *World J of Gastroenterol* 2003;9:2232-2235
- [13] Mustapha SK, Bolori MT, et al. Endoscopic finding and the frequency of *Helicobacter pylori* among dyspeptic patients in North eastern Nigeria, *Interner J Gastroenterology*2007;6:1528-1532.
- [14] Kumar A, Bansal R, Pathak VP, Kishore S, Arya PK. Histopathological changes in gastric mucosa colonized by *H. pylori*. *Indian J Pathol Microbiol.* 2006; 49: 352 - 6.
- [15] Eriksson NK, Färkkilä MA, Voutilainen ME, Arkkila PE. The clinical value of taking routine biopsies from the incisura
- [16] *angularis* during gastroscopy. *Endoscopy* 2005; 37: 532-536.
- [17] Khakoo SI, Lobo AJ, Shepherd NA, Wilkinson SP. Histological assessment of the Sydney classification of endoscopic gastritis. *Gut* 1994; 35: 1172-1175.
- [18] KhanMQ, Alhoms Z, Al-Momen S, AhmadM. Endoscopic features of *Helicobacter pylori* induced gastritis. *Saudi J Gastroenterol* 1999; 5: 9-14.
- [19] Witteman EM, Mravunac M, Beck M J , et al. Improvement of gastric inflammation and resolution of epithelial damage one year after eradication of *Helicobacter pylori* . *J Clin Pathol* 1995; 48: 205-256.
- [20] MisraV, Misra SP, Dwivedi M, Singh PA. Point prevalence of peptic ulcer and gastric histology in healthy Indians with *Helicobacter pylori* infection. *Am J Gastroenterol* 1997; 92: 1487-1491.
- [21] Atisook K, Kachithron U Luengrojanakal P. Histology of gastritis and *Helicobacter pylori* infection in Thailand: a nation wide study of 3776 cases . *Helicobacter* 2003; 8: 132-141.
- [22] Hussein NR, Napali SM, Atherton JC. A study of *Helicobacter pylori*-associated gastritis patterns in Iraq and their association with strain virulence. *Saudi J Gastroenterol* 2009; 15:125-127.
- [23] Gill HH, Desai HG, Majmudar P et al. Epidemiology of *Helicobacter pylori*: the Indian scenario. *Indian Journal Gastroenterol* 1993;12: 9-11