ORIGINAL ARTICLE

Correlation Between Endoscopic and Histological Findings in Different Gastroduodenal Lesion and its Association with *Helicobacter Pylori*

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ABSTRACT

This study had been planned to see the histomorphological pattern of gastric mucosal biopsy, demonstrate the organism *H .Pylori* and correlate with endoscopic findings. The study was carried out in the department of the Pathology Rajshahi Medical College, Rajshahi, Bangladesh, during the period from January 2006 to December 2007. Endoscopic biopsy specimens were obtained from 105 cases of gastro duodenal lesions. According to endoscopic findings out of 105 cases 69(65.7%) had gastric carcinoma, 06 (5.71%) had gastric ulcer, 05 (4.76%) had duodenal ulcer, 05 (476%) had gastritis and 20 (19.04%) had normal mucosa, reported as non-ulcer dyspepsia. Of the 69 endoscopically gastric carcinoma 59 (85.50%) were diagnosed histologically as adenocarcinoma and 10 (14.50%) were diagnosed h-istologically as chronic gastritis in which H.Pylori was positive in 27.5% and 14.50% positive in chronic gastritis. Of 06 gastric ulcer 03 (50%) was diagnosed histologically as gastritis and 03 (50%) were diagnosed histologically as gastric carcinoma. H. Pylori was positive in cent percent of gastritis. Of 05 duodenal ulcer 03 (60%) were diagnosed histologically as gastritis and 02 (40%) were diagnosed histologically as intestinal metaplasia H. Pylori was positive in 66.6% of duodenal ulcer. Of 05 gastritis were diagnosed histologically as gastritis 60% were postive for H.Pylori. Of 20 NUD 18 (90%) were diagnosed histologically as gastritis, 01 (05%) was diagnosed histologically as ulcer and 01 (05%) was diagnosed histologically as normal mucosa, all these cases no H. Pylori was found .

Key Words: Helicobacter Pylori, Gastroduodenal Lesion, Association

Introduction

Helicobacter pylori represent one of the most common and medically important infections worldwide. *Helicobacter pylori* are gram negative, microaerophilic, spiral, motile bacterium that resides in the gastric pits and the overlying mucus blanket. *H. pylori* infection has been established firmly with the development of peptic ulcer, chronic active gastritis, chronic persistent gastritis, atrophic gastritis and gastric neoplasia including gastric adenocarcinoma and gastric mucosa-associated lymphoid tissue lymphomas. [1]

Recent studies have shown an association between long term infection of *H.pylori* and the development of gastric cancers, the second most common cancer world wide. Peptic ulcer disease have shown a very high prevalence in Bangladesh and are important among the major chronic problems encountered by the physicians and surgeons.[2] In a developing country like Bangladesh, overcrowding, bad sanitation and unhealthy practice favor high prevalence of *H. pylori* in the population.[3]

The association of *H. pylori* with gastritis, duodenal ulcer and gastric cancer has been reported by investigators from different countries all over the world.[4,5,6] Similar studies have been done in our country and high association of *H. pylori* in peptic ulcer and gastric cancer has been reported[7,8,9,10].

The risk of ulcer recurrence and associated complications are not diminished unless *H. pylori* infection is cured. Effective antimicrobial treatment depends on sensitive and accurate diagnostic approaches. There are several invasive and non invasive methods for diagnosis of *Helicobacter pylori* infection. Invasive methods requiring endoscopic evaluation include bacteri ologic culture, histopathologic studies, cytological examination of smear, rapid urease test or CLO (Campylobacter like organism) test and molecular studies. Non invasive approaches include serologic testing, fecal antigen detection and urea breath testing.[1]

Although culture is the gold standard, but it is very difficult to perform, requires an enriched transport medium, is expensive and results are delayed. Histology also takes at least 3-4 days and is costly. A further limitation of uses of histology with regard to sensitivity and specificity is the quality of biopsies. If the biopsy is too small, is poorly oriented or is inappropriately fixed or stained, detection of *H. pylori* may not be possible. Among the noninvasive methods, *H. pylori* antibody can be detected in serum by Enzyme Linked Immuno Sorbent Assay (ELISA) and also by Immuno Chromatographic (ICT) rapid spot test. This study had been planned to see the histomorphological pattern of gastric mucosal biopsy, demonstrate the organism *H.pylori* and correlate with endoscopic findings.

Materials and methods

Endoscopic biopsy specimens were obtained from clinically suspected 105 cases of gastro duodenal lesions and who had not taken antibiotics, omeprazole or bismuth salts for at least three weeks prior to endoscopy were selected. . Specimens were collected from the endoscopy unit of the gastroenterology department of Rajshahi Medical College Hospital, Zamzam Islami Hospital and Islami Bank Medical College Hospital. The study was carried out in the department of the Pathology Rajshahi Medical College, Rajshahi, Bangladesh, during the period from January 2006 to December 2007.

Each biopsy from fundus, body and antrum were put into a small-labeled bottle containing 10% buffered formalin and brought to the laboratory in the department of pathology, Rajshahi Medical College, Rajshahi for subsequent processing for histopathological examination It was then processed and embedded in paraffin wax and was cut into sections of 4 micrometer thickness. Two slides were made from each block-one for Haematoxylin and Eosin (H&E) stain and one for modified Giemsa stain. Sections were looked for infiltrations of neutrophils and mononuclear cells, glandular atrophy, intestinal metaplasia, dysplasia, presence of lymphoid follicle and *H. pylori*. All the findings were graded in scale according to the previous investigators[11]

Results

Among the 105 cases, 76 were male(72.3%) and 29 were female(27.6%). The male-female ratio was 2.6:1. The age range of patients was 28 and 90 years with mean age of 54.66 years. The age and sex distribution of the study groups are shown in table I.

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Table I: Age and sex distribution of the study group (according to decades)

Age group (year)	Number	Male	Female	M:F
21-30	02	01	01	1:1
31-40	14	09	05	1.8:1
41-50	28	23	05	4.6:1
51-60	37	26	11	2.3:1
61-70	20	14	06	2.3:1
>70	04	03	01	3:1
Total	105	76	29	2.6:1

Endoscopically diagnosed different gastrodu odenal lesions and NUD with their male female ratio is shown in table II. The highest numbers of patients were with Gastric carcinoma followed by NUD, gastric ulcer, duodenal ulcer and gastritis.

Table II: Sex distributions with their endoscopic diagnosis:

Subjects	Number (%)	Male	Female	M:F
NUD	20(19.04%)	15	05	3:1
Gastritis	05(4.76%)	04	01	4:1
Duodenal ulcer	05(4.76%)	05	00	–
Gastric ulcer	06 (5.71%)	03	03	1:1
Gastric Carcinoma	69 (65.71%)	48	21	2.2:1
Total	105 (100.00%)	76	29	2.6:1

NUD= Non ulcer dyspepsia

Distribution of patients in different age groups is shown in table III. The peak age of incidence of gastritis duodenal ulcer gastric ulcer and gastric carcinoma all were found in the sixth decade.

Table III: Distribution of patients in different age groups :

Age group (year)	NUD	Gastritis	Duodenal ulcer	Gastric ulcer	Gastric carcinoma	Total
21-30	01	00	00	00	01	02
31-40	04	01	00	01	10	16
41-50	08	00	02	01	18	29
51-60	03	04	03	04	22	36
61-70	03	00	00	00	17	20
>70	01	00	00	00	01	02
Total	20	05	05	06	69	105

The correlation between endoscopic findings with histological findings is shown in table IV.

Table IV: Correlation between endoscopic findings with histological findings and association with *H. pylori*:

Endoscopic findings	Number	Histological findings					*H. pylori* association
		Normal	Ulcer	Gastritis	Metaplasia	Carcinoma	
NUD	20	01	01	18	00	00	00
Gastritis	05	00	00	05	00	00	03
Duodenal ulcer	05	00	00	03	02	00	02
Gastric ulcer	06	00	00	03	00	03	03
Gastric carcinoma	69	00	00	09	01	59	19
Total	105	01	01	38	03	62	27

For detection of *H. pylori* infection in 105 cases, Haematoxylin & Eosin and modified Giemsa stained histological sections . *Helicobacter pylori* infection was detected by Haematoxylin & Eosin in 25 (23.80%) cases, by modified Giemsa stain in 27 (25.71%) cases. This is shown in table V.

Table V: Detection of *Helicobacter pylori* infection by different methods:

Methods	Total test	H. pylori positive No.(%)	H. pylori negative No.(%)
Haematoxylin & Eosin stain	105	25(23.80%)	80 (76.19%)
Modified Giemsa stain	105	27(25.71%)	78(74.29%)

Presence of Helicobacter in different gastroduodenal lesions is shown in table VI. The association of Helicobacter pylori was found significant with gastritis, duodenal ulcer and gastric ulcer when compared with non-ulcer dyspepsia (Chi-square test).

Table VI: Presence of Helicobacter pylori in gastric samples of patients with different gastro duodenal lesions (Endoscopically) using Haematoxylin & Eosin and modified Giemsa stain:

Subject	Number	H. P+ve	H. P–ve	H. P+ve%	P value
NUD	20	00	20	00 %	
Gastritis	05	03	02	60%	P<0.001
Gastric ulcer	06	03	03	50%	P<0.001
Duodenal ulcer	05	02	03	66.6%	P<0.001
Gastric carcinoma	69	19	50	27.5%	P<0.02
Total	105	27	78	25.7%	

(x^2= P<0.001); Significant.

The presence of lymphoid follicle, atrophy and intestinal metaplasia were studied in Haematoxylin & Eosin stained sections. Only three (03) patients showed intestinal metaplasia and none of the patient showed any gastric atrophy and lymphoid follicle.

Discussion

According to endoscopic findings out of 105 cases 69(65.7%) had gastric carcinoma, 06 (5.71%) had gastric ulcer, 05 (4.76%) had duodenal ulcer, 05 (476%) had gastritis and 20 (19.04%) had normal mucosa, reported as non-ulcer dyspepsia . Of the 69 endoscopically gastric carcinoma 59 (85.50%) were diagnosed histologically as adenocarcinoma, *H. pylori* positive in19(27.5%) and 10 (14.50%) were diagnosed histologically as chronic gastritis. Of 06 gastric ulcer 03 (50%) was diagnosed histologically as gastritis , *H. pylori* positive 03(50%)and 03 (50%) were diagnosed histologically as gastric carcinoma. Of 05 duodenal ulcer 03 (60%) were diagnosed histologically as gastritis , *H. pylori* positive in 02(66.6%) and 02 (40%) were diagnosed histologically as intestinal metaplasia. Of 05 gastritis were diagnosed histologically 05 (100%) as gastritis and *H. pylori* positive 03(60%). Of 20 NUD 18 (90%) were diagnosed histologically as gastritis, 01 (05%) was diagnosed histologically as ulcer and 01 (05%) was diagnosed histologically as normal mucosa.

In the present study, the range of total 105 patients was from 29 to 90 years with mean age of 54.6 years. The male female ratio was 2.6:1, this can be explained by higher gastric acid level in male.[11] According to endoscopic findings out of 105 cases 69(65.7%) had gastric carcinoma, 06 (5.71%) had gastric ulcer, 05 (4.76%) had duodenal ulcer, 05

(4.76%) had gastritis and 20 (19.04%) had normal mucosa, reported as non-ulcer dyspepsia . Of the 69 endoscopically diagnosed gastric carcinoma 59 (85.50%) were diagnosed histologically as adenocarcinoma in which *H. pylori* positive in19(27.5%) and 10 (14.50%) were diagnosed histologically as chronic gastritis. Of 06 endoscopically diagnosed gastric ulcer 03 (50%) was diagnosed histologically as gastritis in which *H. pylori* positive in 03(50%) and 03 (50%) were diagnosed histologically as gastric carcinoma. Of 05 endoscopically diagnosed duodenal ulcer 03 (60%) were diagnosed histologically as gastritis in which *H. pylori* positive in 02(66.6%) and 02 (40%) were diagnosed histologically as intestinal metaplasia. Of 05 endoscopically diagnosed gastritis were diagnosed histologically 05 (100%) as gastritis and *H. pylori* positive in 03(60%). Of 20 endoscopically diagnosed NUD 18 (90%) were diagnosed histologically as gastritis, 01 (05%) was diagnosed histologically as ulcer and 01 (05%) was diagnosed histologically as normal mucosa and all these cases were *H. pylori* negative.

Haematoxylin and Eosin stain and modified Giemsa stained respectively alone detected *H. pylori* 25 (23.80%) and 27 (25.71%) cases.

Patients with gastric carcinoma, gastritis, duodenal ulcer and gastric ulcer showed significantly higher positives for *H. pylori* when compared with NUD subjects.

In the present study, the sensitivity of H & E stain and modified Giemsa stained sections are 50% and 50 % respectively and specificity are 96.36% and 100 % respectively.

Talukder, 1995; reported the presence of *H. pylori* to be 67.74%, 81.25% and 71.42% in gastritis, duodenal ulcer and gastric ulcer respectively by using H&E stain and endoscopic findings. Hussain,1996; reported 71.70% *H. pylori* positive case in gastritis, 80.95% in duodenal ulcer, 53.60% in gastric ulcer and 60.00% in gastric cancer by using H&E stain and endoscopic findings. Akanda, 2006, reported 47.10% *H. pylori* positive case in gastritis, 54.50% in gastric ulcer and 94.30% in duodenal ulcer by using H&E stain, modified Giemsa stain and CLO test.

Intestinal metaplasia were found in three cases and none of the patients showed any lymphoid follicle, lymphoid aggregates and atrophy. Dixon, 1995 [12] stated that *H. pylori* is absent at the site of intestinal metaplasia.

Conclusion

It was observed that in Haematoxylin and Eosin (H &E) stain, modified Giemsa stain detected *H. pylori* in 25(23.80%) and 27(25.71%) cases respectively of different gastroduodenal lesion. Significantly higher positives were observed in gastric carcinoma and gastritis as compared to non-ulcer dyspepsia. From above data it was revealed that modified Giemsa stain detected *H. pylori* infected patients more accurately than Haematoxylin and Eosin stain.

In the present study histologically diagnosed gastric carcinoma were 59(55.50%) in comparison to endoscopically diagnosed gastric carcinoma, 69(65.7%).

From the findings of this study, it can be concluded that *H. pylori* infection causes a varieties of gastroduodenal lesion. Further studies are required to establish the *H. pylori* positive cases with that of other tests such as serological detection of anti *H. pylori* antibody by ELISA/ICT and culture to establish a diagnosis quickly without any invasive method and institute proper management thus reducing morbidity.

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