ORIGINAL ARTICLE

Histopathological Findings of Gastric Biopsies in Dyspeptic Patients

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ABSTRACT

Aim: To determine the frequency of H. pylori infection in biopsy proven gastritis in Lahore.
Methods: During the period of January 2014 and October 2016, 71 consecutive gastric antral biopsy samples were included because it provides useful information regarding the H. pylori status. The study was approved by the ethical review board of Akhtar Saeed Medical and Dental College. Tissue sections were stained with haematoxylin and eosin for histological examination for severity of gastritis and activity of gastritis. Giemsa stain was used for H. pylori assessment.
Results: The prevalence of *H. pylori* was 53(74.64%) in these dyspeptic patients. Grading of inflammation was decided according to Sydney System. In the present study, histopathological examination of 71 patients revealed mild chronic gastritis in 17(23.9%) and moderate chronic gastritis in 51(71.8%) and severe in 2(2.81%) patients. The association was statistically significant (p value <0.05) by using ANOVA.
Conclusion: Prevalence of *H. pylori* infection in Pakistani population is shocking. Chronic gastritis is the major condition associated with dyspeptic patients.
Keywords: H.Pylori, haematoxylin and eosin, Giemsa stain, lymphoma

INTRODUCTION

Gastritis is the inflammation of the stomach mucosal lining and it is usually classified into acute and chronic gastritis**.** The prevalence and natural history of chronic gastritis has been significantly clarified by the use of endoscopic gastric biopsy[1,2]. Main features of chronic gastritis are infiltration of the lamina propria by inflammatory cells and atrophy of the glandular epithelium. Plasma cells and lymphocytes with few areas of follicles formation predominate, but eosinophils and neutrophils may also be present. In chronic superficial gastritis the inflammatory infiltrate is limited to the foveolar region and unaccompanied by glandular atrophy. In chronic atrophic gastritis the inflammation is more extensive and accompanied by glandular atrophy and it is further classified as mild, moderate, or severe by estimating the thickness of the glandular portion in relation to the thickness of the whole mucosa[3,4]. Thinning of the mucosa in the absence of inflammatory changes is categorized as gastric atrophy. It most probably represents the end stage of a chronic atrophic gastritis in many cases. Pyloric metaplasia of the fundic mucosa and intestinal metaplasia can be seen in chronic gastritis in few cases[5].

Chronic gastritis has been classified into two types with related histological features but with different pathogenic mechanisms. *Type A* or *immune* gastritis[6,7] usually affects the fundus diffusely but spares the antrum. Immune gastritis shows neuroendocrine hyperplasia and is associated with antibodies to parietal cells, achlorhydria or hypochlorhydria, and high gastrin levels in serum. Type B or nonimmune gastritis begins in the antrum and progresses to fundic–pyloric border[8,9]. Previously it is thought that multiple factors like alcohol, tobacco, reflux gastritis, food allergy, and various drugs particularly anti-inflammatory agents are related to pathogenesis of type B chronic gastritis[10,11,12,13]. This view has been modified by the knowledge of the critical role played by Helicobacter pylori[14,15]. It is previously known as Campylobacter pylori, is a curved spirochete-like bacterium, of which two major genotypes exist[16].

It was discovered in 1980s, and it inhabits different areas of the stomach and duodenum[17]. In 1994, the NIH Consensus Conference documented H. pylori as a cause of gastric and duodenal ulcers. The International Agency for Research on Cancer (IARC) declared H. pylori as a member of group I human carcinogen for gastric adenocarcinoma"[18]. It is regarded as the most widespread infection in the world as at least half of world's population is infected with H.Pylori[19]. Third world has higher rates of infection than the west, where the prevalence of H. pylori is declining[20]. Risk factors involved in the pathogenesis of Helicobacter pylori includes socioeconomic status, overcrowding, poor hygiene, alcohol consumption, occupational exposure, diet,

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smoking, family history of gastric diseases and poor water supply. The important route for its transmission is Fecal-oral[21]. H. pylori infection is usually acquired in early childhood[22]. In Pakistan, it is reported to be present in 69% of population presented with gastritis. Out of those (69%) patients, superficial gastritis was observed in 60.87% and atrophy of glands in 19.56% of cases[23].

But limited data is available on morphological changes in gastritis followed by *H. pylori* infection in Pakistan. In this study, we planned to report prevalence of *H. pylori* infection along with associated morphological changes in gastric mucosa. These changes include gastritis, activity, atrophy and intestinal metaplasia in dyspeptic patients of Lahore, Pakistan by histopathological study of biopsies[24].

MATERIAL AND METHODS

During the period of January 2014 and October 2016, 90 consecutive gastric antral biopsy samples were included because it provides useful information regarding the H. pylori status[25]. The study was approved by the ethical review board of Akhtar Saeed Medical and Dental College. After reviewing all the slides, eighteen cases were excluded from the study, of which twelve cases had inadequate material, four were diagnosed as antral polyps and three were those of adnenocarcinoma. Finally 71 cases were included in this prospective study at the Department of Histopathology, Akhtar Saeed Medical College and its affiliated hospitals.

The clinical data was analyzed for patient's age and gender. Four biopsies were collected from each patient (two each from body and antrum of the stomach). All gastric biopsy specimens for histological examination were fixed in 10% formalin, embedded in paraffin wax and cut into 4µm thick sequential sections. All tissue sections were stained with haematoxylin and eosin to document histological examination for severity of gastritis in terms of inflammation, according to Sydney system and lymphoid follicle formation. Giemsa stain was used for further H. pylori assessment. All gastric specimens were evaluated independently by three histopathologists and finally a consensus was made on the multihead microscope for the equivocal cases[25,26]. The histopathological confirmation of *H. pylori* infection was accomplished by H&E and where required by Giemsa staining. The presence of *H. pylori* was graded as absent or present. The grading and severity of gastritis was documented as nil, mild, moderate or severe, based on the Sydney system.10 Gastritis was confirmed on finding increased number of mononuclear inflammatory cells in lamina propria. Activity was recorded as present when an increase in the number of neutrophils was observed. Atrophic changes (damaged gastric lining leading to loss of gastric glandular cells) and intestinal metaplasia (replacement of gastric mucosal cells by resembling intestinal mucosal cells) was also determined[24].

The data was analyzed by using SPSS version 16. Frequencies and percentages were computed for the categorical variables like age, gender, H. pylori, severity and activity of gastritis.

RESULTS

The antral biopsies of 71 dyspeptic patients were tested for *H. pylori* presence, graded for gastritis and other morphological changes like atrophy and intestinal metaplasia. There were 29(40.84%) males and 42(59.15%) females. The mean age of subjects was 39.60±14.41 years. The minimum age was found to be 17 years where as maximum was 82 years. The status of *H. pylori* was positive if bacteria were seen in the tissue samples. The prevalence of *H. pylori* was 74.64% (53 cases) in these dyspeptic patients. Grading of inflammation was decided according to Sydney System. In the present study, histopathological examination of 71 patients revealed mild chronic gastritis in 17(23.9%) and moderate chronic gastritis in 51(71.8%) and severe in 2 (2.81%) patients. Evidence of activity was found in 24(33.8%) patients; mild in 3, moderate in 20 and severe in 1 patients. Atrophic changes were observed in 40(56.33%); mild in 9, moderate in 30 and severe in 1 patients. Intestinal metaplasia with atypia was present in 1 (1.40%) patient. The association was statistically significant (p value <0.05) by using ANOVA (Table 1,2)**.**

Table 1: Ages of patients in different severity groups

	N	Mean	Std. Deviation
Mild	17	39.88	12.499
Moderate	51	38.47	14.310
Severe	2	59.00	1.414
Atypical	1	70.00	.
Total	71	39.83	14.383

Table 2: Comparison of ages (patients) with severity of chronic gastritis

	Sum of Squares	Df	Mean Square	F	Sig.
Between Groups	1739.501	3	579.834	3.049	.035
Within Groups	12740.471	67	190.156		
Total	14479.972	70			

DISCUSSION

H. pylori is considered to be the most important cause of chronic gastritis, duodenal and gastric ulcers and it has been classified as class-1 human carcinogen. [18] The disease progression following *H. pylori* infections is gastritis followed by atrophy, intestinal metaplasia and dysplasia that can lead to carcinoma of gastric mucosa. Gastritis develops in almost all individuals infected with *H.pylori* whereas gastric atrophy and intestinal metaplasia appear more often in *H. pylori* positive than in negative patients[27,28]. Prevalence of 73.61% seems on the higher side as compared to generally reported, but it corresponds to some reports like 84.6%, by another group using PCR in the same region[29] and 83% in another study from Islamabad. It is generally accepted that prevalence differs with the method of investigation employed and socioeconomic status of the patients. Histological examination is the most commonly definitive reliable invasive method employed for *H. pylori* detection[24]. Histopathological sampling does allow for the definitive diagnosis of infection, as well as degree of inflammation or metaplasia, presence and absence of MALT lymphoma and other gastric cancers in high risk patients. Perplexing array of test are available, which include urease breath test, rapid urease test, bacterial culture, serological test, PCR and molecular techniques, but most techniques are weighed down by lack of specificity and sensitivity or are limited in use due to their very expensive cost and inaccessibility . Because of *H.pylori* is difficult to grow in culture media, the role of culture in diagnosis is limited mostly to research and epidemiological considerations. PCR allows identification of organisms in small samples with few bacteria present with major limitation of a few laboratories currently has the capacity to run the assay and it has false positive and negative results. Rapid urease is inexpensive, fast and widely available test. But also limited in value due to the false positive results, decrease urease activity and affection by drugs. Serological test now a day's offer a fast, easy and relatively inexpensive methods of identifying infection with organism. However, this method is not useful means of confirmation and eradication of *H.pylori*[30]. Hematoxyllin and eosin, Giemsa, and silver staining have been used for detection of *H. pylori* in the tissue sections of paraffin embedded gastric mucosa specimens.8 Precision and accuracy of histological reporting for *H. pylori* detection depends on the rightly chosen biopsy sites with adequate sampling and skill of the pathologist. The advantages of histology are detection of *H.pylori* and its colonization density, and information about morphological changes in the gastric mucosa including gastritis, atrophy, intestinal metaplasia, dyspepsia or malignancies[24]. The only disadvantage of this technique is the need for endoscopy to obtain the tissue. However, The finding that gastritis was present in almost all infected patients is in complete agreement with other reports where gastritis was found present in more than 90% of *H. pylori* infected patients.

CONCLUSION

Prevalence of *H. pylori* infection in Pakistani population is shocking. Chronic gastritis is the major condition associated with dyspeptic patients but low prevalence of severe atrophy and intestinal metaplasia predict decreased risk of gastric carcinomas and lymphomas.

Acknowledgments: The authors are thankful to the laboratory staff of Histopathology section of ASMDC for participating in good histopathology processing. Moreover all the consultants are appreciated for their contribution and devotion in the reporting individually and in the committee.

Conflict of Interest: None declared

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