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RESEARCH ARTICLE

The Relation between *Helicobacter Pylori* Density and Gastritis Severity

Serhat Sayin*

Department of Internal Medicine, Training and Research Hospital, Aksaray University, Turkey

***Corresponding author:** *Serhat Sayin, Department of Internal Medicine, Training and Research Hospital, Aksaray University, Turkey*

Abstract

Background/Aims: *Helicobacter pylori* affects many individuals in developing countries. Inflammation caused by *helicobacter pylori* differs depending on the virulence factors, density and host tissue response of the bacteria. This study is intended to investigate the relation between density of *helicobacter pylori* colonization in gastric mucosa in biopsy specimens and gastric mucosal inflammation severity.

Materials and methods: Our study included 685 patients who applied to the hospital with dyspeptic complaints and had gastroscopy between the dates of January the 1st 2017 and January the 1st 2018. Histopathologic results of endoscopic biopsy specimens of these patients were retrospectively screened. Inflammatory severity and *helicobacter pylori* intensity were assessed by Sydney scoring.

Results: 68.6% of the patients were infected with *Helicobacter pylori*. As the intensity of inflammation increased, the intensity of *Helicobacter pylori* increased as well. The relation between the intensity of *Helicobacter pylori* and the severity of inflammation was statistically significant ($p < 0.001$).

Conclusion: *Helicobacter pylori* infection increases gastric inflammation.

Keywords

Helicobacter pylori, Gastritis, Endoscopic biopsy

Introduction

Helicobacter pylori (*H. pylori*/Hp) is a microaerophilic gram negative *bacilli* initially detected in the antral gastric mucosa by Warren and Marshall [1]. Today, around half of the world's population is infected with *Helicobacter pylori* [2]. *Helicobacter pylori* is most commonly found in the antrum. *Bacilli* is placed deep into the mucosa gelatin covering the gastric mucosa or between the mucus layer and the gastric epithelium. *H. Pylori* causes mucosal inflammation with proinflammatory factors that it released and it also spoils mucus layer with released enzymes. The inflammation caused by *helicobacter pylori* depends on the virulence factors, the density and the host tissue of the bacterium [3]. In various studies, *H. pylori* has been shown to be associated with gastritis, peptic ulcer, gastric carcinoma and Mucosal Associated Lymphoid Tissue (MALT) lymphoma [4-6].

Gastritis is an important pathological condition that causes gastric atrophy and cancer characterized by infiltration of inflammatory cells, especially lymphocytes and plasma cells, as well as neutrophils in the lamina propria. Gastritis does not have a specific finding that can be detected by endoscopic imaging [7,8]. However, histopathologic examination of the specimens taken from the tissue by endoscopy is accepted as the gold standard in the diagnosis of gastritis. The Sydney system is a good scoring system for histopathological evaluation and classification of gastritis. Sydney scoring is a classification based on topographic, morphological and etiological criteria of gastritis. It was accepted by consensus of gastroenterologist and pathologists at the World Congress of Gastroenterology in Sydney in 1990. The most important feature of this system is that the changes in the gastric mucosa are graded as five major histologic features (chronic inflammation, neutrophil activity, glandular atrophy, intestinal metaplasia and *H. pylori* density) [9]. This classification facilitates the clinical, endoscopic and morphological association of gastritis. It has been reported that there is a good correlation between polymorphonuclear cell infiltration

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and *H. pylori* infection histologically with the updated Sydney system [10].

This study is intended to investigate the relation between the intensity of *Helicobacter pylori* colonization and the severity of gastric mucosal inflammation in biopsy specimens taken from the gastric mucosa.

Materials and Methods

This cross-sectional analytical study was carried out in Aksaray at Training and Research hospital, Internal Diseases and Gastroenterology outpatient clinics between the dates of January the 1st 2017 and January the 1st, 2018 on 685 patients who were admitted with dissipative complaints and underwent upper gastrointestinal endoscopy. The study data were obtained by retrospectively examining the histopathological results of endoscopic biopsy materials of patients.

The histopathological diagnosis and classification of gastritis in our study was assessed by Sydney scoring. In our study, inflammation grade was evaluated as as mild (< 1/3), moderate (1/3-2/3), and severe (> 2/3) according to the infiltration of neutrophils in lamina propria or surface epithelium.

The existence of a *Helicobacter pylori* was investigated by May Grünwald Giemsa stain in mucosa samples obtained from antrum and its density was evaluated (Table 1).

In evaluation of the obtained data, SPSS (Statistical Package for Social Sciences) for Windows 20.0 program was used for the statistical analysis. Descriptive statistics for continuous variables were given in terms of mean and standard deviation, and descriptive statistics for categorical data were given in frequency and percentage. The Chi-Square test was used to compare the data in the categorical structure. Correlation between parameters was done by Pearson correlation analysis. Correlation coefficient (r) was evaluated as weak between 0.00-0.24, moderate between 0.25-0.49, strong between 0.50-0.74, very strong between 0.75-1.00. The results were evaluated in a confidence interval of 95% and in a significance level of $p < 0.05$.

Table 1: The evaluation of *Helicobacter pylori* density.

Gradation	Description
0	No *H. pylori*
1	Singly or in small groups in less than 1/3 of the mucosa surface
2	Between 1 and 3
3	Wide or in big groups in more than 2/3 of mucosa surface

Table 2: The frequency of *H. pylori* and other histopathological findings.

Parameters	n	%
H. pylori		
Positive	470	68.6
Negative	215	31.4
Pathological diagnosis		
Chronic active gastritis	383	55.9
Chronic gastritis	302	44.1
Inflammation severity		
Mild	235	34.3
Moderate	265	38.7
Severe	185	27.0
Metaplasia		
Yes	94	86.3
No	591	13.7

Results

In our study, 65.8% (n = 451) of 685 patients were female and 34.2% (n = 234) were male. Mean age of all patients was 47.51 ± 15.61 year (47.79 ± 15.79 in women and 46.99 ± 15.28 in men. 68.6% (n = 470) of the patients were infected with *H. Pylori*. Histopathologic results of the patients were presented on the table (Table 2).

No statistically significant relationship was found between *H. pylori* presence and metaplasia status in our study ($\chi^2 = 2.416$, $p = 0.120$). There was a significant relationship between the severity of inflammation and the intensity of *H. pylori* in our study. As the intensity of *H. pylori* increased, the intensity of gastric inflammation increased as well (Table 3). The frequency and intensity of *H. pylori* in chronic active gastritis was statistically significantly higher than that of chronic gastritis (Figure 1). As the severity of inflammation increased, *H. pylori* frequency also increased (Figure 2).

Discussion

The prevalence of *H. pylori* is quite high throughout the world in developed and also in developing countries like our country. *H. pylori* positivity was 68% in our study.

Table 3: The relation of *H.pylori* intensity with degree of inflammation ($x^2 = 3.940$, $p < 0.001$).

	Hp (-)		Hp (+)		Hp (++)		Hp (+++)	
	n	%	n	%	n	%	n	%
Mild inflammation	155	72.1	61	45.2	19	8.3	0	0.0
Moderate inflammation	43	20.0	71	52.6	127	55.7	24	22.4
Severe inflammation	17	7.9	3	2.2	82	36.0	83	77.6
Total	215	100.0	135	100.0	228	100.0	107	100.0

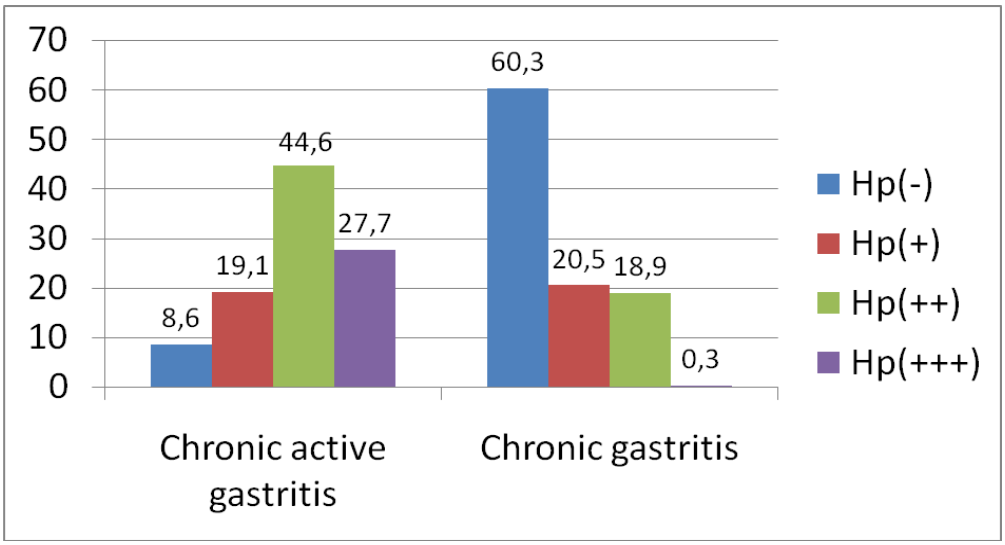

Figure 1: The degree of *H. pylori* intensity in the chronic gastritis and chronic active gastritis ($\chi^2 = 2.092$, $p < 0.001$).

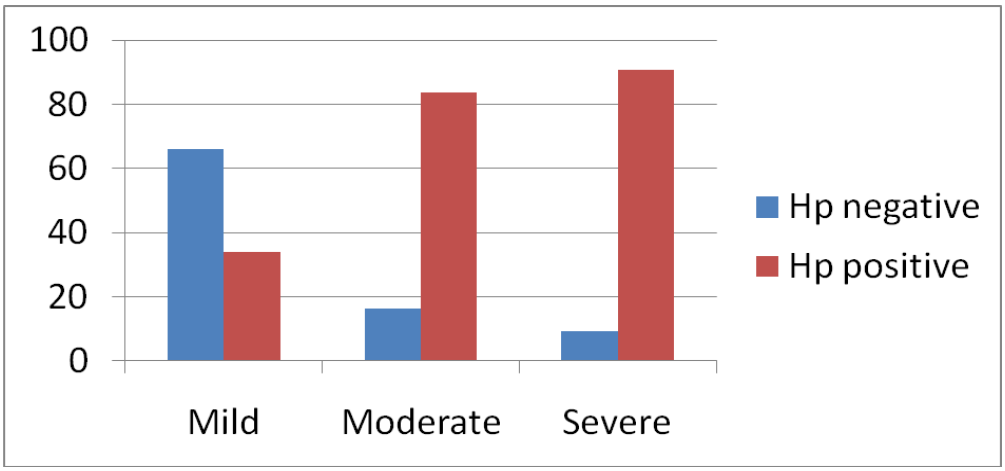

Figure 2: The relation of *H. pylori* frequency with degree of inflammation ($\chi^2 = 2.010$, $p < 0.001$).

In a study by Konakcı, et al. in our country, *H. pylori* positivity was reported to be 50% [11]. In a study performed in Romania by Olar, et al. *H. pylori* prevalence was found to be 63% [12]. In a study in Japan, Nomura, et al. found out *H. pylori* positivity as 62% [13].

H. pylori plays a major role in the pathogenesis of gastric diseases (gastritis, ulcer, cancer) due to the fact that it causes mucosal destruction. However, it is not known exactly how the mucosal damage develops with *H. pylori*. It is thought that proteases released by *H. Pylori* and gastric glycoprotein breaks down mucus structure and as a result, acidic gastric juice passes through this mucous membrane and causes mucosal damage [14]. Cell damage results from the direct toxic effect of ammonia, which is released into the gastric lumen by bacterial urease activity. It is also known that the various cytokines and chemotactic factors secreted by bacteria are also direct cytopathic and inflammatory initiating effects.

While there was no significant relationship between *H. pylori* and inflammation grade in various studies [15,16], there was a significant correlation in some other studies [17,18]. There was a statistically significant relation between the intensity of *H. pylori* and the severity of inflammation in our study. As the intensity of *H. pylori* increased, the severity of inflammation increased as well. In a study by Yakoob, et al. a significant relationship was found between *H. pylori* colonization intensity and chronic gastritis activity [19]. As in our study, Basir, et al. also found a significant relationship between *H. pylori* colonization intensity and chronic gastritis activity [20]. In a study performed by means of Histopathological examination of endoscopic biopsy specimens of 461 patients, Türkay, et al. reported that as the intensity of *H. pylori* increased, the intensity of inflammation increased, too [21]. In a study conducted by Alagöz, et al. a significant correlation was observed between *H. pylori* severity and lymphoplasmacytic cell infiltration and inflammation activation [22]. In contrast, in a study of 272 gastric biopsy specimens by Ardakani, et al. no significant relationship was found between the density of *H. pylori* and the severity of chronic gastritis activity [3]. Again, Choudhary, et al. found no significant

relation between *H. pylori* density and chronic gastritis activity [23].

Conclusion

As a result, studies have shown a relationship between the intensity of *H. pylori* and the severity of gastritis. Histopathological examination and classification of gastritis are important for accurate diagnosis of *H. pylori* infection affecting upper gastrointestinal system by various complications. Proper treatment of *H. pylori* infection can prevent gastritis and its complications.

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