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Original Article

Are colon neoplastic lesions associated with stomach helicobacter pylori infection?

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Abstract. A number of previous studies have shown that there is a correlation between Helicobacter Pylori (H. Pylori) and colorectal neoplasia. However, other studies could not prove whether there is a relation between the H. Pylori infection and the development of neoplastic polyploidy lesions in the colon**.** The aim of this study was to examine whether there is an in increase in the development of neoplastic lesions in the colon depending on the H. Pylori infection. In this case controlled retrospective study, patients who underwent both endoscopy and colonoscopy at the endoscopy unit of our hospital between January 2012 and January 2013 were evaluated. At colonoscopy, patients without any pathology or whose colon mucosa pathologies were reported as "nonspecific colon mucosa" were classified as the first group (n = 85), and those with one or more than one polyps as the second group (n = 75). The groups were compared in terms of the presence and intensity of H. Pylori, and severity of inflammatory activity and intestinal metaplasia. The results of the two groups were analogous with regard to H. Pylori positivity (p>0.05). No statistical difference was observed between the two groups with respect to H. Pylori intensity and severity of inflammatory activity in the H. Pylori positive patients (p>0.05). In conclusion, no relation between the presence of H. Pylori and the formation of polyp in the colon was found.

Keywords: Colon; cancer; Helicobacter Pylori infection; stomach; infection

Introduction

Helicobacter Pylori (H. Pylori) is a spiral shape, gram-negative, microaerophilic type of bacterium that colonizes in the gastric mucosa [1]. It was first discovered by Barry Marshall and Robin Warren in 1982 as the bacterium that causes gastritis and duodenal ulcer in the stomach. H. Pylori contaminate through oral route, and low socio-economic level and age are risk factors [2). It has been found to be more frequent in developing societies [3]. In the world-wide epidemiological research, it has been put forth that in approximately half of the human population H. Pylori is colonized in the stomach. It is known that the incidence of H. Pylori increases with age and the move from the west to the east [4]. In a research conducted in Turkey in 1992, the frequency of H. Pylori was found in 76.8% of asymptomatic individuals aged between 18 and 24 [5]. In another study in 2003 conducted on blood donors, this rate was found to be 85.9% in the group aged between 20 and 29, and 88.6% in the age group between 60 and 69 [6].

HP develops asymptomatically in 80% of humans. At a rate between 105 to 20%, it leads to gastritis, peptic ulcer and even gastric neoplastic lesions by causing chronic inflammation in the gastric mucosa [7].

In some previous studies, it has been observed that there is a correlation between H. Pylori and colorectal neoplasia [1]. However, some other studies could not prove whether there is a relation between the H. Pylori infection and the development of neoplastic polyploidy lesions in the colon [8-9].

The HP infection develops in the carrier individuals an immune-inflammatory response in the gastric cells against the effect created in the gastric mucosa. As a result of this, gastrine release in the stomach increases [10].

Hypergastrinemia has two significant effects. The first is the increase in the acid secretion, and the second is its induction of hyperproliferation in the mucosal cells in the gastrointestinal system [1]. It is still a matter of discussion whether this specific effect of hypergastrinemia may have a role in the development of polyps in the intestinal system.

According to those who support this hypothesis, a possible mechanism is that in the colon mucosa dependent on the hypergastrinemia that occurs in the HP infection, hyperproliferation can lead to the development of neoplasic lesions.

There is no study in Turkey on the exploration of the

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TABLE 1
DEMOGRAPHIC DATA OF THE PATIENTS

	Group 1	Group 2	p
Age	60.1	60.9	>0.05
Gender (F/M)	48/37	43/32	>0.05
H. Pylori presence	44(51.7%)	42(56%)	>0.05
H. Pylori intensity			>0.05
1(+)	23	20	
2(+)	16	15	
3(+)	5	7	
Inflammatory activity intensity			>0.05
0	7	6	
1(+)	47	36	
2(+)	26	22	
3(+)	5	11	
Intestinal metaplasia	16 (18.8%)	13(17.3%)	>0.05
Total	85	75	

TABLE 2
CHARACTERISTICS OF GASTRIC BIOPSY ACCORDING TO THE POLYP TYPE IN GROUP 2 PATIENTS

	Tubular adenoma	Tubulovillous adenoma	Hyperplastic polyp	p
Number of patients	45	14	16	<0.05
H.Pilori presence (%)	24 (53.3%)	7 (50%)	8 (50%)	>0.05
H.Pylori intensity				>0.05
1(+)	14	3	3	
2(+)	7	3	3	
3(+)	3	1	2	
Inflammatory activity intensity				>0.05
0	1	1	0	
1(+)	34	11	11	
2(+)	7	0	4	
3(+)	3	2	1	
Intestinal metaplasia	9(20%)	3(21.4%)	3(18.7%)	>0.05

correlation between the HP infection and colon neoplasias. The aim of this study was to examine whether there is an increase in the development of neoplastic lesions in the colon depending on the H. Pylori infection.

Materials and Methods:

In this study, patients who underwent both endoscopy and colonoscopy at the endoscopy unit of our hospital between January 2012 and January 2013 were evaluated. Demographic data, endoscopy and colonoscopy reports of the patients and the histopathological evaluation of their tissue samples were collected. At colonoscopy, patients without any pathology or whose colon mucosa pathologies were reported as "nonspecific colon mucosa" were classified as the first group and those with one or more than one polyps as the second group.

Histopathological results and demographic characteristics of both groups were compared using statistical methods. Following colonoscopy and endoscopy, the gastric and colon mucosa samples of the patients taken with endoscopic biopsy forceps, or the polypectomy samples prepared as smear were fixated with formaldehyde, and the paraffin-embedded colon polypectomy and stomach endoscopic biopsy tissues were stained with routine hemotoxilyn-eosin following deparaffinization and rehydration. Gastric samples of patients were also histochemically stained with Giemsa with the aim of detecting H. Pylori and PAS/ Alsian blue for detecting intestinal metaplasia. For the evaluation of histopathological colon sample, the WHO criteria were

used [12]. In the evaluation of the gastric samples, the modified Sydney classification was used [13].

In both groups, patients who previously were diagnosed as cancer or had a history of colon resection or received a treatment for H. pylori eradication at least a month ago or those who were diagnosed with familial adenomatous polyposis coli and patients who were younger than 18 and were diagnosed with an inflammatory bowel disease were excluded from the study.

Statistical analyses

The statistical software version 11.5.1.0 (MedCalc, Mariakerke, Belgium) was used. In normally distributed groups, the results were presented with mean and SD. The significance of the differences between groups was determined by Student's unpaired *t*-test for normal distributions, and by the Mann–Whitney U-test in abnormal distributions. Categorical variables were evaluated using Chi-square and Fisher's exact test. A p values less than 0.05 were accepted as significant.

Results

In this study, 85 patients with normal colonoscopic findings in conformity with the criteria stated were classified as the first group and 75 patients with one or more than one polyp as the second group. The mean age in the first and second group was 60.1 and 60.9 respectively. There were 37 male and 48 female patients in the first group and 32 male and 43 female patients in the second group. There was no statistically significant difference between the two groups with respect to age and gender distribution ($p>0.05$). Gastric biopsy samples of 44 (51.7%) patients in the first group were positive for H. Pylori. In the second group, 42 patients (56%) were positive for H. Pylori. The two groups were analogous with regard to H. Pylori positivity ($p>0.05$).

No statistical difference was observed between the two groups with respect to the intensity of H. Pylori and inflammatory activity in the H. Pylori positive patients ($p>0.05$). This was also true for intestinal metaplasia in 16 of 85 patients (18.8%) in group 1 and 13 of 75 patients (17. 3%) in group 2 ($p>0.05$).

In group 2, 45 patients were identified with tubular adenoma, 14 with tubulovillous adenoma, and 16 with hyperplasic adenoma. When these patients were compared according to the types of polyps, no statistical difference was observed with regard to the presence of H. Pylori, H. Pylori intensity, severity of inflammatory activity and the presence of intestinal metaplasia (Table 2). Also no statistical difference was observed between the localization of the polyps and presence of H. Pylori, intensity of H. Pylori, severity of inflammatory activity and the presence of intestinal metaplasia.

Intestinal metaplasia was seen in 29 patients of the 160 patients (18.1%) in both groups. When the patients with intestinal metaplasia were compared to those without intestinal metaplasia, their rates regarding the intensity of H. Pylori, severity of inflammatory activity and presence of H. Pylori were similar to each other, and no significant difference was observed.

There was only one polyp identified in 47 patients in group 2 and 28 patients had more than one polyp. The presence and intensity of H. Pylori were similar between the patients with one polyp and those with more than one polyp with no statistical significant difference.

Discussion:

A number of causes have been envisaged in the etiology of gastric cancer. In epidemiological studies, it has been put forth that many factors like nutrition high in nitrate, salty diet, and smoked food play a role in the development of gastric cancer. On the other hand, it is known that H. Pylori infection has a role in oncogenesis, leading to mucosal atrophy with chronic gastritis [1]).

H.Pylori colonization was investigated on the gastric mucosa samples taken from the patients by using hemotoxylin-eosin and Giemsa stain. Although in some studies which aimed at identifying H. Pylori infection, lgG titre developed against H. Pylori was identified, however in the literature it has been reported that the sensitivity of Giemsa is much higher for the detection of the spiral shaped microorganisms in the gastric mucosa [1, 8].

In our study, the data of the patients who underwent endoscopy and colonoscopy at our clinic were used. Endoscopic biopsies of the stomach were examined with regard to H. Pylori colonization and the intensity of inflammation using the histochemical method. With regard to age, the colorectal polyp group had a higher mean age than the normal colonoscopy group (Table 1). This might be related to the increase in the possibility of polyp formation in the colon with the increase in age. While the number of the male patients was higher in the group with colon polyp, female patients dominated in the group with normal colonoscopy findings. In our study, the rate of colon polyp was higher in male patients than in the females.

In many studies, H. Pylori positivity has been tried to be identified by looking at the serum immunoglobin levels against H. Pylori. However, it is clear that using the histochemical method on the gastric mucosa provides more accurate data for detection of H. Pylori [14, 15]. In our study, while in patients with colon polyp H. Pylori colonization in the gastric mucosa was found in 56 %, in patients with normal colon findings it was in 51.7 %. Thus, no statistical difference was observed between the two groups. Besides, no difference was seen in the rates between the intensities of H. Pylori and inflammation between the two groups.

So far, in many studies it has been explored whether there is a relation between H. Pylori and colon neoplasia or colon cancer. In some studies, no relation has been observed [8-16]. In other studies, a significant relationship has been found [14-15, 17]. In a study on 669 patients conducted by Fujimori et al. [17], it was revealed that the frequency of colon polyp was high in patients with H. Pylori infection. In our study, also the patients were identified as H. Pylori positive with the histochemical analysis of gastric mucosa as well as urease test and urea-breath test. In another study, Lung et al. [16] stated that in patients diagnosed with H. Pylori by the urease test, the possibility of colon polyp increases. In our study, H. Pylori

positivity was not found at statistically significant in patients with colon polyp compared to those without colon polyp (p = 0.63).

It is known that chronic atrophic gastritis and intestinal metaplasia are associated with gastric carcinoma, especially the intestinal type. In our study, no association with colon polyp could be found in the patients with intestinal metaplasia.

When we looked at the polyp localization of the colon in patients, we observed that the polyp was located in the left colon in 30 patients, in the rectum in 14 patients, in the transverse colon in 11, and in the right colon in 26 patients. Considering the polyp localization, no difference was observed in the patients with regard to H. Pylori positivity and H. Pylori intensity.

In our study, the relationship between colon polyp development and the presence of H. Pylori in the stomach was assessed. As a result, no relation between the presence of H. Pylori and the formation of polyp in the colon was found. The absence of any information about the dietary habits of the patients in our study has been a restrictive aspect. Yet, our study is an example to a case-controlled retrospective study on the effect of H. Pylori infection on the development of polyps in the colon. Although there are more comprehensive studies on this topic in the literature, it is still a disputable issue.

Conflict of Interest

The authors declare no conflicts of interest.

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