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The relation between *Helicobacter pylori* and ulcerative colitis

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Background/aim: Besides some genetic explanations of the native course of ulcerative colitis (UC), the most attributable factors are pathogenic bacterial agents. There are some conflicting data about the relationship between *Helicobacter pylori* and the rate of UC in the literature. Therefore, we aimed to investigate the rate of *H. pylori* in UC patients.

Materials and methods: Forty-nine individuals diagnosed with UC who had undergone upper gastrointestinal tract endoscopy for different reasons were included in the study. The presence of *H. pylori* in the stomach was checked by histopathological examination.

Results: *H. pylori* positivity was present in 57.1% of patients with UC. Interestingly, *H. pylori* positivity was lower (11.1%) in pancolitis patients compared to those presenting with more limited illnesses. There were no relationships among the severity of the underlying disease, medication already used, and *H. pylori* positivity rate.

Conclusion: The extension of UC is important for the positivity rate of *H. pylori*. It could not be determined whether the low positivity of *H. pylori* in extended UC cases was due to immunosuppressive drugs or to the UC itself.

Key words: Ulcerative colitis, *Helicobacter pylori*, immunosuppressive drugs

1. Introduction

Ulcerative colitis (UC), characterized by permanent mucosal inflammatory processes, presents with remission and activating periods (1). Although some environmental and genetic causes are attributed to UC progression, the exact cause of the disease is still unclear (1,2). In the recent years, investigators have focused on some pathogens as causes of UC (3,4).

Helicobacter pylori, a pathogen related to chronic gastritis and peptic ulcers, is colonized mainly in the antrum, protecting itself from hyperacidity (5,6). Almost half of the underdeveloped populations are infected by *H. pylori*. Apart from the gastrointestinal system disorders, *H. pylori* is also found to be related to several skin diseases, autoimmune disorders, and iron deficiency (7,8). The association between *H. pylori* and UC is controversial. Some researchers reported that incidence of *H. pylori* is lower in UC patients than in healthy populations (9–12). Possible causes of this low rate of *H. pylori* in UC patients are the immunopathological characteristics of UC and the medications used in UC, such as 5-aminosalicylic acid (5-ASA) and antibiotics (9–12).

We investigated the incidence of *H. pylori* in individuals with UC and determined the impact of several characteristics of UC, including extent and severity of UC, on the incidence of *H. pylori*.

2. Materials and methods

Patients who were diagnosed with colitis and admitted to the outpatient gastroenterology clinic of Numune Training and Research Hospital were included in the study. Specifically, 49 patients with UC who had undergone upper gastrointestinal endoscopy with various indications were included in the study. The presence of *H. pylori* was assessed by taking one biopsy from the antrum in each patient. Patients using antacids or antibiotics in the previous 2 months or who had previously undergone *H. pylori* eradication treatment were excluded from the study. Prior to the study, all UC patients were required to provide an informed consent form and the study design was submitted to local ethics committee for approval.

The density of the colonization of *H. pylori* was assessed by using Sydney classification (13). The demographic data of the patients in each group were obtained from the

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hospital records. The UC patients were also divided into 4 groups according to the extension of the underlying disease (distal type, left-sided type, extensive type, and pancolitis). The clinical activity of UC was scored by the Truelove and Witts criteria (14) and was divided into 3 groups (mild, intermediate, and severe). The endoscopic activity was assessed using the Rachmilewitz scoring system (15).

Statistical analyses were performed with SPSS 15.0 for Windows. The normal distribution of continuous variables was evaluated by histogram and one-sample Kolmogorov–Smirnov test. $P > 0.05$ was considered to be normal distribution. Normally distributed continuous variables were expressed as mean ± standard deviation, continuous variables that were not normally distributed as median (minimum maximum), and nominal variables as number and percentage. The differences between normally distributed independent variables were calculated by independent samples t-test; the Mann–Whitney U test was used when the distribution did not appear to be normal and the Kruskal–Wallis test was used in the comparison of more than 3 groups. The relationship among nominal values was determined by the Pearson chi-square test and Fisher exact test. All calculations were 2-tailed and $P < 0.05$ was considered to be significant.

3. Results

Forty-nine patients were included in the study. The mean age of the individuals was 40.5 ± 10.3 years and 62.5% of them were male.

Overall positivity of *H. pylori* in gastric mucosa was 57.1% in the UC patients. Although baseline features of the patients such as mean age, sex distribution, history of alcohol use and smoking, and severity of underlying UC were similar in each group, the patients presenting with pancolitis had the lowest rate of *H. pylori* (11.1%) among UC patients ($P = 0.005$) (Table 1). On the other hand, the density of *H. pylori* colonization according to the pathological examination was found to be similar in each group (data not shown).

The UC patients were categorized according to the severity of disease. All groups were similar when compared according to baseline characteristics (mean age, sex distribution, history of alcohol use and smoking, and positivity of *H. pylori* and its density of colonization) (Table 2).

Most of the patients were only under the 5-ASA treatment (77.6%), and the rest were under 5-ASA treatment and immunomodulatory medication. Although the positivity of *H. pylori* in the combination group was

Table 1. The comparison of data based on the extension of UC.

	Distal (n = 27)	Left-sided (n = 9)	Extensive (n = 4)	Pancolitis (n = 9)	P-value*
Age, mean ± SD	41.07 ± 10.53	36.88 ± 11.54	47.25 ± 11.44	39.66 ± 7.46	0.178
Male/female, n	18/9	6/3	4/0	3/6	0.113
Alcohol use, n (%)	4 (14.8%)	1 (11.1%)	0 (0%)	1 (11.1%)	0.862
Smoking, n (%)	8 (29.6%)	2 (22.2%)	0 (0%)	2 (22.2%)	0.630
Mild severity of UC, n (%)	21 (77.8%)	5 (55.6%)	2 (50.0%)	5 (55.6%)	0.217
Positivity of *H. pylori*, n (%)	18 (66.7%)	7 (77.8%)	2 (50.0%)	1 (11.1%)	0.015**

*The cutoff value of statistical significance was considered to be <0.05 by using the Kruskal–Wallis test.
**The Mann–Whitney U test, as a post-hoc test, revealed a difference between the pancolitis group and the others, whereas no difference was found among the others.

Table 2. The comparison of variables according to the severity of UC.

	Mild (n = 33)	Intermediate (n = 13)	Severe (n = 3)	P-value*
Age, mean ± SD	42.39 ± 8.73	35.07 ± 12.73	44.00 ± 9.53	0.170
Male/female, n	21/12	8/5	2/1	0.983
Alcohol use, n (%)	4 (12.1%)	1 (7.7%)	1 (33.3%)	0.474
Smoking, n (%)	9 (27.3%)	3 (23.1%)	0 (0%)	0.570
Positivity of *H. pylori*, n (%)	19 (57.6%)	7 (53.8%)	2 (66.7%)	0.918

*$P < 0.05$ was considered to be a cutoff value for statistical significance.

lower than in 5-ASA group (63.2% vs. 36.4%, respectively), the difference did not reach statistical significance (P = 0.114) (Table 3). The density of *H. pylori* colonization was similar in both groups.

Approximately 10% of the UC patients had a history of surgery related to UC; the *H. pylori* positivity was 20.0% in the patients that had a history of surgery, whereas it was 61.4% in patients without surgery (P = 0.150).

4. Discussion

Recent studies have demonstrated that the incidence of *H. pylori* is lower in individuals with UC than in healthy populations (9–11,16). Some studies have indicated that the rate of *H. pylori* is lower in the patients prescribed 5-ASA or sulfasalazine (9,10,12,17). With sulfasalazine therapy for 14 days, there was a mild suppression in the urea breath test, but eradication of *H. pylori* was not observed (6). The inhibiting effect of sulfasalazine on *H. pylori* replication was also shown in vitro, but no bactericidal or bacteriostatic effect was found (9). Sulfasalazine probably blocked the adhesion of *H. pylori* to the gastric mucosa directly over receptors or indirectly by its antiinflammatory effect (18,19). Some investigators reported that the rate of *H. pylori* was lower in UC patients than in healthy controls, but this was not related to any drugs used for UC (20). Another hypothesis suggested that, with aging, both *H. pylori* rate and the use of sulfasalazine increase (20).

Although we did not have any UC patients currently under corticosteroid therapy, the literature revealed no effect of corticosteroids on the prevalence of *H. pylori* (17). However, our results showed that the positivity of *H. pylori* was lower in UC patients presenting with pancolitis than in patients with more limited diseases. On the other hand, despite not reaching statistical significance, the presence of *H. pylori* was found to be lower in patients under 5-ASA and immunosuppressive treatment than in those with 5-ASA therapy alone. This may be attributed to the higher rate of immunosuppressive use in extended diseases, which has a powerful antiinflammatory effect blocking the ability of *H. pylori* to adhere to the gastric mucosa in UC patients presenting with pancolitis. The other possible explanation of these results is that UC has been thought to have an independent protective effect against *H. pylori* colonization (18,20).

The main restriction of our study is that we did not include a healthy group to compare with UC patients. Thus, we did not investigate whether the *H. pylori* rate in UC patients is different from that in healthy controls; we only focused on the assessment of *H. pylori* rate in UC patients in terms of underlying medications and extent of the disease.

In conclusion, our results revealed that the rate of *H. pylori* declined with the elongation of the extent of UC cases; however, it did not differ in terms of severity of UC and the prescribed medications.

Table 3. The comparison of results according to the medications used for UC.

	5-ASA (n = 49)	5-ASA + Immunosuppressive (n = 40)	P-value*
Age, mean ± SD	40.89 ± 10.68	39.36 ± 9.27	0.640
Male/female, n	25 / 13	6 / 5	0.464
Alcohol use, n (%)	5 (13.2%)	1 (9.1%)	0.717
Smoking, n (%)	8 (21.1%)	4 (36.4%)	0.298
Activity of UC			
Mild, n (%)	26 (68.4%)	7 (63.6%)	
Intermediate, n (%)	10 (26.3%)	3 (27.3%)	0.889
Severe, n (%)	2 (5.3%)	1 (9.1%)	
Positivity of *H. pylori*, n (%)	24 (63.2%)	4 (36.4%)	0.114

*: P < 0.05 was the cutoff value for statistical significance.

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