Tropical Biomedicine 35(4): 1148–1153 (2018)

The prevalence of *Helicobacter pylori* in patients with oesophageal stenosis

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Received 5 January 2018; received in revised form 12 February 2018; accepted 13 March 2018

Abstract. Oesophageal strictures often occur following esophagectomy which is performed for cases of oesophageal cancer. These patients require repeated dilation procedures. The aim of this study is to investigate the effects of dietary regimens, which are changed because of stenosis, caused by *Helicobacter pylori (H. pylori)*. In this retrospective study, 28 patients who had operation for oesophageal cancer and underwent dilation due to development of stricture were studied. 30 female and 30 male patients who were admitted to the Gastroenterology Clinic with complaints of dyspepsia and did not receive treatment for the eradication of *H. pylori* were randomly selected and included in this study as a control group. Patients' histopathological records on the hemogram, biochemistry, and endoscopic biopsy were analysed. There were 26 *H. pylori* (+) cases (92.8 %) in the stenosis group consisting of 28 patients, and 37 *H. pylori* (+) cases (61.6%) in the control group consisting of 60 patients. These results were found to be statistically significant (P=0.003). The albumin level in the stenosis group was reported to be statistically low (P=0.002). The incidence of *H. pylori* was found to be significantly higher in patients with stenosis. We ascribed this outcome due to changes in dietary regimens. Our findings showed that the dietary regimens of all patients who underwent esophagectomy should be regulated during the postoperative period considering *H. pylori*. The relationship between *H. pylori* and stenosis was significant, there is a need for further research with a larger sample size to enrich the findings.

INTRODUCTION

Helicobacter pylori (H. pylori) is a spiral-shaped, gram-negative bacteria that colonizes the gastric mucosa (Marshall, 1984). The infection rate of *H. pylori* in the global population was reported to be more than 50.0% (The EUROGAST 1994). Infection with *H. pylori* occurs during the early stages of life and can last forever if untreated (Kuipers *et al*., 2000). Although the majority of individuals remain asymptomatic, some infected individuals, develop gastritis. On the other hand, untreated individuals develop atrophic gastritis and gastric adenocarcinoma following metaplasia. *H. pylori* infection-induced gastritis begins with an acute phase, accompanied by lamina propria and gastric epithelial infiltration of polymorphonuclear leukocytes (PMNs) (Chen *et al*., 2002). Following the acute phase, the *H. pylori* infection-induced gastritis progresses to chronic gastritis, which is characterized by accumulation of lymphocytes or plasma cells, the formation of lymphoid follicles, and the hyperplasia of cells containing gastric glands (White *et al*., 2015).

Oesophageal stenosis is a common complication observed especially among those who underwent esophagectomy with resulting complications in a patient's life. Oesophageal stenosis may develop due to respiratory diseases, cardiac problems, the

surgical techniques used and oesophageal strictures because of anastomotic leakage following surgery (Zhong *et al.*, 2014). The rate of strictures was found to be 42.0% in a study conducted in 607 patients in the Netherlands (Van *et al.*, 2010).

Change in dietary habits in patients who develop oesophageal stenosis, includes a change from solid food to fluid and pureed food. In this study, we aimed to discuss the effects of this changing nutrition on *H. Pylori* infected patients.

MATERIALS AND METHODS

28 patients who had an operation for oesophageal cancer and underwent bougie dilation following the development of stricture at the General Surgery Clinic of Van Yüzüncü Yıl University between 2011 and 2017 were included in the study. 30 female and 30 male patients who were admitted to the Gastroenterology Clinic with gastric complaints and who did not receive treatment for the eradication of *Helicobacter pylori* were studied. The data were retrospectively evaluated. The hemogram, white blood cells, haematocrit, platelet, neutrophil levels, and the upper and lower levels of albumin, creatinine, and glucose values of the patients were obtained. The presence of *H. pylori* was also determined through analysis of pathology results. No blood was obtained from any of the patients. Patients with incomplete results were excluded from the study.

RESULTS

Of the 28 patients with stenosis, 19 were females, and nine were males (age range 23-86, median: 56.3), whereas in the non-stenosis control group there were 30 females and 30 males, totalling 60 patients in all, (age range: 20-78, median: 52.5). In the stenosis group, *H. pylori* was absent in two of the 28 patients (one male and one female), whereas *H. pylori* was detected in 26 patients. No *H. pylori* was detected in 23 (12 males and 11 females) of the 60 patients in the control group but was detected in 37 of the patients (18 females and 19 males). These results were found to be statistically significant (P=0.003) (Table 1). Comparison of the stenosis group with the control group demonstrated that statistical significance was found only the albumin level with regards to biochemistry parameters (P=0.002).

Table 1. *H. pylori* ratio in stenosis and control groups

H. pylori. *stenoz Crosstabulation					
			Stenoz		Total
			Negative	Positive	
H. pylori	Negative		23	2	25
		% within *H. pylori*	92.0%	8.0%	100.0%
		% within stenoz	38.3%	7.1%	28.4%
		% of Total	26.1%	2.3%	28.4%
	Positive	Count	37	26	63
		% within *H. pylori*	58.7%	41.3%	100.0%
		% within stenoz	61.7%	92.9%	71.6%
		% of Total	42.0%	29.5%	71.6%
Total		Count	60	28	88
		% within *H. pylor*i	68.2%	31.8%	100.0%
		% within stenoz	100.0%	100.0%	100.0%
		% of Total	68.2%	31.8%	100.0%
			Ki-kare=1.672 p=0.003 (p<0.01)		

H. pylori positivity rate in stenosis group was 92.8% and *H. pylori* positivity rate in control group was 61.6%.

Statistical Analysis

Descriptive statistics for the continuous variables were presented as Mean, Standard deviation, maximum and minimum values while count and percentages for categorical variables. One-way ANOVA was used to compare group means. Duncan multiple comparison test was also used to determine different group means followed by ANOVA. For determination linear relationships among the variables, Pearson correlation analysis was performed. In addition, chi-square test was performed to determine the relationship between categorical variables. Statistical significance level was considered as 5% and SPSS (ver: 13) statistical program was used for all statistical computations.

DISCUSSION

Epidemiological studies show that *H. pylori* is one of the most prevalent bacterial infections worldwide (Malfertheiner *et al.*, 2017). *H. pylori* causes approximately 50.0% of the global population to have sinus infection. This rate could rise up to approximately 80-90% in developing countries (Salih, 2009). The incidence of *H. pylori* was found to be 56.5% in a study from east of Turkey (Suvak *et al.*, 2015). Infection with *H. pylori* occurs during the early stages of life and can last forever if untreated (Kuipers *et al.*, 2000). *H. pylori* infection is transmitted from one individual to another, and a low hygienic level plays a vital role in its spread (Manfredi *et al.*, 2016).

H. pylori is the etiologic agent of peptic ulcer; 75% of gastric ulcers and 90% of duodenal ulcers are associated to *H. pylori* infection (Ernst *et al.*, 2000), as well as two different types of gastric cancers: adenocarcinoma and mucosa-associated lymphoid tissue (MALT) lymphoma (Venerito *et al.*, 2017). As a result of this correlation, *H. pylori* is the only class of bacteria classified as class I carcinogen by the World Health Organization (Ikezaki *et al.*, 2017). Gastric cancer is still the second most common cause of cancer mortality worldwide, and this high rate may reflect the incidence of *H. pylori* infection (Crew *et al.*, 2006).

The relation between the *H. pylori* and gastric diseases is a known fact. In consequence, many studies have been conducted about bacterial, host and environmental factors which affect the severity of the disease. These studies have mainly aimed to shed light to virulence factors of the bacteria and reveal the relation between dietary habits and *Helicobacter Pylori* infection. Studies investigating the relationship of diet with *H. pylori* showed that *H. pylori* is less prevalent in populations fed mainly on fruits-vegetables and vitamin C (Aditi *et al.*, 2012). In another study, some types of raw fast food types, such as fruit salads, indicated that they might be sources of *H. pylori* resistant and virulent strains (Hemmatinezhad *et al.*, 20016). *H. pylori* was found to be higher in populations fed on carbohydrate-rich foods, processed meat, refined grains and food containing saturated fat (Mard *et al.*, 20014). The high salt concentration in the stomach could destroy the mucosal barrier of the stomach, thereby facilitating the colonization of *H. pylori* and causing inflammation and gastritis. Processed meat contributes to the pathogenicity of *H. pylori* due to their high salt content (Caruso *et al.*, 1990). Zhang *et al.* (2010) reported that acute and chronic alcoholic consumption, as well as salt causes disruption of the gastric mucosal barrier, and results in increased mucosal permeability, chemical inflammation, and density of *H. pylori* colonization. Hosoda *et al.* (2015) suggested that vitamin D3 had antibacterial effects against *H. pylori*, but was ineffective against other bacteria. Treatment with vitamin D3 was shown to result in the collapse of *H. pylori* cell membrane and the eventual destruction of bacteria cells (Hosoda *et al.*, 2015). Chili pepper, Capsaicin, Garlic/Allium, Curcumin, Cumin, Turmeric, Nutmeg, and Cardamom spices, which are frequently used in the kitchen, were shown to reduce

H. pylori colonization through their anti-inflammatory effects and to demonstrate anti-helicobacter properties (Mahady *et al.*, 2012; Holzer *et al.*, 1989; Cellini *et al.*, 1996; De *et al.*, 2009). All these studies suggest that there is a close relationship between nutritional patterns and *H. pylori* infection.

Postoperative stenosis was shown to develop in more than 40.0% of patients who underwent esophagectomy (Van *et al.*, 2010). Our clinical experience showed that stenosis could cause malnutrition by limiting a patient's oral intake. Patients had difficulty consuming solid food due to stenosis, the reason being they tended to deviate to liquid food.

In our study, when the albumin levels of individuals with stenosis and healthy individuals were compared; the level was found to be significantly lower in the stenosis group (P=0.002). Our results showed that stenosis-related chronic malnutrition has developed in the stenosis group.

Our study showed that *H. pylori* was significantly higher (P=0.003) in the *H. Pylori* group compared to the control group. We suggest that the increased level of *H. pylori* in the stenosis group was due to nutritional regimens differed from normal population Due to restricted oral intake that resulted in weakened immune system. In the stenosis group, since solid foods cannot be swallowed, the intake of vegetables, fruits and vitamins is decreased and more liquid foods rich in calories was consumed. Several studies showed that *H. pylori* is found in high dietary regimens with poor intake of antioxidant vitamins and vegetable oils (Kim *et al.*, 2005; Sezikli *et al.*, 2015). This may account for the high rate of *H. pylori* in the stenosis group.

In conclusion, we suggest that the increased incidence of *H. pylori* in patients with stenosis is associated with change in dietary regimen. We recommend that all patients who undergo esophagectomy should regulate their dietary regimens during the postoperative period through considering *H. pylori*. We suggest to provide antioxidant vitamins, fish oils, and folate to lower incidence of *H. pylori.*

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