Co-Occurrence of Helicobacter Pylori and Intestinal Metaplasia in Patients with Dyspepsia

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Abstract

Aim: Helicobacter pylori (HP) is a common gram negative bacteria associated with peptic ulcer disease and malignancy. In this study we evaluated the co-occurrence of helicobacter pylori and intestinal metaplasia (IM) in patients with dyspepsia. Material and Method: We retrospectively evaluated the gastroscopy and antral biopsy results of patients that were admitted to Siverek Government Hospital general surgery outpatient clinic with dyspepsia between November 2013 and January 2015. Pathology samples were investigated with giemsa for HP and with PAS-AB for intestinal metaplasia. Results: We evaluated 682 patients (304 men, 378 women) with a mean age of 38.22 ±14.64 years (range 18-88 years). We diagnosed 555 (81.4%) with antral gastritis, 81 (11.9%) with pangastritis, and 6 (0.9%) with ulcer-vegetant tumor lesion. Based on pathology, we diagnosed 469 (69.6%) with chronic gastritis, 201 (29.5%) with inactive chronic gastritis, and 6 (0.9%) with adenocarcinoma. HP was detected in 475 (69.6%) patients, intestinal metaplasia was detected in 56 (8.2%) patients, and atrophy was seen in 11 (1.6%) patients. Helicobacter pylori was detected in 4 (66.6%) of the 6 patients with adenocarcinoma. HP was detected in 475 (69.6%) patients, intestinal metaplasia was detected in 56 (8.2%) patients, and atrophy was seen in 11 (1.6%) patients. HP was detected in 4 (66.6%) of the 6 patients with adenocarcinoma. In 48 patients with HP, intestinal metaplasia was also found. In 8 patients with intestinal metaplasia, HP was negative. Co-occurrence of HP and intestinal metaplasia was statistically significant (p: 0.006). Discussion: Helicobacter pylori infection promotes intestinal metaplasia. Prevention and treatment of HP prevents precancerous lesions. In particular, patients with dyspepsia resistant to medical treatment should be examined carefully in general surgery practice.

Keywords

Helicobacter Pylori; Intestinal Metaplasia; Dyspepsia; Adenocarcinoma

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Introduction

Helicobacter pylori (HP) infection is associated with atrophic gastritis, intestinal metaplasia (IM), and intestinal type gastric cancer. Atrophic gastritis following long chronic inflammation due to HP progresses to IM, displasia, and gastric adenocarcinoma [1]. Early diagnosis and treatment of patients with HP is crucial for prevention of precancerous lesions. Randomised controlled trials reported that eradication of HP prevents precancerous lesions and gastric adenocarcinoma [2]. The few meta-analyses conducted have shown only that HP eradication is not sufficient to prevent gastric adenocarcinoma in patients with intestinal metaplasia and displasia [3]. Early diagnosis and eradication of HP before the development of precancerous lesions has an important role in gastric cancer prevention. Since WHO and IARC (International Agency for Research on Cancer) have defined HP as a group 1 carcinogen, HP eradication has become more important [4,5]. Half of the population of the world is infected with HP and gastric adenocarcinoma is seen in 75% of these patients [6,7]. Peleteiro et al. showed in their prevalence study that incidence of HP infection has increased in every decade in which it has been tracked [8]. In recent years the prevalence of the disease has increased significantly in developing countries like Turkey. In the 2003 TURHEP study, HP infection prevalence was found to be 82.5% in people older than 18 years [9]. Since the prevalence is higher, early diagnosis and eradication are become more important for our country.

As a consequence of chronic HP infection, diffuse antral gastritis (DAG) or multifocal atrophic gastritis (MAG) evolves. As pathogenesis of the infection is not clear yet, the progression of lesions cannot be predicted. In patients who develop DAG, there is more acid secretion and duodenal ulcers are more common. Hypoacidity is seen in patients who develop MAG, and IM is more common [10]. In our study, we aim to detect HP infection in patients with dyspepsia and to evaluate the IM rate due to HP.

Material and Method

We evaluated the patients admitted with dyspepsia to Şanlıurfa Siverek Government Hospital general surgery outpatient clinic between November 2013 and January 2015. We included the patients who had gastroscopy. We retrospectively investigated gastroscopy findings and antral pathology reports. Pathology samples were investigated with giemsa for HP and with PAS-AB for intestinal metaplasia. Statistical analysis was done using the SPSS 21.0 package.

Results

We evaluated 682 patients (304 men, 378 women) with a mean age of 38.22 ± 14.64 years (range 18-88 years). Inflammation was detected in 677 (99.3%) patients. There were pathologic findings in 455 of these patients (66.7%). We diagnosed 555 (81.4%) with antral gastritis, 81 (11.9%) with pangastritis, and 6 (0.9%) with ulcero-vegetant tumoral lesion. Based on pathology we diagnosed 469 (69.6%) with chronic gastritis, 201 (29.5%) with inactive chronic gastritis, and 6 (0.9%) with adenocarcinoma. HP was detected in 475 (69.6%) patients, intestinal metaplasia was detected in 56 (8.2%) patients, and atrophy was seen in 11 (1.6%) patients. HP was detected in 4 (66.6%) of the 6 patients with adenocarcinoma. In 48 patients with HP, intestinal metaplasia was also found. In 8 patients with intestinal metaplasia, HP was negative (Table 1). Co-occurrence of helicobacter pylori and intestinal metaplasia was statistically significant (p: 0.006) (Table 2).

Discussion

Chronic HP infection plays a particular role in intestinal type gastric carcinogenesis by promoting atrophic gastritis, intestinal metaplasia, and displasia [11]. These precancerous lesions are important risk factors for gastric adenocarcinoma [12]. In 2006, The American Society for Gastrointestinal Endoscopy (ASGE) reported that routine surveillance programmes for intestinal metaplasia are difficult to implement, but they are important and useful in patients at high risk for gastric cancer [13]. The cost effectiveness and feasibility of these surveillance programmes are still debatable. Zullo et al. recommend endoscopic surveillance for patients with gastric atrophy and intestinal metaplasia once every three years [14]. Because early gastric cancer is usually asymptomatic, 75% of patients are admitted to the hospital in the late stages [15]. Atrophic gastritis, intestinal metaplasia, and displasia are precancerous lesions for gastric cancer. Previous studies have shown that HP infection promotes progression of these precancerous lesions [16]. After the publication of reports with similar results, WHO defined HP infection as a class 1 carcinogen. HP infection is reported to increase the risk of gastric cancer by a factor of 2.8 [17]. In HP patients with certain genotypes, the risk of intestinal metaplasia is higher [18]. HP eradication is important because intestinal metaplasia progresses to displasia and gastric cancer. After IM development, mucosal acidity changes and HP colonisation decreases, at which point HP eradication stops being useful [19]. Although local healing following IM can be seen in the antrum, there was no healing in the corpus in some reports [20]. In type 3 IM patients, the gastric cancer incidence is 28% [21]. In the literature, HP eradication has been shown to be important in gastric cancer prevention [22]. In a
study by San et al. 36.31% of 9239 patients were proven to be HP positive by CLO test [23]. In our study we performed gastroscopy in 682 patients; histologically we diagnosed HP in 475 (69.6%) of them. In eastern Anatolia, Olmez et al. detected 560 IM in 4050 patients [24]. In our study we investigated IM development in HP positive patients and diagnosed IM in 48 of 475 HP positive patients. Co-occurrence of helicobacter pylori and intestinal metaplasia was statistically significant (p: 0.006).

In line with other reports, in our study we also detected that HP promotes IM. If HP is positive serologically or histologically, before the development of precancerous lesions, HP eradication should be done to prevent gastric cancer. In HP positive patients, IM may be the irreversible stage in cancer progression. Thus, HP positive patients need close monitoring and aggressive treatment. For prevention of gastric cancer, further studies concerning the healing of IM by HP eradication should be conducted.

**Competing interests**

The authors declare that they have no competing interests.

**References**


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