E-ISSN: 2616-3470
P-ISSN: 2616-3462

www.surgeryscience.com
2019; 3(1): 207-209
Received: 13-11-2018
Accepted: 17-12-2018

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Role of H pylori in GERD- A prospective study

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DOI: https://doi.org/10.33545/surgery.2019.v3.i1d.36

Abstract
Introduction: The role of Helicobacter pylori in esophageal disease has not been clearly defined. To clarify this issue, we analyzed 120 patients with histologically confirmed esophageal disease. *Helicobacter pylori (H. pylori)* causes a long-term infection of the human gastric and duodenal mucosa (1). Mucosal colonisation predisposes for peptic ulcer disease, atrophic gastritis and distal (antral) stomach cancer (2), with various effects on gastric acid secretion. Genetic variability of *H. pylori* is high (3). Several genes have been identified that may play a role in the pathogenicity (4, 5). Most important is the cytotoxin-associated gene A *(CagA),* which is associated with peptic ulcer disease (6), and intestinal type adenocarcinoma of the stomach (7). Patients with duodenal ulcer often have high basal gastrin levels, high peak acid output and high 24-hour intragastric acidity (8-10).
Materials and methods: This investigation was performed at the RL Jalappa Hospital, Tamaka, Kolar in patients who underwent Upper GI Endoscopy. The time of investigation was from January 2016-December 2018.
Results: This study included 30 patients with BE out of 120 patients with GERD. The average age of patients in BE group was 52.4 years. (SD } 10.8 yr.) In the GERD group average age was 40.8 years. In all groups included in this study, men were more represented than women. In BE group, *H. pylori* infection was present in 16.0% of patients. In GERD group, *H. pylori* infection was present in 42.9%.
Conclusion: The prevalence of *H. pylori* infection in patients with BE was lower in comparison with patients with GERD (p<0.01). The prevalence of *H. pylori* infection in patients with BE, especially those with Lower Segment BE was very low, which indicates the possible protective role of this microorganism.

Keywords: H pylori, Barrett's oesophagus, GERD, *CagA*

Introduction
Helicobacter pylori (H. pylori) causes a long-term infection of the human gastric and duodenal mucosa [1]. Mucosal colonisation predisposes for peptic ulcer disease, atrophic gastritis and distal (antral) stomach cancer [2], with various effects on gastric acid secretion. Genetic variability of *H. pylori* is high [3]. Several genes have been identified that may play a role in the pathogenicity [4, 5]. Most important is the cytotoxin-associated gene A *(CagA),* which is associated with peptic ulcer disease [6], and intestinal type adenocarcinoma of the stomach [7]. Patients with duodenal ulcer often have high basal gastrin levels, high peak acid output and high 24-hour intragastric acidity [8-10].
In contrast, patients with *H.* pylori-associated gastric ulcer often have hypochlorhydria [11]. Several reports suggest that the prevalence of *H. pylori* and especially the most pathogenic form-*CagA* might be lower in patients with gastroesophageal reflux disease (GERD), including Barrett's oesophagus (BE) than in the rest of the population [12, 13]. One explanation for the negative association between mucosal colonisation with *H. pylori* and GERD is the effect of *H pylori* on acid production, since extensive gastritis involving the corpus may lower acid secretion by impairing parietal function.
The aim of this study was to determine the prevalence of *H. pylori* infection and its possible protective role in the appearance of GERD and its progression to BE.

Materials and methods
This investigation was performed at the RL Jalappa Hospital, Tamaka, Kolar in patients who underwent Upper GI Endoscopy. The time of investigation was from January 2016-December 2018.
In this prospective study, from 120 patients with GERD, 30 were proved to have Barrett's

Oesophagus (BE). All the patients were interviewed for their age, sex, reflux symptoms, chronicity, medications used, the presence of *H. pylori* infection, weight, family history and Smoking.

Diagnosis of infection with *H. pylori* was made by biopsy and Urease test.

The study excluded patients who did not have typical anamnestic data for gastro-esophageal reflux disease, those who have gastro-esophageal erosive changes during endoscopy, and patients with pre-existing histopathological proven esophageal adenocarcinoma. The results were processed by modern statistical methods. Data processing is performed with InStat 3 statistical package. The difference was considered significant if $P<0.05$.

Results

This study included 30 patients with BE out of 120 patients with GERD. The average age of patients in BE group was 52.4 years. (SD } 10.8 yr.) In the GERD group average age was 40.8 years. (SD } 13.5 yr.). The age difference between groups was significant (One Way ANOVA F = 13.91, $P<0.001$). Among patients with BE, the most represented age group was 50-59 years, in the GERD-group the most represented age group was from 40-49 years.

In all groups included in this study, men were more represented than women. In the group with BE 78.0% were men, in the group with GERD 64.3%. However, the difference was not statistically significant (χ2-test = 4.08, p = 0.130). Average body height of respondents in Group BE was 174, 8cm (SD } 8, 2 cm), in the GERD group it was 168, 5cm (SD } 8, 9 cm). The difference was statistically significant (One Way ANOVA F = 7.45, $P<0.001$).

In patients included in the study, the prevalence of infection with *H. pylori* was analyzed. In BE group, *H. pylori* infection was present in 16.0% of patients. In GERD group, *H. pylori* infection was present in 42.9%. So, in BE group, the prevalence of H. pylori infection showed less significant difference compared to GERD group.

Regarding endoscopic type of BE and the presence of infection with *H. pylori*, there was no significant difference (Fisher Exact Test, P = 0.665).

Discussion

The management of GERD and BE remains a challenging problem and this is partly due to a limited knowledge of its natural history. The relation-ship between GERD, BE and *H. pylori* is very complex (1). There might also be connection between prolonged proton pump inhibition and the rate of progression to atrophic gastritis, leading to hypochlorhydria [12-14]. *H. pylori*, in contrary to overweight and hiatal hernia, may interact with the risk of BE rather in physiological aspect, than anatomically. *H. pylori* can reduce the risk for BE by possible reduction of acidity in the stomach by the action of urease. The fact that *H. pylori* may protect against BE is contrary to the established status of risk factors for peptic ulcer and gastritis.

H. pylori infection was present in 16.0% of patients in BE group, comparing to 42.9% of patients in the group with GERD. Results from one study (28) showed low prevalence of *H. pylori* infection in patients with BE (12%). Data from the literature also showed low prevalence of *H. pylori* infection in these patients. In the same study, of 251 patients who underwent endoscopy, cagA + *H. pylori* was present in 44% of examinations, 36% of 36 patients with GERD. 20% of 10 patients with SSBE, and in 0% of 18 patients with LSBE. A limitation in our study was lacking of the laboratory method for determination of cagA+ types of *H. pylori*.

H. pylori infection has also been implicated in the pathogenesis of GERD. *H. pylori* infection may be associated with increased acid secretion, but in contrast with achlorhydria resulting in atrophic gastritis, depending on the bacterial species and the inflammatory response that causes it. Studies showing that *H. pylori* negative patients have more severe esophagitis compared with H. pylori positive, suggesting that this bacterium may have a protective role in patients with GERD. In fact, infection with *H. Pylori* can induce atrophy and thus reduction of acidic secretion, which ultimately results in reduced risk of developing GERD. In contrast, the eradication of *H. pylori* infection may result in normal acid production and exacerbation of GERD. However, recent clinical studies cannot provide strong enough evidence for a possible role of *H. pylori* infection in the development of GERD and erosive esophagitis. In clinical practice, since *H. Pylori* infection is associated with an increased risk of peptic ulcer and gastric cancer, existing guidelines recommend its eradication, regardless of the potential effect on GERD [28, 29].

H pylori, in particular the *CagA* phenotype, through gastritis and associated hypochlorhydria might be a protective factor against GERD and its complications [30]. In recent years it has become clear that a significant number of patients will develop reflux oesophagitis after apparently successful eradication [30]. The findings are consistent with the hypothesis that the declining infection rates of *H. pylori* in the general population have led to a rise in the occurrence of GERD and associated oesophageal adenocarcinoma [28]. The prevalence of Cag-A phenotype was also lower in patients with complicated GERD (such as BE), than in the rest of population [31].

Conclusion

The prevalence of *H. pylori* infection in patients with BE was lower in comparison with patients with GERD ($p<0.01$). The prevalence of *H. pylori* infection in patients with BE, especially those with Lower Segment BE was very low, which indicates the possible protective role of this microorganism.

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