Role of H pylori in GERD- A prospective study

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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 \( \pm \) 10.8 yr.) In the GERD group average age was 40.8 years. (SD \( \pm \) 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. (SD \( \pm \) 0.8 yr.). In the GERD group average age was 40.8 years. (SD \( \pm \) 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.

**References**

1. Blaser MJ. Ecology of Helicobacter pylori in the human stomach. J Clin Invest. 1997; 100(4):759-762.
2. Kuiipers EJ. Helicobacter pylori and the risk and management of associated diseases: gastritis, ulcer disease, atrophic gastritis and gastric cancer. Aliment Pharmacol Ther. 1997; 11:7l-88.
3. Dunn BE, Cohen H, Blaser MJ. Helicobacter pylori. Clin Microbiol Rev. 1997; 10(4):720-741.
4. Blaser MJ. Intrastrain differences in Helicobacter pylori: a key question in mucosal damage? Ann Med. 1995; 27(5):559-563.
5. Atherton Je. The clinical relevance of strain types of Helicobacter pylori. Gut. 1997; 40(6):701-703.
6. Van Doorn LJ, Figueiredo C, Sanna R, Plaisier A, Schneeberger P, De Boer W et al. Clinical relevance of the cagA, vacA, and iceA status of Helicobacter pylori. Gastroenterology. 1998; 115(1):58-66.
7. Kuiipers EJ, Perez P, Meuwissen SGM, Blaser MJ et al. Helicobacter pylori and atrophic gastritis: importance of the cag A status. J Nat Cancer Inst. 1995; 87(23):1777-1780.
8. Calam J, Gibbons A, Healey ZY, Bliss P Arebi N. How does Helicobacter Pylori cause mucosal damage? Its effect on acid and gastrin physiology. Gastroenterology. 1997;
19. Spechler SJ. Barrett’s esophagus. N Engl J Med. 2002; 346:836-842.
20. Rex DK, Cummings OW, Shaw M et al. Screening for Barrett’s esophagus in colonoscopy patients with and without heartburn. Gastroenterology. 2003; 125:1670-1677.
21. Rastogi A, Puli S, El-Serag HB et al. Incidence of esophageal adenocarcinoma in patients with Barrett’s esophagus and high-grade dysplasia: a metaanalysis. Gastrointest Endosc. 2008; 67:394-398.
22. O’Connor JB, Falk GW, Richter JE. The incidence of adenocarcinoma and dysplasia in Barrett’s esophagus: report on the Cleveland Clinic Barrett’s Esophagus Registry. Am J Gastroenterol. 1999; 94:2037-2042.
23. Van Soest EM, Dieleman JP, Siersema PD, Sturkenboom MC, Kuipers EJ. Increasing incidence of Barrett’s oesophagus in the general population. Gut. 2005; 54:1062-1066.
24. Cameron AJ, Lomboy CT. Barrett’s esophagus: age, prevalence, and extent of columnar epithelium. Gastroenterology. 1992; 103:1241-1245.
25. Spechler S, Goyal R. The columnar-lined esophagus, intestinal metaplasia, and Norman Barrett. Gastroenterology. 1996; 110:614-621.
26. Spechler SJ. Barrett’s esophagus. N Engl J Med. 2002; 346:836-842.
27. Spechler SJ. Intestinal metaplasia at the gastroesophageal junction. Gastroenterology. 2004; 126:567-575.
28. Sharma P, Vakil N. Review article: Helicobacter pylori and reflux disease. Aliment Pharmacol Ther. 2003; 17:297-305.
29. Vaezi MF, Falk GW, Peek RM, Vicari JJ, Goldblum JR et al. CagA positive strains of Helicobacter pylori may protect against Barrett’s esophagus. Am J Gastroenterol. 2000; 95:2206-2211.
30. Labenz J, Blum AL, Bayendorfer E, Meininger A, Stolte M, Borsch G. Curing Helicobacter pylori infection in patients with duodenal ulcer may provoke reflux esophagitis. Gastroenterology. 1997; 112(5):1442-1447.
31. Vicari JJ, Peek RM, Falk GW, Goldblum JR, Easley KA, Schnell J et al. The seroprevalence of cagA positive Helicobacter pylori strains in the spectrum of gastroesophageal reflux disease. Gastroenterology. 1998; 115(1):5.