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Research Article

Virulent Helicobacter Pylori In Dental Plaque and Gastric Specimen Samples

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Abstract

Introduction The aim of study is to investigate the presence of H. pylori in dental plaque and sections of gastric mucosa in patients with dyspepsia and association with oral hygiene statues.

Materials and Methods: Sub gingival plaque specimens (of molar, premolar and incisors) and gastric biopsies from one hundred patients referred to gastrointestinal endoscopy center were collected. Rapid urease test and PCR assays were used to detect the presence of virulent H. pylori.

Results: The presence of H. pylori DNA was confirmed in 96% of sections of gastric mucosa and in 72% of dental plaque samples. The most common H. pylori genotype was vacAs1m1 and cagA positive, either in dental plaque or gastric mucosa. Genomic DNA by H. pylori was also found in 83% of dental plaque samples. Molar regions had the most probability of harboring H. pylori (67% in molar, compared to 25 % in pre molar and 8% in incisors) but there was no difference between molar, pre molar and incisors in isolating cagA+ H. pylori (P>0.05).

Conclusions: The presence of cagA positive strain in oral cavity of dyspeptic patients suggests that dental plaque may act a source of re-infection in stomach. Additionally, according to our findings, detection of H. pylori from dental plaque and gastric biopsy samples were both greater in PCR assay than culturing or the rapid urease test. However, further experiments are necessary to elucidate exact mechanism of H. pylori transmission.

Keywords: Helicobacter pylori; PCR; Dental Plaque; Virulent; cagA.

Introduction

Helicobacter pylori (H. pylori) infection is considered as one of the most prevalent infectious agents worldwide. The prevalence of this gastric microorganism varies between 85-90% in developing countries and 20 -35% in developed countries [1,2]. In past, it has been declared that dental plaque locality does possibility of being a potentially reservoir of H. pylori, beside the stomach and it may act as a source for transmission of this microorganism [3, 4]. Hence, it has been generally stated that infected individuals with H. pylori strains producing 128-kDa proteins cagA (virulent strains) are bound to suffer from

more severe diseases [5,6]. To date, various studies had shown contradictory findings about association of H. pylori cagA positive and diseases outcome in dyspeptic patients [5,7-10]. It seems that dental plaque can play a critical role responsible for re-colonization of the stomach after primary antibiotic therapy [11,12]. While the presence of H. pylori in dental plaque was a common finding in some studies [12,13]; it was not confirmed by others [14,15]. There is a lacking on reports indicating on isolation of cagA positive strains from oral cavity. Undoubtedly, finding an association between virulent H. pylori from dental plaque and certain clinical outcome can disclose a crucial importance of colonized dental plaque with those H. pylori virulent strains. The study aimed to investigate the probability of presence of H. pylori cagA positive in dental plaque of symptomatic patients to find a possible association between those virulent H. pylori strains and gastroduodenal disorders.

Materials and Methods

A total number of one hundred dyspeptic patients referred for upper gastrointestinal endoscopy during the 2007-2009 in Baghiayatallah hospital, Tehran, Iran were recruited. Informed consent was obtained. The study was approved by the ethics boards of University of Tarbiat Modares, Tehran, Iran. Patients received no antibiotics, proton pomp inhibitors, H2-blockers, anti-acids or bismuth compounds, plaque removal or infection in oral cavity three months prior to the sampling. A detailed demographic questionnaire was filled regarding oral care such as teeth cleaning habits, number of visits to the dentist and history of dyspepsia (data not shown). The gingival index scores for the mesial, distal, buccal and lingual gingival were given from 0 (no inflammation) to 3 (sever inflammation, ulceration and spontaneous bleeding). The plaque index measures the thickness of plaque at gingival margin on the buccal, lingual, mesial and distal aspects. The scored used are: 0 (none), 1 (plaque that is not visible to the eye but can be seen on an instrument when scraped along the gingival margin on the tooth surface); 2 (plaque that can be seen by naked eye); 3 (gross accumulation of plaque). Before endoscopy supra gingival and sub gingival plaque samples scraped from molar, pre molar and incisors by sterile curette transferred into tube containing physiological saline and stored at 4°C ,then immediately shipped to the lab for DNA extraction. However, briefly, DNA extraction performed by Genomic DNA Extraction Kit (Bioneer) following by polymerase chain reaction test with specific forward and reveres primers of ureC gene. Forward:5´-CCCTCACGCCATCAGTCCCAAAAA-3´and reveres:5´-AAGAAGTCAAAAACGCCCCAAAAC-3´.Total volume of reactions was 25 µL and solution included 2.5 µL of 10x buffer (PH 8.4) containing 100 mM Tris/HCl, 500 mM KCl and 2 mM MgCl2, o.2 mM dNTP, 1.5 U rTaq DNA polymerase, 2.5 µL bacterial DNA , 0.2 mM primer. 30 cycles performed, 94°C for 5 minutes (primary denaturation), 94°C for 1 minute, 55°C for 1 minute, 72°C for 1 minutes and final extension at 72°C for 10 minutes.

Results

One hundred patients (66 male, 34 female) who had a history of dyspepsia enrolled this study. Overall, 83% of patients had caring H. pylori in dental plaque. According to our findings, molar regions had the most probability for harboring H. pylori (67% in molar, compared to 25% in pre molar and 8% in incisors); but there was no significant correlation was observed between molar, pre molar and incisors in isolation of H. pylori having cagA positive (P>0.05); (34% in molar, compared to 36% in premolar and 30% in incisors). All patients who were positive for H. pylori DNA in their dental plaque had average or poor dental hygiene: 54% scores 2 (inflammation and spontaneous bleeding) and 46% scored 3 (sever inflammation, ulceration and spontaneous bleeding). Seventy three percent of patients, who had H. pylori cagA positive in their dental plaque, had severed clinical complications: 65% duodenal ulcers, 30% gastritis and gastric ulcer and 3% had duodenal deformity.

Discussion

H. pylori infection is one of the most common bacterial infections in world population. The human stomach was considered to be the only reservoir for H. pylori until bacteria were discovered in the human dental plaque, in oral lesions or ulcers, in oral cavity, and in saliva. The results of current study indicate that H. pylori are present in dental plaque of dyspeptic patients (83%). Song Q and et al [16] observed H. pylori in oral cavity of 97% of dyspeptic patients; actually this was the first report indicating such a high prevalence. But there were other studies which gain different results even with using the same pair of primers [13]. This finding may be due to differences in population and sample handling.

Presence of H. pylori cagA positive

DNA was approved by PCR in this study (38.5%) but we find no other similar report indicating such prevalence independent of gastric infection. We found that most of the patients as H. pylori cagA positive in their dental plaque had several complications in their stomach such as duodenal ulcer, gastritis, and duodenal deformity, so it seemed that oral cavity may act as a potential reservoir for infecting and transferring H. pylori to stomach. Remarkably, more studies are necessary to confirm the critical role of oral cavity as a definite reservoir of H. pylori and its importance in transmission of H. pylori. PCR is now widely applied to detect oral H. pylori, maybe due to difficulties of culture method and low specificity of rapid urease test for oral specimens (over growth of oral micro flora and presence of urease producing micro organism in the mouth), but it should be considered that in spite of high detection rate using PCR. A previous study showed

H. pylori had a characteristic distribution pattern in oral cavity [13] with a higher prevalence in molar regions, and our current study showed similar results. It may be due to difficult access to molar sites (especially sub gingival areas) for teeth cleaning and brushing. Furthermore, it is possible that using a mixture of dental plaque caused to increase the rate of bacterial detection [16]. Once H. pylori isolated from oral cavity, the oral health status should be improved to prevent increasing the bacterial load. Collectively, successful bacterial eradication can result in prevention of re-infection. Eskandari et al, reported a relatively lower prevalence of H. pylori in dental plaque, a report which might be affected by different geographical locations [17]. Additionally, plaque removal may be a good suggestion before antibiotic therapy for successful eradication of H. pylori. It is estimated that H. pylori cagA positive can colonize the dental plaque and may transfer to other gastrointestinal sites from this ecological niche. However, this study is a pilot to support more detailed molecular tests to track bacterial identity in oral cavity and dental plaque to elucidate exact mechanism of H. pylori transmission.

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Conflict of Interest

No conflict of interest to declare.

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