Helicobacter Pylori in periodontal pockets of chronic periodontitis patients with and without type II diabetes mellitus: a randomized controlled trial

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Abstract

This randomized controlled study evaluated the association of Helicobacter pylori (H. pylori) with chronic periodontitis patients with and without type II Diabetes Mellitus. H. pylori is considered to be a pathogen responsible for gastritis, peptic ulcers and a risk factor for gastric cancer. The aim of the present study was to evaluate the association of H. pylori with chronic periodontitis patients with and without type II diabetes mellitus before and after treatment. The prevalence of H. pylori in periodontal pockets was determined by rapid urease test in a 36 patients, which were grouped as Group I (Healthy subjects), Group II (chronic periodontitis patients) and Group III (Chronic periodontitis patients with Type II Diabetes Mellitus), 12 in each group before treatment by collecting plaque samples. After treatment, 12 plaque samples were collected and prevalence H. pylori was detected. Group II and Group III had a significantly higher rate of positive results for H. pylori compared to healthy subjects before treatment. After treatment, H. pylori were not detected in Group II and in Group III Only one of 12 chronic periodontitis patients with Type II diabetes mellitus had H. pylori in the periodontal pocket. The prevalence of H. pylori did not differ significantly between the chronic periodontitis patients with and without type II diabetes mellitus. Meticulous scaling and root planning will reduce the prevalence of H. pylori in periodontal pockets.

Introduction

Helicobacter pylori (H. pylori) is a curved, spiral, or gull-wing shaped microaerophilic, Gram-negative, motile bacterium with polar-sheathed flagellae. H. pylori is considered to be a pathogen responsible for gastritis, peptic ulcers and a risk factor for gastric cancer.1,2

The mode of transmission of H. pylori is poorly understood, although the oral-oral, gastric-oral and fecal-oral routes are possible.6

The detection of this microorganism in the oral cavity has been reported by several groups,7-15 who demonstrated the microorganism in dental plaques and saliva, which would implicate the oral cavity as a potential reservoir for H. pylori or a possible route of transmission to other sites. However, other studies reported no detection of H. pylori from dental plaque samples.16-20 Umeda et al. compared the prevalence of H. pylori in patients with and without periodontal pockets and showed a higher prevalence of the bacteria in patients with deep periodontal pockets.21 H. pylori has ability to co-aggregate with periodontopathogenic bacteria such as Fusobacterium nucleatum and Porphyromonas gingivalis.22

Diabetes mellitus (DM) is complex metabolic disturbance, characterized by chronic hyperglycemia. A multivariate risk analysis showed that subjects with type II DM had approximately 3 fold increased odds of having periodontitis and having altered flora in the periodontal pockets of patients with diabetes.23,24 Many studies have evaluated the prevalence of H. pylori infection in diabetes patients with gastritis and the possible role of this condition in glycemic control.25 Therefore the aim of the present study was to evaluate the association of H. pylori with chronic periodontitis patients with and without type II diabetes mellitus before and after treatment. According to our knowledge this is the first study being carried out in this regard.

Materials and Methods

The study population was selected randomly from patients attending the Department of Periodontology, Rajarajeswari Dental College and Hospital, Bangalore from April to June 2010. The grouping was as follows:

Group I: 12 Healthy subjects;
Group II: 12 Chronic periodontitis patients;
Group III: 12 Chronic periodontitis patients with Type II Diabetes Mellitus.

12 plaque samples from healthy subjects (9 males and 3 females, mean age 22.58±4.95), 12 plaque samples from group II (6 males and 6 females, mean age 40.57±12.41) and 12 from group III (10 males and 2 females, mean age 48.67±8.31) were collected before the periodontal treatment. Twelve samples each were collected from group II and group III after treatment.

Chronic periodontitis was diagnosed using the periodontal disease classification system of the American Academy of Periodontology (1999).26 Chronic periodontitis patients with minimum of 20 teeth and average probing depth of >5 mm with and without history of Type II diabetes mellitus were included in the study.

Subjects with healthy periodontium were defined as having probing depth of <3 mm and gingival index of 1 mm. The known cases of type II DM in group III were assessed by random blood sugar level (RBS) and questionnaire. Exclusion criteria were as follows: any history of chronic gastritis, smoking, pregnancy, and periodontal therapy within last 6 months, other systemic conditions that could affect the periodontal status, use of local or systemic antimicrobial agents within 6 months prior to entry into the study. Periodontal evaluation included the Gingival Index (GI),27 and the probing pocket depth (PPD). Ethical clearance for the study was obtained from Institutional Ethical Review Board and subjects who satisfied the inclusion criteria of the study were selected. Informed consent was obtained from all enrolled individuals.

Collection of samples

Supragingival plaque was removed with sterile gauze and subgingival plaque samples were collected from deepest pocket in patients with chronic periodontitis with and without type II DM using sterile curettes. In healthy subjects plaque samples were collected from random sites. Subjects with chronic periodontitis with or without type II DM received full mouth scaling and root planing under local anesthesia and instruction in proper home care procedure and recalled after 2 weeks for post treatment sample collection.

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The rapid urease test
Presence of H. pylori in the samples was detected by rapid urease test. Samples were transferred immediately into vials containing rapid urease test reagent. If the test solution color was changed from yellow to pink within 30 min (as recommended by the manufacturer), the sample was considered to be positive for H. pylori.

Statistical analysis
Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance was assessed at 5% level of significance. χ²/Fisher Exact test was used to find the significance of study parameters on categorical scale between two or more groups. 95% confidence interval was computed to find the significant features.

Results
The demographic and clinical parameters of the subject groups are shown in Table 1. Clinical parameters (PPD and GI) was found to be significantly greater in the Group II and Group III when compared to Group I (P<0.01; Chi-square test). The significant differences were observed between the groups with respect to gender and age.

The presence of H. pylori in periodontal pockets was determined by rapid urease test and result was expressed as either positive or negative. Five out of 12 subjects (41.71%) in group I, 9 out of 12 subjects (75%) in Group II and 10 out of 12 subjects (83.3%) in Group III revealed positive rapid urease test results before treatment. Figure 1 shows the detection rate of H. pylori among the groups by rapid urease test before treatment. The difference in incidence of H. Pylori in group III (83.3%) and group II (75%) was statistically significant (Chi-square test, P=0.001). Figure 2 shows the prevalence of H. pylori in the subgingival plaque of Group II after treatment and Group III after treatment patients. H. pylori was not detected in Group II patients after treatment but was present in only one out of 12 patients of group III after treatment.

Discussion
The periodontal pocket may provide a conducive environment for the colonization of H. pylori. The complex and diverse microbiota together with persistent inflammatory process may provide a wide range of nutrients and binding site for the establishment of this microorganism. Closely related species such as Campylobacter rectus and Fusobacterium were described as key microorganisms in the process of co-aggregation among different genera of facultative bacteria. Colonization of these species may favour the establishment of H. pylori in the periodontal environment. Moreover, the subgingival biofilm can provide significant amount of urea, which can favour the urease-producing bacteria, such as H. pylori. Antagonistic relationship also may occur within subgingival biofilm.

The present study was undertaken to evaluate the association between H. pylori and chronic periodontitis with and without type II diabetes mellitus in 36 subjects before and after treatment. There was increased prevalence of H. pylori in Group II and Group III patients before treatment as compared to periodontally healthy subjects (Group I). The results of the present study suggest the possible association of H. pylori with chronic periodontitis with and without type II diabetes mellitus. Riggio and Lennon studied the presence of H. pylori in the subgingival plaque of adult periodontitis patients. They found that 38% of the subjects with deep periodontal pockets were positive for H. pylori. Also, a study had suggested that poor periodontal health characterized by deep periodontal pocket...
ets may be associated with \( H. \) pylori infection\(^3\) and increased detection of the \( H. \) pylori in the oral cavity of the patients with periodontal disease and other oral conditions. The gender distribution reveals that \( H. \) pylori was detected more in males than in females; the role of the gender as a risk factor for \( H. \) pylori infection was not substantiated. High prevalence of \( H. \) pylori in the dental plaque has been reported using the Campylobacter-like organism (CLO) test and polymerase chain reaction (PCR).\(^2\)\(^,\)\(^3\)\(^,\)\(^4\) However, investigators have tried to detect by the culture method and reported that \( H. \) pylori was not cultivated from culture methods\(^5\) which may be due to low numbers of the organism in the dental plaque samples or existence of non-cultivable forms in the dental plaque. Other investigators employing PCR for the detection of \( H. \) pylori have also failed to reveal the specific amplification-product characteristic of \( H. \) pylori suggesting that periodontal pockets do not constitute a natural reservoir for \( H. \) pylori.\(^6\) Also, high prevalence of \( H \)-pylori in the dental plaque and saliva has been detected using PCR in periodontitis patients.\(^7\) Studies have reported that rapid urease test has a specificity near 100% and sensitivity between 70% and 90%.\(^8\)\(^,\)\(^9\) The sensitivity of using this test on dental plaque to determine \( H. \) pylori status was reported to be 89.7%, with diagnostic accuracy of 86.7%.\(^10\) It has been reported that other urease positive microorganism present in the oral cavity such as \( \text{Streptococcus} \) vestibularis and \( \text{Actinomyces} \) viscosus usually cannot give positive results within an hour.\(^11\) Patients with diabetes mellitus are usually affected by chronic infections. Diabetic patients are at more risk for \( H. \) pylori infection in comparison to non-diabetic subjects.\(^12\) Some studies found a higher prevalence of \( H. \) pylori in diabetic patients and reduced glycemic control while others did not support any correlation between metabolic control and \( H. \) pylori infection.\(^13\) Black pigmented species, especially \( \text{P} \) gingivalis, \( \text{P} \) intermedia and \( \text{C} \) rectus, are prominent in severe periodontal lesion with type II diabetes.\(^14\)\(^,\)\(^15\) These results point to altered flora in the periodontal pockets of patients with diabetes.\(^16\) In the present study after the treatment, all the patients with chronic periodontitis (Group II) were negative for \( H. \) pylori and only one patient in Group III (with diabetes) was positive for \( H. \) pylori suggesting that meticulous scaling and root planning reduces the levels of \( H. \) pylori in periodontal pockets even in diabetes patients. This is the first study conducted to evaluate the association of \( H. \) pylori in chronic periodontitis patients with and without type II diabetes mellitus before and after treatment.

### Conclusions

Periodontal pockets can act as reservoirs for \( H. \) pylori, which can interact with other periodontopathogenic bacteria and might potentially, participate in causing chronic periodontitis and chronic gastritis. The prevalence of \( H. \) pylori \( \text{did not differ significantly} \) between the chronic periodontitis patients with and without type II diabetes mellitus. Meticulous scaling and root planing can reduce the prevalence of \( H. \) pylori in periodontal pockets. Future studies with larger sample size involving other methods of detecting \( H. \) pylori like PCR and serology would help us to assess the potential role of \( H. \) pylori in periodontitis and possible transmission to cause chronic gastritis.

### References