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

Savita Sambashivaiah, Shivaraprasad Bilichodmath, Nanjamannni Nanjaiah, Ritheesh Kulal
Department of Periodontology, Rajarajeswari Dental College and Hospital, Bangalore, Karnataka state, India

**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. The mode of transmission of *H. pylori* is poorly understood, although the oral-oral, gastric-oral and fecal-oral routes are possible. The detection of this microorganism in the oral cavity has been reported by several groups, 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. Ulmeda 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. *H. pylori* has ability to co-aggregate with periodontopathogenic bacteria such as *Fusobacterium nucleatum* and *Porphyromonas gingivalis*.

Diabetes mellitus (DM) is complex metabolic disorder, 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. 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. 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 group II (6 males and 6 females, mean age 22.58±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). 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), 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.
The rapid urease test

Presence of H. pylori in the samples was detected by rapid urease test. Samples were transferred immediately into vial 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. \( \chi^2 \)/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 microorganism 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 pock-

| Table 1. Demographic and full-mouth clinical parameters (mean±SD) of Group I, Group II and Group III subjects. |
|-----------------------------------------------|
| Parameters                  | Group I (n=12) | Group II (n=12) | Group III (n=12) |
| Age (years)                | 22.58±4.95     | 40.57±12.41     | 48.67±8.31       |
| Males (%)                  | 75.0           | 50.0            | 83.3             |
| Females (%)                | 25.0           | 50.0            | 16.7             |
| Gingival index             | 0.32±0.33      | 1.80±0.34       | 1.83±0.29        |
| PPD (mm)                   | 1.33±0.33      | 7.91±0.91       | 8.08±1.31        |
Periodontal pockets can act as reservoirs for \textit{H. pylori}, which can interact with other periodontopathogenic bacteria and might potentially, participate in causing chronic periodontitis and chronic gastritis. The prevalence of \textit{H. pylori} 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 \textit{H. pylori} in periodontal pockets. Future studies with larger sample size involving other methods of detecting \textit{H. pylori} like PCR and serology would help us to assess the potential role of \textit{H. pylori} in periodontitis and possible transmission to cause chronic gastritis.

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[Microbiology Research 2011; 3:e12]

[page 47]
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