Are Dental Plaque, Poor Oral Hygiene, and Periodontal Disease Associated With *Helicobacter pylori* Infection?

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Background: The microorganism *Helicobacter pylori* has been closely linked to chronic gastritis, peptic ulcer, gastric cancer, and mucosa-associated lymphoid tissue (MALT) lymphoma. Despite the current treatment regimens that lead to successful management of *H. pylori*-positive chronic gastritis, the reinfection rate is high. It has been suggested that one of the possible mechanisms of reinfection is the recolonization from dental plaque. The purpose of this study was to determine whether dental plaque, poor oral hygiene, and periodontal disease were risk factors for *H. pylori* infection.

Methods: Among the 134 patients, 65 patients who had a positive *H. pylori* serology or positive rapid urease test or histologic evidence for the presence of *H. pylori* in antral biopsy specimens were categorized as cases. The remaining 69 patients who were negative for *H. pylori* serology, the rapid urease test, and histology were controls.

Results: It was found that the association of periodontal disease and poor oral hygiene with *H. pylori* infection was not significant. There was a higher prevalence of *H. pylori* in the dental plaque of patients with gastric *H. pylori* infection than in controls, but both groups had a surprisingly high positive urease test for *H. pylori* in plaque (89% and 71%, respectively).

Conclusions: *H. pylori* in dental plaque is seldom eliminated by *H. pylori*-eradication therapy, and this may act as a source for future reinfection. Hence, eradication of *H. pylori* from the dental plaque should be made an important part of comprehensive management of *H. pylori*-associated gastric diseases. *J Periodontol 2006;77:692-698.*

KEY WORDS

Dental plaque; *Helicobacter pylori*; periodontal disease.

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Helicobacter pylori is one of the most common bacterial infections of humans[1] and has been closely linked to chronic gastritis, peptic ulcer, gastric cancer, and mucosa-associated lymphoid tissue (MALT) lymphoma.[2,3] Despite the current treatment regimens that lead to successful management of *H. pylori*-positive chronic gastritis, the reinfection rate is relatively high.[4] Dental plaque has been implicated as a potential reservoir of *H. pylori*, and it has also been detected in the saliva.[5-7] The suggested possible mechanism of reinfection is recolonization from dental plaque.[4,8-10] However, some studies have reported no correlation between dental presentation of the microorganism and *H. pylori*-associated gastritis.[11] The hypothesis that oral flora may be a permanent reservoir of viable *H. pylori* is still inconclusive. This study was undertaken to determine whether dental plaque, poor oral hygiene, and periodontal disease were risk factors for *H. pylori* infection.

MATERIALS AND METHODS

This study was approved by a human ethical committee and was designed as a case control study. The study was conducted at the Department of Medical Gastroenterology, Government Medical College, Trivandrum, during a 6-month period from January 2004 to June 2004. Subjects with dyspepsia undergoing endoscopy and who had either a positive

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H. pylori serology or positive rapid urease test on antral biopsy specimens or histologic evidence for the presence of *H. pylori* in antral biopsy specimens were cases, and subjects with dyspepsia undergoing endoscopy and who were negative for *H. pylori* serology, the rapid urease test, and histology were controls. A total of 149 patients were enrolled, but endoscopy could not be undertaken in seven patients. Of the 142 patients who were evaluated, complete data were available for 134 patients. Sixty-five were grouped as cases and 69 as controls. Written informed consent was obtained from the subjects who participated in the study. The study variables were age, gender, socioeconomic status, handling of animals, smoking (duration, type, and amount), alcohol consumption (duration, type, and amount consumed), and use of non-steroidal anti-inflammatory drugs or other drugs.

Oral examination included assessment of oral hygiene status, periodontal disease status, and examination of plaque for *H. pylori* using the rapid urease test.

Oral hygiene status was assessed and classified into good, fair, and poor using the oral hygiene index of Greene and Vermillion.[12]

Periodontal status was assessed based on the criteria employed in previous studies using the third National Health and Nutrition Examination Survey (NHANES III).[13] Patients with one or more sites with a probing depth ≥3 mm and clinical attachment loss ≥3 mm at the same site were classified as having periodontal disease.

Presence of *H. pylori* in the dental plaque was determined using the rapid urease test. A capped tube containing 1 ml freshly prepared reagent was used for the rapid urease test.[14]

Upper gastro-intestinal endoscopy was carried out, findings were recorded, and biopsy specimens taken from the antrum were fixed in 10% formalin for histologic examination and detection of *H. pylori* by half-Gram stain. The rapid urease test was carried out in the antral biopsy as per the method mentioned in earlier studies using the test.[15]

Serological examination for determination of *H. pylori* infection included determination of immunoglobulin G (IgG) antibody levels for *H. pylori*.[‡] A value ≥40 IU/ml was taken as positive and <40 IU/ml was taken as negative.

Data were categorized and analyzed using univariate analysis,[§] and the variables found to be significant in the univariate analysis were then analyzed by multivariate logistic regression analysis.[‖]

RESULTS

Background Characteristics and Sociodemographic Variables

Among the 134 subjects who had complete data, 65 were categorized as cases and 69 as controls. The age among cases ranged from 17 to 69 years, whereas the age among controls ranged from 15 to 76 years. The mean ± SD for cases was 41 ± 11.7 years, whereas that for controls was 39 ± 13.7 years. There were 43 males and 22 females among cases and 47 males and 22 females among controls. There was no difference in the marital status between the two groups.

Table 1.

Occupation and Level of Education of Cases and Controls

	Cases (n = 65)	Controls (n = 69)	*P* Value
Occupation			
Civil servant	9	4	0.580
Private	13	17	
Laborer	16	19	
Student	4	7	
Not employed	23	22	
Schooling (years)			
<10	15	22	0.845
10+	50	47	

The sociodemographic variables examined included occupation (Table 1), level of education (Table 1), monthly income, housing and sanitation (Tables 2 and 3), and possession of accessories (standard of living) (Table 4). The two groups were similar in terms of sociodemographic variables.

Handling of Animals

Handling of animals by the patients was also evaluated. The patients were described as either those who handle animals or those who do not handle animals. It was found that 10 of 55 cases (15.4%) were in the habit of handling animals compared to only two of 69 controls (2.9%). This difference was found to be significant with a *P* value of 0.011.

Lifestyle Variables

Among the lifestyle variables, the habit of smoking and alcohol intake were evaluated. There was no difference in the lifestyle variables between the two groups (Table 5).

Drug History

The history of medication with aspirin, acetaminophen, or other non-steroidal anti-inflammatory drugs was evaluated, and no difference was observed between the two groups in terms of drug history (Table 6).

‡ QBI MAGIWEL, United Biotech, Mountain View, CA.
§ Epi Info software, version 6, Centers for Disease Control and Prevention, Atlanta, GA.
‖ SPSS software, version 11, SPSS, Chicago, IL.

Table 2.

Housing Conditions of Cases and Controls

Variables		Cases (n = 65)	Controls (n = 69)	*P* Value
House	Owned	62 (95.4%)	65 (94.2%)	0.93
	Rented	3 (4.6%)	4 (5.8%)	
Type of house	Concrete	31 (47.7%)	34 (49.2%)	0.70
	Clay	32 (49.2%)	31 (44.9%)	
	Thatch huts	2 (3.1%)	4 (5.9%)	
Water supply	Pipe	4 (6.2%)	4 (5.9%)	0.84
	Local pump	1 (1.5%)	1 (1.4%)	
	General pump	11 (16.9%)	8 (11.6%)	
	Well	49 (75.4%)	56 (81.1%)	
Electricity supply	Present	65 (100%)	67 (97.1%)	0.50
	Absent	0	2 (2.9%)	
Type of cooking fuel	Wood	48 (73.8%)	53 (76.8%)	0.53
	Kerosene	0	1 (1.4%)	
	Gas	17 (26.2%)	15 (21.8%)	

Table 3.

Sanitary Conditions of Cases and Controls

Variables		Cases (n = 65)	Controls (n = 69)	*P* Value
Rooms in the household	<4	44 (67.7%)	45 (65.2%)	0.88
	≥4	21 (32.3%)	24 (34.8%)	
Ventilating facility	Present	62 (95.4%)	64 (92.8%)	0.78
	Absent	3 (4.6%)	5 (7.2%)	
Smoke outlet	Present	63 (96.9%)	65 (94.2%)	0.73
	Absent	2 (3.1%)	4 (5.8%)	
Method of sewage disposal	General	16 (23.2%)	19 (27.5%)	0.85
	Local	49 (76.8%)	50 (72.5%)	
	None	0	0	
Method of waste disposal	General	22 (33.8%)	22 (31.9%)	0.55
	Local	42 (64.7%)	47 (68.1%)	
	None	1 (1.5%)	0	
Type of latrine	None	2 (3.1%)	1 (1.4%)	0.51
	Open pit	0	1 (1.4%)	
	Septic tank	63 (96.9%)	67 (97.2%)	

Oral Hygiene Status

The oral hygiene status of the subjects was assessed and classified as good, fair, and poor using the oral hygiene index of Greene and Vermillion.[12] Six (9.2%) subjects among cases had good oral hygiene compared to eight (11.6%) among controls. The oral hygiene status of 25 (38.5%) subjects among cases was classified as fair compared to 33 (47.8%) among controls and 34 (52.3%) subjects among cases as poor compared to 28 (40.6%) among controls. The observed difference in the oral hygiene status between the two groups was not found to be statistically significant.

Periodontal Status

Thirty (46.2%) subjects among cases had periodontal disease compared to only 20 (29%) among controls. This difference was found to be statistically significant with an odds ratio (OR) of 2.10 (95% confidence interval: 0.96 to 4.60).

Presence of H. pylori *in Dental Plaque*

Presence of *H. pylori* in dental plaque was determined by the rapid urease test. Both groups had a large number of subjects with a positive rapid urease test for *H. pylori* in dental plaque, with a higher prevalence of *H. pylori* in dental plaque of patients with gastric *H. pylori* infection than in controls. Fifty-eight (89.2%) subjects among cases had a positive rapid urease test compared to 49 (71%) subjects among controls. This difference was found to be statistically significant with an OR of 3.10 (95% confidence interval: 1.21 to 9.77).

Multivariate Analysis

The following variables found to be of statistical significance in the univariate analysis were included in the multivariate logistic regression analysis: handling of pets, periodontal disease status, and a positive rapid urease test. In the multivariate analysis, it was found that the observed difference in the handling of pets and the presence of *H. pylori* in dental plaque between the two groups were statistically significant.

DISCUSSION

It has been reported that a high prevalence of *H. pylori* in dental plaque and on the tongue may play an

Table 4.

Standard of Living of Cases and Controls

Accessories		Cases (n = 65)	Controls (n = 69)	P Value
Radio	Yes	64 (98.5%)	68 (98.6%)	0.50
	No	1 (1.5%)	1 (1.4%)	
Refrigerator	Yes	38 (58.5%)	43 (62.3%)	0.77
	No	27 (41.5%)	26 (37.7%)	
Telephone	Yes	31 (47.7%)	39 (56.5%)	0.39
	No	34 (52.3%)	30 (43.5%)	
Scooter	Yes	41 (63.1%)	46 (66.7%)	0.79
	No	24 (36.9%)	23 (33.3%)	
Television	Yes	49 (75.4%)	52 (75.4%)	0.84
	No	16 (24.6%)	17 (24.6%)	
Electric food mixer	Yes	35 (53.8%)	33 (47.8%)	0.60
	No	30 (46.2%)	36 (52.2%)	
Car	Yes	8 (12.3%)	6 (8.7%)	0.68
	No	57 (87.7%)	63 (91.3%)	
Washing machine	Yes	7 (10.8%)	11 (15.9%)	0.53
	No	58 (89.2%)	58 (84.1%)	

Table 5.

Lifestyle Variables in Cases and Controls

Variables		Cases (n = 65)	Controls (n = 69)	χ^2	P Value
Smoking habit	Current smoker	12 (18.5%)	12 (17.4%)	2.22	0.33
	Ex-smoker	11 (16.9%)	19 (27.5%)		
	Non-smoker	42 (64.6%)	38 (55.1%)		
Alcohol consumption	Present	5 (7.7%)	4 (5.8%)	0.46	0.79
	Past	9 (13.8%)	12 (17.4%)		
	Not at all	51 (78.5%)	53 (76.8%)		

Table 6.

Drug History of Cases and Controls

Medication		Cases (n = 65)	Controls (n = 69)	χ^2	P Value
Aspirin	None	62 (95.4%)	68 (98.5%)	4.16	0.12
	<600 mg/week	3 (4.6%)	1 (1.5%)		
	≥600 mg/week	0	0		
Acetaminophen	None	46 (70.8%)	53 (76.8%)	2.02	0.36
	<650 mg/week	12 (18.5%)	13 (18.8%)		
	≥650 mg/week	7 (10.7%)	3 (4.4%)		

important role in the pathogenesis of reinfection.[9] The oral cavity may be a reservoir for *H. pylori* infection, and oral secretions may be an important means of transmission of this microorganism.[10] Some studies have also suggested that poor periodontal health characterized by deep periodontal pockets may be associated with *H. pylori* infection[16] and that there is increased detection of *H. pylori* in the oral cavity of patients with periodontal disease and other oral conditions, including aphthous ulcers[17-20] and mucosal lesions.[21-24] Despite the number of studies conducted, the evidence for the role of dental plaque and oral *H. pylori* in *H. pylori* reinfection is inconclusive.

In the present case control study, we evaluated whether periodontal disease and poor oral hygiene was a risk factor for *H. pylori* infection.

The age and gender distribution of cases and controls in the present study was comparable, indicating that there was little influence of these variables on the disease status of the two groups.

The sociodemographic variables assessed included educational status, income levels, housing conditions, standard of living, and sanitation. These variables were compared individually between the two groups by univariate analysis, and it was found that the two groups were similar in terms of the sociodemographic variables.

Among the numerous studies conducted to examine the role of dental plaque in *H. pylori* infection, very few have accounted for the sociodemographic factors within the study population.

One study based on the data from the first phase of the third National Health and Nutrition Examination Survey reported that periodontal pockets with a depth ≥5 mm were associated with increased odds of *H. pylori* seropositivity after adjustment for sociodemographic factors.[16] It has also been suggested that future investigations should account for race or ethnicity characteristics.[25] In the present study, it was found that the sociodemographic status of the two groups were comparable, indicating a minimal influence of these variables on the disease status.

Among the lifestyle variables, the habit of smoking and alcohol intake were evaluated. The data were analyzed by χ^2 test, and it was found that the difference in the smoking habit and alcohol consumption between cases and controls was not significant (P value = 0.33 and 0.79, respectively).

Handling of animals by the subjects in the two groups was also evaluated. The data were analyzed by χ^2 test, and the observed difference was found to be highly significant (P value = 0.011). Animals have been shown to harbor *H. pylori*. Studies have demonstrated the development of *H. pylori*-induced gastritis in domestic cats with features similar to those occurring in humans.[26] Based on their findings, the authors suggested that cats may act as a reservoir host for the zoonotic transmission of *H. pylori* and that further studies were required to establish this relationship. In the present study, the animals included cats, dogs, and cattle. The high degree of association between handling of animals and *H. pylori* infection observed in the present study suggests that animals may play an important role in the transmission of *H. pylori* infection and that handling of animals may be an important risk factor for developing *H. pylori* infection.

The oral hygiene status of patients was examined using the oral hygiene index of Greene and Vermillion.[12] The oral hygiene status of the patients was classified into good, fair, and poor depending on their oral hygiene scores. Among cases, 34 (52.3%) of 65 subjects had poor oral hygiene compared to only 28 of 69 subjects (40.6%) among controls. Twenty-five (38.5%) patients among cases had fair oral hygiene compared to 33 (47.8%) among controls. Six (9.2%) patients among cases had a good oral hygiene compared to eight (11.6%) among controls. However, this difference was found to be statistically insignificant (P value = 0.396). Few studies have evaluated the oral hygiene status of patients with *H. pylori* infection. Different authors have used different indices to evaluate the oral hygiene of their patients. In a study conducted on 108 subjects,[21] the subjects were evaluated and divided into three groups based on their oral hygiene status as assessed by the oral hygiene index of Greene and Vermillion.[12] These patients had been diagnosed to be positive for *H. pylori* and the associated gastric changes. Of these 108 patients, 21 (19.5%) had good oral hygiene, 51 (47.2%) had fair oral hygiene, and 36 (33.3%) had poor oral hygiene. The study examined the presence of *H. pylori* in dental plaque using a *Campylobacter*-like organism (CLO) test and reported the detection rates to be 100%, 90.2%, and 28.5% for patients with poor, fair, and good oral hygiene, respectively. In another study, the oral hygiene status of gastritis patients was evaluated using the Quigley-Hein plaque index.[9] However, the study did not report on the relationship of plaque scores with *H. pylori* infection in these patients. It has been reported that there was no correlation between *H. pylori* infection and dental hygiene as assessed by the plaque index of Silness and Löe.[10] The results obtained from the present study indicate that there is little correlation between oral hygiene status and *H. pylori* infection.

The periodontal status of the patients was examined as a dichotomous variable with patients described as being either diseased or healthy depending on their periodontal status. Among cases, 30 of 65 subjects (46.2%) had periodontal disease compared to only 20 of 69 subjects (29%) among controls. The data were analyzed by χ^2 test, and the observed difference was found to be statistically significant with a P value of 0.04, OR of 2.10%, and 95% confidence interval ranging from 0.96 <OR <4.60. This was then analyzed by logistic regression, and the difference was not found to be significant. Few studies have evaluated the relationship between gingival and periodontal status with *H. pylori* infection. In a study based on the data from the first phase the third National Health and Nutrition Examination Survey, the investigators reported that poor periodontal health, characterized by deep periodontal pockets, may be associated with *H. pylori* infection in adults in the United States.[16] They reported that periodontal pockets with a depth ≥5 mm were associated with increased odds of *H. pylori* seropositivity after adjustment of sociodemographic factors. However, other investigators have reported that there was no correlation between periodontal disease and *H. pylori* infection.[10] The results from the present study indicate that, although there were more periodontal disease subjects among cases than controls, the observed difference was not statistically significant, indicating that there is no association between periodontal disease attributes and *H. pylori* infection.

The presence of *H. pylori* in dental plaque was examined by the rapid urease test, and the results were expressed as either being positive or negative. Among cases, 58 of 65 subjects (89.2%) had a positive rapid urease test in the dental plaque compared to only 49 of 69 subjects (71%) among controls. On univariate analysis, this difference was found to be statistically significant with a P value of 0.015, OR of 3.38, and

95% confidence interval ranging from 1.21 <OR <9.77. Subsequently, logistic regression analysis was performed, and it was found that this variable had an OR of 2.5 with a *P* value of 0.068. Although the *P* value was slightly more than 0.05, the finding cannot be considered statistically insignificant as the difference in *P* value is only slight and the OR is 2.5. This marginally high *P* value may be due to the small sample size with which the logistic regression analysis was performed, and perhaps a larger sample size would have provided a more significant value. Many of the earlier studies reported a high prevalence of *H. pylori* in dental plaque, whereas some reported a very low prevalence or absence of *H. pylori* in dental plaque; however, the methods of detection varied and included the CLO test, histology, culture, and polymerase chain reaction. High prevalence of *H. pylori* in the dental plaque has been reported using the CLO test and polymerase chain reaction.[5,21,27] However, investigators have tried to detect *H. pylori* in the dental plaque samples by the culture method and reported that *H. pylori* was not cultivated from the samples investigated.[28] The failure of culture methods to detect *H. pylori* from dental plaque may be due to a variety of reasons, such low numbers of the organism or existence of non-cultivable forms in dental plaque. Other investigators employing polymerase chain reaction for the detection of *H. pylori* from subgingival plaque samples in periodontitis patients 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*.[29] In the present study, the rapid urease test was employed to detect the presence of *H. pylori* in dental plaque. Studies have reported that the rapid urease test has a specificity near 100% and sensitivity between 70% and 90%.[30,31] The sensitivity of using the rapid urease test in dental plaque to determine *H. pylori* status is reported to be 89.7%, with a diagnostic accuracy of 86.7%.[32] It has been reported that other urease positive microorganisms present in the oral cavity such as *Streptococcus vestibularis* and *Actinomyces viscosus* usually cannot give positive results within an hour.[33] The results obtained in the present study indicate that there was a high prevalence of *H. pylori* in dental plaque and that this may be associated with *H. pylori* infection.

CONCLUSIONS

From the above discussion, it is seen that *H. pylori* infection was not associated with periodontal status or oral hygiene status in the studied population. It was also found that there was a high prevalence of *H. pylori* in dental plaque, which may be associated with *H. pylori* infection. This possible association of *H. pylori* in dental plaque with *H. pylori* infection may have serious clinical implications. This is because the *H. pylori* eradication therapy commonly employed in the management of *H. pylori*-associated gastric disease has little impact on *H. pylori* residing within dental plaque. Dental plaque, being a biofilm, offers a high degree of protection to the resident microbiota from systemically administered antibiotics and host defense mechanisms. Investigators have reported that after the triple therapy regimen consisting of 1 g amoxicillin, 500 mg clarithromycin, and 20 mg omeprazole for 1 week, there was no change in urease-positive dental plaque and tongue scrapings, although the treatment was very successful in the management of chronic *H. pylori* gastritis.[9] The high prevalence of *H. pylori* in dental plaque suggests that dental plaque can harbor *H. pylori* and can possibly act as a reservoir for *H. pylori* reinfection. However, further studies using larger sample size and sensitive and specific methods of detection of *H. pylori* are required to better assess the relationship of dental plaque, oral hygiene status, and periodontal disease with *H. pylori* infection. Once these relationships are better understood, intervention strategies, including mechanical and chemical plaque control measures, can be designed to better tackle the burden of *H. pylori* infection.

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