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ORIGINAL ARTICLE

The Association Of Dental Plaque And Helicobacter Pylori Infection In Dyspeptic Patients Undergoing Endoscopy

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ABSTRACT

Objective and Background
The aim of this study was to analyze whether there is any association between dental plaque, oral hygiene and periodontal disease and Helicobacter pylori gastric infection. H.pylori, a spiral shaped microaerophilic bacterium, is responsible for peptic ulcer diseases, gastritis and gastric malignancies. Among various reports on the transmission of H. pylori, the faecal oral and oral-oral routes have been suggested to be the most plausible ones. Although it may be transmitted through the oral cavity, it is unknown whether the dental plaque acts as a permanent reservoir of H. pylori.

Methods
In this case control study, 124 dyspeptic patients with dyspepticsymptoms were categorized into the cases (60) and the controls (64) on the basis of the rapid urease test (RUT) and the histopathological results of the antral biopsy specimens. Patients with either of the tests positive or with both the tests positive were categorized as the cases and those with both the tests negative were taken as the controls.

Results
A high prevalence of H. pylori in dental plaque was found among the cases than in the controls. Among the cases, 52 patients out of the 60 (86.6%), had a positive rapid urease test in the dental plaque and among the controls, 12 out of 64 (18.75%) showed positive results. A highly significant association was found between poor oral hygiene status and periodontal disease (probing pocket depth) with H.pylori infection.

Conclusion
Triple / quadruple therapy has no effect on plaque associated H.pylori and it may continue to act as a reservoir. Plaque control measures and pocket eradication therapy are highly beneficial in eliminating and preventing the colonization of H.pylori in the oral cavity.

KEY WORDS
Helicobacter pylori, Dental plaque, Periodontal disease, Oral hygiene

KEY MESSAGES
1. The detection of H.pylori in dental plaque can be a reliable first line diagnostic approach to screen the patients with gastric complaints.
2. Poor oral hygiene and periodontal disease can be the risk factors for H.pylori associated gastric diseases.
3. Plaque control measures and periodontal therapy can be useful adjunctives to systemic antimicrobial therapy for H. pylori eradication.

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INTRODUCTION

Periodontal disease comprises a group of inflammatory conditions of the tooth supporting tissues that result from a complex interplay between the host tissues and aetiological agents. Aetiological agents are pertained to specific bacteria which are found in the dental plaque. Dental plaque is a soft deposit forming the biofilm, which is primarily a collection of microorganisms of more than 600 distinct species which is embedded in an intercellular matrix.

One gm of plaque (Wet wt) contains approximately $2x10^{11}$ bacteria [1]. Dental plaque, a host associated biofilm, provides a protective environment for colonizing organisms and fosters metabolic properties. Microorganisms which are present in the biofilm are resistant to antimicrobials because of the slower rate of growth of the bacterial species in the biofilm, thus making them less susceptible to many, but not all antimicrobials. The biofilm also acts as a barrier to antibiotic diffusion and makes the microorganisms resistant to antibiotics as compared to the planktonic bacteria [2].

The dental plaque typically adheres to the supra gingival and the subgingival tooth surfaces and it quickly forms in the absence of good oral hygiene measures. Many studies have reported the presence of the gram –ve, spiral shaped, urease +ve bacterium, H. pylori in dental plaque, saliva and the dorsum of the tongue. [3],[4]

H. pylori is a microaerophilic bacterium of about 3 μm in length and a diameter of about 0.5 μm, with 4-6 flagella and it is strongly associated with antral gastritis, duodenal ulcers, gastric adenocarcinoma and MALT lymphoma[5]. About half of the world's population is infected with H. pylori [6] and the oral-oral and the fecal-oral modes of transmission have been postulated[7]. Viable H. pylori has been isolated from faeces [8], saliva [7], dental plaque [3],[9] and various oral lesions [10]. However, the transmission and the source of this infection are still unclear. The failure of triple therapy or quadruple therapy to clear H. pylori infection from the dental plaque, despite its clearance from the gastric mucosa [11], raised the possibility that dental plaque is the potential source of re-infection of the gastric mucosa. The detection of this microorganism in the oral cavity has been reported by several groups [4],[12],[13], who demonstrated that the oral cavity acts as a potential reservoir for H.pylori or a possible route of transmission. Periodontal treatment in combination with systemic therapy, has exhibited the successful eradication of gastric H.pylori as compared to systemic therapy alone, with the decreased risk of reinfection. [14]

However, other studies have not detected H.pylori from dental plaque samples [15],[16],[17] If the oral cavity is the reservoir for H.pylori, the eradication of this bacterium from the oral cavity is necessary to prevent gastric infection. The aim of our study was to find out the association between the presence of H. pylori in dental plaque and gastric infections and also, whether oral hygiene and periodontal disease were the risk factors for the H. pylori gastric infection.

MATERIALS AND METHODS

STUDY DESIGN

This case control study comprised of 124 patients with dyspeptic symptoms of at least 6 months of duration. These patients were enrolled and subjected to an oesophago gastro duodenoscopy in the Department of Gastroenterology, Govt. Medical College, Calicut, Kerala, India. This study was approved by the Ethical Committee, Medical College, Calicut, Kerala, India. Informed consent was taken from all patients who underwent the oesophago gastro duodenoscopy. The patients were evaluated by using a detailed questionnaire. The patients were classified into 2 groups i.e., cases and controls on the basis of the rapid urease test and the histopathology results of the gastric biopsies.

Subjects with dyspepsia, undergoing endoscopy, who were either rapid urease test positive or histopathological examination positive or both, were categorized as the cases and those who are both rapid urease test and histopathological examination negative were categorized as the controls.

A total of 130 patients were taken for the study. Among these 130 patients, 6 patients did not undergo the endoscopic procedure because of some technical problems. Therefore, a complete set of data was available for these 124 patients. Data from these 124 patients were used for final analysis. Out of these patients, 60 patients were categorized as the cases and 64 were taken as the controls, depending on the above mentioned criteria.

The patients who were included in this study were of the age group ranging from 16-65 years [11], who were partially or fully dentate ($\geq$8 teeth

excluding 3rd molars). The patients had no systemic conditions modifying periodontal disease manifestations (diabetes, osteoporosis and cellular immunity disorders) and no conditions contraindicating a periodontal examination (increased risk of bacterial endocarditis). There was no history of the intake of antibiotics, H_2 receptor antagonists, bismuth compounds or proton pump inhibitors in the past 3 months and no history of any dental treatment in past 6 months, including oral prophylaxis.

A standard proforma was prepared, consisting of variables like name, age, sex, religion, address, occupation, socio demographic status, education status, diet, source of water supply, habits (smoking / alcohol / pet handling), oral hygiene practices and oral examination. Along with these variables, GIT symptoms (diagnostic criteria) were also included. These were recorded for each patient.

ORAL EXAMINATION

Dentition status, oral hygiene status (OHI-S) and probing pocket depth were assessed under proper illumination by using a mouth mirror, an explorer and William's graduated periodontal probe.

Examination of dental plaque for H.pylori by the rapid urease test:

Supragingival plaque samples were taken from tooth surfaces by using sterile gracey curettes. The plaque sample was squeezed between strips of filter papers to absorb the saliva, which, due to its alkaline pH, can give a false positive result. The dried plaque sample was placed immediately into a capped Eppendorf tube containing 0.5ml of rapid urease solution. A positive result was indicated by the change in colour of the solution from yellow to pink / magenta within the first minute. The urease activity of H.pylori increased the pH to an alkaline value. Few urease positive oral microbes like S. vestibularis and A. viscosus, can give false positive results. But these organisms cannot give positive results within an hour [18]

Gastrointestinal examination

After the oral examination, endoscopic examination was done with the help of a video endoscope. Two gastric biopsy specimens were taken for the detection of H. pylori i.e., one for RUT and the other for histopathological examination (Modified Giemsa staining technique).

Statistical analysis

The study variables were analysed by using univariate analysis and the variables which were found to be significant in the univariate analysis were further analysed by logistic regression analysis by using the SPSS software version 10*. The statistical significance for the tests was set at <0.05.

In the univariate analysis, Chi square test and the odds ratio test were used to find the association between the dental plaque and H. pylori gastric infection in dyspeptic patients and to find the relationship between the oral hygiene status of the patients with H. pylori infection. 't' test was used to find the correlation between periodontal health status and H. pylori infection in the dyspeptic patients.

Results

Age and Sex distribution

In the cases and the controls, age distribution was comparable with a mean age of 42.82 years for the cases and 42.50 years for the controls, with more number of cases in between the age group of 30-50 years. The results were statistically insignificant.[Table/Fig 1]

[Table/Fig 1]: Age distribution

Status	Mean	No.	SD	't' test value	df	P value	95% CI
Case	42.82	60	13.40	0.128	122	0.898	-4.58<CI<5.21
Control	42.50	64	14.11				

Higher levels of the positivity of H. pylori were observed in the stomach samples from the males as compared to the females. The male to female ratio in the cases and controls was approximately 2:1. [Table/Fig 2]

[Table/Fig 2]: Sex distribution

Status		No.	Chi square test	P value
Case	Male	39 (65%)	1.80	0.79
	Female	21 (35%)		
Control	Male	34 (53%)		
	Female	30 (47%)		

Socio demographic status and Pet handling

The effect of variables like income, occupation and education (socio demographic factors) was analyzed and it was found to be statistically non significant in this study. The socioeconomic status of the two groups was comparable in terms of the above variables.

In the case group, only 11.7% (7/60) were pet handlers as compared to 9.4% (6/64) in the

* SPSS software version 10

control group, which showed no statistical significance (Table 3)

Habits (Smoking and alcohol consumption)
No statistically significant association was found between H. pylori infection and smoking / alcohol use in the cases and controls by the Chi square test [Table/Fig 3].

[Table/Fig 3]: Prevalence among Smokers, Alcoholics and Pet handlers

	Smoking		Alcohol		Pet handling	
	Absent	Present	Absent	Present	Absent	Present
Case	53 (88.3%)	7 (10.9%)	56 (93.3%)	4 (6.6%)	53 (88.3%)	7 (11.7%)
Control	56 (87.5%)	8 (12.5%)	61 (93.4%)	3 (4.7%)	58 (90.6%)	6 (9.4%)
Chi square test	0.020		0.711		0.173	
P value	0.887		0.464		0.677	

Oral hygiene habits

Among the cases, 31.6% (19/60) patients used their fingers for cleaning their teeth, as compared to 14.06% patients (9/64) among the control group. 50% (30/60) of the case group patients used tooth paste as compared to 71.87% (46/60) among the control group. The results showed a relationship between H.pylori infection and oral hygiene habits. [Table/Fig 4]

[Table/Fig 4]: Oral hygiene habits

Oral hygiene		Case	Control	Chi square test	P value
Method	Finger	19 (31.6%)	9 (14.06%)	5.490	0.019
	Brush	41 (68.4%)	55 (85.93%)		
Materials	Paste	30 (50%)	46 (71.87%)	6.246	0.012
	Powder	30 (50%)	18 (28.12%)		

The prevalence of H. pylori infection and the oral hygiene status of the patients:

The results showed more prevalence of H.pylori infection in the patients (90%) with poor oral hygiene, which was statistically highly significant. [Table/Fig 5]

[Table/Fig 5]: Prevalence of H. pylori infection and Oral hygiene status of the patients

Oral hygiene groups	Case	Control	Chi square test	P value	Odds ratio	95% CI
Good	0 (0%)	0 (0%)	6.284	0.012	28.988	0.1616<CI<0.7891
Fair	6 (10%)	19 (29.68%)				
Poor	54 (90%)	45 (70.3%)				

The Oesophago gastro duodenoscopy findings:

The Oesophago gastro duodenoscopy (OGD) findings were recorded as patients with normal findings, antral gastritis and gastric ulcers / duodenal ulcers or both. These findings were analysed by the Chi square test. The observed difference between the cases and the controls was statistically significant with a p value of <0.05. [Table/Fig 6]

[Table/Fig 6]: Oesophago gastro duodenoscopy findings

OGD	Case	Control	Chi square test	P value
Normal	6 (10%)	16 (25%)	24.382	0.000
Antral gastritis	28 (46.6%)	44 (68.75%)		
GU / DU	23 (38.3%)	3 (4.68%)		
Both GU & DU	3 (5%)	1 (1.56%)		

The prevalence of gastric H.pylori infection and oral colonization by H.pylori:

86.66% of the cases (52 out of 60) showed +ve RUT / presence of H.pylori in the dental plaque. Among the controls, 12 out of 64 (18.75%) showed the presence of H.pylori in the dental plaque. A positive association was found between the presence of H.pylori in the dental plaque and the gastric infection and it was statistically significant. [Table/Fig 7]

[Table/Fig 7]: Prevalence of gastric H.pylori infection and oral colonization by H.pylori (RUT Dental plaque)

RUT dental plaque	Case	Control	Chi square test	P value	Odds ratio	95% CI
Positive	52 (86.66%)	12 (18.75%)	57.197	0.000	28.167	10.638<CI<74.576
Negative	8 (13.3%)	52 (81.25%)				

Correlation of the number of teeth present and H. pylori infection:

The results showed no statistically significant relationship between the number of teeth present and H. pylori infection. [Table/Fig 8]

[Table/Fig 8]: Correlation of number of teeth present and H. pylori infection

Status	No.	Mann-whitney U	Wilcoxon W	Z	P value
Case	60	1664	3494	-1.292	0.196
Control	64				

The prevalence of the H. pylori infection and probing depth:

The results showed a statistically significant association between the seroprevalence of H. pylori and the probing depth. [Table/Fig 9]

[Table/Fig 9]: Prevalence of H. pylori infection and probing depth

Status	No.	Mean	SD	't' test	df	P value	95% CI
Case	60	3.2043	0.3760	6.178	122	0.000	0.3263<CI<0.6341
Control	64	2.7241	0.4794				

Logistic regression analysis:

Logistic regression analysis showed that pocket depth, RUT dental plaque, OGD findings and the oral hygiene status of the patients were statistically significant. [Table/Fig 10]

[Table/Fig 10]: Logistic regression analysis

	B	SE	Wald	df	P value	Exp (B)
Mean pocket depth	-2.405	487	24.401	1	0.000	0.090
Constant	7.193	1.460	24.264	1	0.000	1330.351

	B	SE	Wald	df	P value	Exp (B)
OGD	-1.398	458	9.319	1	0.002	0.247
RUT	-3.243	0.529	37.624	1	0.000	0.039
Constant	4.676	1.056	19.605	1	0.000	107.382

	B	SE	Wald	df	P value	Exp (B)
Oral hygiene	-0.611	0.215	8.046	1	0.005	0.543
Constant	2.403	0.849	8.006	1	0.005	11.061

DISCUSSION

Dental plaque is a host associated biofilm of incredibly complex communities of microorganisms that lead to periodontal disease and also play a role in affecting systemic health [19]. Recent studies have shown that the H. pylori bacterium which is associated with chronic gastritis, peptic ulcer disease and gastric malignancies, is also present in the dental plaque biofilm. Thus, the dental plaque biofilm may act as a reservoir for H.pylori [20].

Among the various reports on the transmission of H. pylori [7], the faecal oral and the oral-oral routes have been suggested to be the most plausible ones. Few reports have suggested that H. pylori infections are associated with heart disease [21]. The association between H. pylori and oral mucosal lesions and Halitosis also have been reported by recent studies [10],[22]

From the literature, it has been seen that various studies have detected H.pylori in dental plaque and have found the association between dental plaque and H.pylori gastric infection [23],[24],[25].

In contrast to these studies, certain studies have shown that dental plaque may not be a relevant reservoir of H. pylori [17],[26].

Despite the presence of a large number of studies, the role of dental plaque in the transmission of H.pylori induced gastric infections is still controversial.

In the present case control study, we found that the patients were in the age group between 30-50 years. The age distribution of the cases and controls were comparable with the mean age for the cases (42.82 years) and that for the controls (42.50 years). Higher levels of positivity of H. pylori were observed in stomach samples from males as compared to the females (approximately 2:1).

The effect of variables like income, occupation and education (socioeconomic factors) was analyzed and it was found to be statistically non significant in this study. This is in agreement with the observations of a study done by Berroteron et al [27] (2002).

H. pylori infection, smoking and alcohol consumption are risk factors for acid peptic disorders. However, in the present study, no statistically significant association was found between H. pylori infection and smoking / alcohol use in the cases and the controls by the Chi square test.

Similar results have been reported by Hardo et al [28] (1995) who demonstrated that smoking was not associated with a higher rate of infection.

Pet handling by the patients was also evaluated. The patients were divided into two groups; those who handled pets and those who did not. The data were analysed by the Chi square test. In the case group, only 11.7% (7/60) were pet handlers, as compared to 9.4% (6/64) in the control group, which showed no statistical significance.

The Oesophago gastro duodenoscopy (OGD) findings were recorded as patients with normal findings, antral gastritis, andgastric ulcers / duodenal ulcers or both. These findings were analysed by the Chi square test. The observed difference between the cases and the controls was statistically significant, with a p value of <0.05.

The oral hygiene habits in the cases and controls were evaluated by recording the oral hygiene method (finger / brush), frequency (once / twice), timing (morning / night / both) and the material used (paste / powder / charcoal). The data were analysed by the Chi square test. Among the cases, 31.6% (19/60) patients used their fingers for cleaning teeth as compared to 14.06% patients (9/64) among the control group. 50% (30/60) of case group patients used tooth paste as compared to 71.87% (46/60) in the control group. The frequency of tooth brushing and timing had no statistical significance. But the results showed that a positive relationship existed between the

prevalence of H. pylori infection and the oral hygiene technique and the material used. Evidently, the oral hygiene status of the patients was found to be influenced by the brushing technique and the material which was used for tooth brushing.

The oral hygiene status of the patients was examined by using a simplified Greene and Vermillion oral hygiene index. The patients were categorized into good, fair and poor oral hygiene, based on their oral hygiene score. 90% of the cases had poor oral hygiene. The statistical analysis showed a significant relationship between poor oral hygiene and H. pylori infection. H. pylori positivity in the dental plaque was related to the oral hygiene index score. Similar results have been shown by studies done by Avcu et al [29] (2001) and Bruce A Dye et al [30] (2002).

In contrast, fewer studies have found that there is no significant association between H.pylori colonization in the dental plaque and gastric infection [27],[31].

The presence of H. pylori in the dental plaque was examined by the rapid urease test and the results were scored as positive (H. pylori present) or negative (H. pylori absent). Out of the 124 dyspeptic patients, 64 patients (51.6%) showed a positive rapid urease test, thus indicating the presence of H.pylori in the dental plaque. Statistical analyses have shown that there is a positive association between the presence of H. pylori in the dental plaque and gastric infection. From the literature, it has been seen that many studies have evaluated the association between the H.pylori gastric infection and the dental plaque. Our results are in accordance with the results of these studies [11],[23],[32]. The CLO test to detect H. pylori in the dental plaque would thus be a reliable first line diagnostic approach for the gastric H. pylori infection [23].

On the other hand, there are also reports suggesting the absence or lower prevalence of H. pylori in the dental plaque of dyspeptic patients with gastric H. pylori infections [15],[16],[33].

In the present study, we also compared the association of the number of teeth present and H. pylori infection by using the non parametric Mann-Whitney test. The results showed no significant association with respect to the number of natural teeth present and H. pylori gastric infection. These results were in accordance with the studies done by other authors [34],[35].

The probing pocket depth in patients was examined by using William's graduated periodontal probe on 4 sites of the teeth (buccal / labial, lingual / palatal, mesial and distal) and the results were analysed by univariate analysis ('t' test) and logistic regression analysis. The mean probing depth in the cases was 3.20mm, as compared to 2.72mm among the controls and the results showed a statistical significant relationship, with p value of <0.05 by using 't' test. This was further analysed by logistic regression analysis and it was found that the variables had p value of <0.05 and that this was statistically significant. This difference in probing pocket depth among the cases and the controls may be due to the poor oral hygiene status in the cases.

Previous studies also have reported that poor periodontal health characterized by advanced periodontal pockets may be associated with H. pylori infections [30],[36],[37].

But few studies have found no association between periodontal pocket depth and the H.pylori infection [12],[27]

In the present study, we observed that the results shown by the RUT dental plaque examination were comparable with the results shown by the histopathological examination of the gastric biopsies. The clinical implication is that the detection of H.pylori in the dental plaque can be a reliable first line diagnostic approach to screen the patients with gastric complaints before going ahead with endoscopic biopsies.

Regarding the limitation of the rapid urease test to detect the presence of H. pylori in the dental plaque, it has been noted that there is a chance for false positive results. This may be due to other urease positive oral microbial species like S. vestibularis and A. viscosus. Even though these organisms cannot give positive results within an hour as given by H. pylori, further evaluation is required for the confirmation of this finding. To rectify this limitation, more specific and sensitive diagnostic tests like PCR and culture methods are needed.

Conclusion

In our study, we found that there is a statistically significant association between Helicobacter pylori in the dental plaque and Helicobacter pylori associated gastric diseases. Dental plaque, the biofilm may act as a reservoir of Helicobacter

pylori reinfection after successful antibiotic therapy. The maintenance of good oral hygiene, plaque control measures and pocket eradication therapy may play effective roles in the successful management of Helicobacter pylori associated gastric diseases.

Long term studies with a larger sample size are required to assess the efficacy of the plaque control programme on Helicobacter pylori associated gastric diseases.

Acknowledgements

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No.	NAME OF STUDY	AUTHORS' NAME	NO. OF PATIENTS	YEAR	CONCLUSION OF STUD
1.	Identification of Helicobacter pylori DNA in the mouths and stomachs of patients with gastritis using PCR	N P Mapstone, D A Lynch, F A Lewis, A T Axon, D S Tompkins, M F Dixon, P Quirke	13	1993	Positive correlati of H. pylori organism in oral samples with recurrent infectio after eradication bacteria from stomach.
2.	Use of the Polymerase Chain Reaction to Detect Helicobacter Pylori in the Dental Plaque of Healthy and Symptomatic Individuals	N. Banatvala, C. Romero Lopez, R. J. Owen, A. Hurtado, Y. Abdi, G. R. Da Vies, J. M. Hardie and R. A. Feldman	54	1994	H. pylori is prese in dental plaque high frequency a PCR of dental plaque may provi a strategy for studying transmission of H pylori.
3	High prevalence of Helicobacter pylori in saliva demonstrated by a novel PCR assay.	C Li, P R Musich, T Ha, D A Ferguson, Jr, N R Patel, D S Chi, E Thomas	40	1995	Oral cavity reservoirs of H. pylori may serve a potential source transmission and infection in dyspeptic patient
4	A newly developed PCR assay ofH. pylori in gastric biopsy, saliva, and feces Evidence of high prevalence ofH. pylori in saliva supports oral transmission	Chuanfu Li, Tuanzhu Ha, Donald A. Ferguson, David S. Chi, Rongguo Zhao, Nikihil R. Patel, Guha Krishnaswamy and Eapen Thomas	88	1996	Greater prevalen of H. pylori in sa than fecal sample Fecal – oral route may be an impor means of transmission in developing countries.
5	Identification by PCR of Helicobacter pylori in subgingival plaque of adult periodontitis patients	M. P. RIGGIO and A. LENNON	29	1998	Sub gingival plaq may act as a potential reservoi for H. pylori infection
6	Absence of Helicobacter pylori in Dental Plaque Determined by Immunoperoxidase	Enrico Savoldi, Maria Grazia Marinone, Riccardo Negrini, Daniela Facchinetti, Alberto Lanzini, Pier Luigi Sapelli	80	1998	H. pylori is not usually present in dental plaque and oral-oral transmission of t infection could b due to intermitten esophageal reflux only.

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