

Is the presence of *Helicobacter pylori* in the dental plaque of patients with chronic periodontitis a risk factor for gastric infection?

Mohammed Al Asqah BDS¹, Nawaf Al Hamoudi BDS¹, Sukumaran Anil BDS PhD¹,
Abdulrahman Al jebreen MD FRCPC², Waleed Khalid Al-hamoudi MD²

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BACKGROUND: *Helicobacter pylori* is considered to be a pathogen responsible for gastritis and peptic ulcers, and a risk factor for gastric cancer. A periodontal pocket in the teeth of individuals with chronic periodontitis may function as a reservoir for *H. pylori*.

OBJECTIVE: The present study was undertaken to evaluate whether the presence of *H. pylori* in the dental plaque of patients with and without periodontitis correlates with gastric involvement.

METHODS: A total of 101 patients with dyspepsia were included in the present study. Subjects were divided into periodontitis and non-periodontitis groups. For the detection of *H. pylori* in dental plaque, samples were collected from two teeth using a periodontal curette. Subgingival plaque was obtained by inserting two sterile paper points into periodontal pockets for 20 s. This was followed by an upper gastrointestinal endoscopy and antral biopsies.

RESULTS: Sixty-five per cent of patients had dental plaque positive for *H. pylori* and more than 50% harboured the bacteria in their stomach. Periodontitis patients had a significantly higher percentage of *H. pylori* in their dental plaque (79% versus 43%; $P < 0.05$) and the stomach (60% versus 33%; $P < 0.05$) than patients with no periodontitis. Additionally, 78% of patients from the periodontitis group versus only 30% from the nonperiodontitis group had a positive test result for the coexistence of *H. pylori* in both dental plaque and the stomach.

CONCLUSION: Patients with poor oral hygiene have a higher prevalence of *H. pylori* in dental plaque and in the stomach. This finding suggests that the oral cavity may be a reservoir for *H. pylori*, and potentially a source of transmission or reinfection.

Key Words: *Helicobacter pylori*; Oral hygiene; Periodontitis

Helicobacter pylori is a microaerophilic, Gram-negative, spiral and mobile bacterium that is believed to be one of the major factors responsible for gastritis, gastroduodenal ulcers and gastric cancer (1,2). Studies have identified the microorganism in dental plaque and saliva, implicating the oral cavity as a potential reservoir for *H. pylori* or as a possible route of transmission to other sites. The oral cavity can act as an extragastric reservoir for *H. pylori*, which may lead to recurrent gastric infection (3).

Avcu et al (4) observed that patients with poor oral hygiene were most likely to have *H. pylori* in the oral cavity. They also suggested that *H. pylori* could recur in the stomach of these patients after triple therapy more frequently than in patients

La présence d'*Helicobacter pylori* dans la plaque dentaire de patients souffrant de parodontite chronique est-elle un facteur de risque de gastrite?

HISTORIQUE : *Helicobacter pylori* est considéré comme l'un des agents pathogènes responsables de la gastrite et de l'ulcère gastroduodénal et comme un facteur de risque à l'égard du cancer de l'estomac. Chez les personnes souffrant de parodontite chronique, les poches parodontales pourraient constituer des réservoirs d'*H. pylori*.

OBJECTIF : La présente étude a été entreprise afin de vérifier si la présence d'*H. pylori* dans la plaque dentaire de patients souffrant ou non de parodontite est en corrélation avec une atteinte gastrique.

MÉTHODES : En tout, 101 patients souffrant de dyspepsie ont participé à l'étude. Les sujets ont été répartis en deux groupes selon qu'ils souffraient ou non de parodontite. Pour le dépistage d'*H. pylori* dans la plaque dentaire, des échantillons ont été prélevés sur deux dents au moyen d'une curette parodontale. Les échantillons de plaque sous-gingivale ont été obtenus par l'insertion de deux coins de papier stérile dans des poches parodontales pendant 20 s. Cette étape a été suivie d'une endoscopie des voies digestives hautes pour prélèvement de biopsies antrales.

RÉSULTATS : Soixante-cinq pour cent des patients présentaient une plaque dentaire *H. pylori*-positive et plus de 50 % hébergeaient la bactérie dans leur estomac. Les patients atteints de parodontite présentaient un pourcentage significativement plus élevé d'*H. pylori* au niveau de la plaque dentaire (79 % vs 43 %; $P < 0,05$) et de l'estomac (60 % vs 33 %; $P < 0,005$), comparativement aux patients indemnes de parodontite. De plus, 78 % des patients du groupe souffrant de parodontite, contre 30 % seulement des patients indemnes, avaient un résultat positif pour ce qui est de la coexistence d'*H. pylori* au niveau de la plaque dentaire et de l'estomac.

CONCLUSION : On note une prévalence plus élevée d'*H. pylori* dans la plaque dentaire et l'estomac des patients dont l'hygiène dentaire laisse à désirer. Cette observation donne à penser que la cavité buccale pourrait constituer un réservoir d'*H. pylori* et devenir une source potentielle de transmission ou de réinfection.

with good oral hygiene. The findings of Andersen et al (5) justify the suggestion that dental plaque serves as a reservoir for this pathogen. They demonstrated the ability of *H. pylori* to coaggregate with *Fusobacterium nucleatum* and *Fusobacterium periodonticum*, which are early and late colonizers, respectively, of the mouth.

Dye et al (6) found a positive association between *H. pylori* seropositivity and periodontitis in an epidemiological study. They suggested that periodontitis can be a risk factor for *H. pylori* infection. Umeda et al (7) 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. In contrast, other studies (8-10) failed

¹College of Dentistry; ²Department of Gastroenterology, College of Medicine, King Saud University, Riyadh, Saudi Arabia

Correspondence: Dr Waleed Khalid Al-hamoudi, College of Medicine, King Saud University, University Road, Riyadh, Central province 10774, Saudi Arabia. Telephone 966-505-240588, fax 966-146-71217, e-mail walhamoudi@gmail.com

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TABLE 1
Prevalence of periodontitis by sex

	Men	Women	Total
Periodontitis group	35 (56)	27 (44)	62 (61)
Nonperiodontitis group	21 (54)	18 (46)	39 (39)
Total	56 (55)	45 (45)	101 (100)

Data presented as n (%)

TABLE 2
Prevalence of *Helicobacter pylori* in the dental plaque of patients with and without periodontitis

	<i>H pylori</i> in dental plaque		
	Present	Absent	Total
Periodontitis group	49* (79)	13 (21)	62 (61)
Nonperiodontitis group	17 (43)	22 (57)	39 (39)
Total	66 (65)	35 (35)	101 (100)

Data presented as n (%). *P<0.05

to detect *H pylori* in the subgingival plaque of patients with a gastric infection.

Studies regarding the prevalence of *H pylori* in dental plaque, and its correlation with systemic conditions are inconclusive. The present study is one of the very few conducted to evaluate the prevalence of *H pylori* in both dental plaque and the gastric mucosa of symptomatic patients admitted to a gastroenterology department.

METHODS

One hundred one patients attending the gastroenterology unit of the King Saud University Hospital, Riyadh, Saudi Arabia, were asked to participate in the study. The purpose of the study was explained to the patients and consents were obtained. The study was approved by the hospital's ethics committee. All patients had a complaint of dyspepsia and underwent gastroscopy and antral biopsies. Biopsy samples were analyzed immediately using a rapid urease test kit.

All selected patients were nonsmokers, and the following exclusion criteria were used to achieve a more homogenous study population: diabetes, pregnancy, HIV-positive, previous treatment of a peptic ulcer, cancer, antibiotic therapy during the previous two months, periodontal therapy within the past year, signs of severe periodontal infection, severe dental caries and the recent use of nonsteroidal anti-inflammatory drugs.

Based on periodontal status, subjects were divided into two groups (with and without chronic periodontitis). Only patients exhibiting bleeding on probing and at least four teeth with a probing depth of 3 mm or more were assigned to the chronic periodontitis group (n=62). The remainder of the individuals were considered healthy oral cavity subjects (n=39). The probing depth, plaque index, bleeding on probing and clinical attachment level were recorded by the same investigator.

For the detection of *H pylori* in dental plaque, samples were collected from the oral cavity after the clinical periodontal examination. A sterile universal curette was used to collect plaque samples from two teeth with deep periodontal pockets.

The rapid urease test

H pylori was detected by a commercially available rapid gastric urease test kit (Lencomm Trade International, Poland). The samples were immediately inoculated into the rapid urease

TABLE 3
Prevalence of *Helicobacter pylori* in the stomach of patients with and without periodontitis

	<i>H pylori</i> in stomach		Total
	Present	Absent	
Periodontitis group	37* (60)	25 (40)	62 (61)
Nonperiodontitis group	13 (33)	26 (67)	39 (39)
Total	50 (50)	51 (50)	101 (100)

Data presented as n (%). *P<0.05

test gel. If the test gel colour changed from yellow to red within 20 min, up to a maximum of 60 min (as recommended by the manufacturer), the sample was considered positive for *H pylori*.

Statistical analysis

Statistical analysis of data was performed with GraphPad software (InStat, USA). The χ^2 test was used to analyze the association between the detection frequencies of *H pylori* in the dental plaque of patients with and without periodontitis, and the presence or absence of *H pylori* in the stomach. P<0.05 was considered statistically significant.

RESULTS

A total of 101 subjects with a mean (\pm SD) age of 40.77 ± 14.15 years consented and were selected for the study. Of these patients, 56 were men and 45 were women. All patients were subjected to periodontal examination. Sixty-two participants (61%) were diagnosed with periodontitis and 39 (39%) had healthy oral cavities (Table 1).

The prevalence of *H pylori* in the dental plaque of the study cohort was 65% (Table 2). Patients with periodontitis had a significantly higher rate of *H pylori*-positive test results (79%), compared with only 43% of patients without periodontitis (P<0.05).

Among all study participants, 50 (50%) harboured *H pylori* in the stomach (Table 3). The periodontitis subjects had a statistically higher prevalence (60%) of *H pylori* in the stomach than the nonperiodontitis group (33%). Table 4 shows the prevalence of *H pylori* in the dental plaque of patients with and without periodontitis who harboured *H pylori* in the stomach. Twenty-nine (78%) of 37 patients with periodontitis harboured *H pylori* in their plaque samples. However, only four (30%) of 13 subjects without periodontitis harboured *H pylori* in their dental plaque, a difference that was statistically significant (P<0.05).

DISCUSSION

The oral cavity provides a gateway between the external environment and the gastrointestinal tract, and it facilitates both food ingestion and digestion. Poor oral hygiene and tooth loss can potentially affect gastrointestinal flora and nutritional status, and consequently be implicated in the development of chronic gastrointestinal diseases (11). Poor dental health, tooth loss or both, have been associated with an increased risk for gastrointestinal malignancies including oral, esophageal and gastric cancers (12-16). Several studies (4,17-19) have suggested that dental plaque is a reservoir for *H pylori*. The presence of *H pylori* has been universally associated with chronic gastritis and strongly associated with duodenal ulcers. Studies have also identified the microorganism in dental plaque and saliva, implicating the oral cavity as a potential reservoir for *H pylori*, or as a possible route of transmission to other sites.

Riggio and Lennon (20) 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*. In a national survey of 4504 subjects, Dye et al (6) observed that nearly 41% of individuals with periodontitis were *H pylori* seropositive. Moreover, *H pylori* was detected in more than 50% of patients with periodontitis who harboured *H pylori* in their stomach (7). The results of the present study are in agreement with previous studies. Fifty per cent of the study subjects with periodontitis were positive for *H pylori* in dental plaque. Also, among the subjects who were positive for *H pylori* in the stomach, 74% had chronic periodontitis.

Conflicting reports exist in the literature regarding the presence of *H pylori* in the oral cavity and its relation to its presence in the stomach (6,8,9). Asikainen et al (8) were not able to detect *H pylori* in the subgingival plaque of 336 patients with periodontitis, although no analysis for the presence of this bacteria in the stomach of those patients was performed. In another study, Oshowo et al (9) failed to demonstrate any correlation between the presence of *H pylori* in the stomach and dental plaque, and suggested that the oral cavity may not be a reservoir for reinfection. These conflicting observations could be attributed to the diversity of the populations studied, sample collection methods or the specificity of the methods used for the detection of *H pylori*.

Although several studies investigated the prevalence of *H pylori* in the dental plaque of patients with and without

TABLE 4
Prevalence of *Helicobacter pylori* in the dental plaque of patients who harboured *H pylori* in the stomach

	<i>H pylori</i> in dental plaque		Total
	Present	Absent	
Periodontitis group	29* (78)	8 (22)	37 (74)
Nonperiodontitis group	4 (30)	9 (70)	13 (26)
Total	33 (66)	17 (34)	50 (100)

Data presented as n (%). *P<0.05

periodontitis, most did not investigate the simultaneous existence of the bacterium in the stomach (6). In the present study, the presence of *H pylori* was examined in both dental plaque and the gastric mucosa of symptomatic patients presenting to a gastroenterology clinic. A higher number of subjects with periodontitis had a coexistent presence of the bacterium in the stomach and in dental plaque compared with nonperiodontitis patients. The significantly higher prevalence of *H pylori* in the periodontitis group may be related to poor oral hygiene and the presence of periodontal pockets. Miyabayashi et al (3) suggested that *H pylori* in dental plaque may be a risk factor for recurrent gastric infection. The results of the current study support this suggestion and highlight the importance of the oral cavity and dental plaque as reservoirs for *H pylori* as a potential source for reinfection after eradication treatment. A combination of professional medical and dental treatment modalities may be required for individuals with *H pylori* infection.

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