Review Article

Influence of *Helicobacter pylori* Infection on Periodontitis: A Meta-Analysis

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Abstract

Background: *Helicobacter pylori* detection in dental plaque, saliva, or oral mucosa would allow to include this bacteria as a member of the normal oral microbiota and have an etiological role in periodontitis. **Objective:** The objective of this study is to assess the possible relationship of *H. pylori* with periodontitis. **Methods:** A search for studies on *H. pylori* and periodontitis was conducted in the following databases: PubMed (MEDLINE, Cochrane Library), Web of Science, and Scopus. The pooled prevalence was calculated according to DerSimonian and Laird method. For dichotomous outcomes, the estimates of effects of the intervention were expressed as odds ratios using Mantel–Haenszel method, and for continuous outcomes, the estimates of effects of the intervention were expressed as mean differences using the inverse-variance method, all with 95% confidence intervals. **Results:** Twenty-six studies with 4072 participants were included in this meta-analysis. Among periodontitis patients, the estimated prevalence of *H. pylori* detection was 47.93%. Periodontitis patients had 1.89 times more likely to be infected with *H. pylori* ($P < 0.001$) and also, an increase of 2.78 times the probability of *H. pylori* detection on dental plaque ($P < 0.001$). Similarly, *H. pylori* was 2.32 times more likely in the oral microbiota than in the gastric one ($P < 0.001$). Some periodontal parameters (plaque index, probing depth, and clinical attachment level) were significantly worse in positive *H. pylori* patients. **Conclusions:** *H. pylori* infection seems to aggravate periodontitis.

Keywords: *Helicobacter infections, Helicobacter pylori, periodontitis, risk factors*

Introduction

Periodontal disease comprises a group of very frequent inflammatory disorders that involve the supporting tissues of the teeth and are the most important cause of tooth loss in the adult population. The disease arises from oral microbial biofilms that lead to periodontal infection and ultimately destruction of the supporting periodontal tissues.[1] The main periodontal pathogens are *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis, Tannerella forsythia*, *Treponema denticola*, *Fusobacterium nucleatum,* or *Prevotella intermedia*. The imbalance of the oral microbiota (dysbiosis) contributes to the development of both oral and periodontal diseases and other systemic diseases.[2]

Helicobacter pylori is a Gram-negative, helical rod-shaped bacterium commonly resident in the gastric mucosa. This bacterium has been recognized as the main risk factor for chronic gastritis that can evolve into a gastroduodenal ulcer or premalignant and malignant gastric lesions. For this reason, *H. pylori* has been classified as a Group I carcinogen by the International Agency for Research on Cancer.[3] *H. pylori* infection has previously been related to various oral diseases such as oral lichen planus or recurrent aphthous stomatitis.[4] *H. pylori* detection in dental plaque, saliva, oral mucosa, and other parts of the oral cavity raises the question of whether this bacterium can be included as a member of the normal oral microbiota, whether it can cause periodontitis or play a role in its progression.[5]

This study aimed to assess the possible relationship of *H. pylori* bacteria with periodontitis.

Methods

Search strategy

A search for studies on *H. pylori* and periodontitis up to March 2022 was

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conducted in three databases: PubMed (MEDLINE, Cochrane Library), Web of Science (WoS), and Scopus. Search strategies included a combination of the Medical Subject Headings (MeSH) and free-text terms. The search terms were as follows: "Helicobacter pylori" [MeSH Terms] AND ("periodontitis" [MeSH Terms] OR "periodontal diseases" [MeSH Terms]); "Helicobacter pylori" AND "periodontitis"; TITLE-ABS-KEY ("Helicobacter pylori" ["periodontal" OR "periodontitis"]).

The inclusion criteria were as follows: (a) retrospective studies, (b) studies with well-defined *H. pylori* detection diagnostic methods, and (c) articles written in any language and with no restrictions on publication date. The exclusion criteria were: (a) articles with no full-text availability, (b) articles with a relevant risk of bias (score <7 stars on the Newcastle–Ottawa methodological quality assessment scale),[6] (c) articles without clinical data, and (d) studies with unusable data.

Statistic analysis

Data were processed with the statistical programs RevMan version 5.4 (The Cochrane Collaboration, Oxford, UK) and MedCalc statistical software version 20.027 (MedCalc Software Ltd. Ostend, Belgium). The pooled prevalence was calculated according to the DerSimonian and Laird method. For dichotomous outcomes, the odds ratio (OR) with the Mantel–Haenszel Chi-square formula, and for continuous outcomes, the inverse variance for the mean difference (MD) was used, all with 95% confidence intervals (95% CI). Heterogeneity was determined according to the Higgins statistic (I^2) values. In cases of high heterogeneity (I^2 >50%), the random effects model was applied. A P value below 0.05 was considered the minimum level of significance.

Results

Study selection

In the initial search, 369 articles (89 in PubMed, 130 in WoS, and 150 in Scopus) were found between the years 1994 and 2021, 135 of them duplicates, remaining 234 articles for eligibility. Two hundred and eight articles were excluded for: (a) articles with no full-text availability (n = 42), (b) articles with a relevant risk of bias (<7 stars) according to the Newcastle–Ottawa scale (n = 60), (c) articles without clinical data (n = 51), and (d) studies with unusable data (n = 55). Finally, 26 studies were considered in this meta-analysis [Figure 1].

Prevalence of *Helicobacter pylori* in periodontitis patients

Table 1 shows the 26 studies[7-32] that analyze *H. pylori* detection in periodontitis patients. The pooled prevalence of *H. pylori* in periodontitis patients was 47.93% (95% CI: 39.65%–56.26%). The variability by studies ranged from the minimum of 8% (95% CI: 2.22%–19.23%)[31] to the maximum prevalence of 87.5% (95% CI: 71.01%–96.48%).[24]

Detection of *Helicobacter pylori* in periodontitis patients and controls

The evaluation of *H. pylori* detection in patients with and without periodontitis is shown in Figure 2. Sixteen studies[8,9,13,15,17,19-21,23-26,28-30,32] assessed the *H. pylori* detection in the two groups [Figure 2a]. Periodontitis patients were 1.89 times more likely to be infected by *H. pylori* than controls, with a highly statistically significant relationship (OR = 1.89; 95% CI: 1.47–2.44; P < 0.001).

Seven studies[10,16,18,24,26,28,31] examined the detection of *H. pylori* in dental plaque of periodontitis patients and controls [Figure 2b]. Periodontitis patients had a 2.78 times more probability of *H. pylori* detection in dental plaque compared to controls, found a highly statistically significant association (OR = 2.78; 95% CI: 2.06–3.76: P < 0.001).

Detection of *Helicobacter pylori* and periodontal status

Table 2 presents the evaluation of other parameters related to *H. pylori* detection and the periodontal status. Four studies[12,16,18,24] focused on *H. pylori* detection in the oral cavity and the stomach in periodontitis patients. These patients increased 2.32-fold the probability of *H. pylori* detection in the oral cavity concerning the stomach and observed highly statistically significant differences (OR = 2.32; 95% CI: 1.69–3.20; P < 0.001).

Three studies[14,27,33] determined the plaque index (PI) in subjects with and without *H. pylori* infection. *H. pylori*-infected patients had a PI, 0.27 units higher than that of the subjects not infected by the bacteria, showing a highly statistically significant relationship (MD = 0.27; CI%: 0.21–0.32; P < 0.001).

Four studies[14,27,33,34] analyzed probing depth (PD), finding a PD of 0.67 mm higher in *H. pylori*-positive patients against *H. pylori*-negative patients. A statistically significant association between PD and *H. pylori* detection was found (MD = 0.67; 95% CI: 0.22–1.33; P < 0.01).

Three studies[14,27,33] assessed clinical attachment loss (CAL) according to *H. pylori* detection. *H. pylori*-positive patients had a CAL of 0.83 mm. Greater than *H. pylori*-negative patients, with highly statistically significant differences (MD = 0.83; 95% CI: 0.38–1.28; P < 0.001).

Discussion

Data from 26 studies on the possible relationship between *H. pylori* and periodontitis have been included in the present meta-analysis.

In this study, the pooled prevalence of periodontitis patients infected by *H. pylori* was 47.93%. This high prevalence of *H. pylori* in these patients would be justified by its ability to cooperate with some periodontal pathogens such as

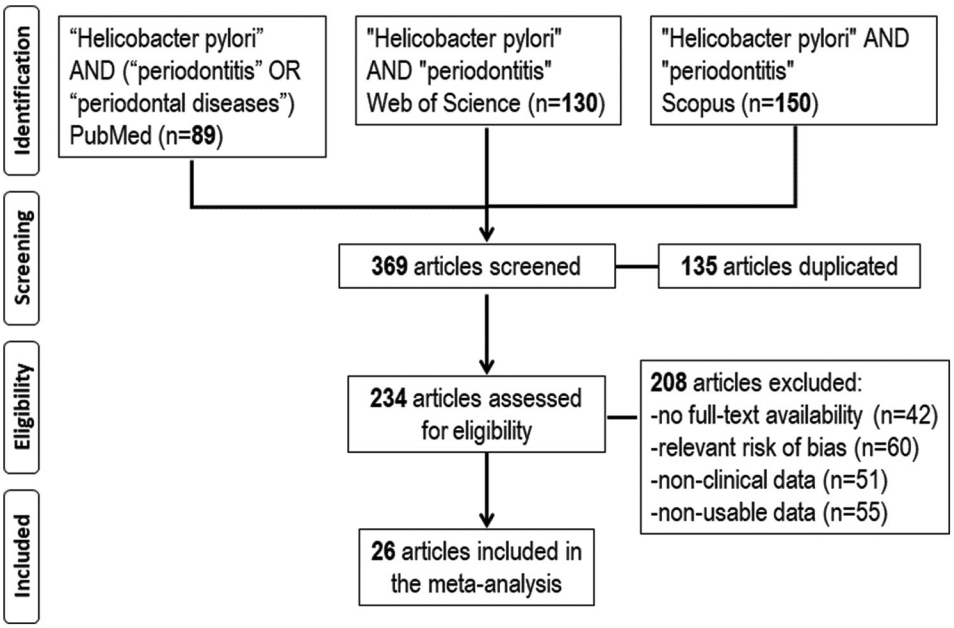

Figure 1: Flow diagram of study selection

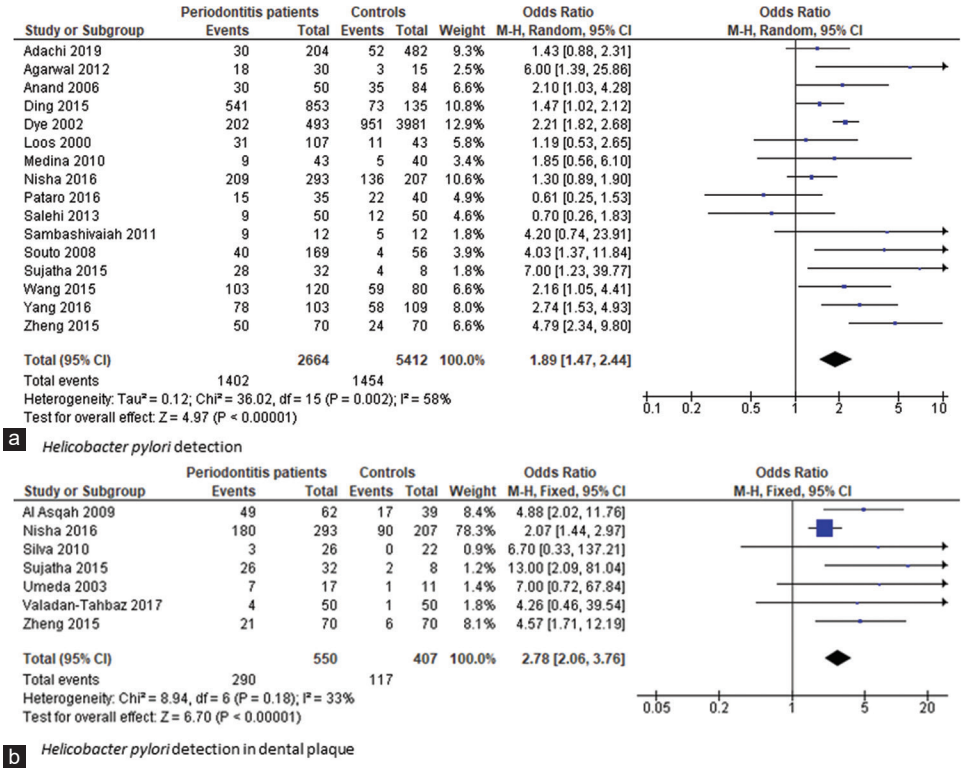

Figure 2: Study data and forest plot graphs for the *Helicobacter pylori* detection (a) and detection in dental plaque (b) in periodontitis patients and controls without the disease

F. nucleatum and *Fusobacterium periodonticum*. These periodontal bacteria are early and late oral colonizers among periodontitis patients, justifying that dental plaque could serve as a reservoir for *H. pylori*. Nevertheless, the presence of *H. pylori* in dental plaque may be intermittent, perhaps as a result of gastroesophageal reflux. The main extragastric reservoir for *H. pylori* is the oral cavity, both dental plaque and saliva could perform as reservoirs of this bacterium who is implicated in the processes of infection and reinfection.[24]

In the present study, periodontitis patients were more likely to be infected by *H. pylori* than controls, with a highly statistically significant relationship ($P < 0.001$). Of the 16 studies that analyzed this variable, 14 of

Table 1: Pooled prevalence (and 95% confidence interval) of *Helicobacter pylori* detection in periodontitis patients

Study, year	Country	Study type	*H. pylori* detection method	*n/N*	Prevalence (%)	95% CI
Riggio and Lennon, 1999[7]	UK	CS	PCR	29/73	39.72	28.45-51.86
Loos *et al*., 2000[8]	The Netherlands	C-C	ELISA	31/107	28.97	20.60-38.53
Dye *et al*., 2002[9]	USA	C-C	ELISA	202/493	40.97	36.59-45.46
Umeda *et al*., 2003[10]	Japan	C-C	PCR	7/17	41.17	18.44-67.07
Gebara *et al*., 2006[11]	Brazil	CS	PCR	13/30	43.33	25.46-62.57
Czesnikiewicz-Guzik *et al*., 2005[12]	Poland	CS	Culture	48/100	48.00	37.90-58.22
Anand *et al*., 2006[13]	India	C-C	RUT	18/30	60.00	45.17-73.59
Teoman *et al*., 2007[14]	Turkey	CS	PCR	27/67	40.29	28.48-52.99
Souto and Colombo, 2008[15]	Brazil	C-C	PCR	40/169	23.66	17.48-30.81
Al Asqah *et al*., 2009[16]	Saudi Arabia	C-C	RUT	30/50	79.03	66.81-88.33
Medina *et al*., 2010[17]	Argentina	C-C	PCR	9/43	20.93	10.04-36.04
Silva *et al*., 2010[18]	Brazil	C-C	PCR	3/26	11.53	2.44-30.15
Sambashivaiah *et al*., 2011[19]	India	C-C	RUT	9/50	75.00	42.81-94.51
Agarwal and Jithendra, 2012[20]	India	C-C	PCR	49/62	60.00	40.60-77.34
Salehi *et al*., 2013[21]	Iran	C-C	PCR	9/12	18.00	8.57-31.43
Zheng *et al*., 2014[22]	China	CS	RUT	430/969	44.37	41.21-47.56
Ding *et al*., 2015[23]	China	C-C	INMC	541/853	63.42	60.09-66.66
Sujatha *et al*., 2015[24]	India	C-C	RUT	28/32	87.50	71.01-96.48
Wang *et al*., 2015[25]	China	C-C	RUT	103/120	85.83	78.29-91.52
Zheng and Zhou, 2015[26]	China	C-C	PCR	50/70	71.42	59.37-81.59
Hu *et al*., 2016[27]	China	CS	PCR	9/14	64.28	35.13-87.24
Nisha *et al*., 2016[28]	India	C-C	RUT	209/293	71.33	65.78-76.44
Pataro *et al*., 2016[29]	Brazil	C-C	PCR	15/35	42.85	26.32-60.64
Yang *et al*., 2016[30]	China	C-C	PCR	78/103	75.72	66.29-83.63
Valadan Tahbaz *et al*., 2017[31]	Iran	C-C	PCR	4/50	8.00	2.22-19.23
Adachi *et al*., 2019[32]	Japan	C-C	ELISA	30/204	14.71	10.14-20.32
Total (random-effects)				2021/4072	47.93	39.65-56.26

Test for heterogeneity Q=628.82, df: 25 (P<0.001), I^2=96.02%, 95% CI: (95.04-96.82). *H. pylori*: *Helicobacter pylori*, USA: United States of America, UK: United Kingdom, *n/N*: Number of positive cases/total number of cases, CI: Confidence interval, CS: Cross-sectional study, C-C: Case-control study, ELISA: Enzyme-linked immunosorbent assay, PCR: Polymerase chain reaction, RUT: Rapid urease test, INMC: Immunochromatographic assay

Table 2: Evaluation of different parameters related to *Helicobacter pylori* detection and periodontal status

Parameter	Reference	Outcome	OR/MD	95% CI	I^2 (%)	*P*
H. pylori detection at different body sites† (mouth/stomach)	[9,11,23,25]	Mouth	OR: 2.32	1.69-3.20	30	<0.001*
PI	[15,26,33]	*H. pylori* (+)	MD: 0.27	0.21-0.32	0	<0.001*
PD	[15,26,33,34]	*H. pylori* (+)	MD: 0.67	0.22-1.13	91	<0.01*
CAL	[15,26,33]	*H. pylori* (+)	MD: 0.83	0.38-1.28	67	<0.001*

*Statistically significant, †Periodontitis patients references. *H. pylori*: *Helicobacter pylori*, OR: Odds ratio, MD: Mean difference, CI: Confidence interval, I^2: Higgins statistic for heterogeneity (percentage), PI: Plaque index, PD: Probing depth, CAL: Clinical attachment level

them[8,9,13,15,17,19,20,23-26,28,30,32] confirmed this higher *H. pylori* detection in periodontitis patients, whereas the remaining two[21,29] observed greater *H. pylori* detection in subjects without periodontitis. Several findings could explain the association between *H. pylori* infection and periodontitis risk. *H. pylori* has been detected in the gingival tissue and may play an important role in the development of periodontal disease. The gastrointestinal mucosa is the primary site of *H. pylori* colonization, although the oral mucosa is also, especially the gingival sulcus, due to the oral–oral or fecal–oral infection routes, with the oral cavity constituting another reservoir of *H. pylori*.[30]

In addition, the *H. pylori* infection rate correlates with the incidence of periodontal disease, increasing the depth of periodontal pockets and the severity of periodontitis. When *H. pylori* is located in the oral cavity, it acts as an additional risk factor for gastroduodenal ulcers and favors gastrointestinal infection through swallowing. Maintaining good oral hygiene with the removal of dental plaque potentially carrying *H. pylori* is an important measure to control infection by this bacterium and, therefore, gastric disease and periodontitis.[26]

In this study, periodontitis patients had higher detection of *H. pylori* in dental plaque compared to controls, with

a highly statistically significant association ($P < 0.001$). All the studies[10,16,18,24,26,28,31] about the detection of *H. pylori* in dental plaque coincided in pointing out this greater detection in periodontitis patients. The presence of *H. pylori* in dental plaque could be associated not only with the oral infection by this bacterium but also with reinfection from the gastric mucosa. A positive association between oral and gastric detection of *H. pylori* has been suggested. Moreover, this higher prevalence of *H. pylori* in dental plaque was more frequently observed in patients with dyspepsia.[20]

The detection of *H. pylori* in dental plaque is very common and is usually associated with greater severity of periodontitis. Furthermore, *H. pylori* in dental plaque is rarely eradicated by antibiotic therapy and may be a source of future reinfection. Because periodontitis is associated with increased *H. pylori* colonization, early diagnosis and management of the periodontal disease are necessary. Therapeutic measures should be implemented for better control of dental plaque in the setting of treating both periodontal disease and *H. pylori* gastric disease.[28]

In the present study, the *H. pylori* detection in the oral cavity of periodontitis patients doubled the probability of detecting *H. pylori* also in the stomach, observing highly statistically significant differences ($P < 0.001$). All the studies[12,16,18,24] that quantified the presence of *H. pylori* in the mouth and stomach corroborated this finding. *H. pylori* infection is considered a primary etiological factor for chronic gastritis, peptic ulcer, and stomach cancer. The eradication therapy is one of the main therapeutic measures for infectious gastric diseases. The *H. pylori* eradication from its oral reservoir could be useful in the prevention of periodontal disease and the treatment of all diseases linked to *H. pylori* infection.[18]

The coexistence of oral and gastric *H. pylori* infection is observed in more than 50% of periodontitis patients. Oral *H. pylori* detection could be a risk factor for recurrent gastric infection. If this is so, the oral cavity and dental plaque, as natural reservoirs of *H. pylori*, could act as a potential source of reinfection even after eradication treatment. This would justify the need for a combination of comprehensive medical and dental treatment modalities for patients with *H. pylori* infections.[16]

In this meta-analysis, *H. pylori*-infected patients had a significantly worse periodontal status by presenting a worse PI, more PD, and greater CAL than subjects not infected by *H. pylori*. All studies[14,27,33,34] that assessed these parameters agreed in pointing out this worse periodontal status of *H. pylori*-positive individuals. The *H. pylori* detection in periodontitis patients leads to an increase in the detection of other periodontal bacteria such as *P. gingivalis*, *P. intermedia*, *Fusobacterium nuclearum,* or *T. denticola* and a decrease in the *A. actinomycetemcomitans* detection. Patients with more severe periodontitis show a higher *H. pylori* detection rate, evidencing the relationship between periodontal disease and infection by this bacterium.[33] Higher PD and CAL have been observed in periodontitis patients infected with *H. pylori*, leading to the assumption that *H. pylori* infection could aggravate periodontal destruction in these patients.[27]

This study has some limitations. The different diagnostic criteria for periodontitis and the different methods for evaluating *H. pylori* infection may have influenced the results and caused heterogeneity. Neither other potentially confounding factors such as smoking or diabetes mellitus closely related to periodontitis could not be considered.

Further studies in larger samples and with longer follow-ups are required to more precisely establish the relationship between *H. pylori* infection and periodontitis.

Conclusions

In this meta-analysis, the pooled prevalence of *H. pylori* in periodontitis patients was 47.93%. Periodontitis patients were significantly more likely to be infected with *H. pylori* and also had an increase in the probability of *H. pylori* detection in dental plaque. Similarly, *H. pylori* detection was relevantly more likely in the oral microbiota than in the gastric microbiota of periodontitis patients. Finally, periodontal parameters (PI, PD, and CAL) were significantly worse in *H. pylori*-infected patients.

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Conflicts of interest

There are no conflicts of interest.

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