

Oral Signs of *Helicobacter Pylori*- Review of Clinical Outcomes



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Abstract

Helicobacter pylori (*H. pylori*) infection is the most common chronic bacterial infection worldwide and it plays a very important role in the pathogenesis of peptic ulcer, gastric carcinoma, and lymphoid tissue lymphoma. High reinfection rates after successful eradication treatments make the oral cavity a possible reservoir area for the survival of the bacterium. Therefore, the relationship between *H. pylori* and oral/gastric diseases is being investigated and has become a remarkable issue in recent years. In the oral cavity, *H. pylori* was first isolated from a dental plaque sample of a patient with gastric *H. pylori* in 1989. Investigation into the relationship of *H. pylori* with oral diseases has shown that oral *H. pylori* may cause halitosis, glossitis, recurrent aphthous stomatitis and dental caries. Hence, the aim of this review is to demonstrate the relationship between *H. pylori* and oral diseases in the context of the previous/latest research.

Keywords: *Helicobacter Pylori*; Dental Plaque; Saliva; Halitosis; Oral Diseases

Abbreviations: PCR: Polymerase Chain Reaction; VSC: Volatile Sulfur Compounds; *P. gingivalis*: Porphyromonas Gingivalis; *T. denticola*: Treponema Denticola; *T. forsythia*: Tannerella Forsythia; *P. intermedia*: Prevotella Intermedia; RAU: Recurrent Aphthous Ulcers; RAS: Recurrent Aphthous Stomatitis; *H. pylori*: *Helicobacter Pylori*

Introduction

Helicobacter pylori (*H. pylori*) is a spiral-shaped, gram-negative, urease-producing and highly pathogenic microaerophilic bacteria which causes development of highly serious gastrointestinal disorders and is mainly acquired in childhood [1-4]. Almost 50% of the global population is affected by the gastric *H. pylori* infection. The infection is more prevalent in developing countries in comparison to developed nations [5]. The prevalence of *H. pylori* infection mainly depends on age, geographic area and ethnicity [6]. Low levels of education and low socioeconomic status lead to an increase in the prevalence of *H. pylori* [7]. Although the pathway of transmission of *H. pylori* is not completely known, it is thought that the transmission could occur by faecal-oral, oral-oral or iatrogenic routes. Diarrhea is often seen in childhood and the transmission of *H. pylori* from the feces to the mouth can be accomplished by sharing infected toys or foods. Additionally, the detection of *H. pylori* in the breast milk suggests that the organism can move through the fingers or nipples to the breast and contaminate the milk. The fecal-oral route of transmission seems to be possible only when there is close contact with infected individuals.

Gastro-oral transmission can occur through vomiting and gastro-oesophageal reflux. Infection can occur through ingestion of food or exposure to vomit-infected objects colonized by *H. Pylori*. Oral-oral

transmission can occur either permanently or temporarily through the dental plaque and saliva. The findings on identical strains of *H. Pylori* in the mouth and stomach strengthen the hypothesis that the oral cavity may be a reservoir for the bacteria. Close person-to-person contact between members of the same family can promote oral-oral transmission. Another mechanism for the oral-oral mode of transmission is through kissing. However, this appears to be an unlikely route of transmission. The increased prevalence of *H. pylori* in the presence of crowded living conditions and poor self-care has also been reported. The higher prevalence of the infection among children whose mothers chew or taste their food before feeding them demonstrates that saliva could be a transmitter medium of the infection [8]. Also, *H. pylori*-like organisms can be seen in other animals, where infections with *Helicobacter* species have been reported in dogs and cats [9].

Oral Cavity as an Extra Gastric Reservoir Area for *H. Pylori* Survival

The oral cavity, which is the entrance of the gastrointestinal system, has a complex flora including 350 different species. The oral temperature varies between 35-37 °C, which provides an ideal environment for the growth of *H. pylori*. Also, dental plaque has a low oxidation-reduction potential and this environment enhances the

reproduction of facultative anaerobic bacteria, suggesting that the acidic pH resulting from the fermentation of carbohydrates creates a very favorable environment for the reproduction of *H. pylori* as a microaerophilic bacterium [10]. *H. pylori* was also detected in periodontal pockets of patients with periodontal diseases, on the dorsum of the tongue and in saliva samples [1,11].

Detection of Oral *H. Pylori*

Cytology, urease test, culture, and polymerase chain reaction (PCR) are the methods used for the detection of *H. pylori* from dental plaque and saliva samples [5]. The use of the urease test for the detection of the *H. pylori* infection in the oral cavity shows low sensitivity due to the presence of a wide variety of bacteria with urease activity in the oral cavity. Streptococcus, actinomycetes and Hemophilus species are some of the urease active bacteria in the oral cavity. For this reason, the urease test is not a suitable method for *H. pylori* detection in the oral cavity [3]. While histological methods using classical hematoxylin and eosin staining methods can be used to visualize *H. pylori*, the use of Warthin-Starry and Giemsa staining can enhance the histological identification of microorganisms. Antimicrobial susceptibility tests can be performed using culture methods. Serological tests detect levels of antibodies such as IgG and IgA in serum raised in response to an infection of *H. pylori*.

A number of different PCR methods have been developed for the diagnosis of *H. pylori* infection, which can help to differentiate *H. pylori* strains [9]. Currently, there is no gold standard method for isolating *H. pylori* from the oral cavity, but it is stated that the best method for the detection of *H. pylori* from dental plaque is the PCR method. The use of molecular methods for the detection of bacteria is becoming increasingly widespread because of the difficulties in culturing bacteria other than gastric mucosa and the necessity of using non-invasive methods [5]. The results of the PCR method need to be examined very carefully as differences in the primers and the genes used in the amplification can affect the results [12].

Helicobacter Pylori in the Dental Plaque and Saliva

The presence of *H. pylori* in the dental plaque and saliva has been investigated in many studies [2,12-25]. The studies conducted thus far with the aim finding an association between oral and gastric *H. pylori* infection have demonstrated conflicting results. Burgers et al. [13] and Song et al. [24,25] conducted a study to determine the association between oral *H. pylori* and gastric *H. Pylori*. Both of the researchers detected oral *H. pylori* in the dental plaque and saliva samples of gastric *H. pylori* negative patients. Therefore, they stated that *H. pylori* may be present in the oral cavity independently from the gastric colonization and may belong to the normal oral flora. Valdez-Gonzales et al. [17] received dental plaque samples from 40 healthy children with no dyspepsia between the ages of 2-11 and examined the presence of *H. pylori*. In total, 35% of these children showed the presence of *H. pylori* in dental plaque samples. In this study, it was demonstrated that the oral cavity of children, particularly dental plaque, may be a reservoir area for *H. pylori*. Rasmussen et al. [18] attempted to demonstrate the presence of *H. pylori* in the oral cavity and the association between oral and gastric presence of the bacterium. A total of 78 adult patients with continuous abdominal pain were included in their study. At the

same time, dental plaque and saliva samples were received from patients before each endoscopic procedure and examined with PCR. The investigators found an *H. pylori* prevalence rate of 70% among saliva and dental plaque samples. Finally, the researchers reported a significant association between gastric and oral *H. pylori*. Liu et al. [19] conducted a study to examine the prevalence of oral *H. pylori* in the Chinese population and the incidence of gastric infection among patients with oral *H. pylori*. Out of the total of 443 adult patients with dyspeptic complaints, 263 (59.4%) had *H. pylori* in their dental plaque samples and 273 (61.6%) had gastric *H. pylori*. Gastric infection was found to be significantly higher in patients with oral *H. pylori*. Investigators found a strong association between the presence of oral/ gastric *H. pylori*, indicating that the oral cavity is an important reservoir for this pathogenic microorganism.

Tiwari et al. [20] received saliva samples from 80 patients suffering from dyspepsia and 20 healthy adult control groups. Seventy-two of the 80 dyspeptic patients (90%) had gastric *H. pylori*. In saliva samples, *H. pylori* was detected in 70 (87.5%) of the 80 dyspeptic patients and in 12 (60%) of the 20 healthy individuals that constituted the control group. Since the investigators detected high levels of *H. pylori* in saliva samples, they noted that saliva has an important role in bacterial infection and may cause re-infections. Assumpção et al. [2] included 99 dyspeptic adult patients in their study. A total of 95 patients (96%) were found to be gastric *H. pylori* positive. Furthermore, *H. pylori* was not detected in the dental plaque specimens of any patient without gastric *H. pylori*. The researchers found a correlation between gastric colonization and oral colonization of *H. pylori*. Bickley et al. [21] received dental plaque and saliva samples from 14 patients before endoscopy and investigated the presence of urease C gene of *H. pylori*. The investigators were unable to detect *H. pylori* in any dental plaque and saliva samples.

Hardo et al. [22] examined dental plaque specimens from 62 adult patients with dyspepsia. They reported an *H. pylori* detection rate of 1.6% in dental plaque samples. No *H. pylori* was detected in any of the dental plaque samples of 61 patients. Hence, the researchers were unable to show a relationship between the presence of gastric *H. pylori* and oral *H. pylori*. Li et al. [23] received saliva samples from 56 patients with dyspeptic complaints and 40 patients were found to be gastric *H. pylori* positive. Among these patients, 30 (75%) of them had *H. pylori* in their saliva samples. *H. pylori* was detected in the saliva samples of 3 gastric *H. pylori* negative patients. The researchers did not find a clear relationship between the gastric colonization of *H. pylori* and the presence of *H. pylori* in saliva. Bharath et al. [16] investigated the genetic presence of the bacterial urease gene using the real-time PCR method among both adult and pediatric patients aged between 10 and 80 years who had dyspeptic complaints. Six of the 56 patients had *H. pylori* in the supragingival dental plaque, while none of the patients had *H. pylori* in gastric biopsy specimens. The researchers suggested that patients may have previously received eradication therapy which was the cause of these outcomes and that this treatment was able to eradicate *H. pylori* in the gastric mucosa but was not effective on the dental plaque.

H. Pylori and Halitosis

H. pylori has an important role in the gastrointestinal diseases that cause halitosis. With the oral colonization of *H. pylori*, the prevalence of volatile sulfur compounds (VSCs) producing periodontopathic microorganisms, such as *Porphyromonas gingivalis* (*P. gingivalis*), *Treponema denticola* (*T. denticola*), *Tannerella forsythia* (*T. forsythia*), *Prevotella intermedia* (*P. intermedia*) are increased [26-28]. On the other hand, Suzuki et al. [26] indicated that the presence of *H. pylori* in the oral cavity may be associated with halitosis caused by periodontal pocketing and inflammation, rather than the bacteria's VSCs producing ability. The researchers found that the prevalence of *P. intermedia* was higher among the *H. pylori*-positive patients, while the prevalence of *P. gingivalis* and *T. denticola* was independent from the presence of *H. pylori* in the patients with periodontal symptoms. *P. intermedia* was detected in various parts of the oral cavity, including supragingival plaque, periodontal pockets, and the dorsum of the tongue. Katsinelos et al. [29] found a positive relationship between the presence of gastric *H. pylori* and halitosis according to the results of organoleptic measuring. The researchers reported that halitosis decreased after the eradication therapy. Chen et al. [30] conducted a subjective halitosis assessment with the organoleptic method and found that patients with gastric *H. pylori* were more likely to have halitosis than those without halitosis. Schubert et al. and other researchers [31-34], performed subjective halitosis assessment using the organoleptic method and could not demonstrate a relationship between the presence of gastric *H. pylori* and halitosis.

H. Pylori and Oral Diseases

Recurrent aphthous ulcers (RAU) are a common disease characterized by the periodic development of painful, single or multiple ulcerations in the oral mucosa. The symptoms of immunocompromised individuals are even more severe. Several possible etiologic factors have been proposed, including focal stimulation of mucosal T-cell functions by endogenous (autoimmune) or exogenous (hyperimmune) antigens. However, evidence of important causal factors is still lacking; for this reason, treatment of RAU with current methods is insufficient and is usually palliative [35]. Previous studies [35-38] have shown an increase in the formation of aphthous ulcers in the oral cavity with the presence of oral *H. pylori*. Also, there have been studies investigating the relationship between oral *H. pylori* presence and other mucosal lesions [39-42]. Iamaroon et al. [43] studied the effects of *H. pylori* in the etiology of RAU formation. They examined the surface of ulcerations and the dorsum of the tongue of 22 patients with RAU, and the dorsum of the tongue of 15 healthy subjects as a control group. The researchers stated that *H. pylori* is not involved in the pathogenesis of RAU. However, they indicated that the tongue dorsum may be a reservoir area for *H. pylori*.

Adler et al., [44] reported that glossitis was seen more frequently in patients who had gastric *H. pylori*. Gall-Troselj et al. [45] found that glossitis and burning mouth syndrome were more common in the presence of *H. pylori*. Tiomny et al. and other researches [27,46,47], reported that the incidence of recurrent aphthous stomatitis (RAS) increases in the presence of *H. pylori*.

Iamaroon et al. [43,48] demonstrated that RAS was not associated with *H. pylori*. Mansour-Ghanaei et al. [48] were only able to isolate *H. pylori* from one oral aphthous lesion thus, they were unable to demonstrate a relationship between a positive *H. pylori* ELISA test and the presence of *H. pylori* in RAS lesions. Iamaroon et al. [43] investigated the association of *H. pylori* and RAS by using a highly sensitive technique, nested PCR, in 22 patients with RAS with ages ranging from 12-36 years. Samples were taken from the lesions and the dorsum of the tongue of each patient. Additionally, samples from the dorsum of the tongue of 15 healthy individuals with ages ranging from 13-40 years who were taken as controls. The results showed that only one sample from a lesion (4.5%) and one sample from the tongue (4.5%) of two different patients with RAS were positive for *H. pylori*. In the control group, only 3 samples (20%) were positive for *H. pylori*. These findings suggest that *H. pylori* does not play a role in the pathogenesis of RAS and the dorsum of the tongue may be a reservoir of *H. pylori* in some individuals.

H. Pylori & Caries, Oral Hygiene Habits, and Periodontal Health

The relationship between periodontal health and oral/gastric *H. pylori* has been studied in many studies [3,19,49-61]. The association between periodontal disease and *H. pylori* infection was tested in a case-control study among 134 dyspeptic patients presenting for upper gastrointestinal endoscopy [50]. The periodontal status of the patients was evaluated as diseased or healthy. Among the cases, 30 subjects out of 65 (46.2%) had periodontal disease compared to only 20 out of 69 (29%) in comparison to the controls. Although the univariate analysis suggested that the relationship was significant, when analyzed by logistic regression, the difference was found to be insignificant. Silva et al. [54] reported that periodontal health was worse in the presence of oral *H. pylori*. However, the researchers did not find a significant association between the presence of gastric *H. pylori* and periodontal health. Al-Asqah et al. [49] examined 62 patients with a gingivoperiodontal disease and 39 patients with dyspeptic complaints without a gingivoperiodontal disease. They reported that 60% (37/62) of the periodontitis patients had gastric *H. pylori* compared to only 33% (13/39) of the patients without periodontitis. Furthermore, they reported that the prevalence of *H. pylori* in the dental plaque was higher among periodontitis patients (79%, 49/62) than in patients without periodontitis (43%, 17/39). They also reported that the presence of the bacterium in both locations was higher among periodontitis patients (46.8%, 29/62) than in patients without periodontitis (10.3%, 4/39). The researchers demonstrated a positive relationship between oral and gastric *H. pylori* presence and periodontal disease.

Souto et al. [55] evaluated the presence of oral *H. pylori* among 56 periodontally healthy and 169 chronic periodontitis patients and found a statistically significant relationship between the presence of chronic periodontitis and bacterial oral colonization. Sambashivaiah et al. [52] found no relationship between periodontal disease and oral *H. pylori*.

Additionally, Butt et al. [56] also failed to demonstrate an association between the oral presence of *H. pylori* and gingival and periodontal health in 78 patients with dyspeptic complaints. Namiot et al. [57] investigated the relationship between oral

hygiene status and oral *H. pylori* presence among 155 dyspeptic patients, but they did not find a relationship. Bali et al. [51] included 124 dyspeptic patients in their study. Sixty patients were found to be gastric *H. pylori* positive and 64 patients were found to be gastric *H. pylori* negative. The researchers did not indicate any relationship between gastric *H. pylori* presence and oral hygiene status. Liu et al. [19] found a positive relationship between *H. pylori* presence in dental plaque and oral hygiene status. They stated that all of the patients who had *H. pylori* in their dental plaque samples were irregularly brushing their teeth [58]. Another study [3] was conducted among Chinese children aged between 3-6 years in order to determine the presence of *H. pylori* in the dental plaque samples and its association with dental caries and oral hygiene habits. In this study, a significant relationship was found between oral *H. pylori* presence and dental caries. The investigators indicated that the oral cavity may be a reservoir for the bacterium; thus, *H. pylori* in dental plaque may play a role in the formation of dental caries, and poor oral hygiene may be a risk factor for *H. pylori* survival in the oral cavity. Few studies have evaluated the relationship between caries and *H. pylori* in the literature. Berroteran et al [59] investigated whether there was a relationship between oral *H. pylori* presence and caries in 32 dyspeptic and 20 asymptomatic healthy subjects. The results did not show a correlation between caries and the presence of oral *H. pylori*. Also, they found that patients with poor oral hygiene and periodontal status had a similar prevalence of *H. pylori* to patients with good-to moderate dental hygiene.

Banatvala et al. [61] found that 58% of 19 healthy children had *H. pylori* in their dental plaque samples, but no relationship was demonstrated between oral *H. pylori* presence and caries. Kolho et al. [58] compared gastric *H. pylori* positive and negative children in terms of caries presence and found no significant difference in caries prevalence between the two groups. The researchers reported that gastric *H. pylori* positive children may have a higher risk of caries development due to medical problems. Wickelhou et al. [60] found no association between *H. pylori* presence and *Streptococcus mutans* count in the dental plaque, but the investigators reported that *H. pylori* in the dental plaque was found at a higher rate in patients with low lactobacilli counts.

Conclusion

H. pylori is a highly pathogenic bacteria that is responsible for gastritis and peptic ulcer formation and causes stomach cancer. For this reason, it is very important to prevent the spread of bacteria among people and the success of eradication treatment must be ensured. Dental plaque is an oral biofilm layer formed on the tooth surface and contains millions of microorganisms. Since *H. pylori* has been isolated on the dental plaque, an answer has been sought as to whether this bacterium poses a risk in terms of re-infection of the gastrium. Poor oral hygiene and dental plaque accumulation may lead to the proliferation of *H. pylori* as a microaerophilic feature that can cause infections. For this reason, patients should maintain good oral care and have regular dental visits in order to prevent possible gastric colonizations.

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