ORAL CAVITY AND SYSTEMIC DISEASES – *HELICOBACTER PYLORI* AND DENTISTRY

Assya Krasteva¹, Vladimir Panov², Adriana Krasteva³, Angelina Kisselova¹
¹ Medical University – Sofia, Faculty of Dental Medicine, Department of Imaging and Oral Diagnostics, Sofia, Bulgaria
² Medical University “Prof. Dr. P. Stoyanov”, Faculty of Dental Medicine, Department of Conservative and Pediatric Dentistry, Varna, Bulgaria
³ Medical University – Sofia, Faculty of Pharmacy, Department of Pharmacology and Toxicology, Sofia, Bulgaria

Correspondence to: Assya Krasteva
E-mail: asyakrasteva@gmail.com

ABSTRACT

*Helicobacter pylori* is a Gram-negative microorganism and an important factor in the pathogenesis of numerous diseases including gastro-intestinal perturbations (peptic ulcer, gastric carcinoma, gastric mucosa-associated lymphoid tissue lymphoma), metabolic, autoimmune, vascular and systemic disorders.

The isolation of *H. pylori* from dental plaque and saliva is of great interest in order for the role of the oral cavity in its transmission to be examined. It is considered that the oral cavity is the main extragastric “reservoir” and an entry of the infection and reinfection. Virulent *H. pylori* strains can be present, although seldom and probably transitory, in oral specimens from patients with chronic dental or gastroduodenal diseases or both.

In the present review we would like to elaborate on the place of *H. pylori* in the oral cavity and the role of the dentists in limiting the transmission of infection and consequently helping to reduce the risk of patient’s reinfection. On the other hand, the possibility dental practitioners to be at a higher risk for *H. pylori* infection is discussed, possible oral manifestations of the disease and some practical recommendations are given.

**Background**

*H. pylori* is an important factor in the pathogenesis of numerous diseases including gastro-intestinal, metabolic, autoimmune, vascular and systemic disorders (atherosclerosis, Raynaud’s disease; headache and migraine; arthritis, immune thrombocytopenia, Henoch-Schönlein disease, Sjögren syndrome, autoimmune thyroiditis; rosacea, chronic urticaria, atopic dermatitis, alopecia areata, iron deficiency anemia, amenorrhea, halitosis, oral ulcer or aphthosis) (3, 5, 16, 17, 19, 22).

**Epidemiology**

This infection affects more than half of the human population. The prevalence is however unbalanced between rural developing areas (more than 80%) and urban developed areas (less than 40%) (25). At birth, the rate of *H. pylori* positivity is 81% in breast-milk and 96% in maternal and infant sera (12).

*H. pylori* is found in gastric mucosa in 90 to 100% of patients with duodenal ulcer and 70 to 90% in patients with gastric ulcer (11).

**Risk factors for *H. pylori***

Risk factors for *H. pylori* infection are:

- poor social and economic development;
- poor hygienic practices;
- absence of hygienic drinking water;
- unsanitary food preparation.

The presence of *H. pylori* in sheep stomach in the absence of associated gastritis and recovery of *H. pylori* from sheep milk and gastric tissue suggest that sheep may be a natural host for *H. pylori* (6). *Helicobacter* DNA was demonstrated in 60% of milk samples and in 30% of sheep tissue samples. Sardinian shepherds have almost a 100% prevalence of *H. pylori* and the prevalence is higher than that of their same-household siblings (6).

**Transmission of *H. pylori***

Main routes of infections are: oral-oral, gastro-oral and faecal-oral transmission, however no predominant mechanism of transmission has been yet identified. Transmission may occur in a vertical mode (e.g. from parent to child) or in a horizontal mode (across individuals or from environmental contamination). In either case, the involvement of water and food cannot be excluded as sources of infection. The likelihood of the transmission pathway in developing rural and developed urban areas appears to be different. In developed areas, person-to-person transmission within families appears to be dominant, while in the rural developing areas the transmission pathway appears to be more complex. In this latter case, the transmission by contaminated food, water, or via intensive contact between infants and non-parental caretakers may have a greater influence than within-family transmission (25).
Oral H. pylori has been detected in periodontal pockets of various depths and the dorsum of the tongue by nested PCR. Overall, 58% of the subjects had a positive fingernail result, with a significant positive relationship between fingernail and tongue positivity. The authors have suggested that oral carriage of H. pylori may play a role in the transmission of infection and that the hand may be instrumental in transmission (7).

**Oral cavity as reservoir of infection**

Recent studies have shown that patients with chronic gastritis are with a higher prevalence of H. pylori in the dental plaque than in the stomach (60% in the gastric mucosa and 90% in the poster) (21). This shows that oral cavity may be the first place for colonization and then the infection may involve the gastric mucosa.

The bacterium has been detected in saliva, supragingival and subgingival plaque, suggesting that these sites may be considered reservoirs for H. pylori not only in urease-positive patients, but in healthy volunteers and thus be involved in the reinfection of the stomach (14).

The eradication therapy is more effective in the absence of H. pylori in the oral cavity (92%), compared to that in its presence (52%). The “reservoir” in the stomach facilitates the reinfection (2, 8, 23).

It is of a great importance that oral H. pylori might cause gastric reinfection even after the eradication therapy (2, 8, 20).

H. pylori has also been found in the palatine tonsils. Some authors have observed that patients with IgA nephropathy had tonsillar H. pylori (P < 0.01) (18). Assumpçao et al. have found that H. pylori was detected in 96% of gastric mucosa samples and in 72% of dental plaque samples. Sixty-three (89%) of 71 dental plaque samples that were H. pylori-positive also exhibited identical vacA and cagA genotypes in the gastric mucosa. The most common genotype was vacAs1bm1 and cagA positive, either in the dental plaque or the gastric mucosa. The conclusion of this survey was that the gastric infection was correlated with the presence of H. pylori in the mouth (1). These data were also seen by Eskandari et al. (9).

It is difficult a niche in the oral cavity, specific to H. pylori, to be defined. The bacteria has been detected in all areas of the mouth, but those data are not comparable because of the different methods performed (24).

Thirty-six specimens from the oral cavity of 25 patients with dental and/or gastroduodenal diseases have been evaluated by culture and rapid urease test and afterward, for the HLO (Helicobacter-like organisms), by immunofluorescence with monoclonal antibodies and PCR. One H. pylori strain was isolated from a 9-year-old child with gingivitis and chronic gastritis. The strain was H. pylori positive by the immunofluorescence and PCR for H. pylori gene ureA and carried the virulence gene cagA as well as the more toxigenic genotype vacAs1m2. Two other HLO strains were isolated from adults and were positive by the immunofluorescence, however without a PCR confirmation, they could not be identified at the species level. The second dental plaque specimen from the previously (two years ago) positive child was negative by the culture and PCR directly in the oral specimen. This can suggest a transitory presence of H. pylori in the oral cavity (4).

**Dental practitioners as a risk contingent for H. pylori**

Dentists and dental professionals may be at increased risk due to the contact with oral cavity of patients with presence of H. pylori in the oral cavity where it may serve as reservoir for gastric infections and participate in the pathogenesis of oral mucosal lesions and ulceration. However, evidence regarding the occurrence of H. pylori infections and colonization in dentists is conflicting, but has been based mainly on serological studies, which carry significant limitations (19).

It has been found that Japanese dentists are at a high risk for H. pylori infection than controls, with the oral-oral transmission route being possibly the most common (13, 19).

**H. pylori and oral health**

Regarding oral health it has been demonstrated that greater plaque index and a higher incidence rate for gingivitis is observed in individuals with gastric H. pylori infection (1). Some authors have found that H. pylori was scarce in patients with periodontitis (9).

Silva et al. (23) studied 115 individuals, divided in 4 groups: (A) with gastric diseases and periodontal disease; (B) with gastric diseases and no periodontal disease; (C) without gastric diseases and without periodontal disease, (D) without gastric diseases and with periodontal disease. H. pylori was detected in the supragingival plaque and there was an association between the supragingival colonization of H. pylori and oral hygiene parameters such as the presence of plaque and gingival bleeding (23).

**Oral manifestations and symptoms in patients with H. pylori infection**

In some patients different nonspecific oral signs of H. pylori infections can often be seen, such as:

- gingivitis (Fig. 1);
- periodontitis (Fig. 2);
- aphthous stomatitis (Fig. 3);
- tongue coating (Fig. 4);
- halitosis.

Many patients complain of subjective symptoms such as month and tongue burning, numbness in the mouth, altered taste, dry mouth, even in the absence of common typical clinical symptoms of infection.
General treatment rules

In 56 patients with chronic periodontitis and gastric *H. pylori* 1-week eradication therapy with amoxycilin 1 g, clarithromycin 500 mg, and proton pump inhibitor 20 mg twice a day was performed. Almost 40% of the patients with gastric *H. pylori* harbored the bacterium in the oral cavity. After the eradication therapy, *H. pylori* was not detected in the oral cavity, which suggests high effectiveness of the therapy protocol in the oral cavity, or it is possible that oral *H. pylori* is of a transient character (2).

In contrast, Zaric et al. (26) communicated that periodontal treatment in combination with systemic therapy could be a promising approach to increasing the therapy efficacy and decreasing the risk of infection recurrence. 77.3% of the patients treated with combined periodontal therapy exhibited successful eradication of gastric *H. pylori*, compared with 47.6% who underwent only triple therapy (26). Similar results
were obtained by Jia et al. (15). Long-term professional dental plaque control was associated with less gastric reinfection by *H. pylori*, suggesting that dental plaque control may help preventing *H. pylori*-induced gastric disease or reinfection. The prevalence of *H. pylori* in the gastric mucosa was 20% in patients who received dental plaque control, which was significantly lower than in those without dental plaque control (84%) (15).

Some studies have shown that *H. pylori* could be transmitted more often through household contact (kissing) than with consumption of contaminated foods. Good nutrition, frequent fruits and vegetables consumption and foods rich in vitamin C, protect against infection with *H. pylori*.

### Practical dental recommendations

Individuals positive for *H. pylori* in their gastric or oral samples, as determined by nested PCR, were treated either with periodontal and triple therapy or with triple therapy alone. Results indicate that 77.3% of those treated with the combined therapy exhibited successful eradication of gastric *H. pylori*, compared to 47.6% who underwent only triple therapy. Analysis of these data suggests that periodontal treatment in combination with systemic therapy could be a promising approach to increase the efficacy of the treatment and to decrease the risk of infection recurrence (10).

Based on the literature data and on our experience we recommend:

1. Professional plaque and tartar removal (by scaling) – at the beginning of eradication, as well as during therapy (5-7 days at the beginning of the treatment) and after medical therapy (10 to 30 days after treatment) to reduce the possibility of reinfection;
2. New tooth brush to be used (in order for further infection to be prevented);
3. Special care to the “removal” of denture plaque – daily brushing with toothpaste, soaking in a disinfection solution, sometimes even replacement with new dentures;
4. Daily care for good oral hygiene: at least 3 min teeth cleaning on all surfaces;
5. Cleaning of the tongue – use of a toothbrush with special equipment (the cracked surface of the tongue can become friendly, except for the healthy bacterial flora, also for harmful bacteria); and cleaning the hard palate;
6. Cleaning the intradental spaces – with dental floss and intradental brushes;
7. Mouthwash usage – twice daily – during the treatment to chemical plaque control (water inhibits the plaque formation, prevents its attachment to the teeth or facilitates its decay, and thus favors the mechanical cleaning);
8. Some studies have shown that chewing gum has an antibacterial effect against *H. pylori*.

### Conclusions

The presence of *H. pylori* in the oral cavity could be considered as main extragastric reservoir and possible source of reinfection. It has been confirmed in some patients with gingivitis, periodontitis, stomatitis aphthosa, halitosis and tongue coating. The dental treatment in combination with systemic therapy could be a promising therapeutical approach.

### REFERENCES


