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Periodontology

HELICOBACTER PYLORI AND PERIODONTAL DISEASE: THE ORAL-GUT-MICROBIAL AXIS

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ABSTRACT Periodontal disease is a collection of bacteria induced inflammatory diseases of the teeth's supporting tissues. Because periodontal disease is frequently accompanied with many systemic disorders that accelerate or predispose the disease's course, periodontitis has a strong link to other areas of medicine. Acute gastritis, chronic atrophic gastritis, gastric atrophy, gastritis ulcers, dysplasia, duodenal ulcer, gastric cancer, and gastric MALT lymphoma are among the most common gastroduodenal disorders associated with Helicobacter pylori infections. Orofecally transferred organisms are the most prevalent. The oral cavity's potential involvement as a way of transferring the microorganism and as an extra-gastric reservoir of H.pylori, which forms inside the oral plaque, the principal etiological agent of periodontal disease, becomes clear through several studies. Given that the oral cavity of a patient with periodontal disease has elevated bacterial plaque indices in connection with infrabony pockets, one could wonder if this represents a favorable environment for H. pylori colonization. Another question to consider is if the presence of H.pylori in the oral cavity is a factor in the recurrence of gastric infections. As a result, may non-surgical periodontal therapy combined with eliminating gastric therapy promote decontamination of the microorganism in the oral cavity, resulting in greater prevention of relapse and re-infection of the gastric cavity? Is it possible that non-surgical periodontal treatment could prevent stomach disorders caused by Helicobacter pylori? By integrating all of the relevant papers, this review aims to answer these questions.

KEYWORDS : Helicobacter pyroli; chronic periodontitis; Gastritis; Nonsurgical Periodontal Therapy

INTRODUCTION

Periodontitis is inflammation of the tooth's supporting tissues that causes gingival detachment and disintegration of alveolar bone.[1] Periodontitis has been linked to illnesses such as obesity, metabolic syndrome, and cardiovascular disease in recent research, therefore prevention and treatment are deemed vital to avoid the incidence of systemic disease.

Helicobacter pylori is an important gastrointestinal pathogen that is strongly associated with gastritis as well as peptic ulcer disease. Antimicrobial therapy frequently fails to cure H. pylori infection, which suggests there may be other sanctuary sites where the organism resides.[2]

It is considered that the oral cavity is the main extragastric "reservoir" of H.Pylori and an entry portal for the bacterium and also plays a role in reinfection.[3] In addition, several investigators have reported detection of the bacterium in dental plaque and saliva samples, and have also showed that periodontal pocket depth ≥5 mm is associated with increased odds of H. pylori seropositivity. Therefore, the presence of H. pylori infection may worsen the degree of periodontitis, while its cure might lead to improvement.[4]

Therefore, the aim of this review is to examine the association between oral and stomach H pylori and periodontal disease by combining all the eligible articles.

Helicobacter pylori is one of the most common bacterial infections in humans. It is a gram negative, microaerophilic, rod-shaped bacterium that colonizes the gastric mucosa. Although its presence in the human stomach has been reported from all parts of the world, the prevalence of H. pylori infection is higher in developing countries than developed countries. First reported in 1983, H. pylori (initially termed Campylobacter pyloridis) is an important human pathogen associated with the etiology of chronic gastritis, peptic ulcer, gastric cancer, and mucosa-associated lymphoid tissue lymphoma (MALT) and has been designated as a Group 1 Carcinogen by the International Agency for Research on Cancer of the World Health Organization (WHO).[5]

Mode of Transmission

The mechanisms of transmission of the microorganism from individual to individual are not yet clear. The possible routes of transmission of H. pylori include iatrogenic, fecal-oral, oral-oral, and through food and water. H. pylori exists in two different morphological forms, spiral and coccoid. The coccoid form is considered a degenerative or dead form of H. pylori, and its role in transmission of disease is negligible. There is ongoing debate about its virulence and transformation. Although the coccoid form of H. pylori is metabolically active, it cannot be cultured in vitro.[6]H. Pylori is most commonly transmitted by orofecal route.[5]

Risk factors for H. pylori Risk factors for H. pylori infection are[3]:

- Poor social and economic development;
- Poor hygienic practices;
- Absence of hygienic drinking water;
- Unsanitary food preparation

DIAGNOSIS

Diagnostic testing for *H. pylori* can be divided into endoscopic and non-endoscopic techniques. The techniques could be direct (culture, histology, or detection of bacterial antigen in the biopsy tissue or stool) or indirect (using urease breath test, or an antibody response as a marker of disease). Serologic test for antibody indicates exposure to bacteria but does not help to assess active infection.[7]

ORAL CAVITY AS A RESERVOIR OF *H.Pylori*

The detection rates of H. Pylori in saliva were generally less than in dental plaque, with only few studies reporting detection rates of 50%.[6] This may be due to the fact that, while dental plaque, being a biofilm, allows the bacteria to adhere to solid surfaces, the constant flow of saliva may contribute to a reduction in bacterial load, making detection difficult.[8]

Palatine tonsils have also been reported as a niche for H.pylori bacteria.

PRESENCE OF H.Pylori IN DENTAL PLAQUE

Dental plaque is a microbial biofilm that adheres tenaciously to teeth and other hard surfaces in the oral cavity, such as restorations.[9]

Umeda et al in 2003 demonstrated that although H. pylori was rarely detected in the oral cavity by culture technique, it was frequently detected by nested PCR in the oral cavity, especially among periodontitis patients who had the bacterium in the gastrointestinal

tract. Among the subjects who harbored H. pylori in the stomach or duodenum, 41.2% of patients with periodontal pockets ≥4 mm.[10]

A significantly higher prevalence of H. pylori was observed in subgingival biofilm samples compared to saliva samples. [11] There are differing points of view in the literature concerning the contention that the oral cavity is a reservoir of H. pylori. Some authors believe that H. pylori has only a transient presence in the oral cavity as they detected low percentage of H. pylori in the mouths of their patients. On the other hand, authors who find this bacterium in almost all of their studied population consider it as part of the normal microbiota of the oral cavity[2]

ASSOCIATION BETWEEN PERIODONTITIS AND *H. PYLORI* INFECTION

An epidemiological study carried out in the United States of America based on data from the National Health and Nutrition Examination Exley III showed that among 4504 periodontitis patients between the ages of 20 and 59 years, all were positive for Hp infection. The results of this study showed that periodontal pockets greater than 5 mm contained the bacterium and that the low periodontal health could be associated with an Hp infection[12]

Byun et al conducted a prospective cohort study which used epidemiological data from the Korean Genome and Epidemiology Study recorded from 2004 to 2016. Among 173,209 participants, 9983 with periodontitis and 125,336 with no periodontitis were selected. This study demonstrated that periodontitis was associated with an increased risk of chronic gastritis/peptic ulcer.[13]

The possible reasons for this association could be:

- Low-grade persistent inflammation caused by periodontitis and/or such infection has been reported to produce inflammatory cytokines, such as tumor-necrosis factor-alpha and interleukin 6, from monocytes and macrophages, which are considered to induce several different systemic disorders including gastritis.[4]
- Periodontopathogens such as Fusobacterium nucleatum and Porphyromonas. gingivalis were found to coaggregate with H. pylori in the dental biofilm[2]
- Some oral bacteria, i.e. Streptococcus and Actinomyces species produce bacteriocin-like inhibitory proteins against H. pylori. The fact that good oral hygiene patients harbor less H. pylori in their mouth could also be explained by this inhibitory activity of the early colonizers of the mouth[14]
- H. pylori survives in moderate to advanced periodontal pockets because the architecture and the microcosm of these periodontal conditions promote a viable habitat for micro-aerophilic and anaerobic microorganisms.[2]

THE EFFECTS OF THE GASTRIC ERADICATING THERAPY ON ORAL HELICOBACTER PYLORI

Treatment protocol for H.Pylori includes[3]:

One-week eradication therapy with:

1. Amoxycilin caps. 1 g, twice daily, after meal;
2. Clarithromycin tabl. 500 mg, twice daily, after meal;
3. Proton pump inhibitor tabl. 20 mg - twice daily, 15 min before meal;
4. Colloidal bismuth subcitrate (120 mg) 4 times daily (3 times before meal and 1 capsule at bed time)

Gebara et al reported an increase in the prevalence of H. pylori in dental plaque in their patients after one week of triple therapy.[15] Wheras Bago et al[16] reported that one week of triple therapy resulted in complete eradication of oral H. pylori in all periodontitis patients who had H. pylori-associated gastric disease.

One major advantage that biofilm bacteria enjoy is an increased resistance to host defense mechanisms and anti-microbial agents. Thus, H. pylori present in the dental plaque, being biofilm-associated, are protected from systemic antibiotics administered for the management of gastric H. pylori infection. As a result, the microorganism may persist in the oral cavity even after successful eradication from the stomach and can cause reinfection.[6]

THE EFFECTS OF NON-SURGICAL PERIODONTAL TREATMENT ON HELICOBACTER PYLORI INFECTION

In Non-surgical periodontal therapy the microbial deposits on the surface of the teeth were removed professionally using ultrasonic instruments and curettes together with other means of controlling plaque such as the use of mouthwashes, after detailed motivation and education of the patient concerning how to successfully carry out oral hygiene at home. This phase of the treatment is defined as being the Etiotrophic one and it is extremely important since this is the phase in which the microbial factors that trigger the disease are removed.

In a study by Zaric et al[17], 43 patients, all positive for oral and gastric Hp, were classified into two groups in which 21 patients received systemic treatment only and 22 received systemic treatment together with periodontal treatment. Three months after completing the treatment 77.3% of the patients who received both the systemic and the periodontal treatment and 47.6% of the patients who received only the systemic treatment showed gastric eradication of the infection. Among the 22 patients who received both treatments Hp was detected in the dental plaque in only 6 patients after completing the treatment, whilst the microorganism was detected in 66.7% of the 22 patients who only received the systemic treatment. The authors also reported that eradication in the stomach coincided with eradication in the oral cavity.

Jia et al[18] evaluated the effect of periodontal therapy on prevalence of H. pylori in the stomach of dyspeptic patients in whom H. pylori was eradicated from the stomach by systemic H. pylori eradication therapy prior to periodontal intervention. They reported that 6 months after periodontal therapy, the prevalence of H. pylori in the gastric mucosa was significantly lower among patients who received periodontal therapy compared with controls who did not receive any form of periodontal therapy.

In a recent study by Adachi et al in 2018 it was observed that the number of subjects positive for periodontitis was decreased among those who underwent successful eradication of H.Pylori infection.[4]

CONCLUSIONS

The oral cavity is an important extragastric reservoir of H. pylori and can serve as both a source of re-infection and route of transmission. Because plaque-associated H. pylori would be resistant to systemic antimicrobial eradication therapy, it can affect the success rates of treatment of gastric diseases. Thus, it is imperative to identify the role of dental plaque, saliva, and periodontal disease in H. pylori infection The initial studies on the role of periodontal therapy in the management of H. pylori infection have shown promising results and should be incorporated in the protocol for the management of H. pylori infections.

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