THE ORAL PRESENCE OF HELICOBACTER PYLORI AND ITS IMPLICATIONS ON ORAL CONDITION AND GASTRIC PATHOLOGY

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Abstract: *Helicobacter pylori detection in oral cavity has lead to the question whether it has a significant importance for the local pathology or for the gastro-intestinal tract. Multiple research conducted in the last twenty years have certified that H. pylori exists in the dental plaque, saliva, aphthous ulceration (RAU), but controversy still exists regarding their contribution as a principal infection or reinfection factor at the gastric level of these reservoirs of Helicobacter. The implications in local pathology seem to be especially in parodontal disease and the pathology of the mucosa.*

Cuvinte cheie:
Helicobacter pylori, tractul gastro-intestinal, parodontopatogen, PCR, cultura

Rezumat: *Detectarea prezenței Helicobacter pylori la nivelul cavității bucale a dus la întrebarea, ce implicații are asupra patologiei bucale dar şi a celei de la nivel gastro-intestinal. Cercetări multiple efectuate în ultimii 20 de ani au certificat existența H. pylori în placa bacteriană, salivă sau ulcerațiile aftoase însă există controverse cu privire la influența ca factor de reinfecție sau primoinfecție la nivel gastric ale acestor rezervoare de Helicobacter. Implicațiile în patologia locală par a fi în special în boala parodontală şi patologia mucoasei.*

INTRODUCTION

Pathogenesis and transmission

H. pylori, gram-negative, a spiral shaped bacteria, has been isolated for the first time from a gastric biopsy by Warren and Marshall in 1983. Soon after this, in 1989, Krajden isolated the bacteria from the dental plaque in the oral cavity.(1,11) H. pylori infection is among the most common bacterial infection worldwide, affecting almost half of the population, although the majority remain symptom-free. Well adapted to the acidic environment of the stomach, it plays an important role in the development of peptic ulcers, gastric ulcers, gastric carcinoma and primary gastric B-cell lymphoma.(1,10) H. pylori produces large amounts of urease, an enzyme that allows the bacteria to metabolize urea present in gastric mucosa, establishing a favourable environment by neutralization of the acid and through the generation of ammonia.

Despite numerous investigations, the modalities of infection and transmission stay unclear. It has been shown that both oral-oral,(2,9) gastric-oral (8,9) and faecal-oral (2,9) transmission occurs in humans together with predisposal factors like close personal contact, lack of hygiene and source of drinking water as well as the low socioeconomic status.(10) Humans appear to be the main reservoir in nature, and so the transfer between individuals seems to be via infected saliva or contaminated food. Taking all this into account, it would be reasonable to assume that the oral cavity plays a critical role in the process of Helicobacter pylori transmission and infection in humans.(2) There is currently no evidence for zoonotic transmission, although H. pylori is found in some nonhuman primates and occasionally in other animals. H. heilmannii is a bacteria that can be transmitted from animals such as dogs, cats, pigs and primates, but causes a mild gastritis and has a prevalence of 0.5% in humans.(12,18)

H. pylori in oral environment

Oral cavity is the gate of the gastro-intestinal tract (GIT) that is of crucial importance for the whole organism.(11) The contribution of H. pylori in the pathogenesis of periodontal disease, recurrent aphthous stomatitis, glossitis, burning mouth syndrome and mucosa lesions remains unclear.(2) The oral acidic pH is suggested to represent a favourable microenvironment for H. pylori despite the presence of an abundant bacterial flora.(3) It is, however, possible that H. pylori is transiently present in oral cavity and leads to a misbalance of the residual flora of the oral cavity. Furthermore, H. pylori might be a part of normal oral microenvironment without being pathogenic for the stomach, remaining in normal immunological balance with the host, or even protecting against other pathogens. In addition, the small number existing at this level might not be a real danger for the gastric infection. However when the immune system of the host becomes impaired, a pathogenic sense is induced.(2,4) Concerning transmission, an important role is played by the gastric acid, such as in gastritis which in acute stage is often accompanied by episodes of reflux that may facilitate the passage of the organisms into the mouth, from where they may be transmitted to other individuals.(8) Due to the fact that the infection implies an oral transit of this pathogen, the hypothesis of direct or indirect correlation between oral health status and gastric infection or reinfection with H. pylori seems to be biologically viable.

H. pylori has been detected in the oral cavity by various methods in: dental plaque, saliva, vomit, supra and sub gingival calculus, mucosal ulceration, tongue dorsum and surface of oral cancer.(4,5,6,7,8,14) Concerning the oral ulcers in recurrent aphthous ulceration (RAU) (40,41,42), as the histological features are similar to those of gastric ulcers, a relationship has been established between RAU and gastric ulcer through H. pylori detection in both cases.(5)

Because acute infection with H. pylori is generally asymptomatic, it is not possible to ascertain when infection occurs on the basis of symptoms or clinical findings.(16) Methods for diagnosis in the stomach are well established, such

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as urease test, urea breath test, histological exam, culture, seric antibodies test, stool antigen presence test and gastric biopsy. (8,17)

Detection of H. pylori in the oral cavity has been done through molecular techniques but rarely by culture.(14,15) The ability to culture H. pylori is greatly reduced when the bacteria is stressed by nutrient depletion, prolonged incubation or antibiotic treatment resulting in conversion from spiral to coccoid form. Coccoid forms are unculturable by conventional techniques, but may still be viable and so pose a risk of infection. The spiral shaped is believed to be responsible for chronic gastric infection, while coccoid form has been implicated in transmission.(13)

Most of the cases of the studies performed for the evaluation of the periodontal status were done by bleeding on probing, clinical attachment level, probing depth or at least OHI (Oral Hygiene Index), all this aspects contributing to division of lots into gingivitis group (probing depth ≤3mm) and parodontitis group(probing depth≥5mm).(2,4,20,21,22,23,)

There are two main methods accepted for detection of H. pylori in oral cavity which are culture and PCR, but each of them has disadvantages. Culture is not as sensitive as PCR because in too many cases, due to the special conditions needed for the sampling process, bacteria dies before transferring into the medium.(24-31) PCR detection however, although highly sensitive, detects the DNA, which is not a viable organism but a part of bacteria genome potentially dead resulting in false positive results.(2,19) Consequently there are many authors that consider culture, while accepting the limitations, as reflecting the presence of clinically relevant data.

CONCLUSIONS

After all these microbiological studies, the conclusion reached is that H. pylori is to be found in the oral cavity of patients with dentures mostly,(11) which have the bacteria in the gastric compartment as well. In these circumstances the question that arises is whether the oral cavity is a reservoir of H. pylori for the stomach or the stomach is the source of infection for the oral cavity. Both ways are theoretically possible taking into account the gastro-oesophageal reflux that exists in gastric infections.

The isolation of H. pylori in dental plaque has been reported by many studies and they suggest that dental plaque might be an important reservoir. Desai et al. postulated that dental plaque is not influenced by the triple antibiotic treatment necessary for gastric eradication meaning that if local treatment is not conducted the gastric reinfection is possible. This fact is due to the small concentration of antibiotics achieved in saliva and dental plaque, insufficient to affect the bacteria.(2,11,32,36-39)

The results of numerous studies revealed that H. pylori is relatively common in the oral cavity, although the character of this oral colonization and the effect on gastric infection and recidivist behaviour remains unclear. It can have a transient presence and can contribute to mucosal lesions and parodontitis as cross-reactions with Campylobacter rectus antibodies have been determined. This does not exclude the possibility that in case of immunodeficiency or disturbed oral balance, the number of bacteria can increase and be sufficient for gastric infection. Loster et al. postulate that there might be a exchanging genetic material process involved and oral cavity is just the first step for better survival of the bacteria in gastric environment.(11)

Some researchers question whether H. pylori's presence in the oral cavity is a residual bacteria composing part of the oral bio film or whether it pathogenic role.(34,35)

The association of moderate to severe parodontitis with deep periodontal pockets is of important consideration. The area around dental plaque has a low reduction-oxidation potential, which promotes the growth of facultative anaerobes. The acidic pH reduced due to the fermentation process of carbohydrates is ideal for H. pylori's growth.(33)

Despite the discordance in the literature and the fact that the problem of H. pylori's presence in the oral cavity and its gastric implications and transmission are not entirely clarified, H. pylori can represent a danger for gastric infection even for a minority of individuals. This aspect should be sufficient in order to implement a preventive approach.

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