Literature Review

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Endodontic infections and Helicobacter pylori

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Abstract

More than 500 bacterial species are found in endodontic infections. There is great variety in the species composition, diversity and quantity of microorganisms. In the presence of endodontic infection, the pulp tissue becomes favorable for the survival of bacteria that reside both permanently and transiently in the oral cavity. Thus, an opportunity is found for some important human pathogens to be able to colonize in the infected dental pulp.

H. pylori is primarily transmitted by the oral-oral and fecal-oral routes. The oral cavity is a major extragastric reservoir of the microbe. The oral Helicobacter pylori may contribute to the progression of periodontal diseases and have been associated with various oral diseases, failed eradication from the stomach and re-infection.

Multiple studies demonstrate the successful isolation of H. pylori from root canals of teeth. There are reports of widespread H. pylori in primary endodontic infection. It has been suggested that the anatomy of permanent teeth favors colonization by this microorganism, assuming the possibility that the microbes were introduced by food, water, or casual contact. Often in root canals, H. pylori has a coccoid form. Dental pulp may be a possible source of Helicobacter pylori infection in children.

Studies have found that H. pylori colonizes inflamed pulp in approximately 40% of all cases by adhesion to human dental fibroblast cells. Periapical lesions with the largest surface area showed higher numbers of gram-negative and rod-shaped species.

Further studies are needed to clarify the role of oral H. Pylori. Its presence in all parts of the oral cavity, including the endodontium, is undisputed, but its role in the course of endodontic infection has not been clarified yet. Its presence in infected root canals could serve as a reservoir for further infections and re-infections.

Keywords: endodontic infections, Helicobacter pylori, reservoir, re-infection

Introduction

Endodontic infections often have a microbial etiology and are among the most common sources of inflammatory diseases in humans. Fungi, archaea, viruses are also associated with inflammatory processes in the pulp. Microbial factors are thought to underlie a significant proportion of inflammatory diseases of both the dental pulp and the periodontium [1, 2]. Bacterial infection of the endodontium is present when the pulp is necrotic or has been removed during treatment, and may reach the peri-apical tissues. Bacteria in the endodontium are usually observed as biofilms attached to the root canal walls. More than 500 bacterial species are found in endodontic infections, but the most commonly present are about 20 to 30 species. There is great variety in the species composition, diversity and quantity of microorganisms [2,3].

The pulp can be irreversibly damaged, regardless of the route of infection penetration into it - most often through a carious lesion, but also in open endodontics, trauma or fracture, through microcracks, lateral canals, after tooth preparation and restoration [4]. However, the route of penetration of the infection into the dental pulp has implications for the type of bacterial strains prevalent in the infected root canal. There are fewer microbial species in endodontic infection compared to periodontal pockets and in the oral cavity due to the limited space and specific conditions in root canals. When the endodontium communicates with the oral cavity, the number of bacteria involved in pathological processes in it increases.

Primary an endodontic infection is caused by microorganisms that initially invade and colonize the necrotic pulp tissue. It is characterized by the presence of mixed nonspecific microflora. Gram-negative bacteria can be isolated in the early phase of periapical inflammation [5, 6]. Obligate anaerobes are often isolated in infected root canals, as in acute apical periodontitis [7]. Obligate anaerobic species are more prevalent in teeth with primary infection, whereas both anaerobes and facultative species predominate after treatment. Interactions between bacteria play a significant role in determining their overall virulence [2].
In the presence of infection in the endodontium, the pulp tissue is unable to conduct effective anti-inflammatory defense against microorganisms, and the environment becomes conducive to the survival of bacteria that reside both permanently and transiently in the oral cavity. Thus, an opportunity is opened for some important human pathogens to be able to colonize the infected dental pulp.
Helicobacter pylori is a gram-negative microorganism and an important factor in the pathogenesis of many diseases, most commonly gastrointestinal disorders (peptic ulcer, gastric carcinoma, gastric mucosal lymphoma). It is also a co-factor of metabolic, autoimmune, vascular and systemic disorders:

- in the cardiovascular system - ischemic heart disease, atherosclerosis, Raynaud's disease;
- in the nervous system - headaches and migraines;
- autoimmune diseases - arthritis, immune thrombocytopenia, Henoch-Schonline disease, Sjögren's syndrome, autoimmune thyroiditis;
- skin diseases - rosacea, urticaria, atopic dermatitis, alopecia areata, prurigo nodularis, purpura, and others - anemia, growth retardation, amenorrhea, halitosis, aphthous ulcers [8, 9, 10].

Helicobacter pylori in the oral cavity.

Data on the isolation of H. pylori from the mouth are sometimes contradictory. The microorganism is primarily transmitted by the oral-oral and fecal-oral routes. The oral cavity is a major extragastric reservoir of the microbe because of the presence of H. pylori-DNA and specific antigens in different niches of the oral cavity. This bacteria in the mouth may contribute to the progression of periodontitis and have been

associated with various oral diseases, failed eradication from the stomach and re-infection. Oral conditions are not good for its survival and little is known about its biological function in the oral cavity. It is crucial to elucidate the survival strategies of H. pylori to better understand the role and function of this bacterium in the oral cavity [11]. The isolation of H. pylori from dental plaque and saliva is of great interest to investigate the role of the oral cavity in its transmission. Virulent strains of H. pylori may be present, although rarely and probably transiently, in oral samples from patients with chronic dental or gastroduodenal disease [12].

Helicobacter pylori and Endodontic Infections

The aim of a 2012 study by Isabela et al. was to look for two human bacterial pathogens - Helicobacter pylori and Chlamydia pneumoniae in primary endodontic infections that are not potential endodontic pathogens and to test whether necrotic tissue in the root canal serves as a reservoir for these pathogens in healthy patients [13]. Neither H. pylori nor C. Pneumoniae have been detected in samples from primary endodontic infections. Bacteria colonizing necrotic root canals are also commonly members of the oral microbiota [14]. Similar results were reported by Tavares in Brazil, in a study published in 2011. Forty specimens from the largest root canal or from one with a periapical lesion present on deciduous teeth with necrosis with or without radiologically detectable peri-radicular or interradicular bone resorption were studied [15]. The results suggest that H. pylori are not endodontic pathogens and that the necrotic root canal does not serve as a reservoir in healthy patients [14].

Multiple studies clearly demonstrate the successful isolation of live H. pylori from root canals of teeth.

Kongulu et al. in 2008 reported the widespread distribution of H. pylori in primary endodontic infection of permanent teeth of children. They suggested that the anatomy of permanent teeth favors colonization by this microorganism, allowing for the possibility that microbes may have been introduced by food, water, or casual contact [15, 16].

A 2012 study examined children with early childhood caries who received dental treatment under general anesthesia. From 10 teeth with pulp necrosis or chronic apical periodontitis, an attempt was made to isolate H. pylori. The presence in the stomach of H. pylori in the children or their parents was not checked, but the patients or their parents had no complaints of gastric or abdominal pathology. Dental plaque and root samples were taken from deciduous teeth. By PCR, DNA fragments were isolated in two root canals and four dental plaque samples, suggesting that H. pylori DNA may be present in some but not all patients. Single colonies were found under microaerophilic growth conditions in two of 10 root canal samples. These two root samples were then subjected to polar-emission scanning electron microscopic examination to see if they had the typical shape of H. pylori and whether the bacteria could be visualized. Coccoid bacteria that may be H. pylori were observed. These morphological findings suggest the presence of live, spiral-shaped H. pylori in the root canals [17].

Brito et al. in 2012 [18] compared the microbiota of endodontic infections in necrotic pulp from 40 HIV-negative and 20 HIV-positive patients. The pulp was subjected to DNA hybridization and polymerase chain reaction was applied. Significant differences were found between the groups with respect to the proportions of taxa and prevalence of the species studied. Among HIV-negative patients, one of the most prevalent taxa was found to be H. pylori, and among HIV-positive individuals, D. pneumosintes, Prevotella tannerae, Porphyromonas gingivalis, Parvimonas micra, Prevotella nigrescens and Corynebacterium diphtheriae. The authors conclude that there are significant differences in the frequency and quantity of specific microbial

taxa between HIV-negative and HIV-positive individuals. The root canal microflora may represent a source of important oral and medical pathogens, mainly in HIV-positive individuals [18].
In a 2018 study Nomura et al. suggested that dental pulp may be a possible source of Helicobacter pylori infection in children. The distribution of H. pylori was investigated in 131 samples of inflamed pulp by PCR. The relationship between the detection of H. pylori and clinical information regarding endodontically infected teeth, the adhesive properties of H. pylori to human dental fibroblast cells have been examined. The results showed that H. pylori was present in 38.9% of the inflamed pulp samples. H. pylori is found predominantly in deciduous teeth, and less frequently in permanent teeth. Samples were taken twice from the same teeth at intervals of 1 or 2 weeks, and H. pylori was detected in most studies in both samples. H. pylori showed adhesive properties to human dental fibroblast cells. H. pylori colonizes inflamed pulp in approximately 40% of all cases by adhesion to human dental fibroblast cells [19].

Murad CF et al. in 2014 investigated the composition of the root canal microbiota in endodontic failures from 36 root canals with persistent endodontic infection to identify and quantify the microorganisms there with DNA hybridization. High mean levels were found for Helicobacter pylori. Periapical lesions with the largest area showed higher numbers of gram-negative and rod-shaped species [20].

Conclusion

Further studies are needed to clarify the role of oral H. Pylori. Its presence in all parts of the oral cavity, including the endodontium, is undisputed, but its role in the course of endodontic infection has not been sufficiently clarified yet. Its presence in infected root canals could serve as a reservoir for infection and re-infection.

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