Research Progress on the Relationship Between Bacterial Biofilm on Implant Surfaces and Peri-implantitis

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Abstract: Peri-implantitis is a common complication after dental implant restoration, and its occurrence is closely related to the dynamic evolution of plaque biofilm on the surface of implants. This article systematically reviews the formation mechanism and influencing factors of plaque biofilm and its pathological correlation with peri-implantitis. Studies have shown that implant surface roughness, material properties, and host immune status jointly regulate the composition and virulence of plaque biofilms. Excessive proliferation of Gram-negative anaerobic bacteria leads to inflammation and bone resorption by releasing virulence factors. Future research should focus on surface modification technology and the development of personalized maintenance strategies to reduce the risk of peri-implantitis.
***Keywords*:** peri-implantitis; dental implant restoration; plaque biofilm; review

1. Introduction

With the popularization of oral implant technology, peri-implantitis has become a major complication affecting the long-term survival rate of implants. Epidemiological data show that the incidence of periinflammation can reach 45% 15 years after implant placement, which directly leads to bone resorption and implant loosening [1]. Different from natural periodontitis, the peri-implantitis lacks the periodontal ligament structure, and the inflammation is more likely to spread to the deep tissues. As the aggregation carrier of microorganisms, plaque biofilm plays a central role in the pathogenesis of peri-implantitis. By analyzing the research results in recent years, we clarify the formation and pathogenic mechanism of plaque biofilm, and provide theoretical basis for clinical prevention and treatment.

2. Plaque biofilm on the implant surface

Plaque biofilm is a three-dimensional structure composed of oral microorganisms, extracellular polysaccharide matrix and inorganic salts, and its thickness is usually 50-200 μm. On the surface of implants, the formation of biofilm begins with the adsorption of salivary proteins, and then early colonizers such as Streptococcus and actinomycetes bind to the surface through adectin [2]. After 48 hours, anaerobic bacteria such as Porphyromonas gingivalis embedded in the deep layer of the biofilm through co-aggregation and formed stable three-dimensional communities. The metabolic activity of mature biofilm is 20-100 times higher than that of planktic bacteria, and the tolerance to antimicrobial agents is significantly enhanced. The formation process can be divided into three stages: the initial stage is 0-24 hours, and the acquired membrane of about 10 nm is formed by the rapid adsorption of proline rich proteins and amylase in saliva. Streptococcus binds membrane components through surface antigens I/II family proteins and forms a monolayer colony. At 24-72 hours after the development stage, the microorganisms secreted exopolysaccharides such as glucan and fructan to construct the community matrix, and Gram-negative bacteria such as Fusobacterium nucleatum joined the community through lectin-mediated co-aggregation. At the mature stage after 72 hours, an oxygen concentration gradient is formed inside the biofilm, and anaerobes proliferate in the deep layer and release virulence factors. At this time, the biofilm has an increased difficulty in removing antibacterial agents [3].

3. Factors affecting the formation of plaque biofilm on the implant surface

3.1 Surface characteristics of the material

The surface properties of materials are the basic conditions that affect the formation of biofilms. Surface roughness is one of the key parameters. When the surface roughness (Ra) of the implant is more than 0.2μm, the micro-structures such as thread depression are easy to form bacterial retention zones, resulting in a significant increase in bacterial adhesion. Clinical data showed that the incidence of periinflammation in rough implants (Ra=1.5μm) was 2.1 times higher than that in smooth implants (Ra=0.1μm), suggesting that surface leveling treatment may reduce the risk of infection [4]. Wettability affects

bacterial colonization by controlling the stability of saliva film. Hydrophilic surface (contact Angle <30°) can increase the amount of bacterial adhesion by 40%, while zirconia and other hydrophobic materials (contact Angle >90°) have lower plaque index than titanium alloy due to low surface wettability [5]. The biofilm-stability of Co-Cr alloy was 35% higher than that of Ti alloy due to its high porosity (>15%). However, due to its chemical inertness, the adhesion amount of Streptococcus mutans on the surface of zirconia is only 1/3 of that of titanium alloy, showing better antibacterial potential.

3.2 Microbial community characteristics

The dynamic change of microbial community is the key step in the pathogenic transformation of biofilm. Streptococcus and actinomycetes are the main flora around healthy implants, and lactic acid is the main metabolite, which maintains a relatively balanced microecology. When peri-implantitis occurs, the structure of subgingival flora is significantly unbalanced, the proportion of Gram-negative anaerobic bacteria increases, and the concentration of Porphyromonas gingivalis increases, and the content of metabolite hydrogen sulfide increases. These changes promote inflammatory progression by destroying the host immune barrier.

3.3 Host-related factors

Host systemic and local factors indirectly affect biofilm formation by regulating the microenvironment. Smoking, as an important risk factor, can increase the concentration of P. gingivalis in subgingival plaque, and its nicotine metabolites can also inhibit the function of neutrophils. The expression of inflammation-related genes is up-regulated in diabetic patients, which accelerates biofilm mediated bone resorption. In addition, the decrease of saliva flow rate will reduce the mechanical scour effect on the biofilm, which will accelerate the biofilm maturation and further increase the risk of infection.

4. The relationship between plaque biofilm on implant surface and peri-implantitis

4.1 The role of microbial virulence factors

Peri-implantitis associated flora can aggravate inflammation and tissue destruction by secreting a variety of virulence factors. Among them, lipopolysaccharide (LPS), as the main component of the cell wall of Gram-negative bacteria, can significantly promote the secretion of interleukin-6 (IL-6) by activating TLR4/NF-κB signaling pathway. Experimental data show that its secretion can be increased, and then accelerate the differentiation and activation of osteoclasts. Gingipain (Rgp), another key virulence factor, has strong proteolytic activity and can directly degrade collagen fibers and fibronectin around implants, leading to a decrease in soft tissue sealing and an increased risk of bacterial invasion into deep tissues. As nano-vesicle structures secreted by bacteria, outer membrane vesicles (OMVs) can carry virulence factors to penetrate the host cell membrane, induce the increase of osteoblast apoptosis rate, and further weaken the stability of bone tissue around implants. Although the dominant pathogens detected in different studies are different, the expression types of virulence factors can still provide an important basis for disease diagnosis.

4.2 Imbalance of host immune response

The occurrence of peri-implantitis is closely related to the imbalance of host immune defense mechanism. Clinical studies have found that the proportion of T helper 17 (Th17) cells in the lesion site is as high as 28.6%, which is significantly higher than that in the healthy control group (9.3%). Its excessive secretion of IL-17A can directly stimulate the generation of osteoclasts and accelerate the process of bone resorption. As an important part of innate immunity, neutrophils excessively release neutrophil extracellular traps (NETs) under inflammatory conditions, which leads to the increase of cathepsin G concentration, and then activates RANKL/OPG signaling pathway, which increases the ratio of RANKL/OPG, and further aggravates the imbalance of bone metabolism. In addition, the abnormal infiltration of immune cells can also lead to the continuous deterioration of the local inflammatory microenvironment, which is manifested as the massive release of pro-inflammatory factors such as TNF-α and IL-1β, forming a vicious cycle of "inflammation, tissue destruction, and aggravation of inflammation".

4.3 Biomechanical synergistic effect

Biomechanical factors play a synergistic role in the progression of peri-implantitis. Occlusal overload, such as a bite force of more than 200 N, can increase the risk of peri-implantitis. The mechanism may be related to the activation of Piezo1 ion channel by mechanical stress. The excessive activation of this channel can promote the apoptosis of osteoblasts and enhance the destructive effect of bacterial toxins on bone tissue. The surface of natural teeth is mainly composed of hydroxyapatite, while the surface of titanium implants forms a unique microenvironment due to local dissolution, and its electrostatic force and ionic bond characteristics are significantly different from those of natural teeth. This niche change

may lead to the abnormality of bacterial adhesion and colonization. This results in the enrichment of specific bacteria such as anaerobes lactic acid and Bacteroides vulgaris at the sites of peri-implant inflammation, which further aggravates the inflammatory response and tissue destruction.

5. Summary

The formation of plaque biofilm on the surface of implants is the result of the interaction of material properties, microbial characteristics and host reaction. Rough surfaces, hydrophilic materials, and systemic diseases such as diabetes significantly increase the pathogenicity of biofilms. Gram-negative anaerobic bacteria cause immune imbalance and bone resorption by releasing virulence factors and metabolites. Future research should focus on the development of surface modification technology with antibacterial function, such as nano-silver coating and photocatalytic materials. To establish a personalized maintenance program based on microbiome detection; To explore the molecular mechanism of material-microbial-host interaction. The incidence of peri-implantitis can be reduced by optimizing the implant design and postoperative maintenance strategy through multidisciplinary collaboration.

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