Review Article

BIOFILMS ON DENTAL IMPLANTS: A REVIEW

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ABSTRACT:
Oral cavity provides a more favourable environment for the growth of the microorganisms as compared to any other part of the human body by exhibiting an ideal non shedding surface. Dental plaque comprises of diverse community of the microorganisms found on the tooth surface. As implants are being placed in large number these days, clinicians may encounter more and more complications. Therefore, understanding the etiology is warranted to establish adequate diagnosis and provide proper treatment. This review highlights the biofilms in relation to the peri-implant region, and the treatment associated with it.
Key words: Biofilms, dental implants, micro-organisms.

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INTRODUCTION

Biofilm is described as relatively undefinable microbial community associated with tooth surface or any hard nonshedding material.[1] Biofilms are ubiquitous and they form on virtually all surfaces immersed in natural aqueous environment, e.g., water pipes, living tissue, tooth surface, implanted medical devices, dental implants, etc.[2] Biofilms formed on the tooth surface is called as dental plaque. Bacteria proliferating in the dental plaque form the main etiologic factors for the majority of the dental ailments, e.g., caries, gingivitis, periodontitis, and peri-implantitis. Microbial attack has been cited as the main cause of the dental implant failure.[3]

The review addresses the pathogenesis, factors affecting implant biofilm, and the treatment associated.

BIOFILM AND TOOTH

The formation of the microbial complex called biofilm in the oral cavity is a multistage journey.[4] Saliva is the main source of nutrients to the bacteria. The acquired pellicle .i.e. thin film covering the tooth, is derived from the salivary proteins and covers the enamel within seconds after brushing. Proteins and the glycoproteins are the molecules binding to the tooth surface, implants, restorations, etc., These molecules promote the adhesion and coaggregation of the oral bacteria. The bacterial adherence to the pellicle is facilitated by the special surface molecules (adhesins) chiefly lectins present on the bacterial cell surface. Intercellular bacterial adhesion and secretion of the extracellular polysaccharides, e.g., levans, dextrans, futher form the multilayered bacterial colonies suspended in the polymer matrix.[5] The microbial load in the saliva constitutes about $10^{[7]}$ bacteria per milliliter.[6] The initial colonizers are the Streptococci (S. viridens, S. mitis, S. oralis). Secondary colonizers comprises predominantly of the Actinomyces species, S. mutans, S. sobrinus. The bacteria multiply and co aggregate with the partner species. Fusobacterium nucleatum has the property to co-aggregate with multiple bacteria hence this species is an important link in the dental biofilms bridging the early and the late colonizers.[7] Two specific signalling molecules have been produced by the oral bacteria. Gram-positive bacteria communicate via small diffusible pepitide channel called as "Competence Stimulating Peptides (CST) and AI-2." AI-2 (autoinducer-2) is a popular signalling molecule exhibited by both gram-positive and gram-negative bacteria responsible for the quorum sensing. The biofilm acts as a barrier for the bacteria against host immunity and the antimicrobial agents.[8,9]

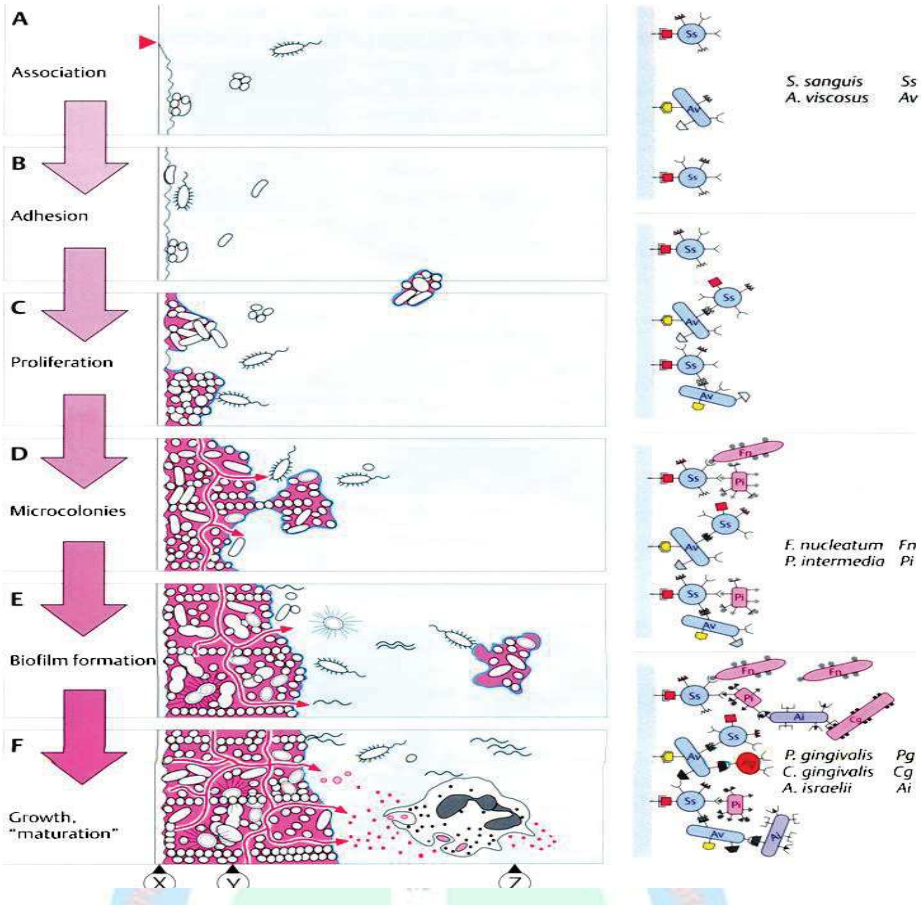

Figure 1: Stages of biofilm formation (ref Wolfe HF. Biofilm plaque formation on tooth and root surfaces. In: Wolfe, H.F. Rateitschak, K.H. (eds). Periodontology,ed 3. Stuttgart: Thieme 2005; 24)

BIOFILM AND IMPLANT

Literature shows that implants with deeper probing pockets had lesser number of coccoid and more levels of the spirochetes.[10] Biofilm formation around natural teeth occurs within minutes and the specific species start colonizing as soon as 2-6 hours. [11] The indigenous microbiota required to set the stage for the complex communities to develop, which is found in normal tooth is absent on the pristine surfaces of the implants.[12] The pellicle starts forming on the implant surface as early as 30 minutes after the implant is exposed in the oral cavity.[13]

The acquired pellicle on the dental implants owing to their lower albumin absorption capacity causes a low plaque formation around implants. Early colonizers are predominantly the gram-positive cocci, rods, and actinomyces species.[14] The periodontal pathogens colonizing on the Streptococci (P. gingivalis, P. intermedia, etc) are the causative microorganisms responsible for peri-implantitis and periodontitis.[15]

Surface roughness largely influenses the osseointegration around the dental implant.[17] However, greater is the surface roughness, higher is the rate of the biofilm formation around the implant.[16]

The attachment of the microorganisms to the hard surfaces, i.e., teeth and implants, besides their interactions with the surface components (roughness) also require certain specific characteristics of these interacting surfaces in terms of their wettability/hydrophobicity and surface free energy (SFE).[18]

The microbiota in healthy peri-implant tissues is dominated with gram positive facultative cocci and rods.[19] A substantial difference in the microbial profile of the peri-implant microflora in certain in vitro studies reveals affinity of the Staphylococcus aureus for the titanium surface but it isn't a common microflora around the teeth. S. aureus has high adhesion for titanium surfaces.[20]

BIOFILM AT THE IMPLANT – ABUTMENT INTERFACE

Implant consists of an implant abutment junction (IAJ). "Microgap" is a joint/gap between the

implant and abutment. Ericsson et al., identified two important microbiologic entities in the implant crestal region: (a) Plaque-associated inflammatory cell infiltrate (PaICT) and (b) implant-associated inflammatory cell infiltrate (IaICT).[21-23] The microgap has been reported to be as high as 40-60 μm[24] It allows micromovement during function[25] and permits microleakage of fluids congenial for bacterial growth. Several studies have reported the bacterial penetration across the implant abutment interface.[26] An in vitro analysis for the possible microleakage at the implant abutment interface was carried out which showed that after 7 days of anaerobic incubation of the partial or completely immersed implants in the medium, the microorganisms were found in both the assemblies indicating bacterial leakage at the implant abutment interface.[27]

Furthermore, when the implants are in contact with plasma or saliva, proteins can direct the attraction or repulsion of bacteria present on external layers since proteins have different degrees of hydrophobic to hydrophilic regions. The main salivary protein adsorbed to titanium in vivo and in vitro is albumin [29,30], and albumin adsorption to titanium occurs through calcium (Ca+2) bridges.[31] The negative charge from titanium dioxide may attract positive ions, such as Ca+2 and its presence thus increases the adhesion of some bacteria species. Hauslich et al.[32] 2012, demonstrated that pretreatment of titanium surfaces with Ca+2 ions increased the adhesion of S. mutans and F. nucleatum to the Ti surfaces, but did not influenced the P. gingivalis adhesion. F. nucleatum possesses Ca+2-dependent binding proteins on the cell surface similar to those of S. Mutans.[33] These findings indicated that the divalent ion Ca+2 may serve as a bridging agent in the adhesion of bacteria to Ti surfaces.

Bacteria can detect the non-biological substrate and express different genes, probably as part of the adaptation to a new microenvironment. The differences in the depth and viability of the biofilms on the different materials are a result of physical and chemical properties that determine gene expression profiling of bacteria, regardless of film formation.[28]

SURFACE ROUGHNESS

Osseointegration of dental implants is associated with increased surface roughness of the dental implant.[35,36] Conversely, a higher surface roughness with a Ra value >0.2μ increases biofilm formation[37,38] and thus contributes to spontaneous progression of periimplantitis lesions.[34,39–40] Berglundh et al[40] showed that increased plaque and faster progression of peri-implantitis were found in rough surface compared with polished machined surface implants. Berglundh et al, Amarante et al also found that machined surface implants harbored significantly less bacteria than plasma-sprayed implants and had increased amount of Streptococcus sp. compared with brushed surfaces. With increasing abutment surface roughness, higher supramucosal plaque accumulation is noted.[41] Quirynen et al showed that abutments with a rough surface harboured more bacterial pathogens and less coccoid microorganisms than that on smooth surfaces. The increase in bacterial pathogens is not observed in submucosal areas, suggesting that periodontal pathogens in this area were more influenced by the patient's oral hygiene rather than surface texture.[41,42]

Anti-infective protocol forms the mainstay of the treatment of dental implant associated infections that can be achieved through mechanical debridement of the implant surface or chemical treatment including local and systemic antibiotics. The selected treatment modality depends on the established diagnosis of peri-implant mucositis or periimplantitis. Treatment success is assessed using outcome measures such as reduction of inflammation, probing depth, and pathogenic bacteria. The presence of specific bacteria had little or no value in predicting treatment failure. Recent literature quotes that nonsurgical mechanical therapy was effective in treating peri-implant mucositis with improved results observed in conjunction with an antimicrobial mouth rinse. A reduction in the proportion of pathogenic species after mechanical therapy has been reported. Implant complications have serious health and financial implications to both the patient and clinician. Oral biofilm forms one of the major etiologic agent in periimplantitis. Studies shows that combination of multiple pathogenic bacteria increases the risk of peri-implant diseases and can better determine disease activity rather than the identification of a single microorganism. The reduction of the bacterial load to a level compatible with health is an important aspect of implant therapy. With the advancement of various diagnostic microbiologic technologies, identification of bacteria in the oral cavity continues to improve. [43]

CONCLUSION:

Increase in surface roughness and surface free energy facilitates biofilm formation on dental implant and abutment surfaces. Future control and

treatment of biofilm research will affect the success rate of dental implants.

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