SCIENTIFIC ARCHIVES OF DENTAL SCIENCES

Volume 2 Issue 7 July 2019

Review Article

Implications of Microbial Biofilm on Dental Implants

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Received: June 17, 2019; **Published:** June 26, 2019

Abstract

Bacteria and fungi have the ability to adhere to abiotic surfaces and form a structured communities called biofilms. Microbial biofilms play an important role in the society, biofilms can thrive on virtually any surface, leading to various detrimental effects, in addition to its impact on healthcare contributing to hospital-acquired infections and infection of implantable medical devices. In this article, the implications of biofilm infection will be highlighted in particular reference to dental implants.

Keywords: Biofilms; Implant-Associated Infection; Peri-Implantitis

Introduction

Bacteria and fungi have the ability to adhere to abiotic surfaces and form a structured communities called biofilms, so these are considered an imperative virulence factors of the microbial pathogens. The interest in microbial biofilm research has dramatically increased in the past few decades motivated by its important role in the society and by the elegant complex features of these multicellular communities. Biofilms can thrive on virtually any surface and may be beneficial or detrimental according to the community's interplay and the surface [1]. The detrimental effects of biofilms may include but not limited to; food and pharmaceutical spoilage, as well as its impact on healthcare contributing to hospital-acquired infections and infection of implantable medical devices [2]. This article aims to discuss the implications of biofilm infection in particular reference to dental implants.

Biofilm formation and dental implants

The majority of bacteria and fungi have the tendency to produce a biofilm. Recent research indicates that several species are particularly prone to form biofilms, such as gram-positive cocci, *Staphylococcus epidermidis*, gram-negative rod, *Pseudomonas aeruginosa* and a yeast-like fungus, *Candida parapsilosis* [3-5].

Biofilm consists of three-dimensional clusters of sessile microorganisms surrounded by hydrated, extracellular polymeric substances which are secreted by the microorganisms following its attachment on the surface. It consists principally of polysaccharides, nucleic acids, proteins and lipids and constitutes between 50 and 90% of the mass of the biofilm. It could be single or multispecies microbial biofilm [6]. Bacteria prefer biofilm formation as a method of growth because they facilitate exchange of nutrients and safeguard the bacterial community against competing microorganisms and adverse conditions [7].

Biofilms are formed on teeth surfaces as dental plaque. Molecular biological techniques have identified about 1000 different bacterial species in the dental biofilm, it is worth noting that not all bacteria in dental plaque contribute to pathologic condition as diseases will only develop when specific pathogens become numerically dominant in dental plaque . Biofilms also form in the paranasal sinuses, on prostheses, and on dental implants, constituting a particular risk for severely immunocompromised patients [7,8].

Microbial cells adhere to surfaces in four main steps:

(I) Random transport of bacteria to the surface,

(II) Initial reversible adhesion,

(III) Irreversible adhesion to the surface,

(IV) Colonization of the surface by multiple microbial cells and biofilm formation (growth and maturation).

The initial phase of biofilm production is adhesion of microbial cells to the surface, either teeth or prostheses. These surfaces are

covered by acquired pellicle derived from components in the saliva, as well as bacterial and host tissue products, allowing reversible non-specific interaction between the microorganisms and the surface due to van der Waals, electrostatic and hydrophobic forces. The major primary colonizers are *Streptococcus mutans*, they have different bacterial adhesins responsible for adhesion to the acquired pellicle [8,9].

In the next phase a specific reaction takes place between the bacterial adhesins and the surface of the acquired pellicle. The adherence of microbial cells to the surface for a long period of time makes this bond irreversible [10]. The degree of adherence depends on microbial species, number of cells, speed of liquid flow, and physicochemical properties of the surface. Subsequently, the microorganisms produce the extracellular polymeric matrix, followed by the maturation and growth of biofilms through the increase in the number of layers of the biofilm depending on the speed of liquid flow, the content of nutrients, availability of iron, pH, osmolarity, oxygen content and ambient temperature. In this manner, microcolonies are formed and maturation of the biofilm follows [11,12]. Quorum sensing is a system of communication between the microorganisms that favours their persistence in the biofilm. It is mediated by a competence stimulating peptide released upon exposure to low pH initiating a coordinated protective response [13].

The basic principles of biofilm formation are equally applicable in context to dental implants as they provide encouraging grounds for bacterial adhesion due to their surface irregularities which facilitate the adherence and maturation of biofilms [14].

After dental implant exposed in the oral cavity through a transmucosal abutment, an acquired pellicle is formed on the implant surface through selective adsorption of the environmental macromolecules such as amylase and serum albumin. Compared to natural teeth, acquired pellicle formed on dental implants has a lower albumin adsorption capability, which may contribute to the lower plaque formation around dental implants [15,16].

The transition of the peri-implant mucosa from health to mucositis and to peri-implantitis is due to the loss of the protective permucosal seal of the soft tissue to the implant surface exposing the base of the sulcus to the penetration of chemical and bacterial substances. This shift from healthy to diseased state is accompanied by differences in bacterial numbers and morphotypes. There is a transition from a gram-positive facultatived ominated flora to a gram-negative anaerobic biofilm. It was found that the failing implant is characterized by a greater proportion of red; *Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythia* and orange; *Prevotella intermedia* and *Fusobacterium nucleatum* complex, in addition to *Aggregatibacter actinomycetemcomitans* and *Eikenella corrodens* accompanied with a reduction in the proportion of microbial flora associated with health. Other pathogens were reported to be associated with peri-implantitis such as *Staphylococcus aureus, Candida albicans*, these opportunistic microorganisms have a high adhesion to titanium surfaces [17-23].

Implications and Future Direction

Microbial biofilms have the ability to cause pathogenic process even in anatomically distant sites due to the detachment of its fragments containing aggregates of bacterial cells, production of endotoxin, evasion of the immunological response of the host, along with formation of a niche for replication of bacterial cells resistant to antibiotics. The most important dilemma of bacterial biofilms is that it is significantly less susceptible to antimicrobial therapy and host defences than the planktonic (free-floating) forms of the same organisms because the structure of the biofilm inhibits the access of antibodies, lysozyme, lactoferrin and granulocytes to the microorganisms forming it, which favours their persistence [24,25]. The penetration of antimicrobial agents into deeper layers of a biofilm is poor. Some antibiotics target the cells which are actively dividing, therefore the metabolically less active cells in the deeper layers of the biofilm may be more resistant to antimicrobial agents, compared to the cells closer to the biofilm surface or planktonic cells. Thus, antimicrobial therapy eradicated only planktonic cells, while cells adhering to the surface are able to replicate within the biofilm and may continue to spread even after antibiotic therapy is completed [26].

This is manifested as therapeutic problems, where antimicrobial therapy may modulate acute exacerbations of disease but once antibiotics are withdrawn the biofilm is rapidly repopulated from cells that persisted during the treatment resulting in recurrent infections and chronic low-grade inflammation, rendering biofilms difficult to eradicate and imposing the removal of the infected implant [27].

At the present time, vigilant attention to surgical technique and the use of peri-operative antibiotics are the mainstay approaches. Nevertheless, there is substantial current and future research

trend on the use of enhanced anti-biofilm surface coatings, and other methods to inhibit biofilm colonisation and adhesion to implant surface in addition to specific anti-biofilm therapies [2,28].

Conclusion

It is imperative to consider the implications of microbial biofilm infection on dental implants, as it is a major cause of therapeutic issues and implants' failure. This resulted in more research dedicated to the use of anti-biofilm surface coatings, and methods to inhibit biofilm adhesion to implant surface, in addition to specific anti-biofilm therapies.

Disclosure

No conflicts of interest.

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