Microbiology of Dental Implants: A Review of the Literature

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ABSTRACT

Root form dental implants have a high success rate and are commonly used for replacement of missing teeth, however failures occasionally occur, such implants must be removed. Like teeth, dental implants also establish microflora soon after placement and stable implants showed no significant shifts in the composition, whereas failing implants showed presence of Gram-negative anaerobic bacteria. This article reviews the microflora associated with dental implants.

Keywords: Microflora, Peri-implantitis.


Source of support: Nil

Conflict of interest: None declared

INTRODUCTION

With the introduction of dental implants, replacement of missing teeth became long lasting treatment modality, providing functional and esthetic integrity, making dental implant treatment more advanced and ameliorated. However, at least 10% of the failures have been suggested to be the result of peri-implantitis. Implant failure has been defined as the inadequacy of the host tissue to establish or to maintain osseointegration, and peri-implantitis has been defined as the inflammatory process affecting the tissue around an osseointegrated implant in function, resulting in loss of supporting bone.

Dental plaque is a diverse microbial community, embedded in a matrix of host and bacterial polymers, growing on teeth as a biofilm. There is sufficient evidence supporting the view that periodontal pathogens, mainly those belonging to the group of Gram-negative anaerobic rods, play an important role in developing peri-implantitis. This article provides a comprehensive review of the studies published in national and international peer-reviewed literature published in English concerning the microflora around dental implants.

A healthy gingival sulcus contains predominantly of gram-positive cocci and rods, principally Actinomyces naeslundii (14%), Actinomyces gerencseriae (11%), Streptococcus oralis (14%) and Peptostreptococcus micros (5%). Gram-negative anaerobic rods account for 13% of the total cultivable organisms on average. With the development of periodontitis, microflora shifts, containing higher number of Gram-negative rods and decreased proportions of Gram-positive species. In an established periodontal lesion, low numbers of cocci and high numbers of motile rods and spirochetes are seen. Increased proportions of Porphyromonas gingivalis, Bacteroides forsythus, and species of Prevotella, Fusobacterium, Campylobacter and Treponema have been detected. However, Danser et al noted that when all teeth are extracted in patients with periodontitis, A. actinomycetemcomitans and P. gingivalis are no longer detectable within a month after full-mouth tooth extraction, but bacteria like P. gingivalis, T. forsythensis, and other pathogenic bacteria that were present before the teeth were extracted reemerge after 6 months of implant placement. These results indicate that bacteria that cause periodontitis also cause peri-implantitis. It is also suggested that the higher the full-mouth clinical probing pocket depth and the greater the full-mouth attachment loss, the higher the attachment loss is to be expected around implants in the susceptible patient. Also, according to a classic postulate of Koch- states that transfer of bacteria from one locus to another can cause the same disease in the other locus, whether this is between or within subjects. Medium of transfer of infection in oral cavity is saliva.

Dental Implant Plaque

Peri-implant microflora is soon established after implant placement and is largely influenced and depends on the presence of teeth. In edentulous patients, the subgingival area around implants consists mainly of Gram-positive facultative cocci and nonmotile rods. On clinically stable implants, S. sanguis and Streptococcus mitis are the most predominant organisms, while motile rods, spirochetes, fusiforms, and filaments are infrequently found. In partially edentulous patients, the total number of peri-implant microorganisms is increased, and the proportion of motile rods, spirochetes, and cocci is increased when compared to edentulous patients. According to Quirynen et al there is an increase in spirochetes and motiles around the implants in proportion of cocci, if the flora of the remaining teeth harbored more than 20% spirochetes. Different implant characteristics might display difference in microbiota (i.e. surface roughness, material, shape), however, studies by Alcoforado et al, Rams et al and Mombelli et al did not show any relation between specific implant system and microbiota around it.
Astrand et al\textsuperscript{14} found that rough-surfaced implants had a higher incidence of peri-implantitis than smooth (turned) surfaces, whereas, Wennstrom et al\textsuperscript{15} reported similar bone level changes for turned and relatively rough surface implants. Nakoa et al\textsuperscript{16} collected microbial samples from patients with 2 to 10-week-old implants and concluded that few microbes like \textit{A. odontolyticus}, \textit{E. corrodens}, \textit{H. actinomycetemcomitans}, \textit{P. micros}, \textit{C. sputorum} and \textit{L. buccalis} are exclusively found in implant related microbiota. Table 1A lists subgingival plaque related to implants and Table 1B lists supragingival plaque associated with implants.\textsuperscript{1} Out of all the microbes \textit{S. mitis} and \textit{S. oralis} are predominant streptococcal and colonize within first 24 hours of plaque formation.\textsuperscript{17}

### Microbiota-related to Failing/Failed Implant

Implants can be either described as failing or failed. Broadly, a failing implant demonstrates progressive loss of supporting bone structure but is clinically immobile, whereas a failed implant is clinically mobile or has explanted spontaneously. They can also be distinguished as late and early failures.\textsuperscript{2} Late failures can be divided into two subgroups, with one including implants failing during the first year of loading (‘soon’ late failures) and one including implants failing in subsequent years (‘delayed’ late failures).\textsuperscript{2} A number of risk indicators such as (i) poor oral hygiene, (ii) a history of periodontitis, (iii) diabetes and (iv) smoking have been identified which cause peri-implantitis.\textsuperscript{18} According to Shaffer et al\textsuperscript{19} (1998), implant sites with a history of endodontic infection or proximal to teeth with endodontic infection may increase the risk of implant failure. According to Malmstrom et al\textsuperscript{20} and Fardal et al\textsuperscript{21} implants placed in patients with a history of refractory periodontitis probably are at an increased risk of failure, as the chance to harbor periodontal pathogens is higher in such patients. Quirynen et al\textsuperscript{22} reported that initial subgingival colonization of implants with bacteria associated with periodontitis can occur within 2 weeks in partially edentate patients. Furthermore, when Shibli et al\textsuperscript{23} compared the microflora around implants that manifested peri-implantitis and those that were healthy, it was noted that the same types of bacteria were present around diseased and healthy implants; but an increased quantity of bacteria was found at diseased sites. Karoussis et al\textsuperscript{24} reported that patients with a history of periodontitis manifested significantly greater probing depths, more peri-implant marginal bone loss, and a higher incidence of peri-implantitis.

Also, implants which display a gap between implant and abutment permits new bacterial colonization.\textsuperscript{25} Although this gap is as small as a dental filling or a crown, there is microleakage, which permits bacterial penetration.\textsuperscript{26} The presence of a microgap between the implant and abutment has a direct influence for crestal bone levels around

### Table 2: Microbiota of failing implant

- \textit{Prevotella intermedia}
- \textit{P. nigrescens}
- \textit{Actinobacillus actinomycetemcomitans}
- \textit{Staphyloccoci, coliforms, candida spp}
- \textit{Bacteroides forsythus}
- \textit{Spirochetes}
- \textit{Fusobacterium spp}
- \textit{Peptostreptococcus micros}
- \textit{Porphyromonas gingivalis}
- \textit{Bacteroides spp}
- \textit{Fusiform bacilli, motile and curved rods}
- \textit{Staphylococcus spp}
- \textit{P. nircens, P. micros}
- \textit{Fusobacterium nucleatum}
- \textit{Actinobacillus actinomycetemcomitans}
- \textit{Capnocytophaga spp}
- \textit{Eikenella corrodens}
- \textit{Porphyromonas gingivalis}
- \textit{Campylobacter rectus}
- \textit{Trepomonema denticula}
- \textit{Tannella forsythia}
- \textit{Streptococcus anginosus (milleri) group}
- \textit{Enterococcus spp}
- \textit{Yeast spp}
implants. King et al27 have shown that a more apical or coronal position of microgap can determine an increase or decrease of bone loss. The reason for this reaction may be related to the presence of microbial colonization at the level of the interface.

Diseased sites harbor a microbiota of Gram-negative anaerobic rods, including black pigmented organisms and surface translocators. In deep pockets of peri-implant tissue A. actinomyctemcomitans and Bacteroidaceae spp. can be commonly found. Failing or failed implants show significantly elevated levels of spirochetes, and also contain P. gingivalis, P. intermedia, Peptostreptococcus micros, Wolinella recta, Fusobacterium sp., A. actinomycetemcomitans, capnocytophaga sp., Treponema denticola and Candida albicans.28,29 Table 2 lists the microbiota related to failing dental implants.

CONCLUSION

Peri-implantitis is multifactorial; however, bacterial pathogens play an important role. Microbiota of periodontitis also causes peri-implantitis, nonetheless a periodontal patient who has been treated and is receiving periodontal supportive therapy can be a candidate to receive dental implants if there are no systemic contraindications for therapy.

REFERENCES


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