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REVIEW ARTICLE

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.

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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.[1] However, at least 10% of the failures have been suggested to be the result of peri-implantitis.[2] Implant failure has been defined as the inadequacy of the host tissue to establish or to maintain osseointegration,[3] 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.[4]

Dental plaque is a diverse microbial community, embedded in a matrix of host and bacterial polymers, growing on teeth as a biofilm.[1] 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.[2] 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.[2] 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[5] noted that when all teeth are extracted in patients with periodontitis, *A. actinomyce-temcomitans* 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.[6] 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.[1]

Dental Implant Plaque

Peri-implant microbiota 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.[7] 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.[8,9] According to Quirynen et al[10] 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,[11] Rams et al[12] and Mombelli et al[13] did not show any relation between specific implant system and microbiota around it.

Astrand et al[14] found that rough-surfaced implants had a higher incidence of peri-implantitis than smooth (turned) surfaces, whereas, Wennstrom et al[15] reported similar bone level changes for turned and relatively rough surface implants. Nakoa et al[16] collected microbial samples from patients with 2 to 10-week-old implants and concluded that few microbes like *A. odontolyticus*, *E. corrodens*, *H. actinomycetemcomitans*, *P. micros*, *C. sputorum* and *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.[1] Out of all the microbes *S. mitis* and *S. oralis* are predominant streptococcal and colonize within first 24 hours of plaque formation.[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.[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).[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.[18] According to Shaffer et al[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[20] and Fardal et al[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[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[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[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.[25] Although this gap is as small as a dental filling or a crown, there is microleakage, which permits bacterial penetration.[26] The presence of a microgap between the implant and abutment has a direct influence for crestal bone levels around

Table 1A: Subgingival plaque associated with implants

Subgingival plaque	
Gram-positive bacteria	*Gram-negative bacteria*
S. sanguis	
Haemophilus spp	*S. songuis*
S. mitis	*H. actinomycetemcomitans*
S. morbillorum	*Copnocytophago spp*
S. milleri	*E. corrodens*
Streptococcus spp	*F. nucleatum*
P. micros	*Bacteroides spp*
A. viscosus	*C. sputorum*
A. naeslundii	*V. parvula*
A. israelii	*L. buccalis*
A. odontolyticus	
Lactobacillus spp	

Table 1B: Supragingival plaque associated with implants

Supragingival plaque	
Gram-positive bacteria	*Gram-negative bacteria*
S. sanguis	HS
Haemophilus spp	HS
S. mitis	*H. actinomycetemcomitans*
S. salivarius	*Capnocytophaga* spp
S. morbillorum	*E. corrodens*
S. cremoris	*F. nucleatum*
S. milleri	*Bacteroides* spp
Streptococcus spp	*L. buccali*
G. haemolysans	*V. parvula*
P. micros	
A. odontolyticus	
Lactobacillus spp	

Table 2: Microbiota of failing implant

- *Prevotella intermedia*
- *P. nigrescens*
- *Actinobacillus actinomycetemcomitans*
- *Staphylococci, coliforms, candida* spp
- *Bacteroides forsythus*
- *Spirochetes*
- *Fusobacterium* spp
- *Peptostreptococcus micros*
- *Porphyromonas gingivalis*
- *Bacteroides* spp
- *Fusiform bacilli, motile and curved rods*
- *Staphylococcus* spp
- *P. nicrescens, P. micros*
- *Fusobacterium nucleatum*
- *Actinobacillus actinomycetemcomitans*
- *Capnocytophaga* spp
- *Eikenella corrodens*
- *Porphyromonas gingivalis*
- *Campylobacter rectus*
- *Treponema denticola*
- *Tannerella forsythia*
- *Streptococcus anginosus (milleri) group*
- *Enterococcus* spp
- *Yeast* spp

implants. King et al[27] 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. actinomycetemcomitans* 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[1] 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

1. Mahesh L, Narayan TV, Kurtzman G, Shukla S. Microbiology of peri-implant infections. Smile Dent J 2011;6:54-57.
2. Heydenrijk K, Meijer HJA, Van Der Reijden WA, Raghoebar GM, Vissink A, Stegenga B. Microbiota around root-form endosseous implants: A review of the literature. Int J Oral Maxillofac implants 2002;17:829-38.
3. Esposito M, Hirsch JM, Lekholm U, Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (I). Success criteria and epidemiology. Eur J Oral Sci 1998;106:527-51.
4. Mombelli A, Lang NP. The diagnosis and treatment of peri-implantitis. Periodontol 2000:1998;17:63-76.
5. Danser MM, Van Winkelhoff AJ, Van Der Velden U. Periodontal bacteria colonizing oral mucous membranes in edentulous patients wearing dental implants. J Periodontol 1997;68(3):209-16.
6. Klinge B, Hultin M, Berglundh T. Peri-implantitis. Dent Clin N Am 2005;49:661-76.
7. Mombelli A, Lang NP. Microbial aspects of implant dentistry. Periodontol 2000;1994;4:74-80.
8. Dharmar S, Yoshida K, Adachi Y, Kishi M, Okuda K, Sekine H. Subgingival microbial flora associated with Brånemark implants. Int J Oral Maxillofac Implants 1994;9:314-18.
9. Papaioannou W, Quirynen M, Nys M, van Steenberghe D. The effect of periodontal parameters on the subgingival microbiota around implants. Clin Oral Implants Res 1995;6:197-204.
10. Quirynen M, Papaioannou W, Van Steenberghe D. Intraoral transmission and the colonization of oral hard surfaces. J Periodontol 1996;67:986-93.
11. Alcoforado GA, Rams TE, Feik D, Slots J. Microbial aspects of failing osseointegrated dental implants in humans. J Parodontol 1990;10:11-18.
12. Rams TE, Roberts TW, Feik D, Molzan AK, Slots J. Clinical and microbiological findings on newly inserted hydroxyapatite-coated and pure titanium human dental implants. Clin Oral Implants Res 1991;2:121-27.
13. Mombelli A, Marxer M, Gaberthuel T, Grunder U, Lang NP. The microbiota of osseointegrated implants in patients with a history of periodontal disease. J Clin Periodontol 1995;22: 124-30.
14. Astrand P, Engquist B, Anzen B, et al. A three-year follow-up report of a comparative study of ITI dental implants and Brånemark system implants in the treatment of the partially edentulous maxilla. Clin Implant Dent Relat Res 2004;6(3): 130-41.
15. Wennström JL, Ekestubbe A, Gröndahl K, et al. Oral rehabilitation with implant-supported fixed partial dentures in periodontitis-susceptible subjects. A 5-year prospective study. J Clin Periodontol 2004;31:713-24.
16. Nakou M, Mikx FHM, Oosterwaal PJM, Kruijsen JCWM. Early microbial colonization of permucosal implants in edentulous patients. J Dent Res 1987;66:1654.
17. A Leonhardt, J Olsson and G Dahlén. Bacterial colonization on titanium, hydroxyapatite, and amalgam surfaces in vivo. J Dent Res 1995;74:1607.
18. Lindhe J, Meyle J. Peri-implant diseases: Consensus report of the sixth European workshop on periodontology. J Clin Periodontol 2008;35:282-85.
19. Shaffer M, Juruaz D, Haggerty P. The effect of periradicular endodontic pathosis on the apical region of adjacent implant. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86: 578-81.
20. Malmstrom HS, Fritz ME, Timmis DP, Van Dyke T. Osseointegrated implant treatment of a patient with rapidly progressive periodontitis. A case report. J Periodontol 1990;61:300-04.
21. Fardal O, Johannessen AC, Olsen I. Severe, rapid progressing peri-implantitis. J Clin Periodontol 1999;26:313-17.
22. Quirynen M, Vogels R, Peeters W, et al. Dynamics of initial subgingival colonization of pristine peri-implant pockets. Clin Oral Implants Res 2006;17(1):25-37.
23. Shibli JA, Melo L, Ferrari DS, et al. Composition of supra- and subgingival biofilm of subjects with healthy and diseased implants. Clin Oral Implants Res 2008;19(10):975-82.
24. Karoussis IK, Kotsovilis S, Fourmousis I. A comprehensive and critical review of dental implant prognosis in periodontally compromised partially edentulous patients. Clin Oral Implants Res 2007;18(6):669-79.
25. Quirynen M, Van Steenberghe D. Bacterial colonization of the internal part of two-stage implants: An in vivo study. Clin Oral Impl Res 1994;5:239.
26. Proff P, Steinmetz I, Bayerlein T, Dietze S, Fanghänel J, Gedrange T. Bacterial colonization of interior implant threads with and without sealing. Folia Morphol (Warsz) 2006;65: 75-77.

27. King GN, Hermann JS, Schoolfield JD, et al. Influence of the size of microgap on crestal bone changes around titanium implants: A histometric evaluation of unloaded nonsubmerged implants in the canine mandible. J Periodontol 2001;72: 1372-83.
28. Leonhardt A, Olsson J, Dahlén G. Bacterial colonization on titanium, hydroxyapatite, and amalgam surfaces in vivo. J Dent Res 1995;74:1607.
29. Tanner A, Maiden MJF, Lee k, Shulman LB, Weber HP. Dental implant infections. Clinical Infectious Diseases 1997;25(2): S213-17.

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