Microbiology of Peri-Implant Infections

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Abstract

The oral cavity has numerous microbes, providing us with a balanced microbial environment, however conditions like periodontitis sometimes prevail leading to bone loss and eventually causing tooth loss if not treated appropriately. By the same token,even with implants, gram positive facultative flora, establishes shortly after implant placement and stable implants showed no significant shifts in the composition, where as failing implants showed presence of Gram-negative anaerobic bacteria, particularly fusobacteria, spirochetes, and black-pigmenting organisms such as Prevotella intermedia. Which leads to destruction of the peri-implant apparatus and eventual loss of the implant if not attended to in a timely fashion. It is important to understand the microbiological aspects of peri-implant disease in order to proffer appropriate treatment.

Keywords: Peri-implantitis, Implant failure, Bacterial flora.

Introduction

Since long man has been searching for 'cell-friendly' materials and with Branemak's discovery of titanium being one of them, dental implant treatment became more advanced and ameliorated. The Industry started searching for new and better implant surfaces and introduced various textured implants, with different degree of roughness,with the aim to improve their interaction with bone and osseointegration. These roughened surfaces unfortunately, were found to attract more plaque and microbial activity. With time pocket formation and soft tissue attachment loss exposes implant surface for further microbial colonization, leading to implant failure.

Dental plaque is a diverse microbial community, embedded in a matrix of host and bacterial polymers, growing on teeth as a biofilm.[1] Antonius Van Leeuwenhoek in 7th century detected mobile and immobile bacteria and the influence of oral hygiene on the bacterial composition of the dental plaque. There are over 700 different species of microorganisms which have been identified as inhabitants of the oral cavity.[1]

Unfortunately, not all of this microflora of oral cavity can be cultured. All surfaces in the oral cavity are continuously covered with a pellicle, which is a selective precipitation of glycoproteins from the saliva onto the hard surfaces including dental implants.[1]

Dental Implant Plaque

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[2] or has explanted spontaneously. Implant failures can also be categorized as early or late. Early failures occur before Osseointegration and prosthetic rehabilitation has taken place while late failure occurs after the implant has been loaded with a prosthesis. Late failures can also be sub-classified as late-early or late-delayed. The cause of late failure may be marginal infection/disease or overload.[2]

Peri-implant infections are classified as peri-implant mucositis and prei-implantitis, depending upon the severity of infection. Peri-implant mucositis is defined as a reversible inflammatory reaction in the soft tissues surrounding an implant. However, prei-implantitis is an inflammatory reaction with loss of supporting bone in the tissue surrounding an implant.[3]

There is vast literature present concluding that microbiota associated with healthy peri-implant tissues closely resembles that of healthy gingiva, and the organisms associated with mucositis are very similar to that of gingivitis and that of peri-implantitis are same as adult periodontitis.[3,4] As soon as the implant is placed there is a sulcus formation around it. As a consequence of the sulcus, oral microbial colonization and biofilm development on dental implants and teeth exhibit shared characteristics, both in health and disease. Also, a classic postulate of Koch – transfer of abscesses supports the

(Table 1) *Showing supra and Subgingival plaque in implant patients*

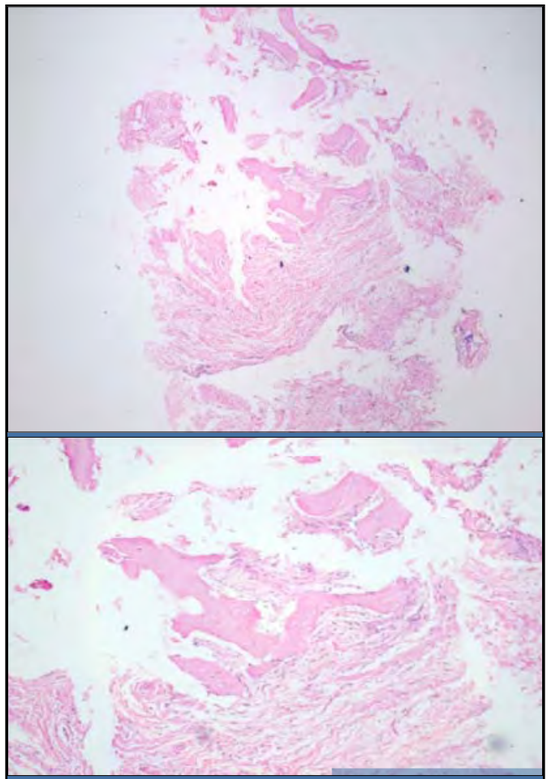
Subgingival Plaque		Supragingival Plaque	
Gram positive bacteria	**Gram negative bacteria**	**Gram positive bacteria.**	**Gram negative bacteria**
S. sanguis		*S. sanguis*	*H.S.*
Haemophilus spp.	*S. sanguis*	*Haemophilus spp.*	*H.S.*
S. mitis	*H. actinomycetemcomitans*	*S. mitis*	*H. actinomycetemcomitan*
S. morbillorum	*Capnocytophaga spp.*	*S. salivarius*	*Capnocytophaga spp.*
S. milleri	*E. corrodens*	*S. morbillorum*	*E. corrodens*
Streptococcus spp.	*F. nucleatum*	*S. cremoris*	*F. nucleatum*
P. micros	*Bacteroides spp.*	*S. milleri*	*Bacteroides spp*
A. viscosus	*C. sputorum*	*Streptococcus spp.*	*L. buccali*
A. naeslundii	*V. parvula*	*G. haemolysans*	*V. parvula*
A. israelii	*L. buccalis*	*P. micros*	
A. odontolyticus		*A. odontolyticus*	
Lactobacillus spp.		*Lactobacillus spp.*	

(Table 2) *Showing shift of microbiota from healthy to diseased pocket*

Healthy Pocket	To	Diseased Pocket
Gram +ve		**Gram -ve**
Cocci		Rods
Immobile		Motile
Facultative anaerobe		Strict anaerobe
Fermentative		Proteolytic

(Table 3)

Most prevalent microbes associated with failing/failed implants
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
Steptococcus anginosus (milleri) group
Enterococcus spp.
Yeast spp.



These two slides show infiltration of inflammatory cells at peri-implant soft tissue due to presence of bacteria

occurrence of same microflora in healthy and diseased gingiva and peri-implant soft tissue. It 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. Klinge *et al.*[5] also support this theory of propogation of infection from periodontopathic bacteria of natural teeth into saliva to the vicinity of implants. Devides and Franco[6] concluded that oral microbial colonization and succession of these microorganisms in the peri-implant sulci occurs as a function of time in the oral environment.

According to the postulate of Koch, biofilm formation will take place on this surface exposed to the oral environment, immediately after surgery. Thus when an implant is exposed to oral cavity certain bacteria accumulate on the implant surface, enabling in its stability. Mombelli[7] identified them as coccoid cells over 85% and Gram positive facultative cocci over 80%. Fusobacteria and black pigmenting Gram-negative anaerobes were found infrequently. Mombelli and Mericske- Stern[8] in edentulous patients found facultatively anaerobic cocci, facultatively anaerobic rods, *Fusobacterium sp.* and *Prevotella intermedia*.

Nakoa *et al.*[9] collected microbial samples from patients with 2-10 week old implants and concluded that few microbs like *A. odontolyticus*, *E. corrodens*, *H. actinomycetemcomitans*, *P. micros*, *C. sputorum* and *L. buccalis* are exclusively found in implant related microbiota. These microbes are potential periodontal pathogens but their early colonization is of importance for the success of implants is unknown. Microflora of Supragingival and subgingival plaque is summarized in **table 1**.[9]

Out of all the microbes *S. mitis* and *S. oralis* are predominant streptococcal and colonize within first 24 hours of plaque formation.[10] Microflora changes when the healthy stable pocket of 3mm changed into diseased pocked leading to failure of implant. **Table 2** shows shift in bacterial flora from healthy to diseased pocket.[1]

Diseased sites harbor a microbiota of Gram-negative anaerobic rods, including black pigmented organisms and surface translocators.[7] 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*. **Table 3** lists some of the microbes associated with failed implants.[9,11]

Endodontic infections are characterized by species belonging to genera Fusobacterium, Prevotella, Porphyromonas, and Actinomyces. Many authors including Shaffer *et al.*[12] (1998), have raised concerns that implant sites with a history of endodontic infection or proximal to teeth with endodontic infection may increase the risk of implant failure. Novaes and Novaes[13] (1995), reviewed the success of immediately placed implants following tooth extraction, and concluded that successful implant integration is highly predictable, at tooth extraction sites with prior periapical lesions, given appropriate preoperative, intraoperative and postoperative management, including meticulous alveolar debridement.

Kalykakis *et al.*[14] found that partially dentate subjects accumulate more plaque, exhibit higher crevicular fluid flow rates, and harbor more frequently *P. gingivalis* and *P. intermedia* than edentulous subjects. Apse *et al.*[15] found a higher proportion of black pigmenting anaerobes on implants in partially dentate than edentulous patients. Implant sites harboring *A. actinomycetemcomitans*, *P. gingivalis*, or *P. intermedia* were found to exhibit greater marginal soft tissue inflammation.[1] It is also seen that incidence of harboring periodontal pathogens namely *A. actinomycetemcomitans* and *P. intermedia* is higher at 4 and 6 months.

Apart from the above mentioned microorganisms, *Candida Albicans* also adheres to the implant surface and has been detected as the oppprtunistic peri-implant lesion.[16,17] it is the most prevalent fungus in the oral cavity, and its occurrence is strongly associated with denture-related stomatitis.[18] According to Burgers *et al.*[19], implant surfaces may be considered a potential reservoir for (re)infection with oral c. albicans, which in turn leads to candidiasis. Conflicting evidences exists if salivary coatings reduce or enhance the adhesion of specific salivary proteins as receptors for fungal adhesion.[20-22] It has been stated that mucin may serve as a receptor for C. albicans adhesion, whereas albumin may act as a blocking agent.

Conclusion

Literature demonstrates that saliva acts as carrier, however, reduction in retrograde peri-implantitis rates are shown when patients delay implant surgery after tooth extraction and rigorous preoperative and postoperative antibiotic regimen as well as improved dental hygiene are incorporated into the post-operative treatment.

Peri-implantitis can be controlled by regular review visits and prophylactic care.

The Cochrane systematic review on the treatment of peri-implantitis concluded that the use of local antibiotics in addition to manual subgingival debridement was associated with a 0.6mm additional improvement for PAL and PPD over a 4-month period in patients affected by severe forms of peri-mplantitis.[23]

To prevent or reduce biofilm formation on biomaterials their surface chemistries can be modified, e.g., by adding surface-modifying end groups (SMEs) or by altering the chemical composition of substrates.[24] According to Bundy *et al.*[25] Titanium has been shown to have bacteriostic effect on *S. mutants*. Zirconia implant materials can also be used as an alternative to conventional monolithic titanium implants, due to their reduced proneness to adhere microorganisms.[26,4]

Biomaterial surface properties, such as surface roughness, surface free energy and chemical properties influecce the quality and quantity of adherence of fungal adhesion.[27,28]

Yet they get infected and failure of treatment occurs. When implant becomes mobile it should be removed, however failing implant can be restored back to health by mechanical debridement, irrigation with Chlorhexidine, treanment with systematic antibiotics and through surgical procedures.[2]

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