MICROBIOLOGICAL ASPECTS DENTAL IMPLANT - A REVIEW

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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.

KEYWORD:

INTRODUCTION
Dental implants like natural teeth are colonized by microorganisms. There are various terminologies associated with ailing and failing implants. Terms such as peri-implant disease, peri-implant mucositis, and peri-implantitis have been proposed that describe pathologic conditions around dental implants. Dental implants are being used more frequently to restore partially edentulous or completely edentulous patients. Clinicians now are facing problems that they encountered with natural teeth.¹ Like natural teeth dental implants are susceptible to inflammation of the supporting tissues by colonization of pathogenic bacteria. Implant failure has been defined as the inadequacy of the host tissue to establish or maintain osseointegration. Implant failures can be classified based on the time of failure as early or late and based on the etiopathogenesis as infectious or noninfectious. Early failures are due to
failure to establish osseointegration and late failure is due to failure to maintain osseointegration.\(^2\)

Osseointegration is defined as a “direct structural and functional connection between ordered living bone and surface of a load-carrying implant”. Early failures can occur as a result of surgical trauma, premature loading of the implant, and bacterial infection. Late failures can occur following prosthetic rehabilitation. Late failures can again be divided as “soon” late failures which occur during the first year of loading and as “delayed” late failures occurring in subsequent years. “Soon’ late failures can be attributed to overloading of the implant placed in bone that is poor both qualitatively and quantitatively. “Delayed late failures can occur when there are changes in the loading conditions in relation to bone quality and volume and also as a result of peri-implantitis”\(^3\) The present review will focus on the microbiota associated with dental implants in healthy and diseased state.

**Microbiota of Successful Osseointegrated Dental Implants**

The predictable long-term success of osseointegrated dental implants used as abutments for prostheses in the treatment of tooth loss has been attributed to a number of factors, including surgical techniques and the use of biocompatible materials. However, the contribution of the peri-implant soft tissue response to the clinical longevity of dental implants is less well understood.\(^4\)

The long term survival of dental implants depends on control of bacterial infection in the peri – implant region. Loss of bone subjacent to peri-implant mucosal tissues has been reported to occur around most implant systems. Although such bone loss is generally very small (mean = 0.1 mm per year after the first post surgical year), sites occasionally demonstrate much greater amounts. Two theories have been proposed to explain rapid loss of peri-implant bone\(^12\) (1) mechanical overloading of the crestal bone; and (2) bacteria initiated disease.

Strong evidence support the fact that microorganisms present in dental plaque in the region of the gingival sulcus or periodontal pocket, or substance derived from them, constitute the primary extrinsic etiological agent in gingival and periodontal disease.\(^5\)

Bacterial infection is one of the primary reasons for implant failure after osseointegration.\(^11\)

The oral cavity comprises a number of ecologically distinct areas, each with a characteristic microbial composition. A number of factrors determine the composition of a specific
microflora: the characteristics of the surface (keratinized surfaces, tongue, buccal mucosa, enamel and dentin) which serves as the site for attachment; the nutrients available from the immediate environment (i.e., from gingival cervicular fluid); and the physiochemical aspects of the area, which are selective for bacterial growth according to the oxygen tension.\[6\]

The oral flora is extremely complex. As many as 200 to 300 different species have been isolated from the oral cavity. However it is generally thought that the indigenous flora consists of approximately 20 species.

**Development of Oral Microflora**

Colonization of the oral cavity of a neonate begins within a few days of birth. *Streptococcus salivarius* predominates at this stage, which is probably due to its enhanced ability to attach its mucosal surfaces. Other streptococci such as *S. Mutans* and *S. Sanguis* have not been isolated until after tooth eruption (consistent with their ability to adhere to hard surfaces only) and it is significant that they disappear from the mouth after the loss of all teeth. Gram-positive rods such as *Actinomyces naeslundii* and *Actinomyces viscosus* also appear very early in life. *A. Naeslundii* can be detected in most neonates and nearly all teenagers and adults. In contrast, *A. Viscosus* usually appears at the time of tooth eruption and is more commonly associated with dental plaque than *A. Naeslundii*. The initial colonizers are generally Gram-positive aerobic and/or facultatively anaerobic bacteria. Anaerobic bacteria such as *Bacteroides* and *Actinobacillus actinomycetemcomitans*, spirochetes and *Fusobacterium* appear later in life and are more frequently isolated after puberty.\[7\]

It is a well established fact that the oral flora varies from one site to another and it appears that certain bacteria have a specific ecological niche within the oral cavity. This specificity appears to be based on their selective ability to attach to various surfaces. At the time of eruption of the complete dentition, Gram-positive streptococci represent a large part of the normal flora, although the actual percentages differ from site to site. *S. Mutans* and *S. Sanguis* are generally found in dental plaque whereas *S. Salivarius* is found in greater numbers on mucosal surfaces and saliva. *Neisseria* has been found to be one of the initial colonizers of cleaned enamel surfaces. Gram positive rods and filaments common to the normal flora are *Actinimyces* species, *Rothia denticola*, *Corynebacterium*, and *Lactobacillus*. Gram negative rods and filaments, such as *Fusobacterium*, black pigmented *Bacteroids*
(BPB’s), and Spirochetes can be isolated from the flora of healthy periodontal sites but represent a very small proportion of the cultivable flora.\(^8\)

With the loss of teeth, bacteria associated with hard surfaces (S. Mutans), strict anaerobes generally found in periodontal pockets (spirochetes), and very fastidious organisms tend to disappear from the oral cavity. Bacteria that are dependent on hard surfaces for attachment and growth will in part recolonize the mouth if a denture is worn. In edentulous subjects the predominant cultivable flora of denture plaque associated with healthy and inflamed palatal mucosa is mainly Straptococci (S. Mitior, S. Sanguis, S. Aureus, S. Mutans), Staphylococci, Actinomyces, Lactobacilli, and gram-negative cocci but very few gram-negative rods. Yeasts such as candida albicans correspond to 0.45% of total viable count.\(^8\)

There appear to be significant changes in the composition of the microflora during the eruption of teeth and again with their loss. The use of implant in the treatment of edentulism has added yet a third set of circumstances that may influence the microbial flora. What lies to be seen is if the placement of implants encourages the re-establishment of the normal microflora as that associated with a dentate state.\(^3,5\)

**Bacterial Composition of Dental Plaque**

Evidence from a number of sources has shown that the primary etiological factor in both gingivitis and periodontitis is based on the quantitative and qualitative characteristics of dental plaque that forms on tooth and tissue surfaces in the gingival sulcus. Dental plaque, a large aggregation of confluent colonies of microorganisms embedded in an organic matrix, is described primarily by its relationship to the gingival margin; supragingival and subgingival.\(^9\)

The primary supragingival and subgingival colonizers constitute gram positive bacteria that are proficient at attachment. Streptococcus sanguis, S.Mitis, actinomyces viscosus, S. Mutans, A. Naeslundii, Rothia dentocariosa, and Veillonella parvula – gram- negative cocci – appear somewhat later. As plaque matures its bacterial content increases in complexity. The metabolic activity of the microflora tends to reduce the oxidation reduction potential and results in an increase in anaerobes in the flora such as bacteroids, fusobacterium, eubacterium, and others. There is no mark difference in the bacterial composition between supragingival and subgingival plaque associated with healthy sites.\(^10\)
Microflora Associated With Gingivitis
Supra and subgingival plaque at inflamed sites is more abundant and the total number of organisms is 10-20 times higher than that in healthy sites. As plaque matures a change in the general pattern in plaque development occurs from predominantly gram-positive coccoid forms to more rod shaped gram-negative forms, particularly in subgingival areas. It has been found that gingivitis is a tissue response to an increase in numbers of organisms rather than qualitative changes in supra and sub-gingival microflora. Although tissue penetration of microorganisms is thought not to occur in gingivitis, the increase in bacterial metabolites and enzymes due to increased bacterial numbers may permeate the junctional epithelium and initiate an inflammatory response.[11]

Microflora Associated With Periodontitis
Although there is large variation in the composition of bacterial plaque, diseased sites are usually associated with increase in gram-negative, motile microorganisms, and spirochetes. A predominance of gram-negative rods, mainly black pigmented Bacteroids, Fusobacterium nucleatum, Selemonas corrodens and Capnocytophaga sp. have been implicated as periodontal pathogens.[12]

Peri-Implant Microflora
The relationship between the peri-implant mucosa and the indigenous oral microflora is in the initial stages of investigation, particularly in terms of pathogenesis of implant failure. There is weak evidence that plaque accumulations cause inflammation of the peri-implant mucosa analogous to gingivitis. Furthermore, it has been observed that rapid loss of bone can occur with concomitant peri-implant sulcus deepening. This in turn has been equated with periodontal disease. Although such associations are hypothetical at this stage, the presence of known periodontal pathogenic organisms in the implant sulcus suggests that these sites are considered “at risk”. [12]

Species associated with periodontal infections such as fusobacteria; spirochetes; Actinobacillus Actinomycetemcomitans; the black pigmented species Porphyromonas gingivalis and Prevotella intermedia; Bacteroids forsythus; Campylobacter rectus have also been identified from implants symptomatic through infection. In contrast, successful implants and inactive periodontal sites are seen to harbor higher percentages of Streptococcus species, Capnocytophaga ochracea and Veillonella parvula than the diseased sites.[11]
The greatest risk factor for periodontal disease is periodontal attachment loss, which is mediated by pathogens in the subgingival microbiota. Subgingival species are grouped into different complexes that are named by color. Different colored complexes appear to have different associations with severity of periodontal disease and various stages in plaque development. **Yellow and green** complexes, which include oral streptococci and several facultative gram-negative rod species, are detected in most healthy gingival sites, and early in plaque development following debridement. Colonization by **orange complex** species appears to depend on the presence of yellow and green complex species. Orange complex species include *Prevotella* and *Campylobacter* species – secondary level periodontal pathogens. The most pathogenic – **red complex** – comprises of *P. gingivalis, B. Forsythus*, and *Treponema denticola*. These species were not detected in the absence of species from other complexes. Other species grouped in a **purple** complex, or not grouped at all, include *A. Actinomycetemcomitans*. Thus, detection of periodontal pathogens in the red complex around successful implants could indicate an increased risk of infection.

Dental plaque on implants can be described by its relationship to the peri-implant sulcus; supracrevicular and crevicular.\[12\] It is clear that plaque forms readily on artificial surfaces placed in the mouth. Plaque accumulation is preceded by the formation of a pellicle, a thin film of complex glycoproteins and proteoglycans. The pellicle formed on tooth surfaces has been extensively characterized, and it is likely that a similar film forms on titanium oxide surfaces. Anodic proteins bind selectively to the hydroxyapatite surface and can hypothetically bind to a cathodic surface like titanium oxide. Binding to the metal surface occurs by means of hydrophilic side chains rotated towards the metal. Such adsorption appears to occur without any serious loss of protein activity. The low surface energy of the titanium surface binds the proteins with a low affinity, which may explain the clinical observation that plaque is less adherent to a titanium surface than a tooth surface. Results from microscopic and cultural investigations of supracrevicular plaque taken from teeth and implants in partially edentulous mouths demonstrates that supracrevicular plaque accumulations are not significantly influenced by a titanium surface.\[13\]

Bacterial flora can be characterized by morphology and motility patterns as viewed in dark field and phase contrast microscopy. Culturing techniques may provide further features of identification by typing the microflora in terms of gram staining, anaerobic growth,
predominance and speciation. These methods have been used to characterize the differences in microflora associated with healthy and diseased sites in periodontal studies.\textsuperscript{[14]}

In a study conducted at the Branemark laboratories, dark field investigations of bacterial morphotypes at healthy implant sites compared favorably with compiled values of similar investigations from periodontal sites.\textsuperscript{[15]} High proportion of coccoid cells and low proportion of motile rods and spirochetes were associated with periodontal health, whereas higher proportions of motile and spirochetal microbes were recovered from diseased sites. Although similarities in the relationship between changes in the microflora and disease progression have not been demonstrated in peri-implant mucosa, the data listed above demonstrate that implant sites yield microflora of similar morphotypic proportions to those found in healthy dentate sites.\textsuperscript{[16]}

Failing implant sites (failure of osseointegration, or fibrous encapsulation) exhibit a significantly different morphotypic microflora from healthy sites. Mombelli et al. showed a significant drop in the number of coccoid cells with a concomitant increase in non-motile rods, motile rods, and spirochetes. A similar change in the microflora has been reported in inflamed fibrous tissue of encapsulated implants and at diseased periodontal sites.\textsuperscript{[17]}

Gram staining provides a method of broad categorization of the microflora. Gram positive organisms are generally associated with periodontal health while gram negative bacteria become proportionately dominant in gingivitis and periodontitis.\textsuperscript{[18]} Healthy peri-implant sites in two studies demonstrated that gram positive organisms dominated in crevicular peri-implant microflora however, it is not known if the ratios of gram positive to gram negative bacteria change with the onset of disease in the peri-implant mucosa.\textsuperscript{[19]}

By altering the partial pressure of oxygen in culture, it is possible to identify bacteria either as capable (anaerobic) or incapable (aerobic) of growth without oxygen. Organisms that can grow under both conditions are facultative. The primary colonizers of supra- and subgingival areas around a tooth are generally aerobic and facultative organisms and are generally associated with healthy tissues.\textsuperscript{[20]} As plaque matures, the proportion of anaerobic organisms’ increases. Some of these organisms include implicated periodontal pathogens. However a ratio of 1:1 aerobic to anaerobic organisms is normally found in periodontal sites. Two studies that examined teeth and implants in partially edentulous patients have demonstrated such a relationship at implant sites.\textsuperscript{[21]}
It is expected that the microflora at implant sites is influenced by the presence of periodontal pockets around teeth, which may act as reservoirs for periodontal microflora. This hypothesis is further supported by comparing two different implant groups; edentulous and partially edentulous patients. Further evidence of significant microbiological differences between edentulous and partially edentulous patients was demonstrated when counts of specific bacteria were compared. Wet spreaders occurred in great numbers and more frequently at implant sites in the partially edentulous patients. B. Gingivalis, B. Intermedius, wet spreader colonies (considered to be typical of the genus Capnocytophaga) have been reported in association with diseased peri-implant mucosa and in periodontitis sites. Although these bacteria were identified at tooth and implant sites the clinical parameters did not indicate progressive bone loss or implant failure. These distinct differences between the edentulous and partially edentulous groups suggest that the presence of pockets around teeth, which harbor a large variety of bacteria, may serve as a reservoir of periodontal pathogens contributing to contamination of peri-implant sites. The longitudinal success of osseointegrated implants in edentulous patients may be due to a reduced intraoral challenge by implicated periodontal pathogens and, conversely, the presence of such pathogens in partially edentulous patients may conceivably hasten subsequent peri-implant disease if patients are not well maintained.

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


