

Understanding Peri-implantitis: A Strategic Review

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The high survival rate of osseointegrated dental implants is well documented, but it is becoming increasingly clear that successfully integrated implants are susceptible to disease conditions that may lead to loss of the implant. Although placement and restoration usually are included in the domain of the periodontal, oral and maxillofacial surgery, or prosthetic specialist, given the increasing numbers of patients treated with osseointegrated fixtures, it is increasingly likely that maintenance of these implants by the general dentist will become much more common. However, the surrounding tissues may be subject to inflammatory conditions similar to periodontal disease and so require maintenance. This article discusses the background, cause, and diagnosis of peri-implant disease, as well as the maintenance, care, and treatment of peri-implant infection in osseointegrated implants.

Key Words: *osseointegration, peri-implantitis, peri-implant mucosa*

INTRODUCTION

As an increasing number of patients choose dental implants over traditional methods of tooth replacement, a corresponding rise in the number of individuals with posttreatment complications can be expected. Consequently, treating peri-implant infection is becoming a more significant factor in the long-term prognosis for implant retention.

It is evident that installation of oral implants is a routine procedure in the reconstruction of fully or partially edentulous individuals. Similar to natural teeth, artificial abutments penetrate the oral mucosa and reach the contaminated oral cavity. When challenged by bacteria within the biofilms formed on implant surfaces, the peri-implant

tissue response seems to follow patterns similar to those of periodontal tissues in a susceptible host.^{1,2} Documentation of implant therapy has so far included only exceptional reports on destructive lesions around implants.³⁻⁵ *Peri-implantitis* is defined as an inflammatory reaction with loss of supporting bone in the tissues surrounding a functioning implant.⁶ The overall frequency of peri-implantitis was reported to be 5%–8% for selected implant systems.⁷

An association has been reported between periodontal and peri-implant conditions. The higher the full-mouth clinical probing pocket depth and the greater the full-mouth attachment loss, the higher is the expected attachment loss around implants in the susceptible patient. In individuals with a history of chronic periodontitis, the incidence of peri-implantitis was 4–5 times higher than in individuals with no history of periodontitis.¹ Longitudinal bone loss around implants was correlated with previous experience of reduced periodontal bone

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support. Thus, periodontitis-susceptible subjects may show increased implant failure rate and marginal bone loss.⁸ Smoking has been reported to significantly correlate with marginal bone loss around implants.

Tissue surrounding implants

The outer surface of the peri-implant mucosa is lined by a stratified keratinized oral epithelium that is continuous with a junctional epithelium attached to the titanium surface by a basal lamina and by hemidesmosomes. The 2 mm long nonkeratinized junctional epithelium is only a few cell layers thick in the apical portion and is separated from alveolar bone by 1–2 mm of collagen-rich connective tissue. This 3–4 mm “biological barrier,” formed irrespective of the original mucosal thickness, protects the zone of osseointegration from factors released from plaque and the oral cavity.⁹ The connective tissue compartment between the junctional epithelium and the alveolar bone consists of scarlike connective tissue, almost devoid of vascular structures, with greater amounts of collagen and fewer fibroblasts.¹⁰ The fibroblast-rich barrier next to the titanium surface has a high cell turnover, and fibroblasts may play an important role in establishing and maintaining the mucosal seal. The peri-implant mucosa is similar to the gingiva around teeth with regard to function and host response to infection.¹¹ An inflammatory cell infiltrate of equal size and composition has been found in clinically healthy tissues of gingiva and in peri-implant mucosa.¹² Results from immunohistochemical and morphologic analyses show that inflammatory cells (eg, neutrophils, lymphocytes, macrophages, plasma cells) are present.

Associated microbiology

Similar to the gingival crevice around the natural tooth, the peri-implant mucosa, which covers the alveolar bone, is closely

adapted to the implant. Microbial colonization and ensuing inflammatory reactions in peri-implant tissues might be analogous to key events in the pathogenesis of periodontitis. In partially edentulous subjects, the developing microbiota around implants closely resembles the microflora of naturally remaining teeth.^{13,14}

Papaioannou et al,¹⁵ using phase-contrast microscopy and DNA probes, determined the prevalence of putative periodontal pathogens in partially edentulous and edentulous patients with a history of periodontal disease. The microbiological profiles were similar around teeth and dental implants of equal pocket depth, which may indicate that pockets around teeth can serve as a reservoir for putative periodontal pathogens. In addition to dark-pigmented, gram-negative anaerobic rods, other bacterial species (eg, *Bacteroides forsythus*, *Fusobacterium nucleatum*, *Campylobacter*, *Peptostreptococcus micros*, *Prevotella intermedia*) are associated with peri-implant infection.¹⁶ Organisms that are less frequently associated with periodontitis, such as *Staphylococcus* spp, enterics, and *Candida* spp, have been found in cases of peri-implant infection.^{17,18}

PERI-IMPLANTITIS

Peri-implantitis is defined as an inflammatory reaction characterized by loss of supporting bone in the tissues surrounding a functioning implant.⁶ It has been described as “a site-specific infection yielding many features in common with chronic adult periodontitis”³ and “an inflammatory, bacterial driven destruction of the implant-supporting apparatus.”¹⁹

During peri-implant breakdown, a complex microbiota is established, closely resembling that found in adult periodontitis.³ When peri-implant tissue breakdown is induced by submarginal placement of plaque-retentive ligatures in animals, a shift in the microflora occurs.^{20,21}

Most available information on the histopathologic features of peri-implantitis lesions has been obtained from experimental studies in dogs and monkeys.²²⁻²⁵ In the experimental models used, plaque formation was allowed and ligatures were placed in a submarginal position around the neck of the implants. Histologic analysis of biopsy material revealed the presence of large inflammatory lesions in the peri-implant mucosa, and that these lesions extended to the alveolar bone. Furthermore, about 60% of the lesions were occupied by inflammatory cells, among which plasma cells dominated. Investigators also described numerous polymorphonuclear cells in connective tissue areas adjacent to the pocket epithelium and in perivascular compartments in more central areas of the inflammatory cell infiltrate. Similar observations were made in a study on the immunohistochemical characteristics of human peri-implantitis lesions.²⁶ This study reported that peri-implantitis lesions consistently exhibited elastase-positive cells (ie, polymorphonuclear cells) in central portions of the infiltrate.

Clinical features of peri-implantitis

Increased clinical probing pocket depth, often accompanied by bleeding and sometimes suppuration, is an indicator of pathology in peri-implant tissues. On the basis of findings of the clinical examination, radiographs of selected areas may be proposed. In peri-implantitis, a bony defect develops around single or multiple implants. The shape of a saucer or rounded beaker is often seen on radiography. Regular check-up visits and life-long supportive therapy are absolutely necessary for the implant patient.

Treating peri-implantitis

It is essential to inform the patient about the need for effective oral hygiene procedures (particularly around implants). The patient should be carefully instructed in the proper use of necessary additional oral hygiene aids.

New data support the need for treatment of peri-implant lesions. Spontaneous progression of experimentally induced peri-implantitis was reported by Zitzmann et al.²⁷ Additional bone loss occurred in most of the implant sites following ligature removal in this experimental model. The reason why some peri-implantitis lesions were associated with extensive bone loss and others with only minor bone loss is currently not understood. In a prospective, randomized, controlled clinical trial, Wennström et al.²⁸ studied the outcomes of restorative therapy in periodontitis-susceptible patients who, after receiving basic periodontal therapy, had been restored with implants. No scientific evidence suggests that periodontal probing affects the integrity of an implant, but it should be noted that a metal probe may damage the implant surface.²⁹ A rigid plastic probe is ideal. Probing the peri-implant sulcus with a blunt, straight periodontal probe allows assessment of peri-implant probing depth, distance between the soft tissue margin and a reference point on the implant for measuring hyperplasia or recession, bleeding, and suppuration. Lang et al,³⁰ investigating the effects of different mucosal conditions around implants, confirmed the excellent sealing effect of the soft tissue collar in health and peri-implant mucositis and reported relatively uninhibited penetration to the alveolar crest in peri-implantitis lesions. Probing around oral implants should be considered a reliable and sensitive parameter for long-term monitoring of peri-implant mucosal tissues.

Implant mobility is an indication of lack of osseointegration, but it is of no use in diagnosing early implant disease; rather it shows the final stages of de-integration. Initially, the bone loss associated with peri-implantitis is observed to be marginal and results in the formation of infrabony defects. The apical portion of the implant will be fully integrated, so an increase in mobility will not be evident. Complete loss of osseointegration

would be reflected in a sudden increase in implant mobility. To arrive at a conclusion in evaluating the outcome of implant treatment in longitudinal studies, complications such as peri-implantitis should always be reported.

Several factors at the time of placement and restoration can improve the long-term prognosis of fixtures. Patient motivation and oral hygiene are paramount. Periodontal health should be achieved before proceeding with implant therapy. Restorations should be cleansable with well-fitting margins. In addition, as much of the mucosal tissue as possible should be preserved in its original position. A maintenance program should be undertaken after successful implant therapy. This should be tailored to the individual and should include regular recalls to provide optimal disease prevention. The recall visit is similar to that of a periodontal patient in maintenance in that each visit includes examination, re-evaluation, diagnosis, motivation, and treatment of infected sites. Before a patient is enrolled in a maintenance program, one should ensure that baseline data have been established. Probing pocket depths and mucosal margin positions are noted, and radiographic crestal bone levels are established.

Incomplete surface decontamination seems to be a major problem in implant maintenance. The screw thread makes scaling difficult, and the presence of periopathogenic bacteria is associated with a poor response to guided tissue or bone regeneration.³¹ As a result, little evidence of true re-osseointegration is found in humans. However, early experimental evidence suggests that re-osseointegration may be possible following completion of appropriate decontamination procedures for sand-blasted and acid-etched implant surfaces.³² If an implant does not respond to treatment, evidence indicates that rather than trying to save the failing implant, it would be better to remove it and place another fixture once the site has healed.

A systemic antimicrobial treatment approach was tested in a study involving 9 patients with marked loss of bone and pocket probing depths of 5 mm around implants.³³ These patients were selected on the basis of microbiological screening; the individuals considered had anaerobic cultivable counts of 10⁶ or more colony-forming units per mL, including 20% or more gram-negative anaerobic bacteria, in diseased sites. Treatment included mechanical debridement, irrigation of all peri-implant pockets 3 mm or larger with 0.5% chlorhexidine, and systemic antimicrobial therapy with an agent specifically effective against strict anaerobes (eg, ornidazole, 1000 mg for 10 consecutive days). After therapy, bleeding scores immediately decreased. Because peri-implantitis lesions usually are well demarcated, controlled delivery devices, originally developed for treatment of localized periodontal infection, may be a successful means of treating peri-implantitis. Such devices can release a sustained high dose of antimicrobial agents precisely into affected sites over several days and may be able to kill bacteria protected in an insufficiently removed biofilm. A controlled case series indicated a beneficial effect in the treatment of peri-implant mucositis and mucosal hyperplasia.³⁴

CONCLUSION

Increasing acceptance of implant placement as a standard treatment option means that more and more dentists will be involved in the long-term care and maintenance of these implants. Peri-implant lesions may develop after several years. Patients who have lost their teeth as the result of periodontal disease seem to be at greater risk. Although several anti-infective treatment strategies have demonstrated beneficial clinical effects in humans (eg, resolution of inflammation, decreased probing depth, gain of bone in the defects), evidence is insufficient to support a specific treatment protocol.

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