



Peri-implant mucositis and peri-implantitis: incidence, etiology, diagnosis and treatment

Commander Brett Metcalf, DC, USN and Commander Matthew J. Gramkee, DC, USN

Introduction

The success of endosseous dental implants has been attributed to their functional ankylosis or bone anchorage. Branemark described this osseointegration as a direct structural connection at the light microscopic level between bone and the surface of a load-carrying implant.¹

Criteria for successful osseointegration were established by Albrektsson and Smith as: 1) absence of persistent signs/symptoms such as pain, infection, neuropathies, paresthesias, and violation of vital structures; 2) implant immobility; 3) no continuous peri-implant radiolucency; 4) negligible progressive bone loss (less than 0.1mm annually) after physiologic remodeling during the first year in function; and 5) patient/dentist satisfaction with the implant supported restoration(s).^{2,3} The clinician should continuously assess these parameters as implants, like natural teeth, are susceptible to periodontal pathogens and a stimulated host inflammatory reaction.⁴

Today, with increasing numbers of patients receiving dental implants, it is imperative that all dental clinicians be competent in monitoring and maintaining dental implant health.⁵ The purpose of this update is to describe incidence, etiology, diagnosis and a treatment strategy for peri-implant mucositis and peri-implantitis.

Incidence of peri-implant diseases

Studies have shown the placement of endosseous implants is a predictable procedure with relatively few biologic complications. The complication rate is difficult to ascertain and may be underreported due to varying assessment procedures, implant systems and protocols. Even so, Berglundh found the incidence of peri-implantitis ranged from 0% to 14.4% and appeared to be related to the number of years the fixtures were in service.^{6,7} Furthermore, there is evidence to indicate the complication rate may be higher in patients with implants replacing teeth lost because of plaque-induced chronic periodontal disease.⁸

The initial inflammatory response to microbial colonization of the implant surface is termed *peri-implant mucositis*. This is a reversible inflammatory condition limited to the soft tissues around the implant (without any bone loss). *Peri-implantitis* results if the inflammation spreads apically with the loss of osseointegrated supporting bone.^{9,10}

Etiology of peri-implant diseases

The mucosa composing the epithelial and connective tissue attachments surrounding endosseous implants provides a tight seal to resist food impaction and microbial invasion and is comparable to the biologic width around teeth described by Gargiulo.^{3,6,11,12} Microbial invasion into implant biologic width and bacterial colonization of the titanium surface leads to mucositis and, if the peri-implant bone levels are affected, to peri-implantitis.^{7,9,10,13} Peri-implant microbiota acquire the patients'

indigenous periodontal microflora.¹⁴ These microbiological findings related to healthy and failing implants are the same as those for healthy and periodontally compromised teeth.^{15,16} Infected sites around failed implants may harbor a complex microbiota with a large proportion of known periodontal pathogens like *Porphyromonas gingivalis*, *Prevotella intermedia*, and *Fusobacterium nucleatum*.⁸ Stated another way, successful implants are sparsely colonized by gram negative cocci while failing implants yield significant amounts of gram negative anaerobic bacteria. These findings support the recommendation that patients with implants be evaluated regularly for any clinical signs and symptoms of peri-implant disease.¹⁷

Diagnosis of peri-implant tissue

Following implant therapy, patients should be placed on a three to four month recall program for the first year. After the first year, their tissue response should be evaluated and then placed on a custom recall schedule. However, this interval should be no longer than 6 months. The clinician should assess: 1) presence of plaque and calculus, (2) peri-implant probing depth (PD) (less than 3mm), (3) presence of bleeding upon probing (BOP), (4) presence of suppuration, (5) width of keratinized gingiva, (6) radiographic evaluation to monitor the crestal bone levels, and (7) fixture mobility.^{18,19} The radiographic evaluation should include a comparison with any previous radiographs. Initial bone loss can be expected to be near the level of the first thread. Additional bone loss of approximately 0.1 mm per year for the first five years (up to a total of 1.5mm) is considered normal.² Complete seating of the associated parts (abutment and/or restoration) should also be verified from the radiograph.¹⁹

A working diagnosis of peri-implant mucositis should be made in the presence of any of the above-assessed factors that result in inflammation around the implant and/or abutment. If the condition worsens to include evidence of bone loss, an appropriate diagnosis would be peri-implantitis.^{7,9,10,13} Regardless of the diagnosis, immediate treatment is recommended.

Treatment protocols

Conventional periodontal therapy should be instituted if inflammation develops around an implant. Lang and coworkers suggested a novel systematic step-wise approach for the prevention and treatment of peri-implant diseases referred to as cumulative interceptive supportive therapy (CIST).¹⁸ This system is based on periodic monitoring with implementation of treatment as thresholds for a particular condition are met. The first step is protocol (A), then (B) and, if conditions continue to worsen, the case should be referred to a specialist with implant training to execute protocols (C) and finally (D).²⁰ Protocol (A) is used to control inflammation in peri-implant mucositis, that is, implants with minimal increase in PD, slight (+) BOP, marginal erythema, plaque and/or calculus. The therapeutic endpoint is to resolve inflammation with cautious mechanical debridement (utilizing plastic curettes and rubber cup prophylaxis), twice daily swabbing with 0.12% chlorhexidine, and

review homecare and patient motivation. Protocol (B) is initiated for conditions that exhibit similar mucositis features but with deeper PD (4 to 5mm); however, there is still no loss of supporting bone. The treatment should include as above plus the addition of a locally delivered antibiotic (minocycline microspheres, doxycycline gel) at the infected implant site(s). Management of peri-implantitis, protocol (C), requires a more robust approach and is employed in conditions with evidence of osseointegrated bone loss (less than 2mm) and PD greater than 5mm. The strategy should comprise the modalities for protocol (A) and (B) with the addition of systemic antibiotic therapy (metronidazole 250mg TID for seven days or amoxicillin 500mg TID for 10 days). Furthermore, periodontal surgical access for surface decontamination (citric acid 1 to 2 minutes or tetracycline 250mg/5ml for 5 minutes) should be considered. Protocol (D) is initiated in circumstances of frank peri-implantitis which reveal probing depths (greater than 6mm), (+) BOP, plaque/calculus and moderate bone loss. This strategy will require periodontal surgical intervention for chemical disinfection, osseous resection and/or guided bone regeneration (GBR). GBR will attempt to salvage the implant through bone regeneration techniques with the use of resorbable or non-resorbable semi-permeable membranes and a bone replacement graft (freeze-dried bone allograft, anorganic bovine bone).

In clinical practice, CIST is aimed at early detection and methodical step-wise treatment which may rescue and reverse the fate of the ailing endosseous dental implant.¹⁸

Summary

Failure of dental implants are detrimental to both patients and dental healthcare providers. Periodic recall should focus on re-enforcement of proper homecare, early detection and, if necessary, immediate treatment of peri-implant mucositis and implantitis. These measures are crucial to long-term success of oral rehabilitation with dental implants. This clinical update discusses incidence, etiology, diagnosis and treatment alternatives for these early complications based on the clinical presentation.

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Commander Metcalf is a third-year Periodontics resident at the Naval Postgraduate Dental School. Commander Gramkee is the Chair of the Department of Periodontics at the Naval Postgraduate Dental School, Bethesda, MD.

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