MANAGEMENT OF PERI-IMPLANT DISEASE: A CURRENT APPRAISAL

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ABSTRACT
While the clinical science of managing peri-implant diseases is progressing, careful monitoring and preventive care of peri-implant tissue health during maintenance is paramount.

Background
Implants have become a routine treatment option for missing dentition. The biological complications of restored dental implants and associated supra-structures share similarities with the biofilm infections of natural dentition. Our existing paradigms of periodontal disease treatment can partially be applied to management of peri-implant disease.

Approach
A critical analysis of the peri-implant disease literature was conducted, anchored by a search on the MEDLINE database (2005 to present) by way of Ovid Medline®. Search terms peri-implantitis, peri-implant mucositis and peri-implant diseases were utilized. Select references within bibliographies of review articles were sought.

Conclusion
The dental team must play a critical role in educating patients to control plaque-biofilm associated with peri-implant tissues and associated restorations. Routine assessments at maintenance appointments allow early treatment intervention to prevent escalation of peri-implant disease. Given the infancy of clinical science surrounding peri-implantitis treatment, further, high-quality evidence based studies are expected.

Key words: Peri-implantitis, peri-implant

With the unfavorable prognosis of a tooth, the clinician and patient may be confronted with decisions of tooth replacement. Dental implants and associated restorations have become an increasingly common treatment option in clinical practice. A recent systematic review has determined the 5- and 10-year implant survival rate to be 97.7% and 92.8%, respectively. Nevertheless, the osseointegrated implant is susceptible to complications. Notably, implants may develop infections similar to the biological complications that afflict natural dentition. Biofilm infections of the peri-implant tissues challenge epithelial health and connective tissue surrounding the implant—and potentially the underlying, supporting bone.

Peri-implant disease is classified in part by anatomical involvement: peri-implant mucositis and peri-implantitis. These specific pathologies can develop subsequent to a normal implant wound healing phase and osseointegration. Peri-implant mucositis is characterized by inflammation of the gingival soft tissue surrounding the implant. Peri-implantitis is defined by loss of crestal bone surrounding the implant in addition to inflammation of the peri-implant tissues clinically noticeable as bleeding on probing. Additional clinical parameters associated with peri-implantitis include suppuration, deepened probing depths, and recession of mucosal tissues. In particular,
peri-implantitis refers to a post-osseointegration event, distinguishing it from dynamic bone level changes associated with remodeling immediately following implant placement.

Peri-implant diseases represent a common finding in contemporary clinical practice. Mombelli and colleagues reviewed peri-implant disease occurrence, focusing on studies spanning a minimum of 5 years; the prevalence of peri-implantitis was reported to be in 10% of implants and 20% of patients. The authors point out, similar to other systematic reviews, that heterogeneity in disease definitions in studies makes it difficult to offer an unequivocal statement on prevalence of peri-implantitis. Another concern is whether the convenience samples of clinical studies can be generalized to the overall population seeking implant therapies. Overall, the prevalence of peri-implant mucositis is higher than that of peri-implantitis; it occurs in about 50% of implants and just under 80% of patients.

Peri-implant diseases are generally thought to represent inflammatory conditions in response to bacterial plaque. Other factors, such as aberrant occlusal forces, may also contribute to the initiation and/or progression of peri-implant disease but remain incompletely understood. The composition of these biofilms is similar to the subgingival bacteria of chronic periodontitis, dominated by Gram-negative bacteria. Notably, studies have generally reported Porphorymonas gingivalis and other red complex bacteria at higher frequencies in peri-implantitis sites than healthy sites. In healthy implants with stable probing depths of 5 mm or less the flora is characterized by gram-positive cocci and small number of gram-negative species.

While the objective of this article is to present the current treatment options for peri-implant disease, it should be prefaced by a discussion on assessment and diagnosis. What will be apparent is that many treatment and management paradigms are in their infancy; consequently, implementing strategies of early detection and maintenance of implants becomes essential.

**IS THE IMPLANT HEALTHY? ASSESSMENT OF PERI-IMPLANT CONDITION**

The clinical assessment tools used in monitoring periodontal health of natural teeth are used in monitoring peri-implant tissues, though some considerations must be made given the obvious structural differences (Figure 1).

Assessment of mucosal inflammation is primarily made by observing bleeding following light probing (0.25 N) of the implant sulcus/pocket. As with periodontitis, the absence of bleeding on probing has a high negative predictive value—providing the clinician a predictor of stable peri-implant conditions. A prospective study detailed the utility of this parameter during the maintenance phase of restored dental implants. Bleeding on probing at implant sites during more than 50% of recall visits over a two-year period was strongly associated with peri-implant disease progression. Suppuration at peri-implant pockets is associated with infection and implies an inflammatory reaction present in the peri-implant tissues. Suppuration has also been correlated to implant bone loss.

Periodontal probing and related attachment level changes represent an essential means of diagnosing and monitoring peri-implant disease. Increased pocket probing depth is a clinical sign consistent with a finding of peri-implantitis, though this finding alone is insufficient to fully establish a diagnosis. Nonetheless, the finding of a probing depth ≥5 mm should be further assessed by the clinician, utilizing current and past radiographs. Note that major epidemiological studies have used peri-implantitis definitions that incorporate a probing depth (either a 4 or 5 mm threshold), radiographic criteria of bone loss, and an inflammatory measure (bleeding on probing, or suppuration). Studies have allayed fears that probing would cause long-term damage to the peri-implant tissues. Gingival mucosa surrounding a dental implant differs from that of natural dentition. The peri-mucosal seal that forms after surgical placement provides a barrier against bacterial invasion from the oral cavity but is lacking some strength of attachment when compared to a natural sulcus. Thus, a plastic probe with minimal force (0.25 N) is recommended around an implant to avoid long-term damage to the peri-implant tissue. Etter et al verified that a complete reformation of the peri-mucosal seal resulted at 5 days following gentle probing. Caution must be applied to probing measurements, as they alone are not indicative of disease around an implant. The presence of prosthetic reconstructions attached to the implant—the profile of abutments or associated crowns may increase probing depth readings. Radiographic interpretation should either confirm or dispute peri-implant diagnosis when bleeding or inflammation is associated with increased probing depth. Use of a radiograph, in conjunction with periodontal probing will greatly aid the clinician in interpreting a ‘large’ probing depth; for example, a radiograph that displays a significant amount of implant supra-structure cantilevered off of the implant should alert the clinician to cautiously consider ‘large’ clinical probing depth results.

Mobility of an implant is a terminal clinical sign—removal of the failed implant is warranted. The osseointegrated dental implant does not have any connective tissue fiber attachments between the bone and the implant. As a result, mobility is a concerning finding suggesting the loss of direct bone-implant contact that leads to the loss of stability. There is minimal clinical science at this time indicating any ability to achieve a stable long-term therapeutic outcome from treating implant mobility.

Radiographic assessment is important in identifying bone loss associated with peri-implantitis. Again, in conjunction with probing, radiographs aid in developing a proper picture of the underlying osseous topography of putative peri-implantitis.
cases (see Figure 3). In the progression of the peri-implantitis, severe bone lesions and crater-like defects may form. Comparisons should be made to a baseline radiograph from the time of prosthetic ‘connection’ to the implant. Thus, stability confirmed by serial radiographs taken on an annual basis during implant maintenance would be congruent with a clinician’s diagnosis of health or peri-implant mucositis.

PERI-IMPLANT MUCOSITIS: NON-SURGICAL MANAGEMENT

The non-surgical management of peri-implant conditions is focused on the reduction of inflammation by control of biofilm. In general, studies have demonstrated the effectiveness of non-surgical mechanical debridement and effective plaque biofilm control in treatment of peri-implant mucositis. Mechanical therapy, whether managing mucositis or peri-implantitis, typically involves the use of curettes to for supra- and sub-gingival biofilm removal. The implant literature describes the use of resin, carbon or titanium curettes and special tips for ultrasonic instrumentation; the general principle is that titanium surfaces should be cleaned with implements less hard than titanium. Other devices used in mechanical approaches include rubber-cup/polishing brushes and air powder flow devices. Air abrasive powder systems, originally used for removal of bacterial biofilm on teeth, have been applied to the treatment of peri-implantitis. Variants of this technique use a slurry of water/sodium bicarbonate or glycine powder delivered by pressurized air and water. A proper angulation of the tip, away from the implant surrounding gingival tissue, is critical to avoid unwanted damage. Available studies indicate some efficacy in reducing probing depths and bleeding at implant sites. The adjunctive use of chemotherapeutics, such as chlorhexidine, has shown minimal or no additional benefit over mechanical debridement alone. Studies indicate that triclosan containing dentifrices effectively reduce the inflammation of peri-implant mucositis. Here, in addition to improvements in plaque index and gingival inflammation, microbiological benefit has been demonstrated; significantly fewer numbers of Gram-negative anaerobes were identified in the subjects using triclosan compared to controls, with more than 90% reduction in P. gingivalis, Campylobacter rectus, Aggregatibacter actinomycetemcomitans, and Tannerella forsythia. Therefore, it may be appropriate to assume that twice-daily use of triclosan-containing toothpaste may enhance dental implant

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**Figure 1.** Components of routine peri-implant tissue assessment. Diagnostics that assess inflammatory status and architectural changes of underlying bone allow clinician to monitor health of peri-implant tissues. (A.) A routine implant assessment protocol involves monitoring inflammation via appearance of tissues and, or, bleeding on probing. Probing depths should be regularly recorded; relative attachment level (RAL) can also be monitored. Similar to clinical attachment level, RAL utilizes a fixed reference point (e.g. implant crown margin) to more accurately monitor peri-implant support. (B.) Peri-implant health is described. Radiographs should be taken bi-annually at minimum with comparisons drawn to time of prosthetic attachment. (C.) Key features of Peri-implant mucositis are listed. Radiographs establish definitive destruction of implant supporting bone.
maintenance and contribute to long-term success. Adequate plaque control by the patient and enrollment into a continuous maintenance program represent a key component of peri-implant disease treatment. Customized hygiene strategies (recommendation of specific brushes or interproximal devices) should consider the prosthetic supra-structure and the patient's manual dexterity (see Louropoulou, Slot, van der Weijden, Mechanical Self-Performed Oral Hygiene Of Implant Supported Restorations: A Systematic Review, this publication).

PERI-IMPLANTITIS: NON-SURGICAL MANAGEMENT

The same mechanical therapies utilized in peri-implant mucositis management are employed in treating peri-implantitis (Figure 4). Again, these modalities aim to reduce bacterial colonization on the surface of the implant; future corrective surgical phases depend on a relatively inflammation and plaque free environment for ideal results. In general, non-surgical mechanical debridement has been reviewed and found alone to be ineffective in the long-term treatment of peri-implantitis. Hence, clinical science has explored adjunctive benefits of local chemotherapeutics. Adjunctive antiseptic agents, such as chlorhexidine, have been suggested for use in peri-implant disease therapy. However, with respect to chlorhexidine as an adjunct, only limited short-term (6 months or less) improvements in clinical parameters have been presented. In studies of non-surgical approaches to peri-implantitis management, adjunctive antimicrobials, such as minocycline microspheres and doxycycline hyclate, in conjunction with mechanical debridement have shown improvements in gingival inflammation and probing depths compared to instrumentation with chlorhexidine irrigation. Further, long-term and randomized controlled investigations are warranted.

Given the untenable long-term results of non-surgical treatment of peri-implantitis, a surgical intervention, flap access seems appropriate as the next step in managing peri-implantitis. The surgeon managing peri-implantitis will rely upon approaches analogous to periodontal surgical approaches—ranging from resection to regeneration.
PERI-IMPLANTITIS: SURGICAL MANAGEMENT

When implant associated probing depth and bone loss is advanced or persists, despite the initial non-surgical treatment provided, a surgical intervention of peri-implantitis is required (Figure 2). Surface decontamination strategies used in non-surgical approaches may be applied now with the benefit of open access, created by an elevated gingival flap. Also, the removal of diseased granulation tissue and access to underlying bony architecture is facilitated. Bone morphology guides the selection and composition of the surgical procedure. With only mild horizontal bone loss around implant(s), an apically positioned flap coupled with implantoplasty is suggested. In implantoplasty, high quality diamond burs and adequate irrigation/evacuation are used to smooth the roughened implant surface to become less plaque retentive, thus reducing the progression of peri-implant bone loss.18

Surgical access will usually involve a full-thickness flap to allow access and cleaning of the contaminated implant surface. Here, the goal is to remove biofilm and create a theoretically compatible surface for re-osseointegration. Literature supports the use of various agents (e.g., saline, abrasive pumice, citric acid, chlorhexidine, and hydrogen peroxide) in surface decontamination as adjunct to surgical debridement. While these supplements had favorable influence on re-osseointegration, no agent was found to be superior19 (see Table 1). Lasers have also been used to decontaminate peri-implant sites once surgical access has been established. Human clinical studies have investigated CO2 and Er:YAG lasers used as a surgical adjunct in

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**Figure 3.** Examples of peri-implant bone loss. (A.) #30 presents with saucer-like defect down to the third implant thread. The defect at #30 was grafted at the time of implant placement into site #29. (B.) Mesial of implant #12 demonstrates bone loss. Clinically, there was a corresponding probing depth of 7 mm and suppuration at the mesial site.

**Figure 4.** A Summary of the management of peri-implant disease.
A summary of current major implant surface decontamination strategies is presented. Photodynamic therapy (PDT), though not commonly employed in private practice, has a growing body of clinical investigations into its use as an implant decontamination strategy. PDT employs a photo-sensitizer dye that reacts to low intensity visible light, between 660 nm and 905 nm, that forms cytotoxic species in the presence of oxygen. DNA or cytoplasmic membrane damage of bacteria are likely mechanisms of PDT. Decontamination; currently, it is difficult to conclude what additional benefit or contribution laser decontamination provides to surgical access.

Regenerative approaches can be used in conjunction with surgical access, drawing upon the paradigms used in managing periodontal defects associated with teeth. Containable bony defects—walled defects associated with the implant—should be more amenable to grafting to favorable gains in bone fill and reductions in probing depth. Debridement and implant surface decontamination are still essential prior to regenerative treatment, as the infection must be completely eliminated before application of bone graft. Patient risk factors, such as smoking, poorly controlled diabetes, and unsatisfactory oral hygiene, may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration. At present, heterogeneity of study designs and study quality may hamper the success of peri-implant defect regeneration.

CONCLUSION
Clinicians actively engaged in an implant maintenance program play a crucial role in peri-implant disease assessment. Furthermore, the entire dental team must aim to rehabilitate the common perception that dental implants are invulnerable. Given the greater clinical success achievable in managing peri-implant mucositis compared to the compromised osseo-integration of peri-implantitis, the contemporary dental practice must establish protocols to assess restored implants and associated peri-implant tissues. Prevention and early interception of etiology and contributing factors associated with peri-implant disease should be emphasized.

REFERENCES


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