

Treatment of Peri-implantitis

There is still no standard protocol for management of this inflammatory process

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During the last decade, replacing missing teeth with implants has become a predictable treatment modality, providing functional and esthetic satisfaction. However, peri-implant tissue destruction sometimes occurs due to poor oral hygiene, resulting in the exposure of a few threads of the implant previously that were embedded in bone.¹ The term “peri-implantitis” was introduced in the late 1980s and was subsequently defined as an inflammatory process affecting the soft and hard tissues around a functioning osseointegrated implant, resulting in the loss of supporting bone.² Given the high number of implant placements each day around the world, a high prevalence of peri-implantitis can be anticipated, which underlines the

necessity for a predictable therapy. In spite of diligent clinical trials and experimental studies, assiduous research is still required for the treatment of peri-implantitis, because there is still no standard protocol for its management.

There are various methods available for the treatment of peri-implantitis, which include mechanical debridement, the use of antiseptics, adjunctive administration of local and/or systemic antibiotics, access flap surgery with or without the use of bone-regenerating procedures, and supportive therapy.³ Each treatment option has a window of effectiveness that seems to be defined primarily by initial probing pocket depth; in addition, certain methods for peri-implantitis treatment produce best results only within a given range of diagnostic parameters.⁴

Implant Maintenance and Mechanical Debridement

A patient with implants must follow proper protocols for their maintenance; this includes an annual visit to the dental office, where clinical and radiographic examinations are conducted to check for implant health and signs of peri-implantitis.⁵

Debridement is accomplished with implant-safe instruments. Plastic, graphite, and gold-tipped instruments can be used to remove deposits. An ultrasonic tip may be used only with a plastic covering that prevents gouging and disturbance of the titanium surface. Polishing the visible portion of the implant can be accomplished with rubber cups and nonabrasive polishing paste or tin oxide⁶ while scaling, which should be done with short working strokes and light pressure. Upon insertion of the instrument, the blade should be closed against the abutment and then opened past the deposit. The deposit should be engaged apically with the stroke extending coronally. A horizontal, oblique, or vertical stroke should be used, depending on the location of the deposit.⁷⁻¹⁰

Mechanical instrumentation to remove bacterial deposits may damage the implant surface if performed with metal instruments harder than titanium.¹¹ According to a report by McCollum,¹² a comparative study that evaluated the surface texture of titanium implant abutments after exposure to plastic scalers and an air-powder abrasive system or polishing with rubber cup and pumice found that none of these methods appeared to roughen the surface; a rubber cup with pumice provided the smoothest polished abutment surface.

Diagnosis

Radiographically, in peri-implantitis, vertical destruction of the crestal bone is present around the implant—which assumes the shape of a saucer—while the bottom part of the implant remains osseointegrated. In some instances, wedge-shaped defects develop along the implant. In addition, there



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is a peri-implant pocket and bleeding after gentle probing with a blunt instrument, and there may be suppuration from the pocket. Tissues may or may not be swollen; however, hyperplasia is frequently seen if implants are located in an area with non-keratinized mucosa or if the suprastructure is an overdenture. Pain is not present.⁹ Both a mean loss of peri-implant bone height amounting to 1 mm to 1.5 mm in the first postsurgical year and vertical bone loss of less than 0.2 mm annually following the implant's first year of service have been proposed as major criteria for success.¹³⁻¹⁵ It should be noted that peri-implant bone loss also occurs in cases of overload and faulty occlusion and may be related to the type of implant used. The implants with the longest smooth surfaces demonstrated the highest amounts of bone resorption 12 months after abutment connection.¹⁶ Mobility of an implant suggests complete bone loss and, therefore, complete failure. To prevent this, peri-implant disease should be recognized earlier, to allow intervention before a substantial portion of the supporting bone is lost. However, mobility in early periods of osseointegration is not a very reliable clinical indicator of peri-implantitis. Therefore, electronic devices such as Periotest® (Medizintechnik Gulden, www.med-gulden.com) should be used.

According to Mombelli et al,¹⁷ peri-implant probing depth measurements are more sensitive to force variation than the corresponding

measurements around teeth. It has been suggested that probing of the implant sulcus is not truly diagnostic, and probing is indicated only in implants where pathology such as bleeding and exudate is present.¹⁸ Etter et al¹⁹ showed that although peri-implant probing disrupts the epithelial attachment to an implant surface, it does not cause permanent damage to the transmucosal seal, and completely new epithelial attachment is re-established within 5 days after peri-implant probing. Therefore, many authors suggest that probing around the implant should be done only when radiographic and clinical signs and symptoms are present. Successful implants generally allow probe penetration of approximately 3 mm; pockets of 5 mm or more signal peri-implantitis.⁴

If pockets of 3 mm are present, the patient's oral hygiene should be improved, with more frequent recall visits. Pockets deeper than 3 mm with no bone loss call for improvements in oral hygiene and correction of unfavorable soft tissue. If pockets of 4 mm to 5 mm are present, cleaning of implants as well as correction of unfavorable soft tissue and use of antiseptic agents should be considered. In pockets deeper than 5 mm with moderate bone loss, treatment with local drug delivery is indicated. For pockets deeper than 5 mm with extensive bone loss, treatment with local drug delivery or surgical intervention should be considered (Table 1).⁴

Local Drug Delivery

Implant surfaces exposed to the oral cavity have rough surfaces, making elimination of infection difficult; as a result, adjunctive use of chemical antimicrobial agents has been advocated. Because peri-implant lesions are well demarcated and contain the same microbiota as that of periodontitis, these agents kill bacteria effectively and have shown improvement in peri-implant lesions, which cannot be removed through mechanical debridement alone. In animal experiments, Ericsson et al²⁰ showed that mechanical debridement—combined with systemic administration of amoxicillin and metronidazole—results in resolution of ligature-induced peri-implantitis lesions.

Topical chlorhexidine has been recommended for the treatment of early peri-implant infections. However, Porras et al²¹ found no difference in improvements following the use of topical chlorhexidine to supplement mechanical debridement compared to mechanical debridement alone; in addition, chlorhexidine gel showed only minor changes in treatment of peri-implantitis. In a clinical study by Mombelli and Lang,²² peri-implantitis lesions were mechanically debrided, pockets were irrigated with chlorhexidine, and adjunctive systemic administration of 1,000-mg ornidazole/day for 10 days was prescribed. This resulted in an improved clinical and microbiological condition for up to 12 months. Renvert et al²³ used adjunctive minocycline microspheres, which resulted in improvements of both probing

TABLE 1

Peri-implantitis Treatment Guidelines

3-MM POCKET	→	Improvement in patient's oral hygiene, with frequent recall visits.
POCKETS > 3 MM	→	With no bone loss, improvements in oral hygiene along with correction of unfavorable soft tissue.
POCKETS > 4 MM TO 5 MM	→	Cleaning of implants along with correction of unfavorable soft tissue and use of antiseptic agents.
POCKETS > 5 MM	┌→	Moderate bone loss → Treatment with local drug delivery
	└→	Extensive bone loss → Treatment with local drug delivery system or surgical intervention.

depth (PD) and bleeding on probing (BOP) scores. At the deepest peri-implant site, the mean PD decreased from $5 \text{ mm} \pm 0.9 \text{ mm}$ to $4.1 \text{ mm} \pm 0.8 \text{ mm}$, and BOP scores were reduced from $100 \text{ mm} \pm 0\%$ to $57 \text{ mm} \pm 35\%$ after an observation period of 3 months. In another study, the same author repeatedly used local administration of minocycline microspheres 1 mg in addition to chlorhexidine gel 1%. Significant PD improvements were shown with minocycline microspheres as an adjunct compared to chlorhexidine alone at days 30, 90, and 180; and at the deepest sites of minocycline-treated implants, the mean PD reduction was 0.6 mm at 12 months.²⁴

Salvi et al³ also used adjunctive local delivery of minocycline microspheres for treatment of peri-implantitis, and at the end of 12 months, he observed a decrease in mean PD from $5.9 \text{ mm} \pm 0.7 \text{ mm}$ to $4.2 \text{ mm} \pm 0.6 \text{ mm}$,

and BOP scores from $92\% \pm 28\%$ to $44\% \pm 51\%$. This clinical study also demonstrated favorable results with the use of minocycline microspheres. Mombelli et al²⁵ achieved comparable results with adjunctive local delivery of tetracycline-impregnated fibers after an observation period of 12 months, which showed PD reduction from $6.03 \text{ mm} \pm 1.54 \text{ mm}$ to $3.85 \text{ mm} \pm 1.49 \text{ mm}$ at sites with the deepest probing pocket depth (PPD) at baseline. In this study, pathogens such as *Tannerella forsythia*, *Porphyromonas gingivalis*, *Prevotella intermedia*, and *Aggregatibacter actinomycetemcomitans* were suppressed but they rebounded during the observation period.

Conclusion

The primary goals of treatment are to eliminate the inflammatory lesion, stop disease progression, and maintain the implant in

function with healthy peri-implant tissues.²⁶ All of the treatment modalities mentioned here have been used by various authors with varying degrees of success, yet there is no standardization for the treatment of peri-implantitis. The most important step in the avoidance of peri-implantitis is maintenance.

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