



Use of Er:YAG Laser to Decontaminate Infected Dental Implant Surface in Preparation for Reestablishment of Bone-to-Implant Contact



Myron Nevins, DDS¹

Marc L. Nevins, DMD, MMSc²

Atsuhiko Yamamoto, DDS, PhD³

Toshiaki Yoshino, DDS, PhD⁴/Yoshihiro Ono, DDS⁵

Chin-Wei (Jeff) Wang, DDS⁶/David M. Kim, DDS, DMSc⁷

The prevalence of peri-implantitis is of concern to all clinicians participating in implant dentistry. Peri-implant inflammation results in the loss of supporting bone for the implant that may or may not be accompanied by bleeding on probing and suppuration. Early diagnosis and intervention are mandated, but there is a paucity of evidence leading to the most effective therapy. There is agreement that one of the challenges in surgically treating peri-implant defects is the process of cleaning and decontaminating the implant surface, which may be contaminated by bacterial aggregates. This preclinical canine study investigates the erbium:ytrium-aluminum-garnet laser to decontaminate the complex rough surface of the implant by stripping the contaminated oxide layer for induction of hard and soft tissue adaptation to a compromised or failing implant. The results provide evidence of new bone-to-implant contact established at a level representative of the size of the defects. The soft tissues contain little or no evidence of inflammation, which can be interpreted as an arrest of the disease progression process. The results can be translated to a treatment goal of stabilizing the prognosis of an implant that has been compromised. (Int J Periodontics Restorative Dent 2014;34:461–466. doi: 10.11607/prd.2192)

Implant replacement of the natural dentition has been incorporated into dental treatment planning as the result of many successful prospective and retrospective reports. It is realistic to anticipate some problems during healing following any treatment regimen, and peri-implantitis has surfaced as a relatively frequently encountered adverse result.^{1–3} It is understood to be a chronic inflammatory invasion of the soft and hard tissues into which the implant is encased and results in the loss of alveolar support or bone-to-implant contact (BIC).^{1–7}

This presents a disappointing discovery for the patient and requires an important decision. If the implant is no longer integrated, it must be removed, but when stable, most patients would prefer a treatment regimen with an endpoint goal of preserving the implant-supported restoration. Of course, the length of the implant and the degree of bone loss influence this decision. The literature is replete with case reports proposing protocols aimed at resolving the microbial contamination of the implant surface and offering a

¹Associate Clinical Professor; Division of Periodontology; Department of Oral Medicine, Infection and Immunity; Harvard School of Dental Medicine; Boston, Massachusetts, USA.

²Assistant Clinical Professor; Division of Periodontology; Department of Oral Medicine, Infection and Immunity; Harvard School of Dental Medicine; Boston, Massachusetts, USA.

³Lecturer, Department of Prosthodontics, Division of Oral Functional Science and Rehabilitation, Asahi University School of Dentistry, Gifu, Japan.

⁴Lecturer, The Japan Institute for Advanced Dental Studies, Tokyo, Japan.

⁵Director, The Japan Institute for Advanced Dental Studies, Osaka, Japan.

⁶Postdoctoral Research Fellow; Division of Periodontology; Department of Oral Medicine, Infection and Immunity; Harvard School of Dental Medicine; Boston, Massachusetts, USA.

⁷Associate Professor; Division of Periodontology; Department of Oral Medicine, Infection and Immunity; Harvard School of Dental Medicine; Boston, Massachusetts, USA.

Correspondence to: Dr Myron Nevins, 90 Humphrey Street, Swampscott, MA; fax: 781-598-8050; email: nevinsperimp@aol.com.

©2014 by Quintessence Publishing Co Inc.

variety of regenerative efforts, but there is a paucity of evidence that would establish a reliable therapeutic approach.⁹⁻¹² The obvious impediment to regenerative therapy is the decontamination of the implant surface now that most contemporary implants have a rough surface to promote osseointegration. Initial reports demonstrate that the erbium:yttrium-aluminum-garnet (Er:YAG) laser will remove the microbial infiltrated oxide layer without deforming the implant or damaging the supporting bone.¹³ This then provides a clean surface to construct new BIC and enable the implant to continue to support the prosthesis.

The objectives of this investigation were to:

1. Determine the Er:YAG laser's ability to treat peri-implantitis by stripping the contaminated titanium oxide layer to promote induction of osseointegration
2. Study the hard and soft tissue adaption to a previously diseased implant surface

Method and materials

This was a prospective preclinical study investigating the use of the Er:YAG laser to decontaminate the surface of a compromised dental implant with a rough surface. The study protocol was approved by the Institutional Animal Care and Use Committee at PARF in Massachusetts, USA. Six foxhounds weighing approximately 25 kg

were selected for the study. They were observed for 14 days for acclimation to their surroundings and then divided into three treatment groups:

- *Group A (2 animals):* Premolars (P2 to P4) and the first molar (M1) were extracted, and the edentulous ridge was allowed to heal for 60 days. Implants were placed, combined with the creation of infrabony defects, resembling bone loss that accompanies peri-implantitis.¹³ Laser decontamination in addition to a bone grafting procedure was planned for this group.
- *Group B (3 animals):* Premolars (P2 to P4) and the first molar (M1) were extracted, dental implants were immediately placed into extraction sockets, and the areas surrounding the implants were prevented from having spontaneous bone fill, thus creating infrabony defects. Laser decontamination in addition to a bone grafting procedure was planned for this group.
- *Group C (1 animal, control):* Similar to group A, except no laser treatment was rendered to treat the contaminated dental implant surface.

All surgical procedures were performed under general and local anesthesia in sterile conditions. Initial intramuscular administration of xylazine hydrochloride (2.2 mg/kg) and tiletamine hydrochloride/zolazepam hydrochloride (10 mg/

kg) was followed by inhalation of 1.5% to 2% isoflurane as a general anesthesia for the duration of the procedure. Local anesthesia (2% lidocaine with 1:100,000 epinephrine) was provided at the surgical sites.

For groups A and C, dental implants were placed after 60 days with intentional infrabony defects filled with retraction cord (Ultrapak, Ultradent) to interfere with healing (Figs 1a and 1b). The group B animals received implants placed in the extraction wounds, and the space surrounding the implants received retraction cord to prevent bone regeneration (Figs 2a and 2b). The implants (Brånemark System Mk III Groovy RP 3.75 × 10 mm, Nobel Biocare) were placed under the same surgical conditions as the tooth extractions. A total of four implants were inserted into each animal (two on each side) according to a randomized distribution pattern generated for each animal before the surgery. The implant osteotomy was performed with torque reduction rotary instruments using sterile saline solution with an insertion device and hand ratchet according to the manufacturer's guidelines. The flaps were adapted for tension-free wound closure with interrupted and horizontal mattress sutures. The animals received the standard postsurgical infection and pain control consisting of intramuscularly administered cefazolin sodium (20 mg/kg) and buprenorphine HCL (0.02 mg/kg). Both groups were then observed for 60 days, at which time the bone defects were treated.



Fig 1a (left) In group A, dental implants were placed 60 days after extraction, and intentional infrabony defects were created adjacent to the implants.



Fig 1b (right) The infrabony defects were filled with retraction cord to interfere with normal bone healing.

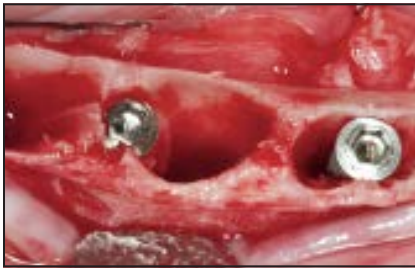


Fig 2a (left) For group B, dental implants were immediately placed into the extraction wounds.



Fig 2b (right) The space surrounding the implants received retraction cords to prevent spontaneous bone regeneration.



Fig 3 Er:YAG laser treatment and augmentation procedures in groups A (a and b) and B (c and d). (a and c) Two months after the implant surgery and peri-implantitis phase, Er:YAG laser was used to decontaminate the implant surface. (b and d) A mixture of 50% autogenous bone and 50% xenograft was used to fill the osseous defects, and the composite was covered with a collagen barrier membrane.



Peri-implantitis treatment

Two months after the implant surgery and the induction of peri-implantitis phase, Er:YAG laser (surface ablation set to 100 mJ/mm², 20 pps) treatment was rendered for both groups A and B. Twenty implants from groups A and B received Er:YAG laser treatment in addition to

bone grafting/membrane treatment (Fig 3), while the remaining four implants from group C received only the bone grafting procedure without Er:YAG laser treatment. Flap access allowed for Er:YAG laser treatment of the implant surface. The micro-explosions from the laser removed the contaminated oxide layer from the implant surface, a mixture of

autogenous bone and 50% xenograft (Equimatrix, Osteohealth) was used to fill the osseous defects, and the composite was covered with a collagen barrier membrane (Collagen Membrane, Dentium) to help contain the bone graft material.¹³ Wound closure and postsurgical animal care protocols were exactly the same as for previous procedures.



Fig 4a Very good BIC, including reosseointegration of previously contaminated implant threads, can be noted on this group A specimen.



Fig 4b Another group A specimen demonstrating partial reosseointegration of previously contaminated implant threads.



Fig 4c Group B specimen demonstrating good BIC, but complete reosseointegration of previously exposed threads has not been achieved.



Fig 4d Another group B specimen demonstrating good BIC, although several threads were still exposed.

Light microscopy

All six animals were sacrificed 3 months after peri-implantitis treatment, and the jaws were resected en bloc using an oscillating autopsy saw. The recovered specimens were immediately immersed in fixative for histologic preparation and evaluation.

Following complete dehydration, the specimens were embedded in ascending grades of ethanol (60%, 80%, 96%, and absolute ethanol) in a light-curing one-component composite resin (Technovit 7200 VLC, Heraeus Kulzer). Polymerized blocks were initially ground to bring the tissue components closer to the cutting surface. A 100- μ m-thick section attached to the second slide was sawed with a diamond blade and 50 to 100 g of pressure. The block sections were sectioned again in a mesiodistal direction, parallel to the long axis of the implant. The final thickness of 40 μ m was achieved

by grinding and final polishing with 1,200-, 2,400-, and 4,000-grit sandpaper. Sections from each block were used for Sanderson RBS staining and acid fuchsin counterstain. Light microscopic overview images of the cores were taken digitally with a Leica M16 stereomicroscope (Leica Microsystems).

Results

Clinical

This investigation was designed to emulate clinical situations, including localized edentulous ridges (delayed implant placement) and implant placement into extraction sites. Both groups presented significant bone defects that were allowed to develop into chronic inflammatory defects through the placement of dental floss in the space between the implants and bone for 6 weeks. All implants were

in place at the conclusion of the study with minimal gingival inflammation. Many of the cover screws were visible, and some had recession of two to three threads. There were no clinical distinguishing features between the two groups.

Histology

Most of the histologic results demonstrated improved BIC to a varying but sufficient degree to enable a patient to continue using the implant. The gingiva was almost depleted of inflammatory cells, indicating the resolution of infection. There was new bone in contact with the implant surface for implants treated with the Er:YAG laser (groups A and B; Figs 4a to 4d), in contrast to the results of the group C implants (Fig 5), where there was a façade of new bone and weak BIC. This may be obscured in the radiographic analysis but is

Fig 5 Group C specimen demonstrating weak BIC and lack of reosseointegration of previously contaminated implant threads.



obvious when assaying the histologic results. Histomorphometric analysis revealed 3.37 ± 0.72 mm of new bone formation for group A, 2.56 ± 0.71 mm for group B, and 1.83 ± 0.75 mm for group C.

Discussion

Peri-implantitis, a chronic inflammatory process resulting in a loss of BIC on osseointegrated implants, has surfaced as an international dental clinical reality.¹⁻³ This is not a condition that one should anticipate for all implants, but is a serious consideration when it occurs. There is considerable evidence of a cause-and-effect relationship between microbial plaque colonization and the peri-implant infections.⁴⁻⁷ This has led to a series of protocols, surgical and nonsurgical, to resolve the problem, but there is a paucity of evidence to suggest any one treatment modality.⁸⁻¹² An ideal therapy

should arrest the disease and promote the regeneration of substantial lost BIC.

The Er:YAG laser system seems to be promising in this application because it possesses the capacity to effectively remove calculus and bacterial colonizations from the titanium implant with no thermal side effects on adjacent tissues due to the high absorption of its emission wavelength (2,940 nm) by water.^{14,15} The Er:YAG laser has potential to demonstrate an important role in treating peri-implantitis due to the microexplosions that are created when the laser energy is absorbed by the water, and the volume can expand by 800 to 1,000 times.¹⁶ The Er:YAG laser is effective at eliminating both the accretions on the implant surface and the contaminated oxidized titanium layer without affecting the potential for reosseointegration. The realistic result relates to the level of bone loss, but the endpoint goal is a treated

implant that will continue to function successfully for the patient.

This study was designed with the understanding that a more aggressive reconstructive protocol with a stable structured scaffold would be necessary to gain complete regeneration of the extensive defects created in this preclinical study. It has been previously demonstrated that vertical defects in preclinical models can be predictably regenerated with a nonresorbable barrier membrane (eg, expanded polytetrafluoroethylene).¹⁶ The present authors selected a regenerative approach to concentrate on the potential positive effect of the Er:YAG rather than the regenerative technique. The regenerative protocols used in this study have been well documented in the treatment of localized edentulous defects using bone augmentation for dental implant placement.^{9-15,17,18} In order to better understand the therapeutic effects of the laser, the present

authors selected a combination of 50% autograft and 50% xenograft together with a resorbable membrane, as these are a common treatment regimen utilized in clinical practice.¹⁹

The overall observations were a reflection of the defect size as a proportion of the threads denuded. Most of the results were similar in regard to arresting progression of disease and the establishment of a strong BIC, but there is a notable difference in the percentage of new bone recovery. The results suggest this would be a preferable option compared with removal of an implant and re-treatment in posterior areas, but its selection would be subject to individual consideration when treating the esthetic zone. The authors have studied the extent and quality of osseointegration on a previously diseased implant surface.¹³

Conclusions

The results of previous research demonstrate the capability of the Er:YAG laser to decontaminate the implant surface by removing the contaminated oxide layer to provide a fresh surface for regeneration.¹⁰ It has also been demonstrated that there is a minimal rise in temperature and, therefore, no visible damage to adjoining bone. The use of the Er:YAG laser for implant surface treatment allowed for regeneration and improved BIC in this preclinical study. New BIC and arrest of the inflammatory process in the soft tissues were observed.

Acknowledgments

The authors reported no conflicts of interest related to this study.

References

1. Peri-implant mucositis and peri-implantitis. A current understanding of their diagnosis and clinical implications. *J Periodontol* 2013;84:436–443.
2. Albrektsson T, Buser D, Chen ST, et al. Statements from the Estepona consensus meeting on peri-implantitis, February 2–4, 2012. *Clin Implant Dent Relat Res* 2012;14:781–782.
3. Klinge B, Meyle J. EAO consensus report: Peri-implant tissue destruction. The third EAO consensus conference. *Clin Oral Implants Res* 2012;23:108–110.
4. Mombelli A, Müller N, Cionca N. The epidemiology of peri-implantitis. *Clin Oral Implants Res* 2012;23(suppl 6):67–76.
5. Berglundh T, Zitzmann NU, Donati M. Are peri-implantitis lesions different from periodontitis lesions? *J Clin Periodontol* 2011;38(suppl 11):188–202.
6. Heitz-Mayfield LJA. Peri-implant diseases: Diagnosis and risk factors. *J Clin Periodontol* 2008;35(suppl 8):292–304.
7. Mombelli A, Décaillot F. The characteristics of biofilms in peri-implant disease. *J Clin Periodontol* 2011;38(suppl 11):203–213.
8. Muthukuru M, Zainvi A, Esplugues EO, Flemmig TF. Nonsurgical therapy for the management of peri-implantitis: A systematic review. *Clin Oral Implants Res* 2012;23(suppl 6):77–83.
9. Renvert S, Polyzois I, Claffey N. Surgical therapy for the control of peri-implantitis. *Clin Oral Implants Res* 2012;23(suppl 6):84–94.
10. Froum SJ, Froum SH, Rosen PS. Successful management of peri-implantitis with a regenerative approach: A consecutive series of 51 treated implants with 3- to 7.5-year follow-up. *Int J Periodontics Restorative Dent* 2012;32:11–20.
11. Froum SJ, Rosen PS. Reentry evaluation following treatment of peri-implantitis with a regenerative approach. *Int J Periodontics Restorative Dent* 2014;34:47–59.
12. Parma-Benfenati S, Roncati M, Tinti C. Treatment of peri-implantitis: Surgical therapeutic approaches based on peri-implantitis defects. *Int J Periodontics Restorative Dent* 2013;33:627–633.
13. Yamamoto A, Tanabe T. Treatment of peri-implantitis around TiUnite-surface implants using Er:YAG laser microexplorations. *Int J Periodontics Restorative Dent* 2013;33:21–30.
14. Aoki A, Sasaki KM, Watanabe H, Ishikawa I. Lasers in nonsurgical periodontal therapy. *Periodontol 2000* 2004;36:59–97.
15. Schwarz F, Aoki A, Sculean A, Becker J. The impact of laser application on periodontal and peri-implant wound healing. *Periodontol 2000* 2009;51:79–108.
16. Schenk RK, Buser D, Hardwick WR, Dahlin C. Healing pattern of bone regeneration in membrane-protected defects: A histologic study in the canine mandible. *Int J Oral Maxillofac Implants* 1994;9:13–29.
17. Nevins M, Heinemann F, Janke UW, et al. Equine-derived bone mineral matrix for maxillary sinus floor augmentation: A clinical, radiographic, histologic, and histomorphometric case series. *Int J Periodontics Restorative Dent* 2013;33:483–489.
18. Nevins M, Cappetta EG, Cullum D, et al. Socket preservation procedure with equine bone mineral: A case series. *J Periodontics Restorative Dent* 2014;34(suppl) s51–s57.
19. Camelo M, Nevins ML, Lynch SE, Schenk RK, Simion M, Nevins M. Periodontal regeneration with an autogenous bone-Bio-Oss composite graft and a Bio-Gide membrane. *Int J Periodontics Restorative Dent* 2001;21:109–119.