

A Rationale for Postsurgical Laser Use to Effectively Treat Dental Implants Affected by Peri-implantitis: Two Case Reports



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Peri-implantitis is a biologic complication that can affect the survival of a dental implant. Most surgical and nonsurgical treatments have been relatively ineffective even when using targeted antimicrobial approaches. A growing number of reports are documenting the presence of titanium granules and/or cement in the soft tissues surrounding peri-implantitis-affected dental implants. Two case reports are presented demonstrating how the Nd:YAG or a carbon dioxide (CO₂) laser used following regenerative surgeries changed failures into successes as measured by radiographic bone fill and improved clinical parameters. These cases suggest that successful peri-implantitis treatment may need to incorporate decontamination of the soft tissues in addition to the implant's surface. Further studies are warranted to determine if each of these lasers would be successful over a larger patient cohort.

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The use of dental implants has been a reliable treatment option for replacing missing or soon-to-be-missing teeth. While dental implants have demonstrated great reliability, their long-term use has uncovered problems that were not necessarily apparent or reported in the early years when they were first implemented.^{1–3} Biologic problems represent one particular category of dental implant complications that have recently received a great deal of attention.^{4,5}

Peri-implantitis has been commonly described in the literature as a pathologic condition occurring in tissues around dental implants, characterized by inflammation in the peri-implant mucosa and progressive loss of supporting bone.^{5,6} The recent World Workshop held by the American Academy of Periodontology (AAP) and the European Federation of Periodontists (EFP) on the classifications of periodontal and peri-implant diseases identified strong evidence that there is an increased risk of developing peri-implantitis in patients who have a history of chronic periodontitis, poor plaque control skills, and no regular maintenance care after implant therapy.^{5,6} The data analyzed by this conference regarding both smoking and diabetes as potential risk factors/indicators for peri-implantitis was found to be inconclusive.⁵ Moreover,

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the literature evidence that the peri-implantitis group reviewed⁵ pointed to there being some limited evidence linking peri-implantitis to other factors, such as postrestorative presence of submucosal cement, lack of peri-implant keratinized mucosa, and positioning of implants that make it difficult to perform oral hygiene and maintenance.

When reviewing the literature regarding treatment approaches for peri-implantitis, most systematic reviews affirm that there is no best approach for effective treatment.^{7,8} Nonsurgical approaches have been consistently ineffective^{9,10} and surgery has been disappointing in its ability to arrest the disease, even when targeted antimicrobial therapy has been employed.^{11,12} It has been speculated that the reason targeted antimicrobial therapy may be ineffective is that no one agent kills all the bacteria and that a dual antibiotic approach, which has been absent in many of the historic approaches, may be necessary.¹³

There have been a number of reports in the literature regarding foreign materials being found in biopsy material obtained around peri-implantitis-affected implants.¹⁴ These materials have included titanium particles and/or residual cement.¹⁵ These two foreign materials may not have been eliminated by the locally or systemically administered antimicrobials or even by surgical approaches that concentrate on mechanical/chemotherapeutic implant-surface decontamination.¹⁶ One possible method for ablating these particles is the use of a laser, which will affect the surrounding

soft tissues.¹⁷ This article presents two case reports demonstrating how the use of either the Nd:YAG or CO₂ laser in the first several months following surgery changed an incomplete treatment into very successful outcomes.

Case 1

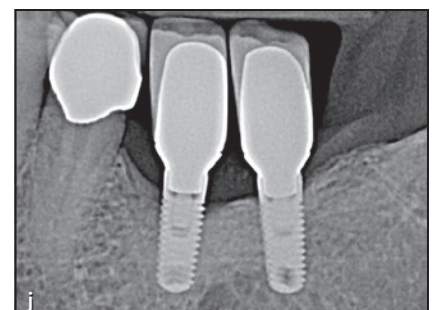
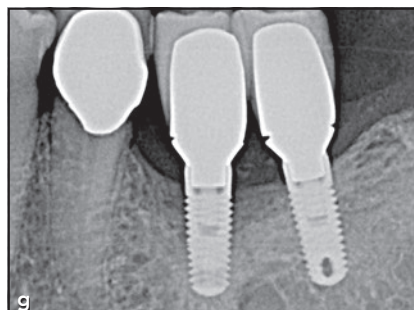
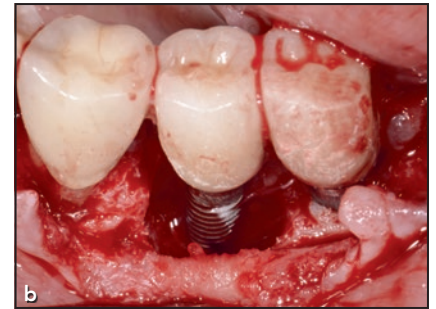
A 63-year-old Caucasian man was referred for evaluation and treatment of several dental implants that were failing due to peri-implantitis. The lesion had reached an advanced level of disease as evidenced by greater than 50% bone loss.¹⁸ The gravest concern was the failing dental implant at the mandibular left first-molar site (Fig 1a). His medical history included hypertension for which enalapril was being taken. The implant under examination had probing depths that reached 9 to 10 mm circumferentially with purulence and bleeding elicited upon both probing and pressure to the tissues. The adjacent teeth and a dental implant had probing depths that ranged from 3 to 4 mm, and the patient was being seen every 3 to 4 months for his periodontal maintenance. Due to the severe bone loss and probing that suggested the lesion was contained, it was determined that a surgical regenerative approach would be performed.

The protocol for this treatment has been previously described.^{19,20} In summary, informed consent was first obtained. Local anesthesia was administered using Septocaine (Septodont) 4% with 1:100,000 epinephrine. Full-thickness flaps were

elevated with periosteal release to allow for adequate flap mobilization for visualization, implant surface access, and coronal advancement at time of closure. Surface debridement was performed using airborne-particle abrasion with glycine (Fig 1b) followed by citric acid (50% solution) applied for approximately 1 minute with cotton pellets. Each step was separated by a vigorous rinse of sterile water. Recombinant human platelet-derived growth factor-BB (rhPDGF-BB) (Gem 21, Lynch Biologics) was then placed on the decontaminated surface of the implant, and the circumferential lesion received a composite graft of freeze-dried bone/demineralized freeze-dried bone allografts in a 70:30 ratio (Creos Allo.Gain, Nobel Biocare) (Fig 1c) hydrated by the rhPDGF-BB and layered/contained by a collagen membrane (Creos Xenoprotect, Nobel Biocare) (Fig 1d). The graft was hydrated with rhPDGF-BB together with enamel matrix derivative (Emdogain, Straumann). The site was sutured with 5-0 polytetrafluoroethylene sutures (Omnia) (Fig 1e). Postoperative pain management was achieved with 600 mg ibuprofen, and infection control included systemic use of amoxicillin for 7 days (875 mg twice daily) and an anti-inflammatory triple botanical rinse (PeriActive, IZUN Oral Care). The patient was seen every 2 weeks for follow-up.

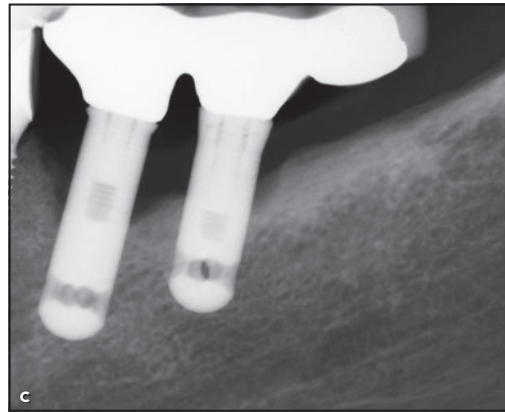
At 5 months, the outcome of treatment appeared to be suboptimal, as evidenced by highly inflamed soft tissues (Fig 1f) and poor osseous profile/bone fill (Fig 1g). The decision was made to treat the area

Fig 1 Case 1. (a) Pretreatment radiograph suggests severe bone loss on the anterior implant in the mandibular left first-molar site. (b) The lesion seen is a circumferential moat around this anterior implant at the first molar. There is some residual glycine powder on the dental implant following airborne-particle abrasion. (c) Following implant surface decontamination, the lesion is filled with an allograft of mineralized:demineralized bone in a 70:30 ratio. The graft has been hydrated with rhPDGF-BB. (d) A collagen membrane has been adapted over the composite graft in an effort to contain it. (e) The flaps have been advanced to completely cover the graft membranes and were secured with 5-0 polytetrafluoroethylene sutures using an interrupted technique. (f) The clinical view at 5 months demonstrates that the soft tissues are still fairly inflamed. When lightly cleaning and probing the area, there is bleeding with this provocation. (g) The 5-month postoperative radiograph suggests some improvement with incomplete healing. (h) The site has been treated using an Nd:YAG laser with two passes. Intervening these two passes, the site received curettage of the soft tissue along with postoperative use of amoxicillin. (i) Clinical view 1 year after the initial treatment. The soft tissues surrounding the dental implant are greatly improved. Probing depths were consistent with good health and there was an absence of bleeding. (j) The radiograph taken at the 1-year follow-up suggests a substantial improvement in the hard tissues surrounding the dental implant, which starkly contrasts both the pretreatment and 5-month radiographs.





Figs 2a to 2c Case 2. (a) Preoperative clinical view of an implant-supported fixed partial denture showing recession around dental implants with lack of gingival tissue. (b) Probing depth is 7 mm with bleeding and suppuration. (c) The preoperative radiograph suggests severe peri-implantitis with greater than 50% bone loss around the implants.



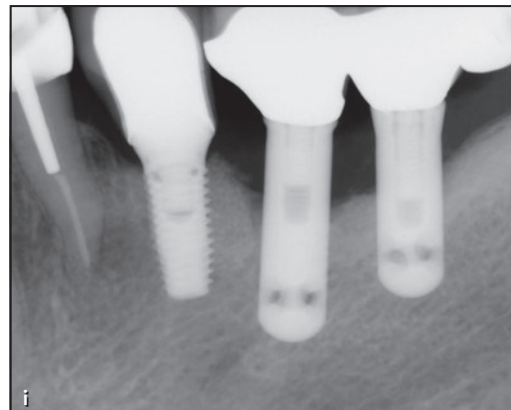
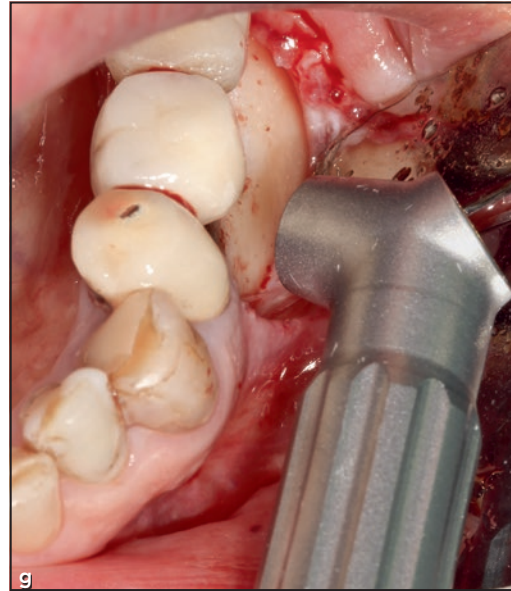
with the Nd:YAG laser (PerioLase, Millennium Dental Technologies). A first pass was performed with a setting of 3.6 watts, 100 milliseconds, and 20 Hz delivering 65 J, followed by curettage of the tissue and then a second pass with a setting of 3.6 watts, 650 milliseconds, and 20 Hz for 77 J (Fig 1h). Amoxicillin (875 mg, twice daily) was again given for 7 days as a postoperative antibiotic. The patient was seen at 1 week, 4 weeks, and 3 months postoperative and every 3 months thereafter. Probing depth measurements were performed at 6 months and were reduced to 4 mm with an absence of bleeding. Two years af-

ter the initial surgery, there was radiographic evidence of a favorable gain in bone, and the soft tissue parameters were consistent with good health (Figs 1i and 1j).

Case 2

A 67-year-old woman, with a non-contributory medical history taking a multivitamin with no stated food or drug allergies, was referred for evaluation and treatment of a cantilevered implant partial denture in the mandibular left quadrant that was failing due to peri-implantitis. She had hydroxyapatite-coated

dental implants (Calcitek, Zimmer Biomet) placed over 20 years ago at the mandibular left first- and second-molar sites that were splinted with a cantilever distal pontic at the third-molar site (Fig 2a). These implants were affected by severe peri-implantitis¹⁸ with probing depths greater than 7 mm, over 50% bone loss (Figs 2b and 2c), and keratinized tissue measuring less than 1 mm in width and thickness. In addition, she had a rough-surface implant (Osseotite, Zimmer Biomet) placed at the second-premolar site approximately 10 years earlier with evidence of early to moderate peri-implantitis.¹⁸ The dentition of this



Figs 2d to 2i Case 2. (d) Regenerative graft material (anorganic bovine bone collagen hydrated with rhPDGF-BB) was placed around the implants at the time of surgery. (e) A xenograft collagen soft tissue graft was placed over the bone-replacement graft material mixed with enamel matrix derivative. (f) Soft tissue healing at 3 months after the initial surgery appears poor as ongoing suppuration is present. (g) Surgical reentry procedure allowing for soft tissue laser ablation of the flap using a 9.3-micron CO₂ laser. (h) Favorable soft tissue healing is seen at 3 years after the initial procedure, demonstrated by both an increase in the gingival tissue and the lack of bleeding on probing. (i) The radiograph taken at the 3-year follow-up shows bone fill of the hard tissue defect, suggesting improved health of the implants.

patient was periodontally stable, with probing depths ranging from 2 to 4 mm. She was seen at 3-month intervals for maintenance visits. Due to the severe bone loss and lack of keratinized tissue, it was determined that a surgical hard and soft tissue regenerative approach would be performed.

The protocol for treatment has been previously described.^{19,20} In summary, informed consent was first obtained. Local anesthesia was administered using Septocaine (Septodont) 4% with 1:100,000 epinephrine. Full-thickness flaps were elevated with periosteal release to allow for adequate flap mobilization ensuring visualization, implant surface access, and coronal advancement at the time of closure. Implant-surface debridement was performed using airborne-particle abrasion with glycine followed by mechanical debridement with a titanium brush (RotoBrush, Salvin Dental) followed by citric acid (60% solution) applied for approximately 1 minute with cotton pellets. Each step was separated by a vigorous rinse with sterile water. rhPDGF-BB (Gem 21) was then applied to the decontaminated surface of the implant and layered with anorganic bovine bone with 10% collagen (Bio-Oss, Geistlich) (Fig 2d). Because of the limited keratinized tissue, a bovine collagen soft tissue matrix (Mucograft, Geistlich) (Fig 2e) was placed over the bone replacement graft. The graft was hydrated with rhPDGF-BB along with enamel matrix derivative (Emdogain). The tissue was coronally advanced and secured using a sling suture tech-

nique with 5-0 polygalactin sutures (Vicryl, Ethicon). Postoperative pain was managed with 800 mg ibuprofen, and infection control included systemic use of amoxicillin (500 mg, tid) for 10 days and a homeopathic antibacterial/anti-inflammatory rinse (VEGA Oral Rinse, StellaLife). The patient was seen every 2 weeks for follow-up.

At 3 months, healing appeared suboptimal as the soft tissues showed high inflammation with suppuration (Fig 2f). The decision was made to treat the area with the 9.3-micron-CO₂ Solea laser (Convergent Dental). A mucoperiosteal full-thickness flap was reflected, and both the implant surface and the soft tissue flap were ablated using a setting of 1.0 watts, lower power mode, 1.2-mm spot size, 30% cutting speed, 100% mist (13 mL/minute), with a contra-angle hand-piece (Fig 2g). Amoxicillin (500 mg, tid) was again given for 10 days as a postoperative antibiotic. The patient was seen at 1 week, 3 weeks, and 3 months postoperative and every 3 months thereafter. Probing-depth measurements were performed at 1 year and were reduced to 3 mm with an absence of bleeding. Three years after the initial surgery, there was a favorable gain in both hard and soft tissues, indicative of good health (Figs 2h and 2i).

Discussion

Dental implants differ from teeth in their ability to handle inflammation, which stems from their differences in the interfacial soft tissue interfaces.

Teeth have both connective tissue fibers inserting into their root surface cementum in addition to a hemidesmosomal adherence of the sulcular epithelium. Implants, on the other hand, have only a hemidesmosomal adhesion of the supracrestal epithelium to act as a barrier to the oral/sulcular environment, since the supracrestal connective tissue fibers run parallel to the surface of the implant. Thus, anything that might impact oral sulcular epithelial homeostasis would be of great concern.

The most recent World Workshop held by the AAP and EFP reaffirmed plaque as the primary etiology to peri-implantitis.⁶ At the time of the conference, they also felt that the available evidence did not allow an evaluation of the role that titanium or metal particles play in the pathogenesis of peri-implant diseases. Hence, the concept of implant tribocorrosion as an inflammatory initiator is a departure from the long-held bacterial pathogenesis model.¹⁴ This newer perspective may help explain why many of the treatment efforts have failed, and it may well be the reason clinicians have had to continually explore other treatment methods to better manage this challenging problem. One possible solution has been the additive use of a dental laser to the regenerative treatment for peri-implantitis. Both the Nd:YAG and CO₂ lasers have an impact on the soft tissues. Their ability to ablate residual titanium and/or cement may help enable the sites to successfully heal. In both case reports, a modification of a regenerative surgical algorithm was used and met with

high success.^{19,20} This begins with the algorithm's ability to decontaminate the infected implant surface²¹ but may in some instances fall short if soft tissue decontamination is not achieved.

The cause(s) of these titanium particles to be present in the surrounding soft tissues is currently unknown. There are several possible scenarios, with several having some supporting evidence from the literature. First, titanium particles might be produced during the insertion of the dental implant into the bone.²² Second, there may be dissolution of titanium from the dental implant into the submucosal plaque as a result of the peri-implantitis disease itself.²³ Third, titanium particles might come from the micromotion present at the abutment-implant interface.^{24,25} Fourth, titanium particles might be produced when performing certain professionally administered hygiene procedures around the dental implant.²⁶ The ability of both the Nd:YAG and CO₂ lasers to ablate the soft tissue of this particulate material while killing some of the residual translocated/invasive bacteria may facilitate soft and hard tissue healing.

Suárez-López Del Amo et al²² evaluated as part of their study the impact of the titanium debris that may be created. They took titanium debris that they created from dental implants and cultured the material with normal oral keratinocyte spontaneously immortalized cells. The authors determined that the particles/debris may contribute to the disruption of epithelial homeostasis and potentially compromise

the oral epithelial barrier by damaging the cellular DNA. They also speculated that the particles could be the result of corrosive forces triggered through surface degradation and leaching of metal ions and debris. Nevertheless, further investigative work needs to be performed on this growing area of concern.

The results achieved in these two case reports are just a sample of a growing number of patients treated by these authors. To date, there are no controls to determine how the lasers are truly impacting the treatment outcomes. However, it is the authors' collective observation from a number of their patients treated for peri-implantitis that it is a complex disease entity with many factors associated with its initiation and progression. The merit of adding laser treatment in those patients who have been appropriately treated with surgery and are not completely responding to care should be evaluated in controlled trials.

Conclusions

These two cases are, to the authors' understanding, the first patient case reports in which the use of a laser following regenerative surgery provided a successful solution for residual pathology. Further case reports and controlled trials are needed to determine if each of these lasers would be successful with a larger patient cohort who were treated by a greater number of clinicians.

Acknowledgments

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