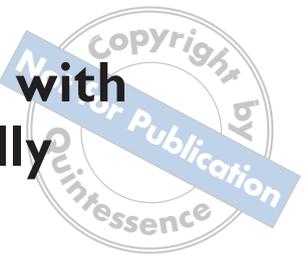# Treatment of Peri-implantitis Lesions with Laser-assisted Therapy and a Minimally Invasive Approach: A Case Report

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ABSTRACT: In addition to conventional treatment modalities, the use of lasers has been increasingly proposed for the treatment of periodontal and peri-implant infections. A 56-year-old female patient had 5 implants with severe and painful peri-implantitis lesions. Local and systemic anti-infective therapies were performed as an adjunct to mechanical treatment but suppuration still persisted. This condition resolved after 3 treatments with diode laser (810 nm) irradiation (1.0 W, CW). During the operation, 2 failed implants were removed, and Er,Cr:YSGG (2780 nm) laser was used for decontamination of implant sockets (1.0 W, 20 Hz) and implant surfaces (0.5 W, 20 Hz), then the implant sockets were augmented. After ten months, radiographic findings showed bone fill in the area of removed implants and no lesions around remaining implants. Laser decontamination provided us with the opportunity to augment the sites without losing soft and hard tissues.

Peri-implantitis has been defined as an inflammatory process affecting the tissues around an osseointegrated implant in function, resulting in loss of supporting bone.[1] It has been reported that the microbiota associated with peri-implantitis corresponds to that observed at sites with advanced periodontitis, and it has been suggested that periodontal pathogens present in the periodontal pockets of teeth may colonize newly inserted implants and give rise to tissue breakdown.[2] This is very common in the case of partially edentulous patients with active periodontal disease and who also bear implants. In this group of patients, colonization of the peri-implant sulcus is observed in one month following the connection of the implant to its prosthetic abutment.[3,4] This colonization does not necessarily imply that peri-implantitis will develop with the subsequent rapid loss of bone height; hence, it is suggested that, in addition to the presence of these periodontal pathogens, other local, systemic, and genetic factors must coexist in order for prolonged, active infection to actually take place. The most remarkable of these factors include smoking, non-parallel implant direction, excessive mechanical stresses on the implant, systemic conditions, long-term treatment with corticoids, and radiation and chemotherapy.[5]

Diagnosis is based on changes of color on the peri-implant mucosa, bleeding on probing, increased probing depth, suppuration, peri-implant radiotransparency and gradual loss of bone height around the implant.[6]

Clinically unhealthy implants are classified as "ailing" or "failing".[7,8] It is necessary to distinguish between an ailing vs a failing implant to determine the treatment steps necessary to salvage the unhealthy implant. Implants exhibiting soft tissue problems exclusively are classified as ailing and have a more favorable prognosis.[7,9] Peri-implant mucositis involves inflammatory

changes confined to the soft tissue surrounding an implant.[10] Therefore, an implant exhibiting peri-implant mucositis is an ailing implant. In some instances, the ailing implant may have exhibited early bone loss along with soft tissue pocketing.[7,9]

Clinically, lack of osseointegration leads to implant mobility and subsequent failure. Therefore, a mobile implant is a failed implant. Non-functional, failed implants must be removed to prevent the associated bone loss from continuing. The option of removing the implant, allowing for healing, and then placing a new implant should not be ruled out.[11]

Treatment options for ailing and failing implants are varied. The clinician should start conservatively and progress to more invasive therapy, based on a system of comulative interceptive supportive therapy (CIST).[12] The overall goal of therapy is to establish a functional restoration and acceptable esthetics. Therefore, any therapy provided should arrest further loss of bone support and re-establish a healthy peri-implant mucosal seal.[7-9]

Various therapies have been advocated for the resolution of the peri-implant infection and in the restoration of the peri-implant tissues. The most conservative approach to treatment involves nonsurgical therapy. This kind of treatment modality includes three subcategories: pharmacological therapy, occlusal therapy, and mechanical debridement.[8,10] Pharmacological therapies include subgingival irrigation with disinfecting agents, and topical and systemic antibiotics.[9,10] Bacteria associated with failing implants have been found to be sensitive to the following antibiotics: penicillin G, amoxicillin, combination of amoxicillin and metronidazole, and amoxicillin-clavulanate.[13, 14]

Mechanical debridement of tissues surrounding an implant using either plastic hand instruments or ultrasonic instruments with a plastic tip has been suggested. Plastic instruments are necessary to debride plaque from titanium dental implants without damaging the soft titanium surface.[15, 16]

The clinician may use a surgical approach when nonsurgical therapies are not indicated or are unsuccessful. In an implant affected by "infectious failure," the assumption is implant surfaces exposed to periodontopathogens have become contaminated with endotoxins that may interfere with the repair process.[16] The first step in surgical therapy is exposing and treating the bacterially contaminated implant and bone tissue surface.[17]

In the literature, efficacy studies of surgical lasers as a method of decontamination on different implant surfaces depending on power intensities show that bacteria kill rates of up to 99.4% have been attained.[18] The semiconductor 809-nm, the CO_2 and Er:YAG lasers are recommended, since it appears that they do not exert a negative impact on the implant surface. The Er:YAG laser generates the least amount of heat in the bone tissue surrounding the implant.[19-21] The decontamination mechanism of the lasers is based on their thermal effect, which denatures proteins and produces cell necrosis.[21] Another type of laser with a low thermal effect on the bone and implant surface is the Er,Cr:YSGG, which represents an improvement over the technical properties of the Er:YAG.[6]

Further regenerative treatment will depend on the amount of bone loss and the esthetic impact of the implant in question. The main problem is augmentation of the area in intrabony defects surrounding the implant and implant socket after removing the implant; if bone loss is advanced, it will be necessary to surgically debride the soft, peri-implant tissues affected by the chronic infection, decontaminate the microimplant surface and, finally, apply bone regeneration techniques aimed at recovering the lost bone.[22] Bone substitutes should not be placed in infected sites. The clinician should be confident that any active or recent infection has been properly treated.

CASE PRESENTATION

In this case report, a 56-year-old female patient with severe and painful peri-implantitis lesions is presented (Fig 1).

Her chief complaints were continuous and extensive pain in the mandibular anterior region, extraoral redness, and swelling, which made it painful for the patient to touch this area. However, there was no lymphoid involvement observed. Intraoral examination revealed an edentulous maxilla and a complete denture. This examination further revealed five implants in the anterior mandible, which were inserted five years ago, two of which failed with pus discharge and inadequately attached gingiva (Fig 2), one of the implants was dysfunctional, and the last two were observed to have moderate and severe peri-implantitis. In the first and second molars of the posterior mandible, gingival inflammation with deep pockets and Class III furcation defects were seen. Moreover, mobility on left second premolar and molars were observed.

The patient was prescribed combined antibiotic therapy amoxicillin-clavulanate (1000 mg) and ornidazole (500 mg) for five days and instructed to apply home care. The mechanical periodontal therapy with

Fig 1a Clinical view at first visit.

Fig 1b Radiographic view at first visit.

Fig 2 Inadequate gingival attachment and diffuse inflammation of peri-implant and lip mucosa.

Fig 3 Povidone-iodine irrigation.

an ultrasonic device, plastic curettes, and intrapocket povidone iodine (10%) irrigation was performed for several appointments (Fig 3). However, pus discharge was persistent around the two failed implants; therefore local metronidazole gel (25%) was applied as an additional treatment. Slight improvement and gingival recession was observed, but pus discharge was still present.

As all of these conventional treatment approaches failed to stop the suppuration, it was agreed upon to support the treatment with laser decontamination therapy. The condition resolved after three exposures to subgingival diode laser (wavelength 810 nm) irradiation with energy outputs of 1.0 W, continuous wave, and 600-µm flexible fiber delivery system (Fig 4).

Subsequently, the surgical phase was planned to improve the success of the augmentation of the implant sockets with the help of Er,Cr:YSGG laser (wavelength 2780 nm) decontamination. During the operation, two failed implants were removed (Fig 5), intrabony granulation tissues were eliminated (Fig 6), and Er,Cr:YSGG laser irradiation was used to decontaminate implant sockets and implant surfaces with a Z3 probe (320 µm) in a noncontact mode (Fig 7). The energy settings on the control panel were 1.0 W, 20 Hz, 11% air, 10% water for implant sockets and 0.5 W, 20 Hz, 11% air, 10% water for implant surface, and energy densities of 37.5 and 25 J/cm^2, respectively.

Decontaminated implant sockets were augmented with bone substitutes and collagen membrane (Bio-Oss® , Bio-Gide®) (Fig 8).

Fig 4 Diode laser irradiation.

Fig 5a Removal of the bridge and spontaneous extraction of 2 implants.

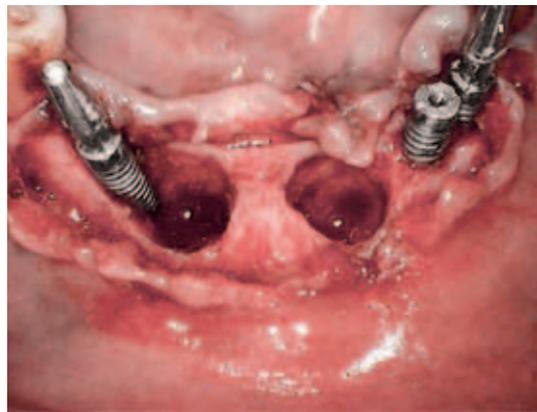**Fig 5b** The axes of the implants were not parallel.

Fig 6 Infected implant sockets and peri-implant bone defect.

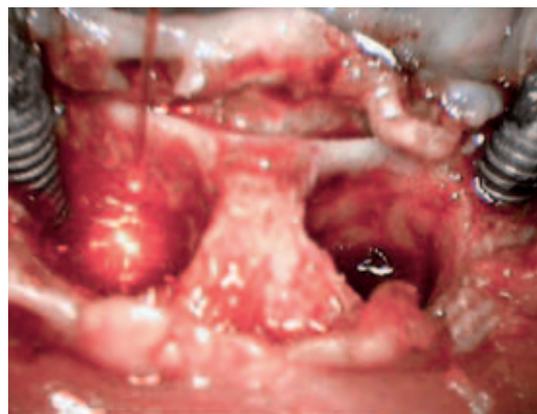**Fig 7a** Implant surface decontamination with Er,Cr:YSGG irradiation.

Fig 7b Socket decontamination with Er,Cr:YSGG irradiation.

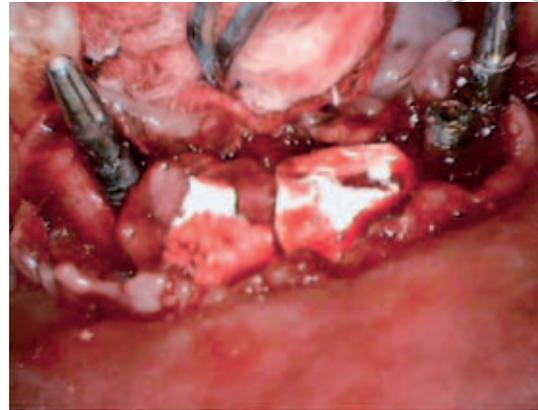**Fig 8a** Alveolar augmentation with bone substitutes.

Fig 8b Placement of collagen membrane.

Fig 9 Placement of the e-PTFE membrane over the collagen membrane.

Fig 10 Appearance of the augmented area after six weeks.

Due to inadequately attached gingiva, a second layer consisting of e-PTFE membrane was placed for protection of the augmented area (Fig 9). The healing period was uneventful (Fig 10).

After ten months, radiographic findings showed bone fill in the area of the removed implants and there were no lesions around the remaining implants (Fig 11). Consequently, the first phase of the main therapy was completed. The patient preferred to postpone the complete treatment.

The mobile premolars and molars on the left side of the mandible were splinted with fiber-supported composite restorations (Fig 12). The patient's current fixed partial denture with implant abutment was not replaced, but was cemented again temporarily.

Occlusal adjustment was performed (Fig 13), due to combination syndrome. This part of the therapy was satisfactory for the patient in terms of function and esthetics (Fig 14). Follow-up is planned after the first phase of treatment, with intervals of three months.

DISCUSSION

This case presents the possible clinical consequence of incorrect treatment planning, including keeping periodontally affected teeth, improper prosthetic restorations, and non-parallel implant direction on the long-term stability of dental implants. Fardal et al[4] also presented a case with severe peri-implantitis in a patient with refractory periodontitis. They suggested that the remaining teeth had acted as a reservoir for periodontal pathogens involved in the peri-implantitis lesions.

There is considerable evidence to support a cause and effect relationship between microbial colonization

Fig 11 Bone fill and preservation of bone level at tenth months.

Fig 12a Fiber splint material.

Fig 12b Fiber-supported uni-composite restoration to stabilize mandibular left pre-molar and molar.

Fig 13 Occlusal adjustment to eliminate primary contacts.

Fig 14 Esthetic appearance.

and the pathogenesis of implant failures.[23-25] Deconta-mination of structured implant surfaces is difficult to achieve, since conventional mechanical treatment ap-proaches, such as plastic curettes, sonic/ultrasonic scalers, and air-powder flow, have been proven to be insufficient for obtaining a complete removal and elimi-nation of both plaque biofilms and bacteria on rough-ened implant surfaces.[26,27] The use of topical antiseptic agents and locally applied antibiotics is recommended for implant detoxification. Povidone iodine is widely used as a topical antiseptic in medicine and potentially beneficial in the management of some periodontal dis-eases.[28,29] Controlled release devices that contain metronidazole (25%) are also beneficial agents in an-timicrobial periodontal therapy.[30] In this case, povi-done iodine (10%) was also used as an adjunct to sys-temic antibiotics, and although subsequent local metronidazole gel failed to stop the suppuration, it did somewhat decrease it. At the same time, mechanical debridement was performed at every appointment. During the nonsurgical phase of therapy, ceasing the disease activity and elimination of infection was thus achieved by diode laser (810 nm) irradiation.

During surgical-phase planning, it is essential for the clinician to recognize unhealthy implants and to deter-mine whether they are ailing, failing, or failed. Ailing and failing implants are amenable to therapy. Implants diagnosed as failed should be removed. Extraction of implants may result in a severely deformed alveolar ridge, and restoration of these cases is usually difficult,

because of oral hygiene, esthetic, and prosthetic problems. The most important measure to this end is to decontaminate implant and tissue surfaces beforehand, in order to enable bone regeneration to take place. With the help of Er,Cr:YSGG laser, decontamination was provided without producing any increase of temperature on the irradiated area.

The combination of bone substitutes and a collagen membrane seems to be preferable in the surgical treatment of peri-implantitis defects and socket augmentation. In this context, it must also be emphasized that membrane exposure has been reported to be a frequent complication.[31] In the present case, successful decontamination was achieved by laser. Subsequently, augmentation of peri-implantitis defects was performed with bone substitutes and double-layer membrane, which resulted in decreased radiolucency within the implant sockets and gain in the alveolar bone height.

CONCLUSION

The development of laser assisted techniques in periodontology and implantology is directed towards minimally invasive procedures, with high success rates, which result in shorter healing periods, high patient comfort, and predictability of the final result. In the presented case, both diode and Er,Cr:YSGG lasers in the nonsurgical and surgical steps of the treatment were used. The highest risk factor in the augmentation procedure and in regenerative periodontal surgery is infected implantation defects/sockets. Placement of the bone substitutes and membranes into infected alveolar sockets would result in an unsuccessful treatment. In this case, laser decontamination was the only option for a minimum-risk treatment, without losing soft and hard tissues and without the need for a second surgical operation. Finally, proper attention to personal and professional plaque removal will allow the maintenance of a successful treatment outcome. Within the limits of the present case, it can be concluded that laser decontamination may be a favorable choice for elimination of persistent infection.

ACKNOWLEDGMENT

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