Review Article

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Peri-Implantitis and Laser Therapy: Review

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ABSTRACT

Background: Implants in present time are being used extensively for missing teeth replacement. In contrast to a natural tooth has a robust junctional epithelium and direct connective tissue attachment to the tooth, the cementum, and the alveolar bone as well as a periodontal ligament, a dental implant has a long, thin junctional epithelium with connective tissue attachment to the alveolar crest of bone and no direct connective tissue attachment to the implant restoration. The direct implant-to-bone interface and the lack of an interposed tissue ligament makes a dental implant less susceptible to the traditional disease mechanisms of periodontitis, however the long, thin junctional epithelium is a less resilient tissue barrier and a point of penetration for chronic inflammation which can lead to peri-implantitis. Various treatment modalities have been advised for the prevention, treatment and maintenance of peri-implantitis.

Keywords: Peri-implantitis, Lasers, LANAP, LAPIP.

INTRODUCTION

Peri-implant diseases are present in two forms: peri-implant mucositis (PIM) and peri-implantitis (PI). The term peri-implantitis was introduced in 1987 by Andrea Mombelli to describe a destructive inflammatory process affecting a soft & hard tissues around osseo-integrated implants leading to formation of Peri-implant pocket and loss of supporting bone. Peri-implant mucositis is a plaque induced reversible inflammation of the soft tissues around the implant and can be treated with non-surgical periodontal therapy. Although there is no correlation to marginal bone loss, peri-implant mucositis may present with bleeding on probing and increased probing depths. Peri-implantitis, which presents around the implant with clinically significant crestal bone loss, is the progressive inflammation of periimplant mucositis.[1]

Biofilm have always been associated with periodontitis and Peri-implant diseases.[2,3] Therefore, removal of the biofilm is essential to prevent, manage and control peri-implant infections.[4] Based on the diagnosis made patients should be on a regular recall schedule, and maintenance programs (CIST) proposed by Lang and co-workers should be designed on an individual basis.[5]

LASER

The term "laser" originated as an acronym for "light amplification by stimulated emission of radiation". Laser dentistry is one of dentistry's latest advances. The laser delivers energy in the form of light. The use of lasers in dentistry has received much attention, in both clinical practice and research. Their unique properties have shown to produce favorable clinical results and encourage patient's acceptance. Lasers can be applied to almost any clinical situations, but their efficacy versus conventional techniques in many cases is not clearly known.

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Various types of lasers have been investigated as an adjunct to periodontal therapy including carbon dioxide (CO2), diode, neodymium: yttrium-aluminium-garnet (Nd: YAG) and erbium: yttrium-aluminium-garnet (Er: YAG) lasers.[6]

PERI-IMPLANTITIS

According to *Albrektsson and Isidor et. al.,* 1994, Periimplantitis is referred to as an inflammatory process effecting the tissues around an osseointegrated implant in function resulting in loss of supporting bone.[7]

Classification of peri-implantitis

Stuart J. Froum, and Paul S. Rosen, proposed a classification which is based on three distinct clinical stages of peri-implantitis: early, moderate, and advanced.[8]

Classification of peri-implantitis	
Early	PD ≥ 4 mm (bleeding and/or suppuration on probing*) Bone loss < 25% of the implant length
Moderate	PD ≥ 6 mm (bleeding and/or suppuration on probing*) Bone loss 25% to 50% of the implant length
Advanced	PD ≥ 8 mm (bleeding and/or suppuration on probing*) Bone loss > 50% of the implant length

For the 3rd EAO Consensus Conference in Pfäffikon, Switzerland – February 2012, a systematic review was undertaken to determine peri-implantitis prevalence and incidence and it was estimated that "five to ten years after implantation, approximately 10 % of the implants and 20 % of the patients were affected by periimplantitis."[9]

Lekholm and coworkers and Holt and associates have demonstrated that the supra and subgingival microflora cultured from titanium endosseous implants are similar to organisms cultured from natural tooth.[10] However, experimental studies and the analysis of human biopsy materials, shows that lesions of peri-implantitis are larger and extend closer to the bone crest than periodontitis lesions. In addition, peri-implantitis lesions contain larger proportions of neutrophil granulocytes and osteoclasts than periodontitis lesions.[11-13]

In recalls following implant placement, the peri-implant tissue should undergo careful clinical and radiological monitoring so that changes will be promptly noted. Diagnosis is based on: a) probing peri-implant soft tissue, b) clinical signs of inflammation and bleeding on probing, c) radiographic images, d) Implant mobility is an indication of a complete loss of osseointegration, and therefore cannot be used for early diagnosis of peri-implantitis, e) suppuration.[14]

Predisposing risk factors for peri-implantitis include smoking[15-17], genetic factors, periodontal disease history, poor oral hygiene[19], poor access reconstructions,[18] insufficiently wide (< 2 mm) keratinized gingiva is linked with elevated plaque accumulation, inflammation and recession[22] and iatrogenic factors such as cement residue.[21]

TREATMENT MODALITIES

Depending on the severity of the peri-implantitis lesion, surgical or nonsurgical procedures should be implemented. Along with mechanical debridement, combined with antiseptic/antibiotic therapy[23], the adjunctive use of the laser have been shown to be effective for treating peri-implantitis[24-25]. The rationale behind nonsurgical and/or surgical approach for treatment of peri-implantitis are[4]

- To reduce the total amount of microorganisms on the titanium surface,
- To reduce and eliminate bleeding on probing (BOP),
- To decrease probing pocket depth (PPD),
- To prevent re-infection,
- To enhance self-performed oral hygiene and peri-implant health,
- To improve implant longevity.

Various treatment protocols for peri-implantitis are described in the literature6. There is

no single measure for resolving peri-implantitis but rather a sequence of steps.

Step 1 – Assessing the situation
Step 2 – Non-surgical debridement
Step 3 – Re-assessment
Step 4 – Surgical intervention
Step 5 – Post-surgical care
Step 6 – Maintenance care

The present review is written to assess the role of non-surgical periodontal therapy in the treatment of peri-implantitis. Non-surgical debridement using appropriate instruments, such as titanium curettes, air-powder abrasive devices, ultrasonic devices, photodynamic therapy, or Er: YAG laser, should precede surgical intervention. Systemic antibiotics, local antimicrobials and/or the use of topical antiseptics (e.g., chlorhexidine) may be concomitantly prescribed. Individual oral hygiene instruction should be provided to ensure good plaque control. Several recent studies have found laser therapy a promising treatment of periodontal disease and now peri-implantitis.

Specifically, a pulsed Nd: YAG laser has been investigated and its efficacy determined for achieving bacterial ablation without damaging the surface properties of titanium implants. Another study found that the use of an Nd: YAG laser was able to totally reduce contamination on irradiated implants.[26, 27] Contributing to the positive effects that the Nd: YAG laser has on treating peri-implantitis are its ability to penetrate the soft tissue to achieve an effective kill of bacteria, and its ability to promote effective hemostasis. The Nd: YAG laser penetrates several mms into soft tissue and dentin. This is a significantly favorable and distinguishing characteristic of the Nd: YAG laser, since the great depth of penetration of the free running, pulsed laser energy allows for a greater kill rate of black pigmented bacteria and the ability of the laser energy to affect deeper blood vessels, creating excellent hemostasis. In contrast to above results, study conducted by Kreisler M et. el., have stated that Nd: YAG and Ho: YAG lasers are not suitable for use in decontamination of implant surface irrespective of power output.

While using CO2 and ER: YAG laser, the power output should be limited to avoid damage. Both laser types have wavelengths that are highly absorbed by water and consequently very shallow penetration depths in tissues. Thus, they cannot access pathogens or affect hemostasis below the surface.

LANAP AND LAPIP

The Nd: YAG laser is at the heart of the Laser-Assisted Peri-Implantitis Procedure (LAPIP) that is based on the successful LANAP (Laser-Assisted New Attachment Protocol) therapy. The LANAP therapy is an FDA-approved protocol that provides cementum-mediated new periodontal ligament attachment to root surfaces in the absence of long junctional epithelium. It treats the periodontal pocket walls to remove diseased epithelium, then seals them with a laser-generated blood clot. The therapy results in greater probing depth reduction and clinical probing attachment level gains, as well as induces periodontal regeneration.[28, 29]

The LAPIP technique is basically an implant-specific modification to the LANAP procedure.

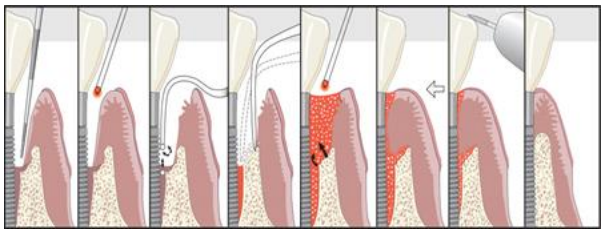

Fig 1: Lapip Protocol.

Both utilize an ablation step to remove inflamed sulcular tissue and decontaminate the root/implant surface, followed by a scaling step using an EMS piezo scaler. A laser induced hemostasis step further decontaminates the tissue and causes the blood to clot, creating a closed system. This seals the area, preventing the down growth of the gingival epithelium and allowing the area to heal from the base of the defect coronally.

A. Perio probe indicates excessive pocket depth. **B.** Laser vaporizes bacteria, diseased tissue, pathologic proteins, and titanium corrosion contaminants in soft tissue. **C.** Ultrasonic scaler tips are used to remove surface accretions.

D. Bone is modified at time of surgery. **E.** Laser is used to form a stable fibrin blood clot containing stem cells from bone. **F.** Adhesion to clean surface, with a stable fibrin clot at the gingival crest to create a 'closed system'. **G.** Occlusal trauma is adjusted. **H.** New attachment is regenerated.

Renvert et. al.,[30] evaluated the 6-month outcomes following treatment with an Er: YAG laser (21 patients with 55 implants) compared with an air-abrasive device (21 patients with 45 implants). There were no patient withdrawals, no implant losses, and no adverse effects of treatment reported.

At baseline, 31% of the implants in the laser group and 38% of the implants in the air powder abrasive group had suppuration on probing. After 6 months, both treatment groups reported 11% of implants with suppuration on probing.

At the patient level, 25% of the patients in the laser group had a mean PD reduction ≥ 1 mm, whereas 38% of the patients in the air-abrasive group had an average PD reduction ≥ 1 mm. The mean BOP was reduced from 100% to 75% in the air-powder abrasive treatment group and from 100% to 70% in the Er: YAG laser treatment group. Radiographic evaluation showed a mean bone loss of 0.1 mm in the air-powder abrasive group and a mean bone loss of 0.3 mm in the Er: YAG laser treatment group. The authors reported that none of the implants in either group had a positive outcome (defined as having PD ≥ 5 mm with BoP and suppuration at baseline, but no PD ≥ 5 mm and no BoP or suppuration at 6 months).

Schar et.al.,[31] evaluated the 6-month treatment outcome of photodynamic therapy compared to local delivery of minocycline (20 patients with 20 implants in each group). There were no withdrawals and no implant losses. At 6 months, the minocycline group had a mean PD reduction from 4.4 mm to 3.9 mm, with a reduction in mean number of sites with BOP from 4.4 to 2.1 sites. The photodynamic group had a mean PD reduction from 4.2 mm to 3.8 mm, with a reduction in mean number of sites with BOP from 4.0 to 2.3 sites.

At 6 months, 15% of the implants in the minocycline group had complete resolution of mucosal inflammation compared to 30% in the photodynamic treatment group. There were no data on radiographic bone levels, suppuration on probing, or complications reported.

CONCLUSION

The treatment of peri-implant diseases must always include anti-infective measures. As with chronic or aggressive periodontitis, the therapy contains of two essential components: professional subgingival instrumentation for the reduction and/or elimination of the pathogenic microbiota6 and oral hygiene instructions for the supra-gingival plaque control. A recent systematic review concluded that in most studies peri-implantitis treatment resulted in an improvement in clinical conditions for the majority of patients. However, in some patients, despite treatment, there was a recurrence or progression of disease requiring re-treatment or removal of the implant.

With respect to the choice of treatment modality, the clinician should choose the most appropriate treatment method based on the individualized needs of the patient.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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