Peri-Implant Disease, a Consensus for Treatment: A Case Study

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The aim of this case report was to find common treatment with the use of laser energy to treat failing implants. This article discusses definition of peri-implantitis and how-to diagnosis peri-implantitis. The article shows a case report of treatment with the use of two different lasers.

Key Words: peri-implantitis, mucositis, photobiomodulation, Erbium, 9300 nm CO_2 laser, 10600 nm CO_2 laser

Introduction

Peri-implant mucositis and peri-implantitis are both caused by bacterial plaque.[1–3] Peri-implantitis is defined as a plaque-induced inflammation that leads to progressive crestal bone loss adjacent to a dental implant. Peri-implantitis is analogous to periodontitis and shares many similarities in etiology and pathogenesis but has some distinct differences.[4] It is estimated that peri-implantitis affects 28%–56% of all implant patients and 12%–43% of all implants.[5] This disease entity is distinct from mucositis, which is also defined as plaque-induced inflammation only without associated bone loss. Peri-implant mucositis is analogous to gingivitis and is a precursor to peri-implantitis. Mucositis is believed to affect about 80% of all implant patients and 50% of all implants.[5] Risk factors for developing both peri-implant mucositis and peri-implantitis include poor oral hygiene, diabetes, a history of periodontitis, and smoking[1,4]—a list that, not surprisingly, is similar to risk factors for developing gingivitis and periodontitis.

When the breadth of the dental implant market is considered, peri-implantitis is potentially a significant global health care problem. In 2012, an estimated 1 260 000 dental implant procedures were performed in the United States alone, at with an estimated market value of $900 million. The dental implant market is expected to grow to more than $2 billion by 2021 in the United States and to $6.8 billion by 2024 worldwide.[6,7] If 28%–56% of all implant patients and 12%–43% of all implants develop peri-implantitis, this potentially adds millions (if not billions) of dollars in additional treatment costs. More importantly, given our understanding of the potential adverse effects of periodontal disease on systemic health, peri-implantitis may also increase the risk for various systemic diseases, given its similarities with periodontitis.

This clinical problem is also exacerbated by the challenges in diagnosis as well as a lack of high quality and long -term treatment data. An accurate diagnosis is an absolute requirement to successfully treat any clinical condition. Crestal bone loss around implants caused by peri-implantitis must be differentiated from bone loss that occurs from a variety of other reasons unrelated to plaque or inflammation. This includes physiologic bone remodeling,[8] establishment of biologic width,[9] occlusal trauma,[10,11] and faulty surgery.[12] Differentiation between bone loss caused by peri-implantitis and other potential etiologies is often difficult and can lead to inaccurate diagnosis and subsequent ineffective treatment protocols. Mucositis, for example, could be associated with an implant that has bone loss and deep pocketing caused by occlusal trauma but was instead misdiagnosed as peri-implantitis.[11] Treatment could therefore focus on surgically reducing deep pockets and removing plaque when the appropriate protocol may be prophylaxis and a minor occlusal adjustment.

Diagnosis

Peri-implant mucositis is defined as a plaque-induced inflammation without associated progressive loss of crestal bone. The clinical findings include mucosal inflammation, bleeding on probing and suppuration, and pathologic pocket formation. Clinical probing of the pocket is essential for making a diagnosis. along with radiographs, when deep pockets are

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encountered to rule out crestal bone loss beyond what is considered normal bone remodeling and adaptation.[1,2,5,13]

Peri-implantitis by definition is a plaque-induced inflammation that leads to progressive loss of crestal bone around an implant. The clinical findings include mucosal inflammation (as demonstrated by bleeding upon probing or suppuration), pathologic pocket formation, and radiographic evidence of bone loss beyond what is expected from physiologic remodeling and adaptation. Therefore, clinical probing of the peri-implant pocket and radiographs are essential for making a diagnosis. Diagnosis of peri-implantitis, however, can be challenging, because there are many theories that can explain crestal bone loss around implants other than plaque-induced inflammation. In addition, the crestal bone loss threshold between what is considered to be normal and what is considered to be disease has no clear consensus.[2,5,13]

Physiologic adaptation and bone remodeling can cause peri-implant crestal bone loss up to 1.5 mm in the first year of function and 0.1 mm each subsequent year.[8] This is widely known and accepted as a normal consequence of osseointegration and occlusal loading. This small degree of bone loss is not surprising, given our understanding of wound healing and associated physiologic bone loss commonly seen with other oral surgical procedures, including tooth extraction[14] and even mucoperiosteal flap reflection.[15] In addition, studies evaluating the characteristics of peri-implant health with disease often use the objective value of 2 mm of bone loss or more as a threshold to diagnose peri-implantitis.[16] Therefore, 2 mm of marginal bone loss appears to be a reasonable clinical threshold for clinicians to distinguish between physiologic bone remodeling and a pathologic condition such as peri-implantitis.[13]

Biologic width establishment is another physiologic phenomenon implicated in crestal bone loss[9] around an implant, although this may be broadly related to physiologic adaptation. Despite differences in the physical attachment between teeth and implants, there is a histologic presence of junctional epithelium and connective tissue adjacent to both teeth and implants with similar histologic dimensions.[17] Following implant placement, the biologic width reestablishes and can lead to crestal bone loss to create the required space. Implant design, depth of placement, and micro-gap position appear to influence the amount of bone loss seen during biologic width establishment.[9] Once again, 2 mm of marginal bone loss appears to be a reasonable clinical threshold to distinguish between normal physiology and pathology.[13]

Occlusal trauma, although controversial, is also accepted as a potential cause of peri-implant bone loss.[10,13,18,19] Crestal bone loss due to occlusal trauma can be conceivably induced when forces exceed the adaptive capacity of the bone-implant interface. A physiologic quantity of internal bone stress is required for normal bone remodeling.[20] Bone loss including loss of osseointegration can occur when these forces are exceeded.[10] Diagnosis of occlusal trauma is therefore difficult due to the inability to quantify these forces, as well as the lack of knowledge of the specific physiologic requirement for that force in a given patient. Bone changes due to occlusal trauma may or may not be associated with inflammation, which further complicates the diagnosis.[10,21] Spontaneous bone repairs around implants with severe crestal bone loss without any intervention other than occlusal adjustment has been documented in multiple case reports.[11,22,23] Despite the lack of clarity due to contradictory findings in the literature, an occlusal analysis and elimination of excessive functional and parafunction forces is at least prudent—if not an absolute requirement—when non-physiologic crestal bone loss is noted around an implant. An exclusive diagnosis of occlusal trauma can be made with some confidence when bone loss is present without inflammation, bleeding upon probing, and pathologic pocket formation.[22,23] However, mucositis associated with inflammation and bleeding on probing can be superimposed on occlusal trauma, which could lead to a misdiagnosis of peri-implantitis[11] and subsequent ineffective treatment.

The concept of a foreign body reaction has been recently introduced as another possible cause of crestal bone loss around implants.[24,25] The authors, many of whom were the earliest researchers to study the phenomenon of osseointegration, postulate that osseointegration itself is a foreign body reaction equilibrium that results in rigid implant fixation. When disturbed, that equilibrium leads to marginal bone loss and eventual implant loss referred to as disintegration by Albrektsson[25] or "osseoseparation"[26] by Koka and Zarb. Dissolution of titanium, metal corrosion, or the release of titanium fragments into the tissues may initiate this disturbance and lead to an adverse foreign body reaction histologically characterized by the presence of multinucleated giant cells.[26] Although further studies are required to validate this theory, it is reasonable to believe that a titanium-fragment-induced foreign body inflammatory reaction can be treated similarly to a bacterially induced inflammatory reaction. Both theories implicate an antigen (titanium fragments vs bacteria) that induces an inflammatory response and leads to bone loss. This would potentially make the diagnostic distinction between the two entities clinically insignificant, as treatment would likely be similar.

The key factor, therefore, in diagnosing peri-implantitis is detection of inflammation associated with pathologic pocket formation and progressive crestal bone loss. Inflammation and pocket depths are best confirmed by the presence of bleeding upon gentle peri-implant probing and/or suppuration. Loss of crestal bone can be detected through probing and confirmed with radiographs. However, it must be understood that only interproximal bone can be visualized radiographically, while the periodontal probe can detect crestal bone loss circumferentially around the entire implant. This validates the periodontal probe as the most important instrument for peri-implant disease detection. Recognition that peri-implantitis may not be the primary cause of the bone loss but may have occurred as a consequence to initial bone loss and pocket formation through another mechanism and is equally important and vital in making a diagnosis to determine effective treatment.

Treatment modalities have consisted of traditional treatments with chemical means using phosphoric acid, citric acid, and tetracycline. These means have been inconsistent at best. In 2006, Dr Robert Miller published an article in "Implant Dentistry" on the use of Er,Cr:YSGG to disinfect the implant surface with the use of laser for disinfection. The use of laser therapy has shown to have a greater effect than do chemical means, and with Miller's publication, it has been shown to

disinfect to depths of over 1 000 microns, compared to depths with the use of chemicals of around 100 microns.

Treatment with lasers has not consistently worked well solely by itself. In April 2017 at the annual meeting of the Academy of Laser Dentistry (ALD), invited companies were asked to participate in a consensus for treatment of peri-implantitis. The committee was asked to find consistent treatment with the use of light energy as an adjunct therapy to treat peri-implantitis. The following criteria were discussed by this group:

- Cause of peri-implantitis
- Wall defects that are treatable
- How to clean the implant surface
- Protocol to treat implant surface with laser energy that has worked consistently and what has not worked consistently
- Use of biologics
- Use of membranes
- Post-operative care
- Use of photobiomodulation
- Home care post-operatively

The following is each company's champion for treating peri-implantitis with their laser device.

Discussion

Lasers that ablate hard and soft issues are Erbium lasers (2780 nm and 2940 nm) and 9300 nm CO_2 laser. The 10600 nm CO_2 laser ablates soft tissue only. Photobiomodulation does not ablate but rather is used to aid in healing of damaged tissues.[27]

Case study treated with 9300 nm CO_2 and 2780 nm Erbium lasers

Presentation using the 9300 nm CO_2 laser (Solea, Convergent, Boston, Mass) featured Kusek. Dr Kusek showed eight cases treated with the CO_2 laser at different energy levels. Two cases showed that treatment using lower energy values had failures and recurrences of infection. Using energy levels of around 14W for at least 40 seconds or over 550 J of energy showed success in all cases. Dr Kusek has subsequently stated that the two failure cases have been retreated with higher energy values and have now been successful, with one case at post-op of over 1.5 years. Kusek's treatment protocol is as follows: extension of the flap two teeth beyond the infected site with only one releasing incision in a non-aesthetic position, with the use of the 9300 nm CO_2 laser. Figure 1 depicts this.

The second step is to elevate the site to expose the granulation tissue over the implant body; Kusek uses a surgical hand-piece to remove the tissue to expose threads of the implant body, as shown in Figure 2.

It is noted that treatments of 3 or 4 wall defects have a good prognosis with at least half of the implant body into solid alveolar bone.[28] The third step is to use a titanium brush (Salvin Dental Specialties, Charlotte, NC) to scrub the surface of the implant to the point that visually, with loupes, there are no tissue tags on the implant surface, as shown in Figure 3.

The fourth step is the use of the 9300 nm CO_2 laser (Convergent Dental) with the setting of 14W of average energy to the surface of the implant body. The laser tip is approximately 15 mm away from the implant surface with 1 mm spot size and 100% water mist; as reported by Mang,[29] it has been shown that the use of a water-cooling system is necessary to keep the implant and bony walls from overheating the site, which would cause surrounding bone to develop necrosis (Figure 4).

The fifth step is to use the 2780 nm ER,Cr:YSGG laser (Biolase Technology, Irvine, Calif) around the bony walls with a setting of 4.5W 31/21 air/water with a 800 micron-sized tip to create bleeding points around the bony walls to obtain fibroblast to aid in the healing process (micro-RAP phenomenon).[30] If an Erbium laser is not available, then either a Piezo to create bleeding points or the use of small round drill used in surgical hand-piece is required. The sixth step is to place a combination of Bio Oss (Geistlich Pharm AG, Wolhusen, Switzerland) material that is soaked in platelet rich fibrin (PRF) plasma and cut into small pieces to place around the implant body. Place over this a slowly resorbing collagen membrane (Biomend Extend, Zimmer, Carlsbad, Calif), fashioned to cover interproximal portion of the implant (Figure 5).

The seventh step is to place a PRF membranes (Intra-spin, Intra-lock, Boca Raton, Fla) to cover the collagen membrane and implant body, as shown in Figure 6.

In the eighth step, the area is sutured with 3.0 PTFE (Salvin) sutures with a deep horizontal mattress for a coronally repositioned flap, followed by interrupted sutures to get tight primary closure. Finally, use of 2780 nm Er,Cr:YSGG (Biolase) laser 4.0W 20Hz 31/0 air/water is used to deepithelialize the tissue in an area of 1 cm around the entire surgical site[31] to aid in healing (Figure 7).

Sutures are removed at 2 weeks. Photobiomodulation using an 800 nm diode is applied to the surgical site for 30 seconds), done at the time of surgery, 1 week after surgery, and then at 2 weeks or at the suture-removal appointment. The patient needs to keep the area clean by manual toothbrush and must not use oral irrigators. The clinician waits for 2 months of healing before an oral irrigator or electric toothbrush is used (Sonic Care, Philips Andover, Mass; Oral B Cincinnati, Ohio). A radiograph should be taken at the 6-month recall appointment.

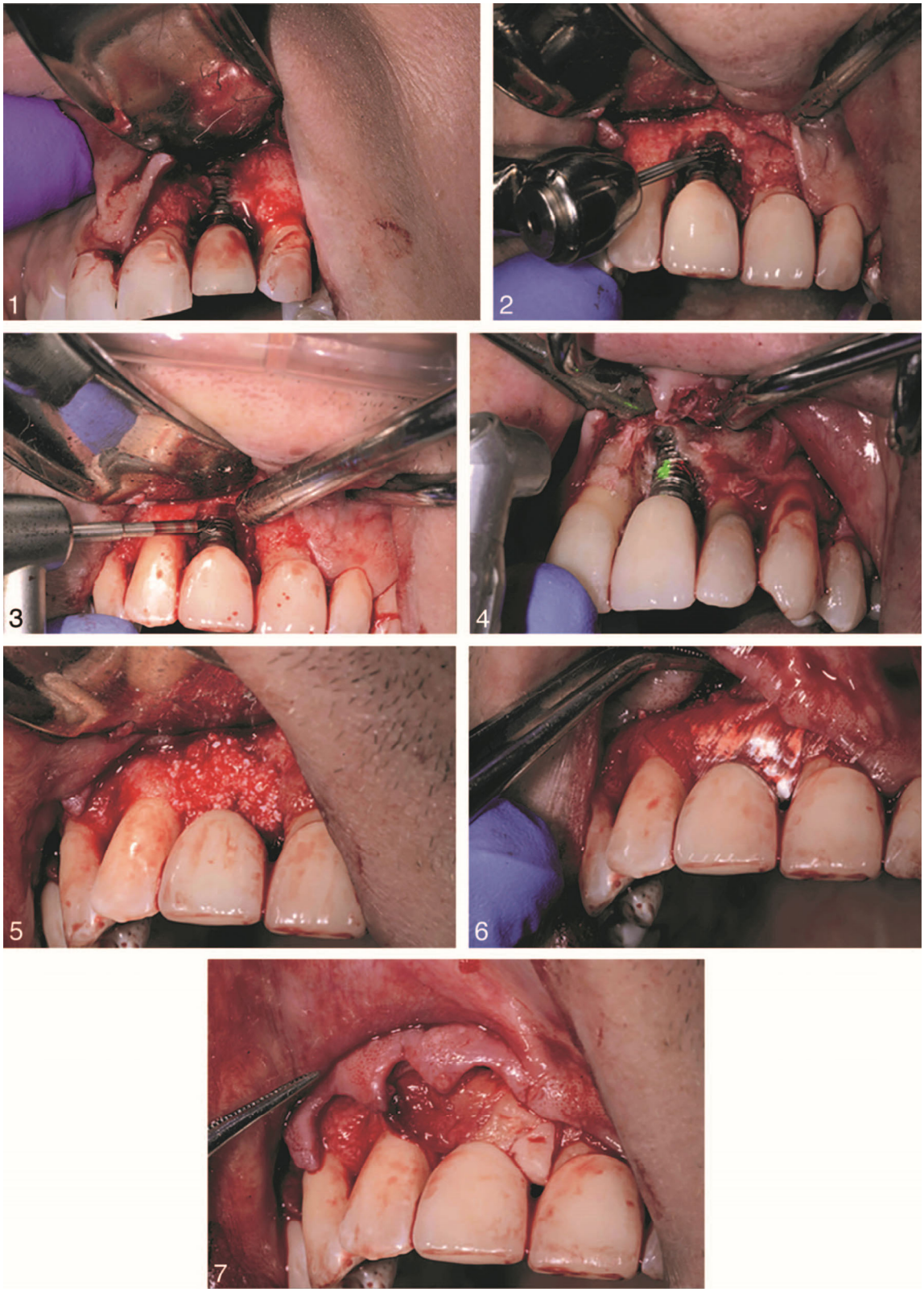
Using this protocol, Kusek has reported 100% success rate for treatment of these 3 and 4 wall defects for a period of 4 years.

Peri-implantitis treatment utilizing closed flap with 10600 nm CO_2 laser

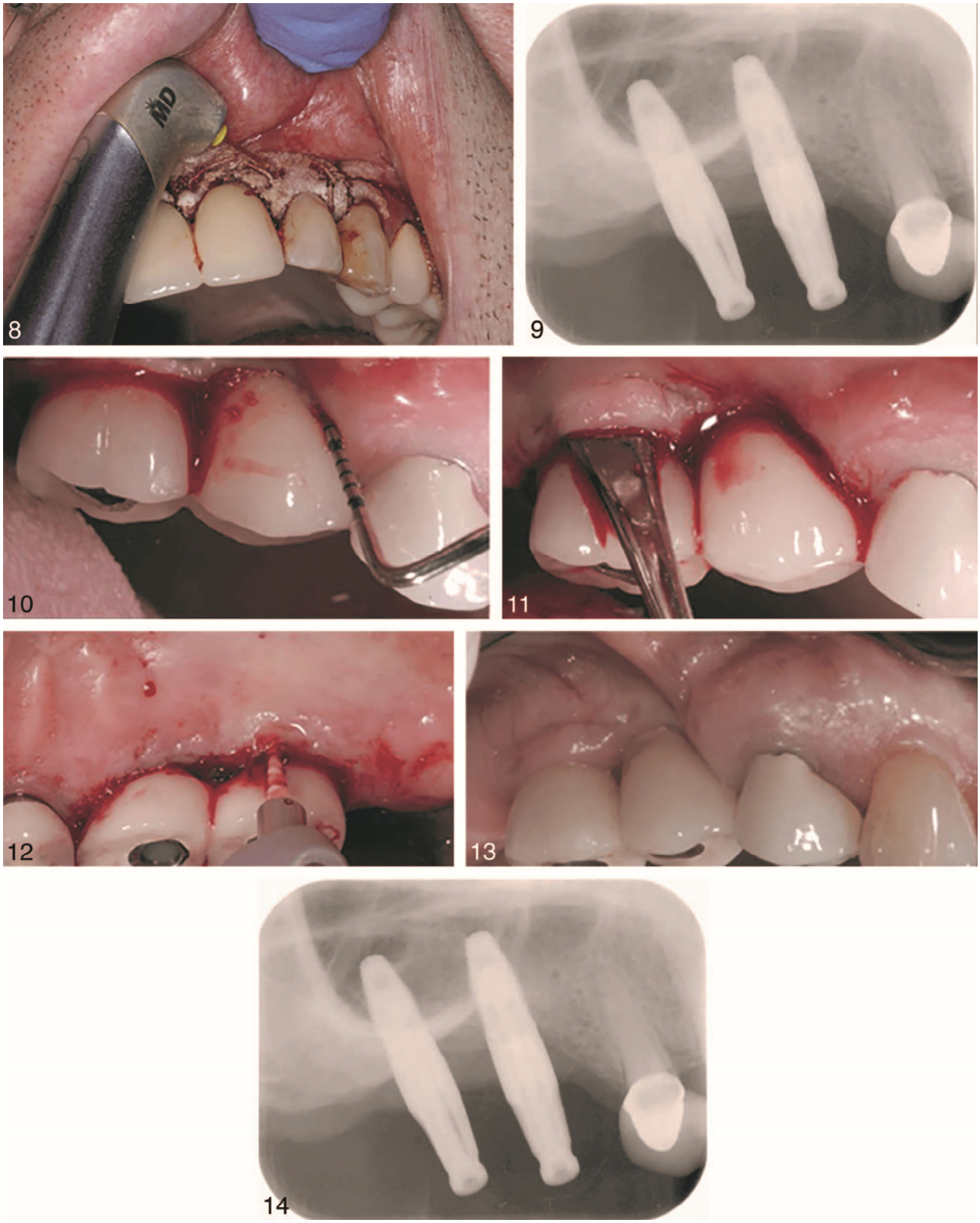
Dr Linden utilizes the 10600 nm CO_2 laser in assisted closed flap treatment for the treatment of peri-implantitis. He has found that laser energy is efficiently deposited in the biofilm, laser heat is confined to biofilm, there is no thermal conduction into the body of the implant, and laser-generated heat inside the biofilm is sufficient to vaporize the biofilm. He concluded that there is a bacterial reduction of 95%–100% on the implant surface without heating or damaging the implant.

Protocol for use of 10600 nm CO_2 Super Pulsed laser (LightScalpel, Bothell, Wash, USA) is as follows:

1. Closed procedure under local anesthetic
2. Bone sounding to determine accurate bone topography
3. Modified fiberotomy/closed elevation
4. CO_2 for de-epithelization of the soft tissue surrounding the implant surface



Figures 1–7. Figure 1. Flap reflection. **Figure 2.** Removal of granulation tissue. **Figure 3.** Titanium brush. **Figure 4.** 9300 nm CO_2 laser. **Figure 5.** Bio Oss + platelet rich fibrin plasma for graft. **Figure 6.** Interwoven collagen membrane. **Figure 7.** Platelet rich fibrin membrane.



Figures 8–14. Figure 8. De-epithelialization with Erbium laser. **Figure 9.** Preop radiograph. **Figure 10.** Bone sounding with probe. **Figure 11.** Modified fiberotomy. **Figure 12.** 10600 nm CO_2 laser. **Figures 13 and 14.** 6-month postop.

5. Piezo ultrasonics applied to the apical cortical base of the implant
6. CO_2 use for laser hemostasis circumferentially around the implant
7. Occlusal adjustment
8. Oral antibiotics for 7 days
9. Adapted oral hygiene regimen
10. Dietary restrictions for 4–5 days following the treatment.

Case Study

A 50-year-old patient presented with two implants with suppuration and pocket depths of 5–9 mm circumferentially around all implant surfaces (Figure 8). Radiograph examination showed circumferential bone loss around both implants. A diagnosis of peri-implantitis was made, with the bone loss extending to 3–5 threads from the implant platform.

Bone sounding is crucial with the closed technique. The data obtained the clinician as to how far the laser needs to go into the pocket (Figure 10).

After the bone sounding is completed, a modified fiberotomy is performed. A small elevator is used to sever the gingiva around the affected implants (Figure 11) to open up the coronal aspect of the gingival tissue (2–4 mm deep). This step is important as it helps to introduce the laser tip into the pocket.

Step 1

The technique is performed in a top-down manner, using the super-pulsed setting of 4W with 0.25 mm periodontal tip. The average power is 0.4W. The crest of the epithelium is removed using a sideways circumferential movement to the full width of the pocket but short of the apical 1–2 mm of the pocket (Figure 12). Hand speed is crucial for this procedure, as it should be slower (1 mm/sec for thicker epithelium and slightly faster for thinner epithelium of 2–3 mm/sec).

Clean the implant with ultrasonics using the Piezo Scaler. The goal of this procedure is to remove the calculus, residual tissue, and cement from the implant and pocket region.

Step 2

The technique is performed in a bottom-up manner, using the super-pulsed setting of 2W with a 0.4 mm periodontal tip (Figure 12). The average power was 0.4W. The laser is reintroduced into the peri-implant pocket with the coagulation setting. The technique is similar to the de-epithelialization steps described earlier, except it starts 1–2 mm from the base of the pocket and works coronally. The spacing of laser firing is in 2–mm increments around the entire circumference of the implant pocket.

After the laser treatment, the patient was prescribed doxycycline 100 mg twice a day for 7 days. The patient was put on an adapted oral hygiene regimen that included no flossing for 10 days. A 6-month follow-up showed 3-4 mm pockets circumferentially around each implant (Figures 13 and 14).

Treatment for peri-implantitis with traditional methods has been marginal at best. The dawning of laser dentistry has changed treatment from guarded to hopeful to success. In 2017, the Academy of Laser Dentistry took the challenge to bring together a group of clinicians, giving them the task of developing a protocol to treat this growing disease. The laser companies invited for this symposium were Biolase, Convergent, Fotona, Lighschapel, and Millennium. Millennium elected not to participate. The authors would like to thank ALD for having the foresight to find a solution for the treatment of peri-implantitis with light energy.

Conclusion of the Consensus for Diagnosing Peri-Implantitis From ALD 2017 Annual Meeting

- Occlusal overload can be an initiating portion to this disease
- Need to use a metal probe for diagnosing of pockets
- Presence of suppuration is a major indicator for need to treat
- Corrosion of metal can be an initiating factor for peri-implantitis
- Surface texture appears to create a better chance for disease factors
- There is a definite bacterial connection with periimplantitis.

Conclusion of the Consensus for Treatment of Peri-Implantitis From the ALD 2017 Annual Meeting

- Flap reflection to gain access to treating a 3- or 4-wall defect. These are only ones that have shown to be treatable consistently
- Biofilm must be removed by either a Glycerinn Air Polisher (Air Flow Brenchley, NY; Hu-Friedy, Chicago, Ill), or titanium brush (Salvin)
- Either Erbium or CO_2 lasers have been shown to be the best lasers to detoxify an implant surface without creating excess heat, which would lead to de-bonding of the implant/bone contact.[32] The worst lasers to use are Diodes and Nd:YAG as they create excess heat and can even melt a metal implant[32,33]
- Decortication of the bone surrounding the implant (Erbium) or Piezo and de- epithelialization of soft tissues inside and outside of the flap (CO_2 or Erbium)
- Use of biologics (plasma rich fibrin PRF) in combination with a slowly resorbing particulate graft (usually xenograft) or slow resorbable graft material
- Collagen membrane that is interwoven to prevent tissue invagination, then placing PRF membrane over the collagen membrane before closing
- Use of photobiomodulation will aid in the healing process.
- Occlusal adjustments at the time of surgery and 2 weeks after surgery. Consider use of an occlusal guard.

Abbreviations

ALD: Academy of Laser Dentistry
PRF: platelet rich fibrin

Note

Authors report no conflicts of interest.

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