Treatment of Refractory Apical Peri-Implantitis: A Case Report

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INTRODUCTION

Dental implants have become a highly successful and routine treatment for the loss of teeth, with implant survival rates reported as greater than 89% at 10–15 years of follow-up.1 Although the success rates of dental implants are high, several reports have demonstrated implant failures.2 The etiology and mechanism of implant failures are multifactorial and categorized as failures due to infection (ie, peri-implantitis or retrograde peri-implantitis), or failures due to trauma (ie, excessive overloading or implant fracture).3 Peri-implantitis is defined as “an inflammatory process affecting the soft and hard tissues surrounding an osseointegrated implant resulting in rapid bone loss around the apex of the implant.”4 A rare event, retrograde peri-implantitis (periapical implant lesion, implant periapical pathology, apical peri-implantitis, abscess formation around the apex of implant), has also been reported as 1 possible cause for dental implant failures.5,6 Retrograde peri-implantitis was first described in 1992 by McAllister et al.7 It was defined as bone loss limited to the apical segment of an otherwise osseointegrated implant by Reiser et al8 in 1995, and was often diagnosed as a radiolucency surrounding the implant apex.

The prevalence of implant apical lesions is 0.26%, but they are always unpleasant, and exacerbation of the lesions may lead to the mobility of the implant and even implant removal.9 Different treatments have been introduced; however, the phase, level, and size of the lesion were not considered in these managements. The underlying causes may include bacterial contamination during insertion, vascular impairment, overheating of bone during drilling, and vascular ischemia, all of which can result in bone loss around the apex of the implant.10 Other etiologic factors may include overdressing of the site, implant surface contamination, preexisting bone pathology (bacteria, inflammatory cells, and/or remaining cells from a cyst, granuloma), the presence of root remainings, and implant placement close to an infected maxillary sinus.11

Sussman et al12 classified implant periapical lesions (IPLs) into 2 case types according to the main infection pathway: (1) implant to tooth, which occurs during osteotomy preparation either by direct trauma or indirect damage, causing the adjacent pulp to undergo devitalization; and (2) tooth to implant, which occurs shortly after implant placement when an adjacent tooth develops periapical pathology, either because of operative damage to the pulp or the reactivation of a prior apical lesion. In both types, the resulting periapical pathology may hinder implant healing.12

The literature proposes different management approaches for implant periapical pathology, such as systemic antibiotic therapy,13 periapical surgery with or without resection of the implant apex, guided bone regeneration, or implant extraction. Monotherapy via systemic antibiotics usually cannot achieve complete lesion resolution, because of difficulties in the eradication of bacterial colonies from the IPL.14 The infected lesion typically requires surgical intervention and elimination of the infection. Implant apical resection or implant removal depends on the extent of infection and the stability of the implant.15

The purpose of this case report was to describe a surgical treatment procedure based on the concept described earlier with the addition of root therapy of adjacent tooth. The possible etiology and treatment approaches of refractory periimplantitis are discussed.

CASE REPORT

A 42-year-old female patient, with no history of systemic disease, was referred to the Department of Periodontology for dental implant treatment regarding tooth #10. The patient’s dental history revealed that the left maxillary lateral incisor (#10) had fractured 1 month ago. The tooth was asymptomatic. A periapical radiograph showed incomplete root canal treatment with apical radiolucency. Written informed consent was obtained from the patient prior to treatment. Based on prosthodontic evaluation, the extraction of tooth #10, followed by ridge preservation with xenograft (Gen-Os, Tecnost, Torino, Italy) covered with a collagen membrane (Evolution Standard, 20 × 20 mm, OsteoBiol, Tecnost, Coazze, Italy) was considered (Figure 1).

Six months after the ridge-preservation procedures, residual buccopalatal bone thickness was 6 mm and mesiodistal width was 7 mm at the coronal part of alveolar bone. A screw-shaped titanium dental implant, 3.30 mm wide and 13 mm long (Tapered Screw-Vent, Zimmer Dental, Carlsbad, Calif) was placed at the tooth #10 region in a 2-stage approach. The postoperative course of healing was uneventful, and the
patient had no particular complaints after surgery. Six months after implant placement, prosthetic rehabilitation was performed with a cemented porcelain crown. Three months after the rehabilitation, the patient reported pain at the apical region of the implant. A clinical sinus tract was visible at the apical region (Figure 2). Periapical and panoramic radiographs showed a radiolucency at the apical portion of the implant, and a computerized tomography (CT) scan also confirmed the presence of the apical lesion unrelated to the adjacent teeth (Figures 3 and 4). A radiolucency of about 4 mm in diameter was identified at the implant apex, and a diagnosis of IPL was established. Adjacent central and canine incisors were vital.

In subsequent follow-up visits, the lesion demonstrated an increase in size, and consequently, 4 months after prosthetic rehabilitation, surgical intervention was performed. Periapical surgery was performed under local anesthesia articaine (Ultracain D-S Forte, 4% articaine with 1:100 000 adrenaline, Sanofi-Aventis, Frankfurt, Germany), an incision was made at the mucogingival junction with a number 15 surgical blade, and a full-thickness flap was elevated. A large formation of inflamed tissue was noted at the apex of the implant (Figure 5). The curettage of inflamed tissue was performed, and, the apical portion of the implant was cleaned with ultrasonic scalers under abundant irrigation with sterile saline solution (Figure 6). After debridment, the defect was cleaned with chlorhexidine digluconate and sterile saline irrigation. Xenogenic cortico-cancellous grafting material (Gen-Os, Tecnoss) was packed into the bony defect for guided bone regeneration (Figure 7). A resorbable collagen membrane (Evolution Standart, 20 x 20 mm, OsteoBiol, Tecnoss) was placed over the graft material (Figure 8), and the wound closure was observed with 4-0.
sutures. Ten days of healing were uneventful; however, 4 weeks after surgery there was a recurrence of the buccal sinus tract.

Periapical radiograph showed extended radiolucency including the adjacent canine tooth (Figure 9). First, root therapy was performed for the canine tooth. Following the endodontic treatment, a surgical treatment phase was initiated. Resective apical surgery was planned for the second treatment protocol. A full-thickness mucoperiosteal flap was raised, and the lesion was debrided. The affected apical part of the implant (4 mm) was resected by a fissure diamond bur through irrigation with an external cold sterile saline solution (Figure 10).

Guided bone regeneration therapy supported with platelet-rich fibrin (PRF) was performed. The patient’s venous blood sample was taken before surgery. The blood was placed in 10-mL glass test tubes, without anticoagulant, and immediately centrifuged at 3000 rpm for 10 minutes. The protocol for PRF
preparation was applied according to Choukroun’s procedure. The PRF obtained was used in 2 ways: a part of it was used as a filling material, with xenogenic graft material (Gen-Os, Tecnoss) (Figure 11), and the remaining part was shaped to form a resistant fibrin membrane. Resorbable collagen membrane (Evolution Standard, 20 x 20 mm, OsteoBiol, Tecnoss) was placed over the graft material, and the PRF membrane was placed over it (Figure 12). The flap was repositioned coronally, and primary closure was achieved with 4-0 sutures. The patient received antibiotics, metranidazole 500 mg, 2 times daily for 10 days. One week after periapical surgery, the pain and inflammation had subsided, and uneventful healing was observed.

The third- and sixth-month (Figure 13) follow-up visits demonstrated no pain or discomfort for the patient, and no sign of infection was noted. A periapical radiograph at 6 months (Figure 14) and a panoramic radiograph at 12 months after surgery presented new bone regeneration at the apical region of the implant (Figure 15).

DISCUSSION

The implant periapical lesion is the infectious-inflammatory process of the tissues surrounding the implant apex. It may be caused by different factors, and a previous study summarized the proposed etiology of IPLs. Microbial contamination is the predominant causative factor, which includes reactivation of an endodontic lesion from an adjacent tooth, residual infection from previously failed endodontic therapy, a retained root tip, residual infection in a healing socket, and a maxillary sinus infection. On the other hand, there are some controversial etiologies that have also been suggested, including bone microfracture, buccal plate fenestration, development of osteomyelitis, overheating, implant surface contamination from intraoral sources, overloading, and poor bone quality.

Diagnosis is achieved by studying the presence of symptoms such as pain, swelling, suppuration, or fistula; radiographs also assist in diagnosis, as an implant periapical radiolucency may appear. Treatment of an infection at the apex of an implant can be very difficult, and there is no clinical protocol for the management of an IPL. Treatment varies according to the type of lesion.

The therapies for an IPL are similar to those for peri-implantitis, and include the following: (1) nonsurgical treatment via systemic antibiotics; (2) resective treatments including debridement along with detoxification of the implant surface using a chemical agent (chlorhexidine gel, stannous fluoride, tetracycline hydrochloride, hydrogen peroxide, citric acid, polymyxin B, or chloramine T), and intraoral apical resection of the implant apex; and (3) regenerative treatments including debridement, detoxification of the implant surface, intraoral apical resection of implant apex, and guided bone regeneration.

The clinician should remember that monotherapy via systemic antibiotics do not always achieve complete resolution of the IPL because of the difficulty in eradicating bacterial colonies from the lesion area, and definitive surgical intervention is advised within 1 month of IPL onset. Most authors recommend the exposure of the implant apex, degranulation of the defect, detoxification of the implant surface, and regeneration of the lost bone.

Several methods were proposed to detoxify the implant surface. Chlorhexidine digluconate and tetracycline hydrochloride are most frequently used for this process. Simple saline irrigation was also used in some cases but only for cleaning the defect after debridement, not for detoxification. In our case, we used chlorhexidine digluconate and saline irrigation for detoxification of the region in the first surgery and only saline irrigation in the second surgery.

According to the literature, some researchers suggest that curettage of the infection at the implant apical area was sufficient treatment for periapical lesions; however, in other studies, especially cases in which curettage of the palatal or lingual side of implant apex was difficult, implant apicectomy was recommended for facilitating complete removal of lesion. In our case, the first treatment protocol was curettage of the lesion; however, recurrence of the infection developed after suture removal. Resective surgery was planned for the second operation, and apical side of the implant that existed in the defect area was resected. The decision to use a resective technique was based on the fact that complete debridement of the infected implant portions and defect walls would be impossible given the extent of the lesion. It was stated that, after resection, access to the defect area could be achieved exactly, and bacteria colonized over the implant surface could be completely eliminated. The hypothesis for
retrograde peri-implantitis generally focused on apical residual infection caused by the lost tooth; and prevention of recurring infection involves completely cleaning the apical region with resection and curettage.\textsuperscript{24}

Guided bone regeneration therapies are suggested in the literature for implant apical lesions to regenerate lost bone; however, it can not be determined whether regeneration was achieved or only a repair was formed, because there is no histologic information for successfully treated implant periapical lesions.\textsuperscript{25} Bretz et al\textsuperscript{20} successfully treated a case of implant periapical lesion with periapical surgery, curettage, chlorhexidine irrigation, placing demineralized bone, and covering the field with a reabsorbable collagen membrane. In our case, guided bone regeneration therapies were performed in both surgeries; however, in the second surgery, PRF was used to support healing in soft and hard tissues. PRF may be considered a second-generation platelet concentrate, and is widely used to accelerate soft and hard tissue healing because of the presence of its many growth factors.\textsuperscript{26} Localization of the implant is in the esthetic zone, and using PRF could also prevent deformities at soft tissue, which could occur because of recurring surgeries. At control visits, soft tissue healing was uneventful at the apical region of the implant.

At the beginning of the infection symptoms, the implant was clinically stable, and the neighboring teeth were considered healthy. A vitality test was positive for central and canine teeth; however, the radiographs, taken at the recurrence of the lesion, showed that the periapical lesion was extended to the apex of the maxillary left canine tooth. Root therapy was performed before surgery. A test of vitality of the adjacent teeth and a quality assessment of their endodontic treatment should be part of routine implant treatment planning.

**CONCLUSION**

This case report suggests that refractory IPL lesions may successfully be treated by apical resection of the implant, and regenerative surgeries could be efficient instead of implant removal, as it would avoid excessive bone loss. Platelet-rich fibrin is a newly developed platelet concentrate that has successfully been used in a number of surgical procedures to optimize wound healing. Several studies indicate that it may also have the ability to stimulate bone formation. In this article, we present a case where PRF was used to stimulate bone formation to facilitate implant survival. Further research should investigate the healing tissue, at the apical side of implant, histologically for determining the type of the new regenerated tissue.
REFERENCES


