Nonsurgical Endodontic Treatment of Necrotic Teeth Resolved Apical Lesions on Adjacent Implants with Retrograde/Apical Peri-implantitis: A Case Series with 2-year Follow-up

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Abstract

Retrograde (or apical/periapical) peri-implantitis (RPI) presents with radiographic signs of bone loss at the apical area of endosteal implants and may also present with clinical signs of abscess formation or a sinus tract traceable to the implant periapex. The lesion may form days up to several years after the initial implant placement. In contrast to marginal peri-implantitis, which has a prevalence of 19.83%, RPI may be underreported because many clinicians are currently not aware of this type of lesion. The etiology, although not fully understood, may be attributed to endodontic infection of an adjacent tooth or residual microorganisms present after the extraction of an infected tooth at the implant site. There are several treatment modalities available for the management of RPI. Nonsurgical root canal treatment may be an option if the implant RPI etiology is suspected to be related to an adjacent endodontically involved tooth. In a previous report, surgical treatment modalities to correct RPI were described. This current case series presents 2 cases of RPI in which nonsurgical root canal treatment of the necrotic adjacent teeth resulted in full radiographic and clinical resolution of the adjacent apical peri-implant lesions with 18-month and 2-year follow-ups, respectively. RPI may be prevented by evaluating the endodontic status of natural teeth adjacent to the implants and by addressing endodontic infections near the implant sites. Certain types of implant RPI may successfully be resolved nonsurgically by addressing adjacent endodontic infections as shown by this case series. (J Endod 2019;45:645–650)

Key Words

Bone regeneration, dental pulp necrosis, periapical diseases, peri-implantitis

Peri-implantitis is defined as a pathological inflammatory condition occurring in the mucosal tissues surrounding the coronal portion of dental implants leading to progressive loss of the supporting bone (1). It may be preceded by a localized soft tissue reaction to plaque and manifest as peri-implant mucositis, which resembles gingivitis around natural teeth. Currently, the mechanisms pertaining to the progression of peri-implant mucositis to peri-implantitis have not yet been fully identified (1, 2). Failing implants because of infection appear to have a similar bacterial profile and mechanisms of host response to teeth with chronic periodontitis. Several opportunistic pathogens such as Pseudomonas aeruginosa, Staphylococcus aureus, and some fungal organisms and viruses have been linked to peri-implantitis (3–6). In a recent review and meta-analysis (7), the subject-based prevalence of peri-implantitis and peri-implant mucositis was reported to be as high as 19.83% and 46.83%, respectively. Various treatment modalities for peri-implantitis have been suggested with variable success, and a recent survey from practicing periodontists in the United States revealed that peri-implantitis is a relatively frequent occurrence resulting in the removal of up to 10% of implants that are placed (8). A history of chronic periodontitis, poor plaque control, and a lack of regular peri-implant maintenance treatment have been identified with an increased risk of developing peri-implantitis (1).

In addition to the peri-implant diseases described previously that occur around the marginal peri-implant tissues, another type of peri-implantitis at the apical tissues termed retrograde (or apical/periapical) peri-implantitis (RPI) has also been reported in the literature, mainly in case reports and case series (9–28). RPI limited to the periapex of osseointegrated implants typically does not result in complete osseodisintegration of the implant. RPI, in contrast to marginal peri-implantitis, presents itself with radiographic signs of bone loss associated with the apical area of the osseointegrated implant that in most reported cases resulted in clinical signs of inflammation with or without the development of an abscess or a sinus tract, which may be noted between 1 and 8 weeks (10, 23, 24) and up to 4 years after the initial implant placement (29). The prevalence of RPI is 0.26% (30) and may be underreported because many clinicians are currently not aware of this type of lesion. The etiology

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of RPI is currently not fully understood but in the current literature is mainly attributed to (1) an endodontic infection of an adjacent tooth, (2) residual microorganisms present after the extraction of the infected tooth, (3) excessive heat or compression at the time of implant placement, or (4) implant apex contamination at the time of implant placement. Other reasons, such as viral infectious sources, have also been discussed in the literature (12, 16, 17, 31, 32).

Case Report 1

A 58-year-old male patient was referred to a dual-trained endodontist-periodontist for possible implant therapy on severely resorbed posterior mandibular partially edentulous ridges. The patient’s medical history was contributory for hypertension, hypercholesterolemia, and mitral valve prolapse, and he was classified as American Society of Anesthesiologists (ASA) II according to the ASA physical status classification. He reported having an angioplasty 10 years ago. The patient’s list of medications included valsartan (angiotensin II receptor antagonist), hydrochlorothiazide (diuretic), pitavastatin (statin), clopidogrel (blood thinner), and vitamin C and fish oil supplements. His dental history included multiple restorations, generalized slight to moderate chronic periodontitis, cervical abrasions, and a history of tooth loss. A comprehensive treatment plan was established for the patient after clinical and radiographic evaluation including advanced imaging with cone-beam computed tomographic (CBCT) imaging of the edentulous ridges in the posterior maxilla and mandible. CBCT imaging revealed severe atrophy of the posterior mandible and the need for ridge augmentation of sites #18 and 19 in preparation for dental implant therapy. It was also decided that tooth #20 adjacent to the future implant site, which had a buccal noncarious cervical lesion, would be treated with a subepithelial connective tissue graft to attempt root coverage. Tooth #20 was asymptomatic, tested normal to cold, and clinical and radiographic evaluations were within normal limits at the time of this initial comprehensive examination. All treatment options were discussed with the patient, the risks and benefits were reviewed, and informed consent was obtained. A guided bone regeneration (GBR) procedure was performed at sites #18 and 19 using a freeze-dried bone allograft, autologous platelet-rich plasma, and a resorbable barrier membrane (Optimatrix; DSM Biomedical, Exton, PA). Tooth #20 concurrently received a buccal subepithelial connective tissue graft from a palatal donor site in order to cover the cervical defect and achieve root coverage. The patient returned for postoperative appointments, and healing was within normal limits; the patient remained asymptomatic. The patient returned for implant placement at sites #18 and 19 after allowing for 7 months of healing after the GBR procedure. Two bone-level endosseous implants (3.5 × 14 mm, Ankylos; Dentsply Sirona, York, PA) were successfully placed following the manufacturer’s protocol. Healing abutments were placed, the tissue flap was sutured with 4-0 Vicryl (Ethicon Inc, Somerville, NJ), and full tissue closure over the implant sites was achieved. The postoperative follow-up was within normal limits and without symptoms. The patient returned after 9 months of healing for uncovering of the implant sites in preparation for prosthetic restoration of implants #18 and 19 by placement of healing abutments. As part of the evaluation of the implants, advanced imaging and periapical radiographs were taken that revealed a periapical radiolucency that had developed around the apex of tooth #20 as well as the implant apex of #19. Tooth #20 was subsequently tested and diagnosed with pulp necrosis and asymptomatic apical periodontitis, and implant #19 was diagnosed with class 2 RPI (Sarmast and Wang RPI Classification). The patient exhibited no symptoms or pain, and there were no clinical signs of infection or sinus tract formation.

Procedure

After discussion with the patient about the periapical radiolucency at the apex of #20 and implant #19, treatment alternatives were discussed, and it was decided that a nonsurgical treatment approach would be the first option in an attempted treatment of these lesions. Informed consent was obtained for orthograde root canal therapy (RCT) for tooth #20. Profound anesthesia of tooth #20 was achieved by 3.4 mL lidocaine 2% with 1:100,000 epinephrine via an inferior alveolar nerve block. A dental dam was placed on tooth #20, and the aseptic technique was observed throughout the treatment. Access was created, and 1 canal was located. Rotary and hand files were used to clean and shape the canal to the working length (ProTaper Gold; Dentsply Tulsa Dental Specialties, Tulsa, OK) via a modified crown-down technique. Irrigation was performed with 6% sodium hypochlorite and 17% EDTA. The patient was asymptomatic at the second visit 4 weeks after the initial RCT visit. The canal was reinstrumented and reirrigated as previously described and subsequently dried and obturated with BC Sealer via a single gutta-percha cone technique (Brasseler USA, Savannah, GA). A resin orifice barrier was placed and light cured, the tooth was subsequently temporized with Cavit (3M ESPE, St Paul, MN), and occlusion was checked. Subsequent postoperative visits were within normal limits, and the patient did not report any symptoms. Tooth #20 was restored with a crown a few weeks after completion of the RCT. Follow-up with radiographs revealed evidence of healing through resolution of the periapical radiolucency both around the apex of tooth #20 and the implant apex of tooth #19. The implants #18 and 19 were also permanently restored with crowns. After 2 years of follow-up, there is still no radiographic evidence of persistent lesions at the apices of #19 and 20, and the patient remains asymptomatic. The resolution of both radiographic lesions was attributed to the nonsurgical endodontic treatment of tooth #20 (Figs. 1A–H and 2A–E).

Case Report 2

A 66-year-old male patient was referred to a periodontist for comprehensive implant therapy on bilaterally resorbed mandibular and maxillary edentulous ridges. The patient’s medical history was contributory for hypercholesterolemia, and he was classified as ASA II physical status classification. The patient’s list of medications included baby aspirin 81 mg (antiagulant therapy), Crestor (statin), and multivitamins. His dental history included multiple restorations, generalized slight to moderate chronic periodontitis, attrition, and a history of tooth loss. A comprehensive treatment plan was established for the patient after clinical and radiographic evaluation including advanced CBCT imaging of the edentulous ridges in the posterior maxilla and mandible. CBCT imaging revealed severe atrophy of the posterior mandible and the need for ridge augmentation of sites #19, 20, 29, and 30 in preparation for dental implant therapy. Tooth #28 was asymptomatic and tested normal, and clinical and radiographic evaluations were within normal limits at the time of this initial comprehensive examination. All treatment options were discussed with the patient, the risks and benefits were reviewed, and informed consent was obtained for orthograde root canal therapy (RCT) for tooth #20. After discussion with the patient about the periapical radiolucency, the procedure was performed at sites #19, 20, 29, and 30 using a freeze-dried bone allograft, tenting screws (Zimmer, Palm Beach Gardens, FL), autologous platelet-rich fibrin, and a resorbable barrier membrane (Optimatrix). The patient returned for postoperative appointments, and healing was within normal limits. The patient returned for implant placement at sites #29 and 30 after allowing for 5 months of healing after the GBR procedure. Two bone-level endosseous implants (3.7
10 and 4.7 × 10 TSVT, Zimmer) were successfully placed following the manufacturer’s protocol. Healing abutments were placed, the tissue flap was sutured with 4-0 Vicryl, and full tissue closure over the implant sites was achieved. The postoperative follow-up was within normal limits and without symptoms. The patient returned after 3 months of healing for uncovering of the implant sites in preparation for prosthetic restoration of implants #29 and 30 by placement of healing abutments. Advanced imaging and periapical radiographs were taken at this appointment in order to evaluate healing and osseointegration of implants, which revealed a periapical radiolucency around the apex of tooth #28 as well as the implant apex of #29. Tooth #28 was tested and diagnosed with pulp necrosis and asymptomatic apical periodontitis, and implant #29 was subsequently diagnosed with class 2 RPI (Sarmast and Wang RPI classification). The patient exhibited no symptoms or pain, and there were no clinical signs of infection or sinus tract formation.

**Procedure**

After discussion with the patient about the periapical radiolucency at the apex of tooth #28 and implant #29, treatment alternatives were discussed, and it was decided that a nonsurgical treatment approach would be the first option in an attempted treatment of these lesions. Informed consent was obtained for orthograde RCT for tooth #28. Profound anesthesia of tooth #28 was achieved by 3.4 mL lidocaine 2% with 1:100,000 epinephrine via an inferior alveolar nerve block and local infiltration. A dental dam was placed on tooth #28 via single tooth isolation, and the aseptic technique was observed throughout the treatment. Access was created via the occlusal surface of tooth #28, and 1 canal was located. Rotary and hand files were used to clean and shape the canal to the working length (Dentsply Tulsa Dental Specialties). Copious irrigation with 6% sodium hypochlorite and 17% EDTA was performed. The patient was asymptomatic at the second visit 4 weeks after the initial RCT visit. The canal was reinstrumented and reirrigated as previously described and subsequently dried with sterile paper points and obturated with a single gutta-percha cone and BC Sealer (Brasseler USA); a resin orifice barrier was placed and light cured, the tooth was temporized with Cavit, and occlusion was checked after removal of the dental dam. The subsequent postoperative visits were within normal limits, and the patient did not report any symptoms. Tooth #28 was restored with a crown a few weeks after the completion of RCT. Follow-up with radiographs revealed evidence of healing through resolution of the periapical radiolucency both around the apex of tooth #28 and implant apex #29. The implants #28 and 29 were also permanently restored with crowns.

Figure 1. Case 1: (A) endosseous bone-level implants (3.5 × 14 mm Ankylos, Dentsply Sirona, York, PA) placed at sites #18 and 19 after 7 months of healing after the GBR procedure. (C and D) Placement of healing abutments after 9 months revealed periapical radiolucent lesions (PARLs) at the apex of tooth #20 and the implant apex of #19 on periapical radiographs. (E and F) Nonsurgical root canal treatment completed on (D) necrotic tooth #20 with postoperative periapical radiographs a few weeks after completion of RCT on tooth #20 and crown placement on implants #18 and 19. (G and H) Two years of follow-up showing resolution of PARLs around apex #20 and implant apex #19.
After 6 months of follow-up, there is still no radiographic evidence of remaining lesions at apices of #28 and 29, and the patient remains asymptomatic (Figs. 3A–F and 4A–C).

**Discussion**

Previous studies have shown that the chance of RPI developing on an implant placed adjacent to a tooth with an apical lesion is about 25% (odds ratio = 8.0) (12). As described in a previous study, 4 distinct classes of RPI have been identified along with their respective treatment options based on currently available evidence (32). Class 1 is applicable when implant placement results in devitalization of a previously vital adjacent tooth. Class 2 is when the implant apex is infected by a persistent periapical lesion on an adjacent tooth or implant. The authors would recommend that an adjacent necrotic tooth should fall into this category as well. The suggested treatments for class 1 and 2 based on the current evidence in the literature includes nonsurgical and/or surgical RCT of the affected tooth, with or without previous use of antibiotics for class 2. Class 3 describes when the implant apex is placed or angulated outside of the envelope of bone either lingually or labially. Class 4 is when the implant apex lesion developed because of residual infection at the implant placement site. In this case, antibiotic therapy and/or surgical debridement with possible grafting may be treatment options. Grafting may also be an option for a class 3 RPI. Removal of the aberrant implant is usually an option for class 1, 3, and 4 depending on the clinical situation (32). Both cases showed successful outcomes based on radiographic follow-up of the lesions. This indicates that nonsurgical endodontic treatment of the tooth suspected to be involved in the development of RPI on adjacent implants may result in full resolution of both lesions and should therefore be considered as the first line of treatment in similar cases. The prognosis for RPI once properly diagnosed and when appropriate treatment is provided is in most cases favorable, with 75%–90% success based on available information from previous case series (25, 33).

**Conclusion**

Because the etiology of RPI may in some cases be endodontic implant related, it is important that endodontists are able to properly diagnose RPI and provide treatment when possible or engage in management with other specialist colleagues. Endodontists together with their specialist colleagues should advocate for careful examination of the dentition adjacent to any planned implant site. Adjacent tooth/teeth should be evaluated for the presence or absence of pulpal and

**Figure 2.** Case 1: (A–C) preoperative intraoral pictures of the partially edentulous ridge with severely deficient bone height and width at sites #18 and 19 and the cervical defect and recession on buccal aspect of vital tooth #20. (D) The postoperative occlusal view of grafted site (GBR) #18 and 19. (E) The postoperative view of restored implants #18 and 19 also showing complete root coverage of the cervical defect on tooth #20 after guided tissue regeneration.
Case 2: (A and B) the initial radiographs from the lower right quadrant; tenting screws are in place as part of ridge development for implant placement (GBR) of 2 implants (3.7 × 10 and 4.7 × 10 TSVT Zimmer Biomet, Warsaw, IN) at sites #29 and 30. Implant #29 showing radiographic evidence of radiolucency (PARL) at the visit 3 months after placement. (C) The adjacent tooth (#28) tested necrotic, and nonsurgical RCT was initiated. (D and E) The follow-up periapical radiography almost 3 weeks after RCT on tooth #28. Crowns placed on implants #29 and 30 and tooth #28. (F) The periapical radiolucency on implant #29 was almost completely resolved at 2 months after a root canal on tooth #28.

Figure 3.

Case 2: (A) the initial panoramic image showing edentulous maxillary ridge and partially edentulous posterior mandible, bilaterally pneumatized maxillary sinuses, and existing restorations at the treatment planning visit. (B) The 6-month follow-up after completion of RCT of tooth #28. (C) The 18-month follow-up showing placement of mandibular restorations with implant-retained maxillary prosthesis and no signs of PARLs on tooth #28 or on adjacent implant #29.

Figure 4.
periapical pathology, and appropriate RCT and infection control should be performed before implant placement. Implants that are placed adjacent to natural teeth with signs of infection of endodontic origin should be monitored for the development of RPI. RPI may successfully be resolved by addressing the endodontic infection as shown by this case series.

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References