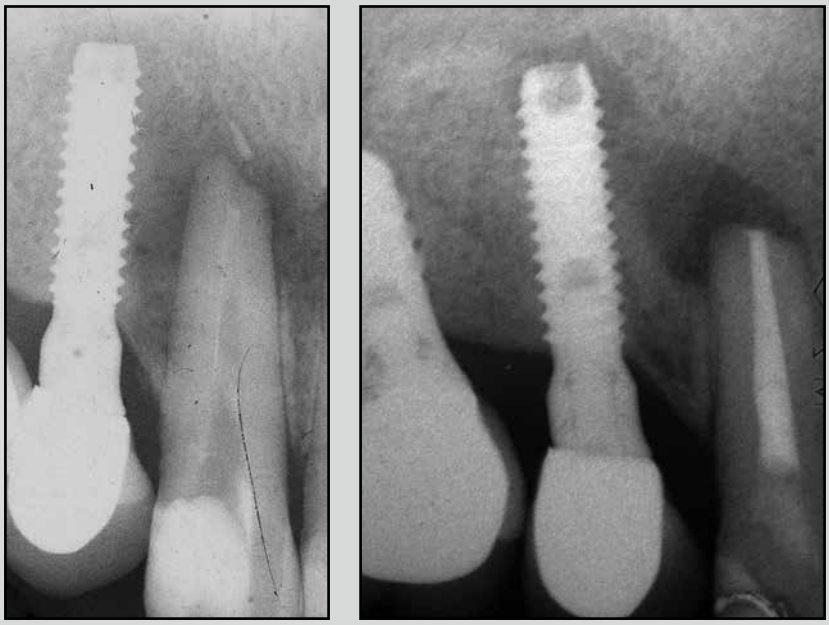


Retrograde Peri-implantitis: Report of a Case Successfully Treated by Resection of the Implant Apex

Debora Franceschi, DDS[1]
Valentina Giuliani, DDS[1]
Veronica Giuntini, DDS[1]
Giovan Paolo Pini Prato, MD, DDS, PhD[2]
Leandro Chambrone, PhD, MSc, DDS[3]

Retrograde peri-implantitis (RPI) is a periapical lesion that develops after implant insertion in which the coronal portion of the implant achieves a normal bone-to-implant interface. The most common etiology of RPI is the presence of an adjacent endodontic lesion. In most of the case reports available in the literature, the diagnosis of RPI occurred between 1 week and 4 years after implant placement. This case report illustrates the treatment of RPI that occurred more than 15 years after implant loading, caused by endodontic infection of the adjacent tooth.
Int J Periodontics Restorative Dent 2021;41:443–448. doi: 10.11607/prd.4837

[1]Department of Experimental and Clinical Medicine, University of Florence, Florence, Italy.
[2]Tuscany Academy of Dental Research (ATRO), Florence, Italy.
[3]MSc Dentistry Program, Ibirapuera University, São Paolo, Brazil; Unit of Basic Oral Investigation (UIBO), Universidad El Bosque, Bogotá, Colombia.

Correspondence: Dr Debora Franceschi, Department of Experimental and Clinical Medicine, University of Florence, Via Ponte di Mezzo, 48, Florence, Italy.
Email: debora.franceschi@unifi.it

Submitted January 22, 2020; accepted March 15, 2020.

Retrograde peri-implantitis (RPI) is defined as a clinically symptomatic periapical lesion (diagnosed as radiolucency) that develops shortly after implant insertion in which the coronal portion of the implant achieves a normal bone-to-implant interface.[1] It should be distinguished from a clinically asymptomatic periapical radiolucency, which is usually caused by implants that are shorter than the drilled implant site or by heat-induced aseptic bone necrosis.

Several etiologies have been proposed in the literature. Sussman[2] proposed two pathways that might lead to RPI: type 1 (implant to tooth), which occurs when the osteotomy preparation causes direct damage to the adjacent tooth; and type 2 (tooth to implant), which occurs when an adjacent tooth with a periapical pathology contaminates the implant. The prevalence of RPI was reported in a retrospective study of 539 implants, with 1.6% of maxillary and 2.7% of mandibular teeth exhibiting this condition before abutment connection.[1] Incidence is reported to increase to 7.8% when teeth adjacent to the implant site have a previous history of root canal therapy, and it is correlated with the distance between implant and adjacent tooth and/or with time from endodontic treatment of adjacent tooth to implant placement.[3] The most common radiographic and

clinical findings are the presence of lesions around the implant apex and sinus tract, often accompanied by symptoms of pain, tenderness, and swelling.

Treatment options were suggested in some reviews of case reports.[4–7] The protocol usually included elevation of a full-thickness flap, complete removal of granulation tissue, and curettage of the bony cavity walls.[1] Some studies reported the use of grafting materials alone or in association with a membrane,[8–14] while others did not use any grafting material or membrane.[15–17] Some authors reported the use of detoxification agents, and very few cases included apicoectomy of the implant.[10,17] Three literature reviews reported an RPI treatment decision tree.[4,5,7] Although there have been many reports discussing the treatment of RPI, clinical decision-making guidelines are still lacking.

The aim of this case report is to illustrate the treatment of an RPI lesion that occurred more than 15 years after implant loading, caused by an endodontic infection of the adjacent tooth.

Case Report

A 45-year-old systemically healthy woman with an American Society of Anesthesiologists classification of I was referred for consultation in April 2014 regarding her maxillary right canine. The patient complained of swelling and pain to percussion. The canine had a composite restoration, and fixed crowns on implants restored the maxillary right first and second premolars. The radiographic examination showed radiolucency apical to the canine (Fig 1a). The electric and thermal pulp test confirmed necrosis of the tooth. Endodontic therapy was performed with ProTaper Universal (Dentsply Maillefer) to an ISO size of #40.

The root canal was filled using gutta-percha (Mynol, Block Drug) and the warm vertical condensation technique. At the 6-month and 1-year clinical and radiographic follow-ups, there was incomplete resolution of periapical lesion, and the patient did not report any signs or symptoms. At 17 months postoperative, the patient presented with swelling at her maxillary right buccal site. Radiographic examination revealed a radiolucent lesion on the apical and distal sites of the right canine, and no pathologic periodontal probing depth (PPD) was recorded. In agreement with the patient, an endodontic surgery was planned. The surgery was performed under magnification using diamond drills associated with constant sterile water irrigation. A complete debridement of the pathologic tissue was performed after root resection, approximately 3 mm from the apex with no bevel angle, in order to have a complete visualization of bone cavity that extended close to the apical part of the implant. A retrograde cavity was prepared with ultrasonic tips, the cavity obtained (3- to 4-mm depth) was dried with microtips applied to air and sealed with mineral trioxide aggregate (ProRoot MTA, Dentsply Sirona; Fig 1b).

Nine months later, a large abscess developed on the buccal aspect of the canine, with an associated 12-mm PPD on the central buccal site. The radiographic examination showed radiolucency around the most apical part of the implant distal to the canine (Fig 1c), but the implant was clinically stable with no signs of pathologic probing. It was decided to extract the tooth (Fig 1d), where a vertical fracture had recently been confirmed. The alveolar socket was cleaned with sterile saline solution, and a systemic antibiotic therapy was prescribed for 6 days (875 mg amoxicillin and 125 mg clavulanic acid every 12 hours).

Wound healing was uneventful 1 month after tooth extraction, but a fistula was still present on the buccal side (Fig 2a), and the patient still had soreness and swelling. A 3D sectional CBCT was done to exclude sinus communication (Figs 2b and 2c). The CBCT confirmed an area of radiolucency around the most apical part of the implant, and the sinus floor was intact. Therefore, it was decided to surgically access the area to allow for debridement of the bone cavity and decontamination of the implant surface (Fig 2d).

First, a mucoperiostal flap was elevated to access the lesion, which was a small cave under the cortical bone, 5 mm in diameter, containing pus and granulation tissue. The granulation tissue was removed, and curettage of the bony cavity walls was performed. The apical implant surface was irrigated with sterile saline solution, and the implant surface was decontaminated by means of the gentle and controlled use of Air-Flow Perio powder (EMA Dental).

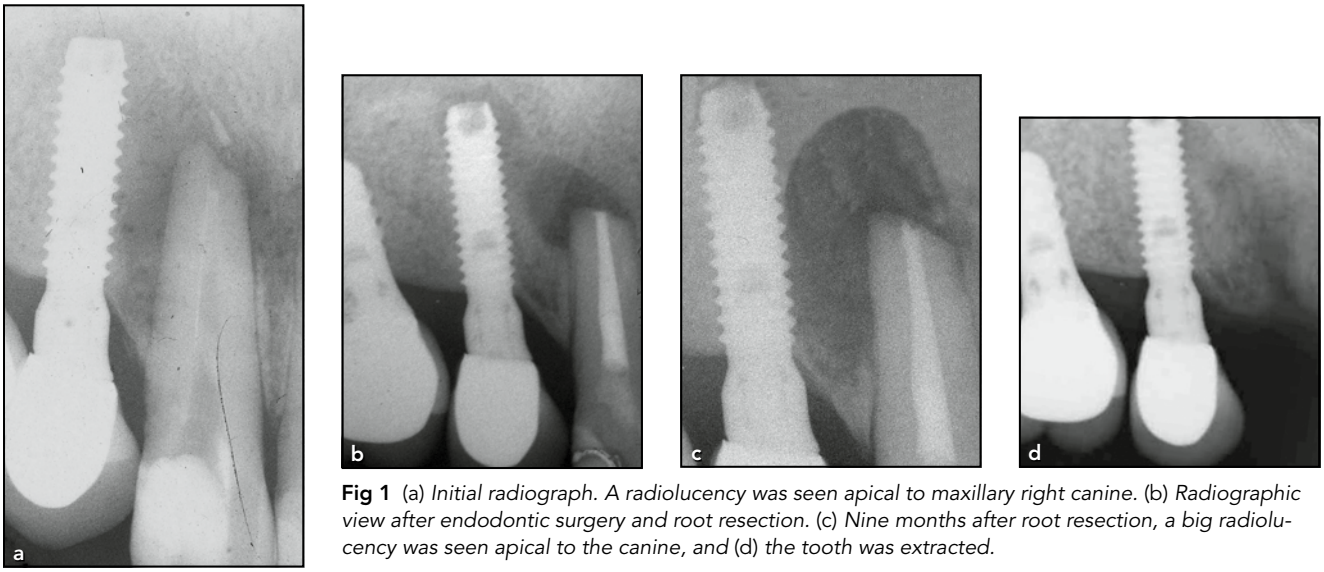
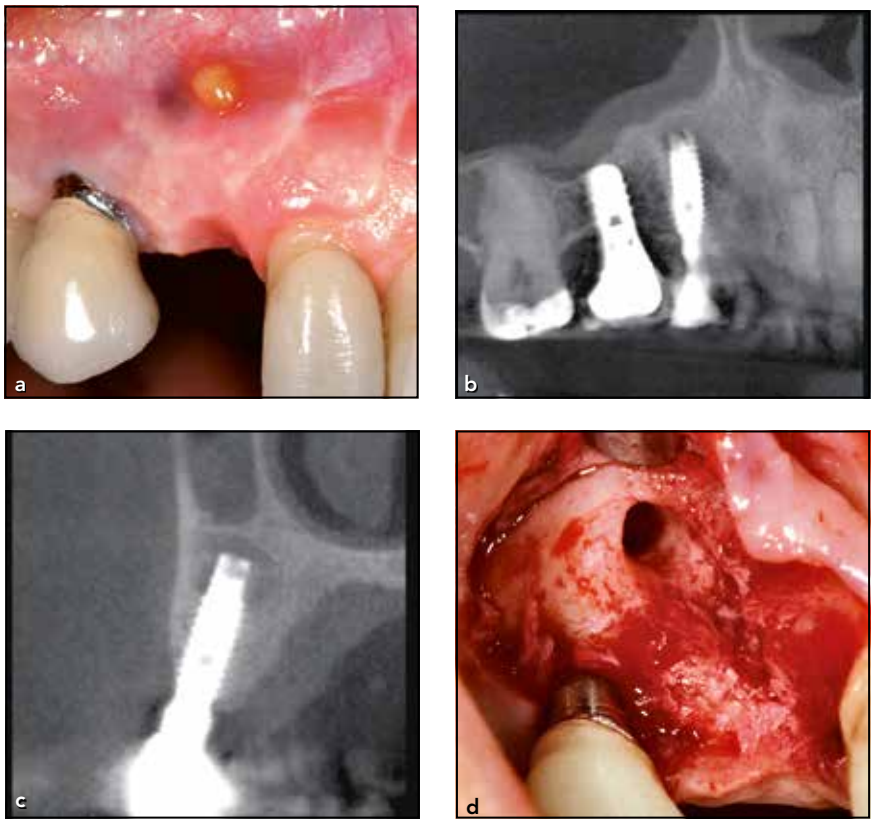


Fig 1 (a) *Initial radiograph. A radiolucency was seen apical to maxillary right canine.* (b) *Radiographic view after endodontic surgery and root resection.* (c) *Nine months after root resection, a big radiolucency was seen apical to the canine, and* (d) *the tooth was extracted.*

Fig 2 (a) *A fistula was still present after tooth extraction.* (b and c) *CBCT sections of the right maxilla, confirming an an intact sinus floor and an area of radiolucency around the most apical part of the implant.* (d) *First access surgery. The bone cavity was debrided, and the implant surface was decontaminated.*



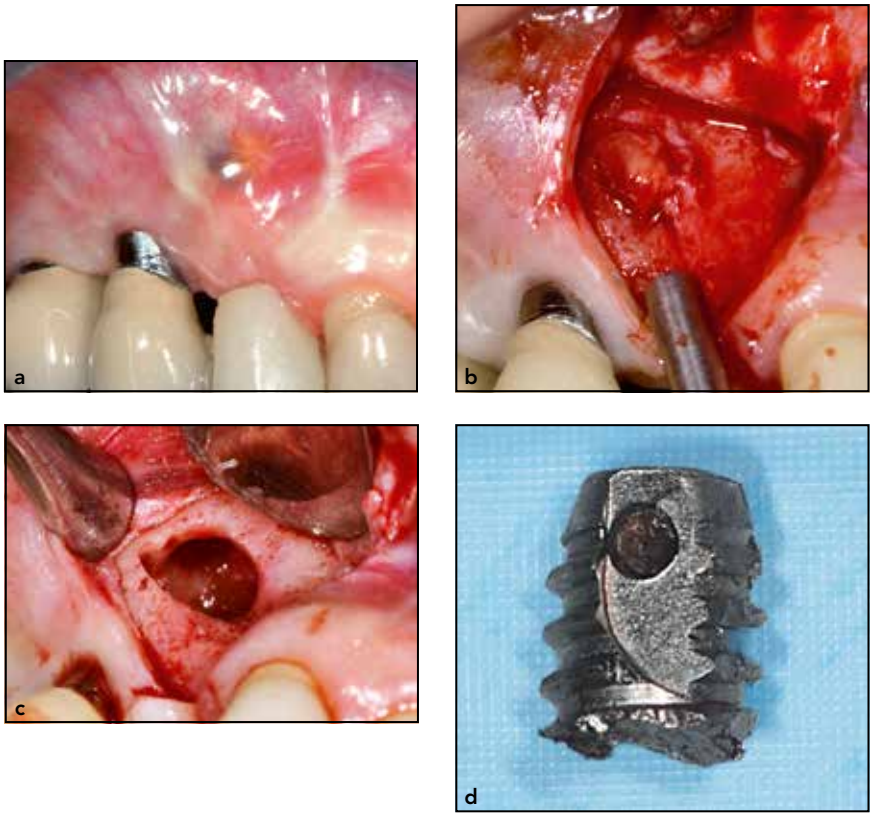


Fig 3 (a) *Six months after the first access surgery, a small fistula was present under the mucosa.* (b and c) *A second paramarginal flap was elevated to remove* (d) *the apical portion of the implant.*

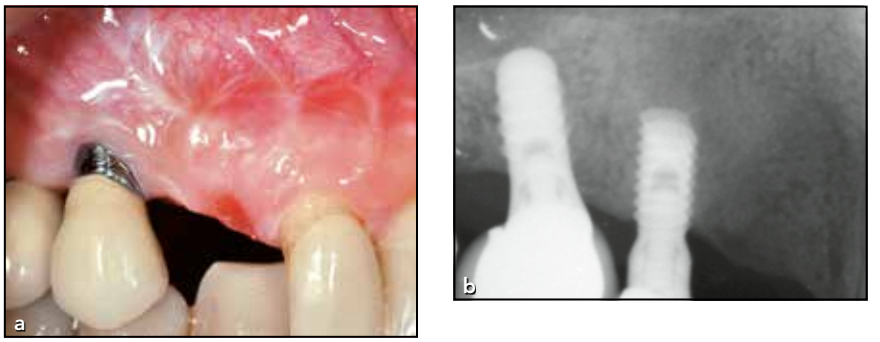


Fig 4 (a) *Clinical and* (b) *radiographic views of the resected implant at the 2-year follow-up.*

At the 6-week follow-up, a small fistula was present under the mucosa, and the infection was still active (Fig 3a). It was thus decided to perform a second surgical procedure to treat the RPI lesion. A full-thickness paramarginal flap was elevated to remove the apical portion of the implant by means of a multiblade bur. The exposed implant surface was cut up to the bone margin (Figs 3b to 3d).

At the 1-month follow-up clinical examination, the patient showed a good wound healing without pain or swelling. The patient was enrolled in a stringent oral hygiene protocol[18] and adhered to follow-ups for 2 years postoperatively. Further bone healing progress was observed (Fig 4).

Discussion

Peri-implantitis is becoming a very important issue in implant therapy. Peri-implantitis is defined as a pathological condition occurring in tissues around dental implants, characterized by inflammation in the peri-implant connective tissue and progressive loss of supporting

bone.[19] In the present case report, a multisurgical therapy involving a tooth extraction, two surgical accesses for implant surface debridement, and implant apicoectomy was required for the treatment of an RPI lesion (ie, reestablishment of peri-implant tissue health).

Most RPI cases are caused by infections of peri-implant soft tissues that develop from the gingival margin through the sulcus; in some cases, the contamination of the implant surface can occur through an endodontic lesion of adjacent teeth.[5] Prevalence of peri-implantitis on an implant adjacent to an endodontically treated tooth is reported as 7.8%,[3] which was higher than the overall reported incidence. The most common etiology of RPI described in the literature was the presence of an adjacent endodontic lesion.[5,7]

Other etiologies include residual bacteria in the implant site, overheating, implant surface contamination, fenestration of vestibular bone, and development of osteomyelitis.[5,7] For most of the case reports available in the dental literature, the diagnosis of RPI occurred between 1 week and 4 years after implant placement.[4] The RPI diagnosed in the present case report occurred 15 years after implant loading, confirming the importance of the endodontic conditions of teeth adjacent to implants over time. Moreover, the present case report confirmed the possible contamination of the implant surface by means of an endodontic infection, even after many years of implant loading. In the present case, there was no loss of supporting bone and the peri-implant soft tissues were stable, except for the presence of a buccal fistula and the radiographic evidence of radiolucency around the implant apex.

Many surgical approaches have been described to treat RPI.[4,5,7] These should provide an adequate access to the defect, but at the same time be as conservative as possible.[6] Most studies include debridement of the defect and detoxification of the implant surface using different agents. None of the studies published described a specific protocol to treat the exposed implant surface, which is a crucial component to achieve resolution of the infection.

In the present case report, two considerations might be drawn. With regard to accessing the defect to provide complete detoxification of the implant surface, the debridement of the bone defect performed in the first surgery was not enough to solve the infection. Consequently, subsequent resection of the implant apex was necessary to allow complete resolution of the infection and new bone formation at the 2-year follow-up. Although guided bone regeneration (GBR) procedures have been proposed in the literature, no GBR procedure was performed in the present case after resection of the implant apex, as the implant surface was under a thick cortical bone and the residual bone defect had the configuration of a four-wall defect.

Conclusions

Within the limits of this case report, the proper diagnosis and treatment of teeth with endodontic lesions adjacent to implants may prevent the development of RPI, even after many years of implant loading. Once an RPI lesion is established, the implant surface debridement should be effective in order to reestablish peri-implant tissue health.

Acknowledgments

The authors declare no conflicts of interest.

References

1. Quirynen, M, Gijbel F, Jacobs R. An infected jawbone site compromising successful osteointegration. Periodontol 2000 2003;33:129–144.
2. Sussman HI. Periapical implant pathology. J Oral Implantol 1998;24:133–138.
3. Zhou W, Han C, Li D, Li Y, Song Y, Zhao Y. Endodontic treatment of teeth induces retrograde peri-implantitis. Clin Oral Implant Res 2009;20:1326–1332.
4. Sarmast ND, Wang HH, Solsatos NK, et al. A novel treatment decision tree and literature review of retrograde peri-implantitis. J Periodontol 2016;87:1458–1467.
5. Chan HL, Wang HL, Bashutski JD, Edwards PC, Fu JH, Oh TJ. Retrograde peri-implantitis: A case report introducing an approach to its management. J Periodontol 2011;82:1080–1088.
6. Romanos GE, Froum S, Costa-Martin S, Meitner S, Tarnow DP. Implant periapical lesions: Etiology and treatment options. J Oral Implantol 2011;36:53–63.
7. Ramanauskaite A, Joudzbalys G, Tozum TF. Apical/retrograde peri-implantitis/implant periapical lesion: Etiology, risk factors, and treatment options: A systematic review. Implant Dent 2016;25:684–697.
8. McAllister BS, Masters D, Meffert RM. Treatment of implants demonstrating periapical radiolucencies. Pract Periodontics Aesthet Dent 1992;4:37–41.
9. Bretz WA, Matuck AN, de Oliveira G, Moretti AJ, Bretz WA. Treatment of retrograde peri-implantitis: Clinical report. Implant Dent 1997;6:287–290.

10. Shaffer MD, Juraz DA, Heggerty PC. The effect of periradicular endodontic pathosis on the apical region of adjacent implants. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86:578–581.
11. Jalbout ZN, Tarnow DP. The implant periapical lesion: Four case reports and review of the literature. Pract Proced Aesthet Dent 2001;13:107–112.
12. Flanagan D. Apical (retrograde) periimplantitis: A case report of an active lesion. J Oral Implantol 2002;28:922–96.
13. Ataullah K, Chee LF, Peng LL, Lung HH. Management of retrograde periimplantitis: A clinical case report. J Oral Implantol 2006;32:308–312.
14. Tözüm TF, Sençimen M, Ortakoğlu K, Ozdemir A, Aydin OC, Keleş M. Diagnosis and treatment of a large periapical implant lesion associated with adjacent natural tooth: A case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101:e132–e138.
15. Ayangco L, Sheridan PJ. Development and treatment of retrograde periimplantitis involving a site with a history of failed endodontic and apicoectomy procedures: A series of report. Int J Oral Maxillofac Implants 2001;16:412–417.
16. Brisman DL, Brisman AS, Moses MS. Implant failures associated with asymptomatic endodontically treated teeth. J Am Dent Assoc 2001;132:191–195.
17. Dahlin C, Nikfarid H, Alsén B, Kashani H. Apical peri-implantitis: Possible predisposing factors, case reports, and surgical treatment suggestions. Clin Implant Dent Relat Res 2009;11:222–227.
18. Nieri M, Giani M, Pagliaro U, Picciullo A, Franceschi D, Rotundo R. Efficacy and preference of manual toothbrushes: A randomised, single blind, controlled trial. Eur J Oral Implantol 2013;6:181–188.
19. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. J Clin Periodontol 2018;45(suppl 20):s246–s266.