APICAL (RETROGRADE) PERI-IMPLANTITIS: A CASE REPORT OF AN ACTIVE LESION

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KEY WORDS

Dental implant
Apical peri-implantitis
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Dental implant treatment can be complicated with infection. There have been reports of infections that are limited to the apical portion of a root form implant.[1–3] These infections have been called apical or retrograde peri-implantitis. A list of possible causes includes overheating during the osteotomy,[4] bacterial contamination from an adjacent tooth,[5] residual bacteria from the infected tooth that previously occupied the site, bone microfractures from overloading or loading too soon, and residual space left after not seating the implant to the full length of the osteotomy.[1–3,6] Most treatments entail surgical debridement of the lesion and surface treatment (detoxification) of the apical or exposed portion of the implant with tetracycline or chlorhexidine gluconate.[7,8] The etiology and treatment of apical or retrograde peri-implantitis remain a topic for discussion.[1–3,6,9–13] The following case of peri-implantitis was treated with surgical debridement and a paste of calcium hydroxide in water and no implant surface detoxification. This resulted in resolution of the associated signs and symptoms of infection.

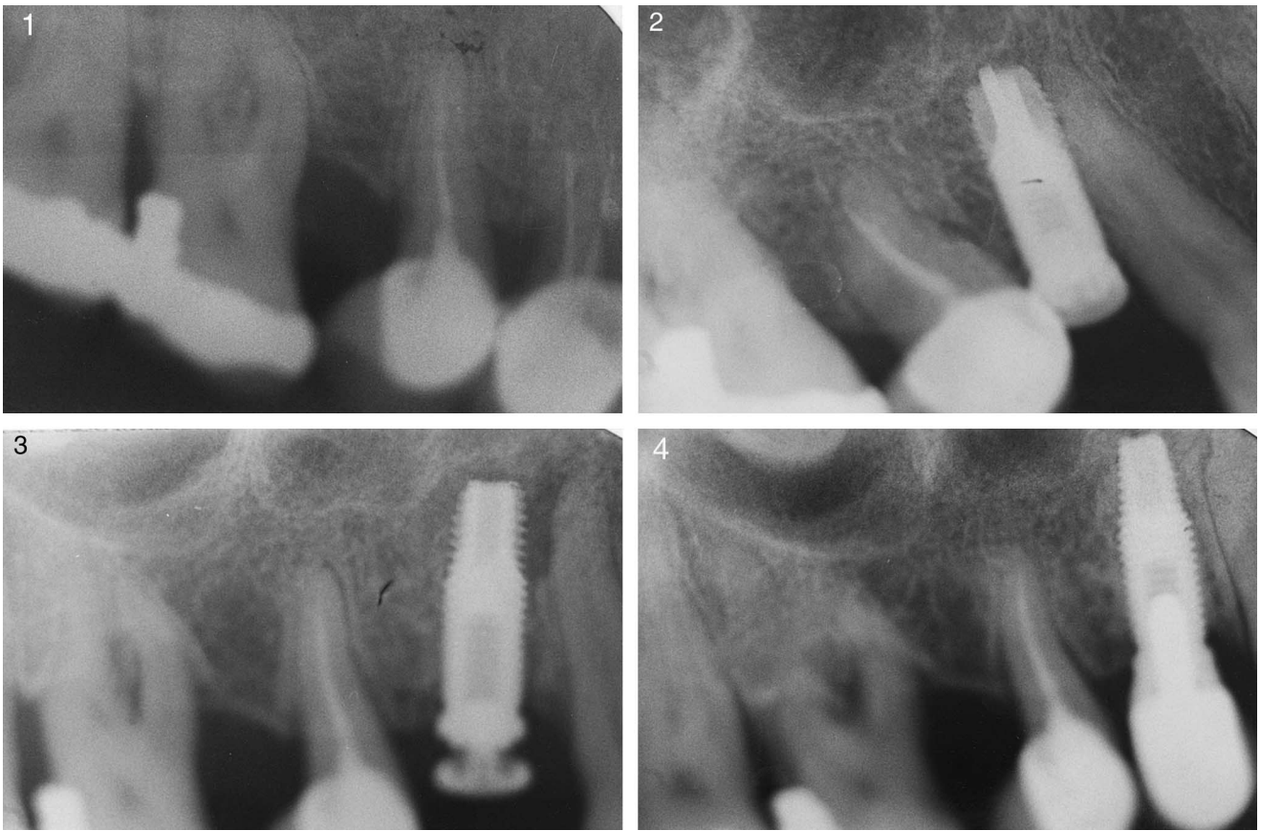
CASE REPORT

The patient, a 46-year-old female, had a history of cervical vertebral fusion and was a 2-pack-a-day smoker. She takes carisolprodol (Soma), celcoxib (Celebrex), and hydrocodone (VicodinES) as needed for relief of symptoms from her cervical vertebral fusion. Her last medical exam was 2 years before (1998). Her physician deemed her a candidate for dental implant treatment. She denied any allergies to medications or foods.

She was being treated for periodontitis. She wore a Nesbit-type maxillary right partial denture, replacing tooth 3. Her occlusion is an Angle Class II with a 6-mm incisor-to-incisor overjet and an open bicuspid occlusion.

She was seen for an emergency visit for acute pain at tooth 12. This tooth was painful to percussion. It had been endodontically treated 8 months prior by another practitioner. A full crown was cemented with zinc phosphate cement. There was no sinus tract at this time. Local anesthesia was obtained with facial and lingual infiltrations. The crown was entered occlusally and the gutta percha seal was removed mechanically with Gates–Glidden drills and then the apical portion removed with an endodontic file dipped in chloroform. The lingual canal was found to be ledged at about 1.5 mm from the root apex. The canal space was filled with calcium hydroxide (Pulpdent) and cotton and closed with Cavit (ESPE). She was prescribed penicillin, 500 mg qid (4 times a day) for 7 days, with a 2-g loading dose.

She was seen on the third day and was still experiencing pain. Options and risks were explained. She opted for extraction and implant treatment. Local anesthetic (1.8 cm^3 Xylocaine)



FIGURES 1–4. FIGURE 1. Failed endodontic treatment of tooth 12. FIGURE 2. Implant in tooth 12 site. FIGURE 3. Apical peri-implantitis lesion. FIGURE 4. Fourteen months after apical treatment.

was infiltrated and the tooth was atraumatically extracted and the socket curetted. She was advised to quit smoking and the complications of habitual smoking explained. There was relief of pain, and the extraction site healed uneventfully. No graft materials were placed in the extraction socket. Study casts were made, a surgical guide was made, and a flipper-type appliance was constructed for site 12.

Three months later, the site was evaluated and the bone volume estimated for implant size and position.[14] After the site was locally anesthetized (1.8 cm^3 Xylocaine and 0.9 cm^3 Marcaine), she was given 800 mg of ibuprofen and a chlorhexidine (Peridex) oral rinse. A full-thickness flap that avoided the interdental papillae was raised and an osteotomy prepared by first marking the site with a #6 round burr, then drilling the site in sequence with 2-mm, 2.8-mm, and 3.2-mm drills (Figure 1). All these were internally and externally irrigated with normal saline. All drills were used at 900 rpm. Misch bone type D-2 was encountered.[15] A 4.0-mm × 13-mm fixture (Osseotite, Implant Innovations Inc, Palm Beach Gardens, Fla) was placed into the full length of the osteotomy (Figure 2, X ray). The site was primarily closed with 4-0 Vicryl (Ethicon). Blood loss was approximately 5 ml. Her blood pressure ranged between 149/94 and 137/87. Her pulse ranged between 89 and 59 beats per minute. The flipper appliance was relieved and adjusted to remove the site-12 denture tooth from occlusion. She was prescribed doxycycline, 100 mg twice a day (bid) for 10 days (she complained of diarrhea from penicillin), chlorhexidine gluconate (Peridex) oral rinse bid, and hydrocodone (Vicodin ES) for pain relief. The sutures were removed 10 days later, and the site had healed well. The patient was reappointed in 4 months for evaluation of the site.

Ten weeks postoperatively, she was seen on an emergency basis for pain at the implant site. A periapical radiograph revealed a loose cover screw and periapical radiolucency on the implant (Figure 3, X ray). She was prescribed penicillin, 500 mg qid for 7 days, and hydrocodone (Vicodin ES) for pain control.

Five days later, she was seen for definitive treatment. A draining sinus tract was noted at the apical area of the implant. Ibuprofen, 800 mg, was orally administered and a chlorhexidine gluconate (Peridex) rinse used. The implant was immobile, and there was no pocketing. The area was locally anesthetized (1.8 cm^3 Xylocaine and 1.8 cm^3 Marcaine), and a full-thickness semilunar mucosal flap was raised. The apical lesion

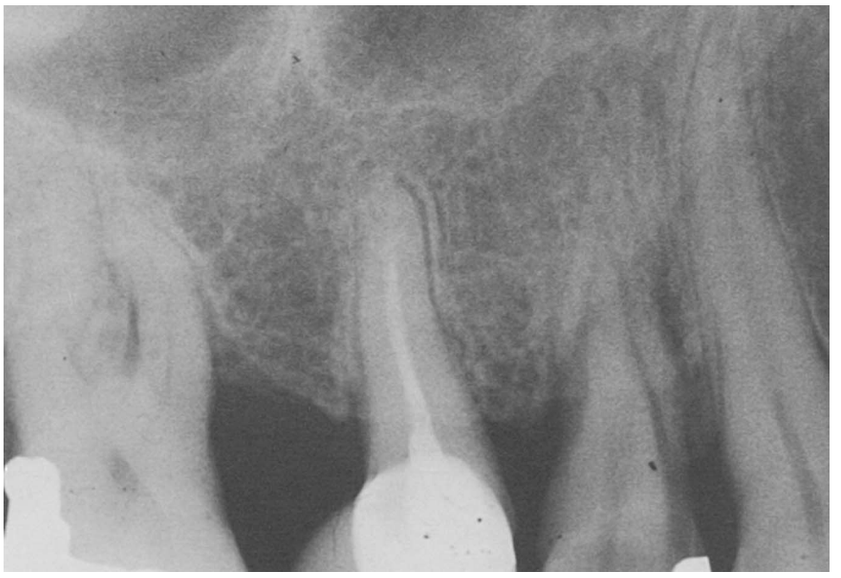


FIGURE 5. Preendodontic treatment.

was debrided and curetted, vigorously avoiding contact with the implant surface. The implant surface was not detoxified. A paste of calcium hydroxide and water (TempCanal, Pulpdent Corp, Watertown, Mass) was placed into the bone defect. This material contains barium sulfate, which is radiopaque. No membrane was used. Primary closure was obtained with 4-0 Vicryl. She was seen 2 days later. The site was slightly swollen and the pain was mostly relieved. At 1 week, the sutures were removed and the patient stated that she had no pain or discomfort.

At 7 weeks (4 months postimplant placement), she was seen and reported no symptoms and, in fact, ''felt great.'' At that time, a straight abutment was placed, the retaining screw tightened to 32 Ncm and slightly prepared and impressed for a full crown cemented restoration. A provisional acrylic crown was placed. A porcelain-fused-to-noble alloy crown was cemented 6 weeks later and made to avoid any centric or eccentric contacts, as was the position of the natural tooth. The crown was cemented with zinc oxide and eugenol paste (Opotow) and then, 4 months later, with zinc oxide and eugenol cement (Temerex). No signs or symptoms of infection were noted.

The patient was seen at 11 months after the apical procedure. A periapical radiograph showed radiographic resolution of the apical lesion, although the presence of barium sulfate in the preparation used was noted (Figure 4). The patient had no symptoms at this time and there were no signs of infection.

DISCUSSION

This case report describes an apical peri-implantitis that was treated with surgical debridement and calcium hydroxide in water paste. The origin of the periapical infection is open to question. An enumeration of possible causes is listed at the start of this article. Each is now discussed with etiologic speculations.

The osteotomy was prepared under copious saline irrigation at 900 rpm. This should preclude any osseous heating, although maxillary premolars are prone to periapical lesions.[1,4] The implant was seated to the bottom of the osteotomy, verified by radiograph. However, a small cortical perforation or thinning from the medullary side of the cortex by the drill at the apical end of the osteotomy could provide a path of least resistance for an infectious process. The infection did not push its way occlusally into the implant-bone interface, causing exfoliation of the implant, but moved through the cortex and into the soft tissue and formed a drainage tract. Without this transcortical path of infection, it is likely the implant would have become totally infected, lost its osseointegration, and become mobile.

Implant loss due to infection may come from either of 2 directions, a gingival or an apical (intraosseous) origin. Early or overloading is not probable in this case because the implant was not loaded at the time of infection and the flipper appliance was relieved and infrequently used by the patient. The adjacent teeth did not have symptoms of infection, but consideration must be made for subclinical necrosis or partial necrosis of an adjacent tooth as a source of bacteria.[11] Tooth 13 had endodontic treatment that seems radiographically inadequate, but there are no symptoms and no discernible communication to tooth-site 12. However, black-pigmented pathogens have been detected in symptomatic as well as asymptomatic endodontically treated teeth, but virulence is variable and bacterial synergism may be important for infectivity.[16,17] Tooth 11 is virgin, asymptomatic, and tested (and has been recently tested) positively to an electric pulp test. There was no discernible radiograph apical lesion on tooth 11 before the endodontic treatment (Figure 5) or after (Figure 4).

The apical peri-implantitis likely came from residual infection of the failed orthograde endodontic therapy. Periapical implantitis is associated with previously failed endodontic and apical procedures at the site.[14] Root canal treatment cannot eradicate all of the microorganisms, especially those associated with failed treatment, and this may allow colonization in the surrounding periapical tissue.[18] These bacteria probably remain after extraction in small islands that escape the acuity of intraoral radiographs.

Encapsulation is a bacterial survival mechanism.[19] The bone at the implant site may have contained residual encapsulated bacteria, which were then

activated by the implant osteotomy, reinstating the infection at the apex of the implant. The antibiotic used at time of placement was a bacteriostatic agent, doxycyline. The use of a bactericidal antibiotic at the time of extraction and implant placement may have prevented or only delayed this episode.[20] Penicillin is a bactericidal antibiotic. Most streptococci, a common dental infectious agent, are very susceptible to penicillin.[21] *Streptococcus* spp encapsulate and can account for 30% to 90% of the bacteria in a dental infectious population.[22,23]

Bacteroides are inhabitants of tooth periapical lesions.[24] These bacteria are susceptible to metronidazole,[25] cefoxitin,[26] chloramphenicol,[27] and clindamycin.[28] Many anaerobic infections are caused by a mixture of organisms. Most anaerobic pathogens are susceptible to penicillin, but most of the *Bacteroides fragiles* group is resistant to penicillin.[29] *Bacteroides* also encapsulates itself in a polysaccharide that probably promotes its virulence, survival, and importance in mixed infections.[30–34] *Bacteroides forsythus* has been shown to persist in asymptomatic periradicular endodontic lesions[35] and may persist and survive in the bone in an encapsulated form after an extraction and infect a newly placed implant.

Penicillin, clindamycin, and erythromycin are first-line antibiotics for dental infections.[36] Penicillin is bactericidal in high dosages. Clindamycin is bactericidal or bacteriostatic depending on the concentration, site, and organism.[37] It has been shown to be superior to penicillin in treating and reducing beta-hemolytic streptococci, a common agent of oral infection.[38] Erythomycin is usually bacteriostatic, but in high concentrations becomes bactericidal against susceptible organsims.[39] Penicillin or higher doses of clindamycin or erythromycin should be considered for routine prophylaxis at implant surgery, especially after failed endodontics to prevent apical peri-implantitis. Other factors such as smoking and periodontitis may be contributory. One respected implantologist has suggested that apical entrapment of gingival epithelial cells during the placement phase of treatment may be a cause.[13] In this case, a full-thickness flap was raised, keeping the gingiva away from the drilled site. It is the operator's opinion that no gingiva was engaged with the drill or implant during placement. The use of chloroform for removal of gutta percha can be called into question, but this material has not been reported to be associated with endodontic infections.

Patient compliance can be a problem with antibiotic administration. A minority of patients take medications as directed.[40] This may affect bacteria survival in bone and behoove the surgeon to impress the patient with respect to compliance. An opportunity was missed in this case by not obtaining a culture or doing sensitivity and a microscopic examinations of the apical lesion. It is difficult to obtain a culture from these sites. A culture report can return as ''normal oral flora'' by the lab.

Anachoresis, the theory that bloodborne pathogens colonize in areas of previous injury and on implants, has been discussed in the literature but has not been proven in some studies.[41–43] There is growing doubt that implants become infected through hematogenous routes and that, instead, late infections are caused by bacteria present at the time of surgery.[44]

The implant surface was not detoxified with tetracycline or chlorhexidine. These materials are used and removed from the site. The calcium hydroxide was left in direct contact with the implant surface and was not removed. Some pathogens can withstand the acid attack of agents such as chlorhexidine and citric acid.[45,46] Calcium hydroxide has been shown to be a better inhibitor of growth activity of bacterial species commonly involved in endodontic infections than chlorhexidine.[47] Any periapical implant radiolucency should be addressed as soon as possible to prevent an acute exacerbation and total loss of implant integration.

Conclusions

A case of apical (retrograde) peri-implantitis has been described with speculations on etiology. The case was successfully treated with surgical debridement and placement of a commercially available calcium hydroxide paste without implant surface detoxification. There are suggestions for the use of bactericidal antibiotics, such as penicillin, or higher dosages of clindamycin or erythromycin for implant placement in failed endodontic extraction sites. A culture and sensitivity and microscopic examinations of the apical implant lesion are beneficial for treatment and diagnosis.

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