10.5005/jp-journals-10004-1032

CASE REPORT

# Recurrent Peri-implant Infection: A Case Report suggesting Origin from an Infected Palatal Root of a Clinically Asymptomatic and Radiographically Normal Appealing Adjacent Molar Tooth

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## ABSTRACT

A case is herewith reported in which a dental implant was immediately placed in an extraction socket of tooth no. 4 and, shortly later, exhibited signs of infection. The local area and implant were treated with standard local measures in addition to completion of a root canal procedure on an adjacent suspect tooth no. 5. In spite of this treatment scheme, the area again exhibited obvious clinical signs of infection during the ensuing weeks. At this point, although previously thought to be within normal limits, the asymptomatic and radiographically normal tooth no. 3 was then considered as the probable source of local infection, and even though pulp testing of no. 3 suggested vitality, the tooth was nonetheless opened revealing a necrotic and fetid palatal pulp. Root canal procedures for tooth no. 3 were then initiated but not completed. The patient then returned to her original dentist who subsequently referred the patient to an endodontist for completion of the root canal treatment on tooth no. 3.

It is important to evaluate adjacent teeth when peri-implantitis occurs. In this case, no radiographic or clinical evidence was present to alert the clinician that the source of the implant-associated infection was the palatal root of tooth no. 3.


**Keywords:** Recurrent peri-implantitis, Endodontic failure, Infection.

**How to cite this article:** Nordquist WD, Krutchkoff DJ. Recurrent Peri-implant Infection: A Case Report suggesting Origin from an Infected Palatal Root of a Clinically Asymptomatic and Radiographically Normal Appealing Adjacent Molar Tooth. Int J Clin Implant Dent 2015;1(1):44-47.

**Source of support:** Nil

**Conflict of interest:** None

## INTRODUCTION

Numerous and various factors have been proposed to explain clinical failures that occur following placement of dental implants, and many, if not most, of these involve bacterial contamination and subsequent infection. Traditional causes have included those attributed endodontic failure, a periapical lesion of an adjacent tooth, impingement by the implant onto an adjacent tooth root, periodontal pathogens seeding into the area, overheating of bone during surgery and implant contamination during surgery, including impaction of infected epithelial cells into the peri-implant area.[1-9]

Although, usually considered clinically insignificant, the possibility of low-grade and totally asymptomatic periapical infection of adjacent teeth looms as an underappreciated source of potential complication. As such, it may well be underestimated as the putative contaminant source of bacteria that infect and subsequently cause failure of oral implant procedures. We herewith present a case in which peri-implant infection occurred that was ultimately attributed to a clinically silent, low-grade infection within the palatal root of an adjacent molar tooth no. 3. The latter was both radiographically and clinically within normal limits and, as such, very difficult to diagnose. In fact, the correct diagnosis of low-grade palatal root infection of tooth no. 3 was initially missed, not only by the author but also the referring general dentist and the endodontist who previously performed the endodontic procedure on the other adjacent tooth no. 5. Through a host of conflicting professional opinions and resultant confusion, the patient ultimately decided to have the implant removed even though, at that point, the infection had probably been treated adequately leading to conditions favoring implant survival. A variety of lessons were learned from this case and are herewith presented in the interest of hopefully leading to improved patient care in clinically similar cases.

## MATERIALS AND METHODS

A 53-year-old female patient presented to the author's (WN) office for tooth removal and implant replacement of tooth no. 4 (Fig. 1). The patient had been under the care of her general dentist and had no other significant dental or health issues. A treatment plan that included extraction of the involved tooth and immediate placement of an

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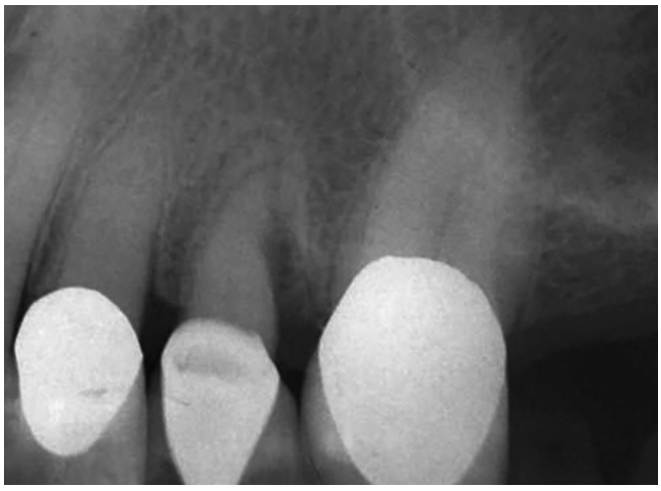

**Fig. 1:** Pretreatment X-ray

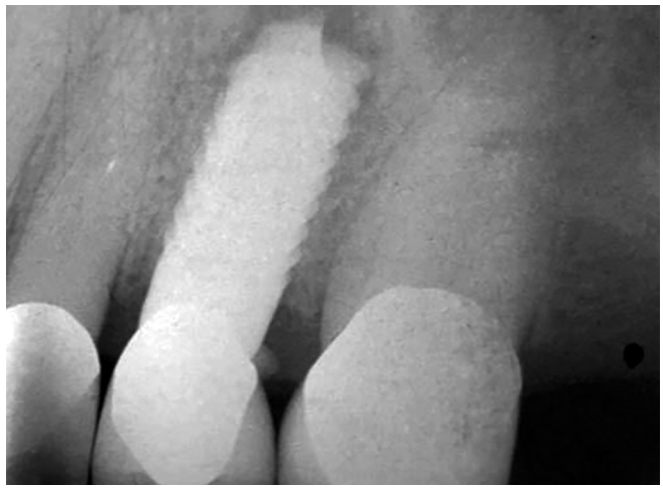

**Fig. 2:** Peri-implant lesion

implant into the extraction socket was presented to the patient and subsequently accepted. The extraction and immediate placement of an implant was performed and proceeded uneventfully.

At the time of uncovering surgery, a peri-implant infection with a draining fistula was noted adjacent to the implant in the periapical region (Fig. 2). Periapical surgery was performed and included both curettage of infected granulation tissue in the affected area plus sectioning of an approximately 2 mm portion of the implant tip. The spherical bone void was then grafted with synthetic resorbable HA mixed with a solution of lincomycin. It was noted that the remainder of the implant was solidly surrounded and integrated with dense normal bone, showing no sign of infection nor any evidence of acute osteomyelitis. Also, there was no visible track pointing to any site of suspicious origin. With no other credible evidence, it was assumed that the source of infectious bacteria was contaminated material impacted into the peri-implant area during surgery. The area healed normally and the problem was thought to be mitigated.

However, soon after the healing phase from prior surgery, the sinus track once again opened and began to drain. Local treatment was instituted and consisted of local irrigation and placement of Arestin into the infected area. After a short period of time, in which there was no clinical resolution, it was decided to re-enter the area to determine whether there might be something that was previously missed. No changes were seen except that the augmentation material was no longer present. The lesion size was exactly the same, and there were no clues as to why the drainage persisted. The previously cut apical surface of implant was 'freshened' to remove any residual surface contamination that might be present. In other respects, the implant was totally surrounded by solid bone on all sides. Removal of the additional metallic material revealed the interior threaded cavity of the implant. Since, the threads and screw-seat of the abutment-implant junction were initially sealed with Bis-GMA resin (Scotch Bond); at the time, the implant abutment was placed, the author was confident that no microleakage would occur. Therefore, the exposed void in the implant was sealed with a light-cured composite resin and the area was reaugmented with a mixture of synthetic bone and an antibiotic. The site healed uneventfully.

Several weeks later, the area once again developed a draining fistula. At that time, it was determined that there was nothing about the implant itself that had caused the infection. The two adjacent teeth no. 3 or 5 became suspect, even though there was no clinical or radiographic evidence of any problem. Since, both of these teeth had been previously crowned, testing for vitality was challenging. With the patient's permission, the vitality test was performed by cutting through the occlusal of both crowns with a no. 700 carbide bur and into the dentin without use of an anesthetic. Tooth sensitivity when penetrating into the dentin would indicate vitality of the tooth. Tooth no. 5 tested nonvital, however, tooth no. 3 did respond to the stimulus and it was concluded that it was vital. A composite restoration was placed in the preparation of tooth no. 3 and endodontic treatment was commenced on tooth no. 5. The canal was completely calcified, but the root canal treatment was completed successfully by the patient's endodontist. Unfortunately, shortly thereafter, the apical area of the implant once again developed a draining fistula. At that point, it was immediately suspected that tooth no. 3 might be the culprit even though it tested vital. This suspicion was explained to the patient. She was willing to take the risk and allow endodontic treatment on tooth no. 3 to rule out tooth no. 3 as the source of the infection. The tooth was opened revealing a dead necrotic pulp within the palatal canal. Additionally, the mesial and distal buccal canals were calcified. Necrotic tissue from the lingual canal was

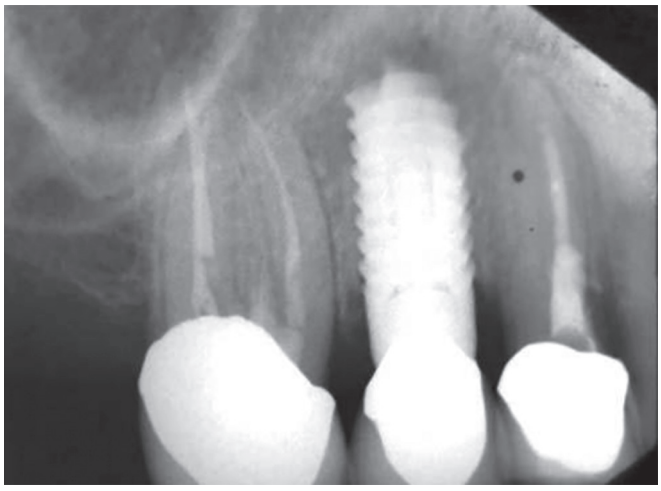

**Fig. 3:** The implant area after two endodontic procedures. Notice no bone loss occurred on the implant during this long treatment of the periapical infection

easily removed intact using a barbed broach intact and exhibited both a dark gray color and a very fetid odor.

Given the fact that the diagnosis of this situation was so difficult and protracted, the final treatment resolution took several months. The patient, thus, lost faith in her implant even though it had been treated successfully (Fig. 3). Accordingly, under the advice and encouragement of her previous dentist, his endodontist and an oral surgeon; she opted to have the implant removed to assure no future problems with the area despite advice to the contrary.

## DISCUSSION

In this case, the many instances of recurrent local infection after placement and local treatment of a dental implant were determined to have resulted from a necrotic palatal canal of an adjacent molar, which required a very difficult and lengthy diagnostic procedure to uncover. In a prolonged sequential therapeutic process, several other possible sources of infection were gradually eliminated leaving the unsuspected, innocent-appearing molar tooth as the sole remaining logical culprit. Even though care must be taken to diagnose these potential problems before implants are placed, this case illustrates the difficulty in reaching proper diagnosis, and thus determining the source of the infection. This is particularly true when adjacent teeth are both clinically and radiographically within normal limits. Further, when implants are contemplated, the treating dentist must be suspicious of adjacent teeth as potential sources of bacteria that can infect and thus compromise future implants, particularly when such teeth are crowned, heavily restored or have prior root canal treatment. Proper diagnosis in these situations is often difficult as demonstrated here when infection of the palatal canal of tooth no. 3 was initially missed, not only by the author, but also the patient's general dentist and endodontist.

Chronic, low-grade infection of asymptotic teeth, especially when crowned or periodontally involved, is probably more common than what most dentists appreciate. Implant dentists are becoming more aware of this problem due to the propensity of implants to fail in the vicinity of previously compromised teeth, but other dentists are apparently less suspicious. It has been well documented that the surfaces of dental implants are vulnerable to bacterial contamination during the early integration process. Experience has taught us that the implant itself serves as a 'dip-stick' or indicator of subclinical, low-grade chronic infection in the alveolar bone. In this case, tooth no. 5 had calcifications which indicate long standing chronic injury of that tooth. It was later discovered that tooth no. 3 also had calcifications plus necrotic palatal root tissue. It is highly unlikely that the implant procedure caused calcification of these teeth, since peri-implant infection occurred within 5 months of its placement. Rather, the calcifications were likely due to pre-existent, long-standing chronic injury.

An additional source of the problematic infection here could have been bacterial debris associated with the extracted tooth no. 4. The tooth was not only previously treated by root canal but was also loose and periodontally involved. However, in removal of this tooth, the source of infective material is likewise removed leaving any residual bacteria at the surgical bed as subject to effective neutralization by the inflammatory response. Further, the re-entry surgery verified that the vast majority of the implant had osseointegrated, which is additional evidence that the source of the infection was not associated with the extracted tooth itself since, otherwise, the implant would not have integrated. Therefore, the preponderance of evidence indicates that tooth no. 4, including its associated bacterial load, is a highly unlikely source for the observed repetitive infection in the implant site.

It must be emphasized that even though low-grade infection is not clinically obvious (and thus deemed absent), there is no guarantee that it does not exist in subclinical form. Even though others might disagree, the authors feel that subclinical chronic infection existed in the vicinity long before the implant was placed. This infection was well tolerated by the patient until the implant was placed. Once the implant was inserted, it became contaminated by bacteria present in the bone originating from adjacent sites (palatal apex of tooth no. 3). This procedure altered the local environment to the extent that it resulted in mild exacerbation of infection and formation of a draining fistula (sinus tract). However, since the patient had already built up an immune defense against this particular bacterium over the years, an acute

osteomyelitis did not occur. The infection was localized and was evidently insufficient to cause extensive bone destruction even though the therapeutic procedure took several additional months to resolve.

Chronic infection in the oral cavity can be caused by one or more of the hundreds of bacteria known to exist within the mouth. Often these infectious foci can be subtle, asymptomatic and extremely difficult to diagnose. Ordinarily, such infections are clinically insignificant, but when subjected to superimposed trauma associated with placement of an implant, they can become activated and lead to local bone loss which threatens implants and often causes them to fail. Whether originating from an endodontically treated tooth, an asymptotic necrotic nerve canal, or from periodontal disease, these foci need to be taken more seriously as potential sources of implant failure. In that these seemingly innocuous infections may also be implicated in the pathogenesis of several systemic diseases in addition to their role in implant failures, they need to be studied more extensively and eliminated, if possible, prior to placement of advanced dental implant prostheses.

## SUMMARY

An elusive, asymptomatic necrotic palatal root canal of tooth no. 3, with no evidence of previous radiographic abnormality, was judged to be the source of an infection within an adjacent implant site. Infection presented as a peri-implant draining fistula in the site of a previously extracted tooth no. 4. Ultimately, this difficult situation lead to loss of the implant even though this implant could well have survived. Progress in this case was delayed due to the extreme difficulty in reaching the proper diagnosis, and this delay played a role in the patient's loss of confidence, the resultant disagreement among involved practitioners and the ultimate decision to remove the implant that otherwise, may have survived. Details of the case are presented in hopes that other practitioners can avoid difficulties encountered here when dealing with similar cases.

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