Case Report

Retrograde Peri-Implantitis: A Case Report

Abstract

Retrograde peri-implantitis describes a lesion that is periapical to an osseointegrated implant. The condition is identifiable by radiological examination and from clinical symptoms such as pain, tenderness, or the presence of a sinus tract. Multiple surgical methods have been used to resolve this disorder. This case report describes a successful treatment of retrograde peri-implantitis and the associated follow-up. The study implant was restored to health with full functionality.

Keywords: Dental implant, retrograde peri-implantitis

Introduction

During recent decades, the use of dental implants have become a popular method to replace missing teeth. However, the treatment carries the possibility of implant failure with multifactorial etiologies; for example, retrograde peri-implantitis is a potential cause of failure. Retrograde peri-implantitis is also referred to as "implant periapical lesion," "periapical implant pathology," "periapical implant lesion," "retrograde peri-implant infection," and "apical peri-implantitis."

The condition was first described by McAllister et al. as a clinical symptomatic periapical lesion. Diagnosis is made by a radiolucency develops shortly after implant insertion, in which the coronal region of the implant forms a normal bone-implant interface.

Retrograde peri-implantitis is often diagnosed by radiographic imaging of periapical radiolucency around the implant's apical region. The patient might experience pain, redness, tenderness, and swelling, and may present with a sinus tract. The prevalence of retrograde peri-implantitis was assessed in a retrospective study of 539 implants of which 1.6% were maxillary and 2.7% were mandibular teeth.

Zhou et al. recently reported a 7.8% incidence of retrograde peri-implantitis adjacent to an endodontically treated tooth, which is greater than the overall reported incidence.

There are three etiologic factors that lead to retrograde peri-implantitis. The first, implant factors, include contamination of the implant, and poor biocompatibility with the implant surface. The second factor, patient factors, include residual bacteria at the implant site, an adjacent endodontic lesion, residual root particles or foreign bodies, or poor bone
Case Report

A 59-year-old male patient suffered from palpation pain and intermittent fistula in the lower right area (tooth 46) for 1 year. No relevant past medical history was recorded, except that the patient was allergic to penicillin. The tooth had a history of endodontic treatment and was restored with a 44-x-46 PFM bridge more than 7 years previously.

Clinical examination revealed an ill-fitting 44-x-46 bridge, and the presence of a fistula tract over the 46 buccal mucosa. Periapical radiographic assessment revealed incomplete 46 root canal treatment with apical radiolucency and furcated bone resorption (Fig. 1).

The tooth was diagnosed with a poor prognosis and extraction was advised. The patient opted for treatment with 45, 46 implants. The 46 extraction was performed on July 18, 2010 (Fig. 2) and the tooth socket was completely debrided. Two months later, two dental implants were placed in the 45–46 region on September 20, 2010 (Fig. 3). The patient was placed on clindamycin HCl (150 mg) four times a day for seven days.

At the two week postoperative follow-up, a mild-pain sensation was noted in the area of tooth 45. Radiolucency extended to the apical portion of 45 dental implant (Fig. 4). The apical lesion steadily increased in size during the first month (Fig. 5A), second month (Fig. 5B), to the seventh month (Fig. 5C). Increased
bone was visible by radiography until the 9th month (Fig. 6). Additionally, symptoms subsided throughout this period. Stage II of the procedure was carried out at three weeks (Fig. 7), and finally, the tooth was restored with a permanent crown on September 25, 2011 (Fig. 8).

Discussion

Most case study reports conclude that the endodontic pathology of an extracted tooth, or that a possible endodontic pathology arising from a neighboring tooth, are the main causes of retrograde peri-implantitis. Endodontic bacteria can be reactivated during an implant osteotomy, and this may lead to implant infection.10

The patient in this case report had previously received incomplete endodontic treatment of tooth 46, and presented with a large apical lesion. Two months following tooth extraction, dental implants were inserted into areas 45 and 46. Although area 45 had developed retrograde peri-implantitis, 46 did not show signs of the condition, thus, we excluded the presence of an adjacent endodontic lesion as the source of implant infection because of the successful implant in area 46. There was no indication of biocompatibility issues with the implant, as there were no residual root fragments or foreign bodies present in the bone. Because implant and patient factors were excluded, the remaining "dentist factor" should account for the emergence of retrograde peri-implantitis in the 45 area. For this factor, bone overheating or bone compression are the most probable causes.

Recently, various treatment strategies have been used for the management of retrograde peri-implantitis, including debridement alone, a combination of debridement and grafting material with or without membrane, detoxification of the infected implant surfaces, and apicoectomy.12 However, our patient received successful treatment and only required regular follow-ups. The patient’s symptoms and radiographic radiolucency completely disappeared after nine months.

Conclusions

Although many articles reported high success rates for surgical treatment of retrograde peri-implantitis, there was no scientific validation of such procedures. In addition to the various treatments available, regular follow-ups could improve the prognosis for patients. Additional research is needed to provide greater understanding of the etiology and clinical symptoms related to retrograde peri-implantitis.
References