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REVIEW ARTICLE

RETROGRADE PERI-IMPLANTITIS – A REVIEW OF LITERATURE AND AN UPDATE

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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. Retrograde peri-implantitis constitutes an important cause for implant failure. Retrograde peri-implantitis may sometimes prove difficult to identify and hence institution of early treatment may not be possible. This paper aims to briefly discuss etiology and treatment strategies of retrograde peri-implantitis available in the literature.
Keywords: Retrograde peri-implantitis; Dental implant; Periapical implant pathology.

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INTRODUCTION

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. These infections have been called apical or retrograde periimplantitis.[1]

Retrograde peri-implantitis (RPI) is defined as a clinically symptomatic periapical lesion (diagnosed as a radiolucency) that develops shortly after implant insertion in which the coronal portion of the implant achieves a normal bone to implant interface.[2] This lesion occurring at the periapical area of an osseointegrated implant, has recently been described as a possible cause for dental implant failure.[3] According to Mellonig et al., implant failures can be placed in two categories; namely, failure due to infection (periimplantitis or retrograde periimplantitis) and failure due to trauma (excessive overloading or implant fracture). [4]

This condition was first described by McAllister et al. in which microbial involvement from the implant site, the extracted teeth or adjacent teeth, generation of excess bone heat during implant placement and premature loading from inadequate relief of interim prostheses were considered the probable causes.[2]

The condition is also referred to as implant periapical lesion, periapical implant pathology, endodontic-implant pathology, periapical implant lesion, retrograde peri-implant infection, apical peri-implantitis.[5]

The prevalence of retrograde peri-implantitis was assessed in a retrospective study of 539 implants, with 1.6% of maxillary and 2.7% of mandibular teeth that exhibited this condition prior to abutment connection.[6]

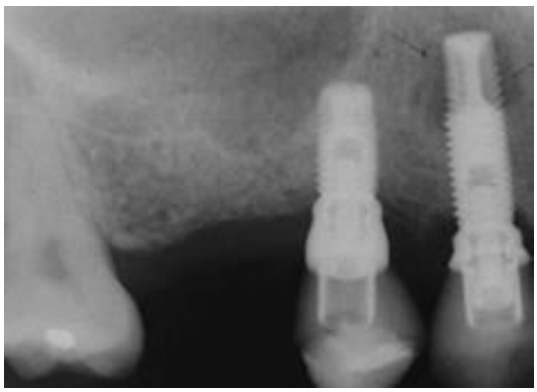

Figure 1: Radiograph showing Retrograde perio-implantitis. Periapical radiograph showing evidence of periapical bone loss surrounding the implant in the right maxillary premolar region.[7]

ETIOLOGY

As a retrograde peri-implantitis is often accompanied by symptoms of pain, tenderness, swelling and/or the presence of a fistulous tract, two types of lesion can be distinguished: the disease active periapical implant lesion; and the disease-inactive periapical implant lesion. Lesions are called 'inactive' when the radiological findings are not comparable with the clinical findings and/or the patient's symptoms. A clinically asymptomatic, periapical radiolucency (which is usually caused by placement of implants that are shorter than the prepared osteotomy) is to be considered as inactive. When an implant is placed next to a pre-existing, detectable radiolucency, which is related to scar tissue, this also can lead to an inactive lesion.[8,9] An inactive lesion can also be caused by aseptic bone necrosis, frequently induced by overheating the bone during osteotomy preparation. Overheating is mentioned as a risk factor for bone necrosis. Uncontrolled thermal injury can result in the development of fibrous tissue, interpositioned at the implant–bone interface, compromising the longterm prognosis of the implant. Implant insertion in a site with pre-existing inflammation (caused by bacteria, viruses, inflammatory cells and/or cells remaining from a cyst or a granuloma) can also lead to an active periapical implant lesion. These lesions are initiated at the apex of the implant but have the capacity to spread coronally and facially. [10]

Sussman proposed two pathways that may lead to retrograde peri-implantitis: type 1 (implant to tooth) and type 2 (tooth to implant). Type 1 RPI occurs when the osteotomy preparation causes direct or indirect damage to the adjacent tooth, resulting in devitalization of the tooth pulp and periapical pathology. Subsequently, the periapically infected tooth inhibits osseointegration of the implant. Type 2 RPI occurs when an adjacent tooth with periapical pathology contaminates the fixture and interferes with osseointegration of the implant.[11]

Thus, there are mainly 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 quality. The third, the dentist factor, includes bone overheating, bone compression, and premature loading.[5]

TREATMENT

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. [5]

All risk factors for periodontal diseases should be assessed and controlled as far as is possible. Periodontal treatment should be completed and there should have been a sufficient period of supportive therapy in order to assume stability. [7]

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.[12,13]

In some case reports, nonsurgical treatments have been discussed. Chang et al. treated one patient without surgical intervention. They used amoxicillin in combination with clavulanic acid, prednisolone and mefenamic acid, after which the patient's symptoms completely subsided and radiographically the lesion disappeared. After a follow-up of 2 years the implant remained stable.[14]

Waasdrop & Reynolds also treated one patient nonsurgically with the use of antibiotics. The radiographic lesion gradually resolved during the following 9 months without further treatment. However, other authors reported that antibiotics were not effective in controlling active lesions. [15]

The literature also supports a surgical approach that may include debridement only or bone replacement grafts (BRGs) with or without the use of membrane barriers.[3]

Quirynen et al. performed treatment on 10 cases with periapical implant pathology (out of a total of 426 solitary implants). The protocol for the treatment of retrograde lesions in the maxilla included elevation of a full-thickness flap, complete removal of all accessible granulation tissue using hand instruments (with special attention to reach both apical and oral parts of the implant surface) and curettage of the bony cavity walls. In half of the defects, deproteinized bovine bone mineral was used as bone substitute (at the discretion of the surgeon), whereas the other defects were left empty. In the mandible an explorative flap mostly revealed an absence of a perforation of the cortex so that a trepanation of the bone had to be performed. They concluded that the removal of all granulation tissue is sufficient to arrest the progression of bone destruction.[10]

The use of different types of BRGs with or without occlusive membranes is not universally accepted; however, their application may provide several advantages. First, BRG can act as a scaffold for new bone cells to grow into the bony defect. Second, they can maintain the space and prevent soft tissue from repopulating the defect. It has been demonstrated that the use of bone regeneration materials for apicoectomy surgeries improves the predictability of clinical, radiographic and histological healing.[16]

CONCLUSION

Post placement for every implant patient a preventive programme should be tailored to each individual patient's risks, with regular maintenance and close monitoring to detect early disease. 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.

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