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Implant Periapical Lesions: Literature Review and Two Case Reports

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Review

History

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ABSTRACT

Objectives: Implant peri-apical lesion is a peri-implant complication described by radiographic periodontal bone loss that occurs around the implant's apical region. Different approaches have been suggested for addressing implant peri-apical lesions, involving peri-apical surgery, regenerative therapy, or a combination of regenerative surgery with the resection of the implant apex.
Materials and Methods: In this systematic review, we searched the available literature on implant peri-apical lesions using the PubMed database and described the possible aetiologies and treatments of two implant peri-apical lesions that occurred at different stages of osseointegration.
Case Report: Two patients presented to our clinic with pain and swelling 16 days and 7 months after implant surgery. In both patients, the operation site was opened, granulation tissues were excised, and the area was rinsed with sterile physiologic serum solution. In the second patient, graft and membrane application was performed additionally. Both sites were primarily closed.
Conclusions: No complications were observed at either site during the follow-up period. Therefore, both approaches can be considered effective in the treatment of implant peri-apical lesions.

Keywords: Dental implants, postoperative complications, oral surgery peri-implantitis.

İmplant Peri-apikal Lezyonları: Literatür İncelemesi ve İki Olgu Sunumu

Derleme

Süreç

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ÖZ

Amaç: İmplant peri-apikal lezyonu, implant apeksi çevresinde radyografik kemik kaybı ile karakterize bir peri-implant komplikasyonudur. İmplant peri-apikal lezyonlarının tedavisi için peri-apikal cerrahi ve implant apeksinin rezeksiyonu ile birlikte veya rezeksiyon olmaksızın rejeneratif cerrahi dahil olmak üzere farklı yaklaşımlar önerilmiştir.
Gereç ve Yöntemler: Bu sistematik derleme için, PubMed veri tabanını kullanarak implant apikal lezyonları hakkındaki mevcut literatürü araştırdık ve osseointegrasyonun farklı aşamalarında meydana gelen iki implant peri-apikal lezyonunun olası etiyolojilerini ve tedavilerini tanımladık.
Olgu Sunumu: İki hasta implant cerrahisinden 16 gün ve 7 ay sonra ağrı ve şişlik şikayetiyle kliniğimize başvurdu. Her iki hastada da operasyon bölgesi açıldı, granülasyon dokuları çıkarıldı ve bölge steril serum fizyolojik ile irrige edildi. İkinci hastada ek olarak greft ve membran uygulaması yapıldı. Her iki bölge de primer olarak kapatıldı.
Sonuçlar: Takip süresi boyunca her iki bölgede de herhangi bir komplikasyon gözlenmedi. Bu nedenle, her iki yaklaşımın da implant peri-apikal lezyonlarının tedavisinde etkili olduğu düşünülebilir.

***Anahtar Kelimeler**:* Diş implantları, komplikasyonlar, peri-İmplantitis, cerrahi tedavi.

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Introduction

Implant dentistry has significantly changed the way patients are treated with fixed and removable prosthetic solutions that restore chewing function and aesthetics.[1] Dental implants offer several advantages over traditional prostheses, including a high success rate, a reduced risk of caries, endodontic complications, and sensitivity in the adjacent teeth.[2]

The popularity of dental implants has been associated with an increase in various mechanical and biological complications.[3,4,5] The 1999 periodontal disease classification excluded peri-implant disorders and conditions. However, peri-implantitis, peri-implant mucositis, and peri-implant health were acknowledged by the 2017 World Workshop. Peri-implant health was described in clinical practice as having no evidence of clinical indicators of inflammation, absence of bleeding and suppuration on probing, stable probing depth compared to previous examinations, and no destruction of bone.[6] Peri-implantitis is an irreversible inflammatory condition associated with plaque and is the primary reason for late implant failure.[7] This disorder is outlined by the pathologic inflammation of the peri-implant tissues accompanied by a progressive loss of radiographically observed surrounding bone.[8] Initially owing to the case description used, the occurrence of peri-implantitis is still up for discussion. According to a systematic review, mucositis affects 43% of dental implants, and peri-implantitis affects about 23%.[9]

Implant peri-apical lesion (IPL) is a peri-implant complication first recognised as a distinct condition by McAllister et al.[10] IPL is a symptomatic issue that typically arises within the first six months following implant placement. It is identified radiographically by bone loss around the implant's apex, without signs of marginal bone loss or pathological peri-implant probing depths. This condition is fundamentally different from marginal peri-implantitis, which affects the coronal portion of the implant and leads to progressive bone loss in the surrounding structures.[11,12]

With the growing number of individuals undergoing implant treatment in recent years, the incidence of IPL has also increased. According to the literature, the prevalence of IPL ranges from 0.26% to 9.9%.[13,14] Reports indicate that its occurrence varies between 0.28% and 5.8% in the maxilla and 0.42% and 2.7% in the mandible. Additionally, studies have indicated that IPL is more frequently observed in maxillary premolars.[15-17] The development of an IPL may be influenced by the endodontic state of the tooth that was removed from the implant site and its neighbouring teeth.[18] Studies have shown an increased risk of IPL when adjacent teeth have failed endodontic treatment or when peri-apical lesions are present before implant placement.[17] In the early stages of implant treatment, IPL incidence was relatively low, as implants were primarily placed in fully edentulous areas. Consequently, due to the limited number of studies on the topic, IPL remains a relatively unfamiliar pathology among clinicians.[19] However, as implants are now more commonly used to replace single or multiple teeth requiring extraction, a rise in IPL cases is expected.[18]

Different hypotheses have been proposed regarding the possible aetiology of IPL. These hypotheses include early loading, the bone's overheating caused by drilling, implant placement in an infected area, bone over-preparation, residual root fragments or foreign bodies that are present, implant surface contamination during instrumentation, and insertion into a contaminated maxillary sinus area.[20-23]

A retrospective study reported that a tooth with a history of endodontic lesions can cause IPL with a probability of 7.2%, even in the absence of peri-apical lesions. While the study identified a correlation between endodontic treatment and IPL in four cases, IPL was also diagnosed in two instances where implants were placed in areas adjacent to teeth that had either never undergone endodontic treatment or had previously been treated.[24]

Acute dental peri-apical pathology typically presents with pain that intensifies upon percussion on the affected tooth. Conversely, acute IPL does not provoke heightened pain from percussion due to the lack of periodontal ligament innervation. Instead, patients experience spontaneous pain. During the chronic phase, a dental peri-apical abscess may develop a fistula through bone and soft tissue, facilitating the drainage of purulent material through the partially consolidated interface between the implant and surrounding bone.[25]

IPL is identified by employing clinical and radiographic evaluations. However, symptoms and clinical signs are not always present. When they do occur, they may include persistent and severe pain (which may not respond to analgesic treatment), inflammation, swelling, abscess formation, fistula development in the buccal apical region of the implant, tenderness, and redness.[26]

Determining the stage of lesion evolution is essential for designating the most suitable therapeutic strategy. In non-suppurative acute IPL, an inflammatory infiltrate is present, characterised clinically by spontaneous and localised pain. The mucosa may appear swollen, and percussion of the implant produces a tympanic sound, though radiographic imaging does not reveal alterations in bone. An IPL or apical abscess may develop, characterised by a buildup of purulent collection surrounding the implant apex. During this suppuration phase, the accumulating pus seeks drainage through the path of least resistance, leading to bone destruction around the implant. When drainage pathways are established, the condition transitions into subacute IPL. If the coronal bone-implant connection is preserved, a fistulous tract may develop towards the buccal cortex, potentially leading to a buccal abscess. If the coronal bone-implant interface is poorly structured, drainage will occur along the path of least resistance, resulting in the coronal destruction of the peri-implant bone.[27]

Reiser and Nevins[28] categorized IPL into passive and active types. The lesion, in its active state, is symptomatic and necessitates treatment to prevent more bone destruction. The dormant state of the IPL is asymptomatic and is usually detected via radiographic examination, appearing as a radiolucent area around the apex of the implant. This radiolucent area is an apical scar resulting from bone necrosis caused by excessive implant bed preparation or overheating during placement. Dormant lesions typically do not require treatment unless the radiolucent area enlarges. However, they should be monitored radiographically.[26]

Histologic analysis of IPL has indicated the presence of osteonecrotic bone, inflammatory cell infiltration, and/or cysts in the apical region. This indication may be linked to vascular impairment and bone fracture.[29] These findings are similar to those of the granuloma or abscess in the root apex of non-vital teeth.[30] Furthermore, the pathological analysis of IPL revealed gram-positive rods, gram-negative rods, cocci, and filamentous microorganisms.[10] Bacterial culture of IPL is difficult to obtain.[29] The prevalence of anaerobic bacteria exceeds that of aerobic bacteria due to intraosseous lesions.[24] Various microbial species have been identified in IPL, including *Porphyromonas gingivalis, Staphylococcus aureus, Corynebacterium, Prevotella intermedia, Klebsiella pneumoniae, Enterococcus faecalis,* and actinomycotic colonies, along with a range of anaerobic and facultative bacteria. Among these, *Porphyromonas gingivalis* is the most prominent species, while *Candida albicans* has been rarely detected.[24,29] Therefore, microbiologic findings should be considered for antibiotic selection.[24]

Treatment modalities for IPL include antibiotic administration, degranulation with or without apical implant resection, implant surface treatment with or without chemical detoxification, and bone grafts with or without a barrier membrane.[11,31-33] In a retrospective study, Di Murro et al.[19] reported that the cumulative survival rate of IPL cases has been reported to fall between 67.5% and 97.4%**,** having a 72-month to 4.54-year mean follow-up period.

This study aimed to present two cases of IPL diagnosed during the early and late phases of osseointegration, review potential aetiologies, and retrospectively assess the effectiveness of two different treatment approaches.

Materials and Methods

This publication was prepared as a narrative review and case report. A literature search was conducted by the authors. The PubMed database was used for manuscript preparation. The authors evaluated the literature selected for this publication, and appropriate original articles were included. Ethics committee approval is not necessary for this study. Patients' informed consent was obtained in the case report before the procedures.

Case 1:

A systemically healthy 49-year-old female patient who smoked 25 cigarettes a day was admitted to the clinic because of missing teeth in the left maxillary region. The patient was diagnosed with generalised chronic periodontitis on a clinical and radiographic examination. After the non-surgical periodontal treatment of the patient was completed, CBCT was taken at the implant site, and the surrounding tissues were evaluated by an experienced periodontologist and radiologist (M.M.A.- M.H.K.) (Figure 1). Due to the radiolucent appearance in the apical part of tooth number 25, a vitality test was performed, and the tooth was determined to be vital. It was decided to extract tooth number 25 due to low bone support and grade 3 mobility. In the same visit, an immediate implant was placed in tooth number 25 and a delayed implant was placed in tooth number 27. Informed consent was obtained from the patient before surgeries. Implants (Implance, AGS Medical, Turkey) were placed in the relevant areas by an experienced physician (M.M.A.). Sixteen days after the implant surgery, the patient presented to the clinic with severe pain and swelling. Radiographs showed a peri-apical lesion (Figure 2), and antibiotics (amoxicillin + clavulanic acid 1000 mg, 2x1, 7 days), a non-steroidal anti-inflammatory drug (dexketoprofen 25 mg, 2x1), and an antiseptic rinse (120 mg 0.12% chlorhexidine gluconate + 150 mg 0.15% benzidamine hydrochloride, 2x1, 14 days) were prescribed. Medication was started preoperatively and continued postoperatively. The area was surgically reopened. A defect was observed at the implant's apical region (Figure 3). The granulation tissue in the defect area was completely excised with titanium curettes and irrigated with sterile serum physiologic solution. Graft and membrane applications were not performed due to the patient's financial constraints. After creating bleeding points to increase blood supply in the area, the flap was primarily closed. Patients were advised to apply cold compresses for 24 hours after surgery to reduce swelling, to maintain a relatively soft diet, and to avoid the use of tobacco products. The sutures were removed a week later. The patient was followed up every 6 months. Patients did not perform any procedure during the follow-up period. After six months, peri-apical radiographs of the relevant area were obtained for prosthetic loading (Figures 4-5). The results were evaluated by a radiologist and showed significant bone filling with re-trabeculation in the defect area.

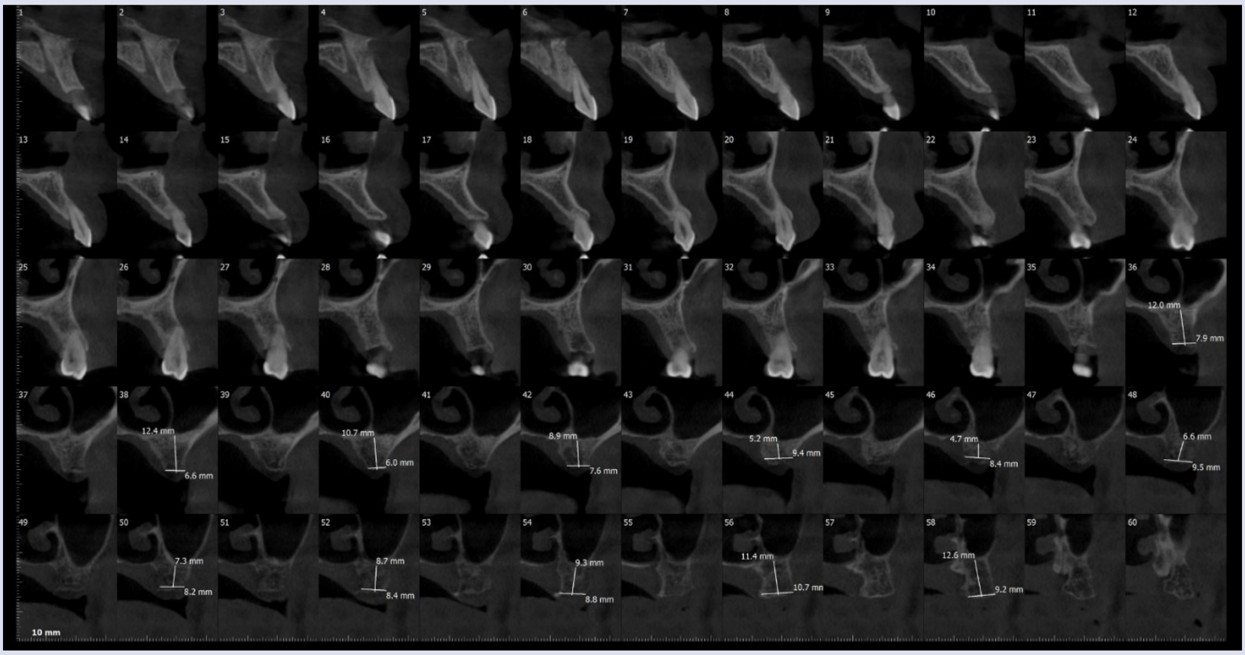

Figure 1. CBCT image of the area before surgery.

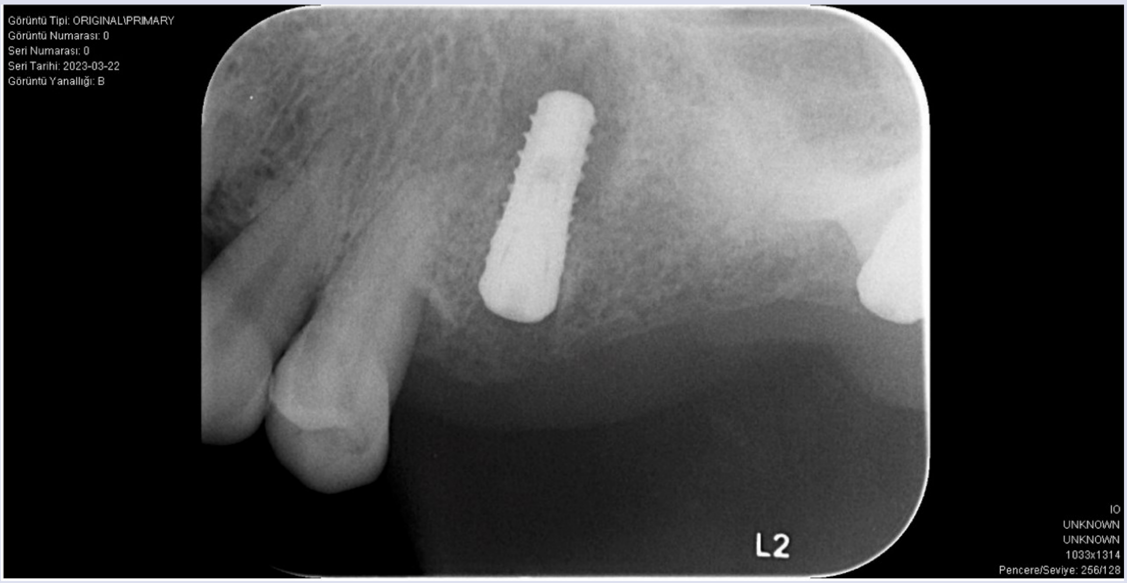

Figure 2. 16th day after implant surgery.

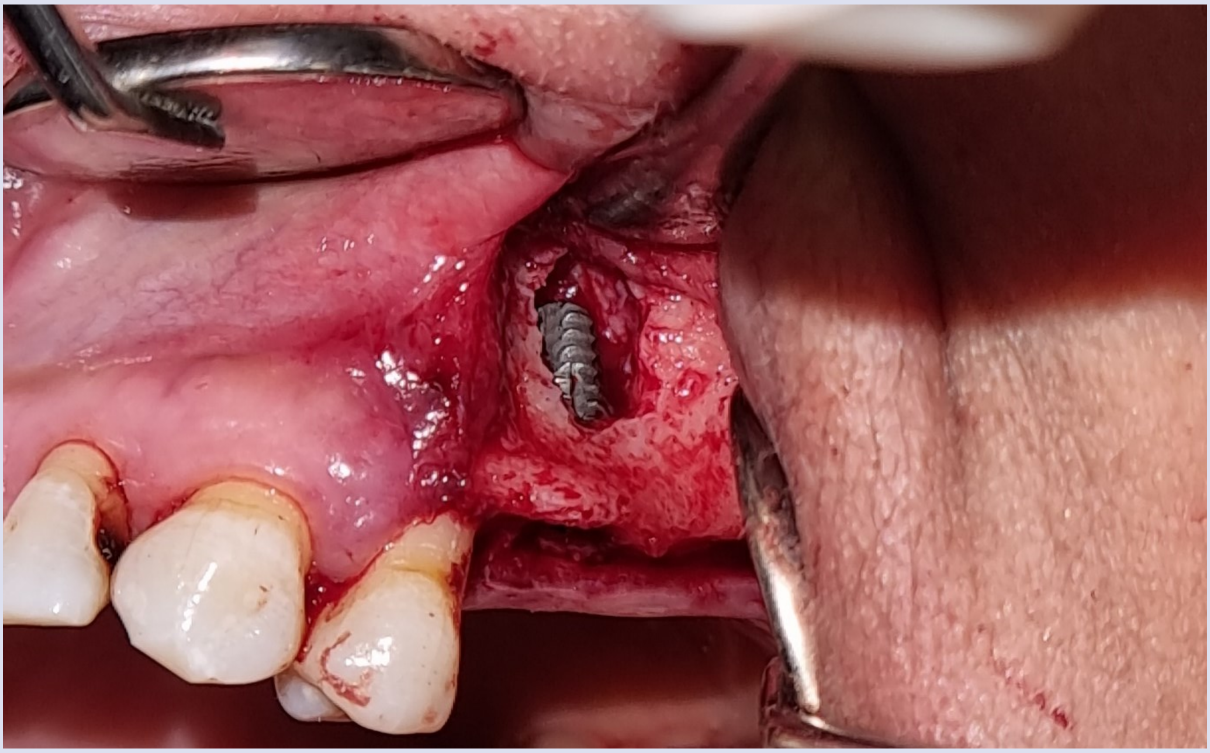

Figure 3. Surgical opening of the region and view of the defect area.

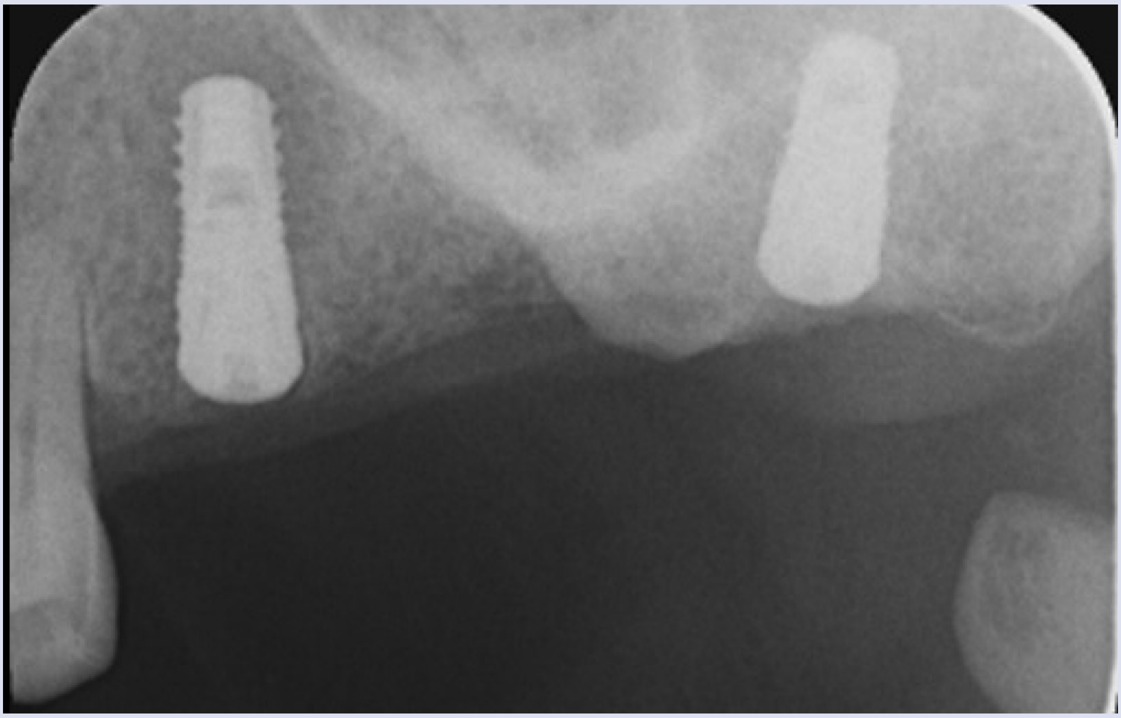

Figure 4. Peri-apical radiographic images 6 months after surgery.

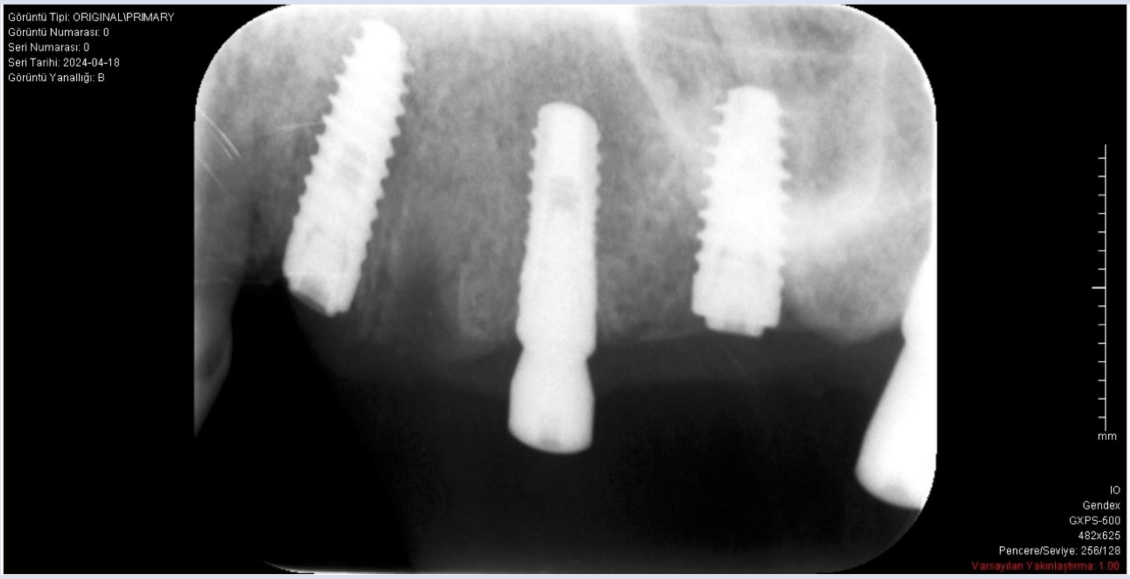

Figure 5. Peri-apical radiographic images a year after the surgery.

Case 2:

A systemically healthy 69-year-old woman presented to the clinic with one missing tooth, number 16. Non-surgical periodontal treatment of the patient was completed. The relevant area and surrounding tissues were evaluated with a panoramic radiograph (Figure 6). No conditions were found that would contraindicate the implant application. An implant (TRI Dental Implants Int AG, Switzerland) was placed in the relevant area (M.M.A.). The patient presented to the clinic with severe pain and swelling 7 months after implant surgery. Radiographs revealed a radiolucent lesion affecting the peri-apical region of the implant and the adjacent tooth number 15 (Figure 7). Antibiotics (amoxicillin + clavulanic acid 1000 mg, 2x1, 7 days), a non-steroidal anti-inflammatory drug (dexketoprofen 25 mg), and an antiseptic rinse (120 mg 0.12% chlorhexidine gluconate + 150 mg 0.15% benzydamine hydrochloride, 2x1, 14 days) were prescribed. Medication was started preoperatively and continued postoperatively. Tooth number 15 underwent an endodontic treatment. The patient's informed consent was obtained before surgery. The area was then surgically opened. A defect was observed in the implant's apical region. The granulation tissue in the defect area was completely removed with titanium curettes and irrigated with sterile saline solution. Tooth number 15 underwent apical resection, and an apical plug was created with retrograde filling material. Due to the large size of the defect area, the area was filled with deproteinised bovine bone graft (Cerabone, Botiss biomaterials GmbH, Zossen, Germany) and covered with a collagen membrane (Jason membrane, Botiss biomaterials GmbH, Zossen, Germany) (Figure 8). The site was primarily closed. Patients were advised to apply cold compresses for 24 hours after surgery to reduce swelling, to maintain a relatively soft diet, and to avoid the use of tobacco products. The sutures were removed a week later. Prosthetic loading was completed at the third month after surgery. No lesion was observed on radiographs taken at the sixth month, first year and seventh year (Figures 9-10). Patients did not perform any procedure during the follow-up period.

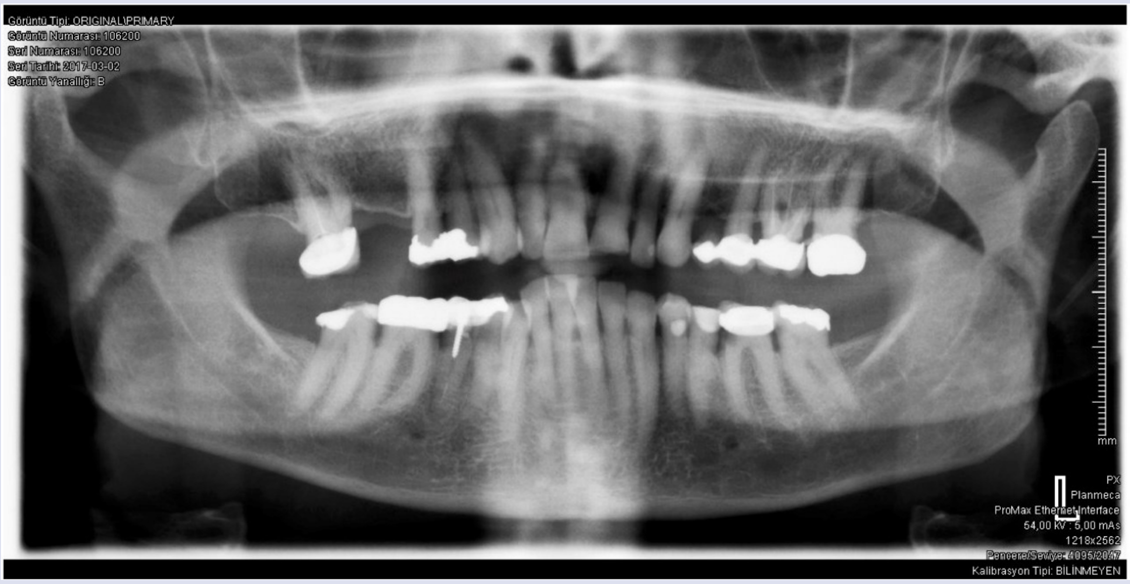

Figure 6. Panoramic image before surgery.

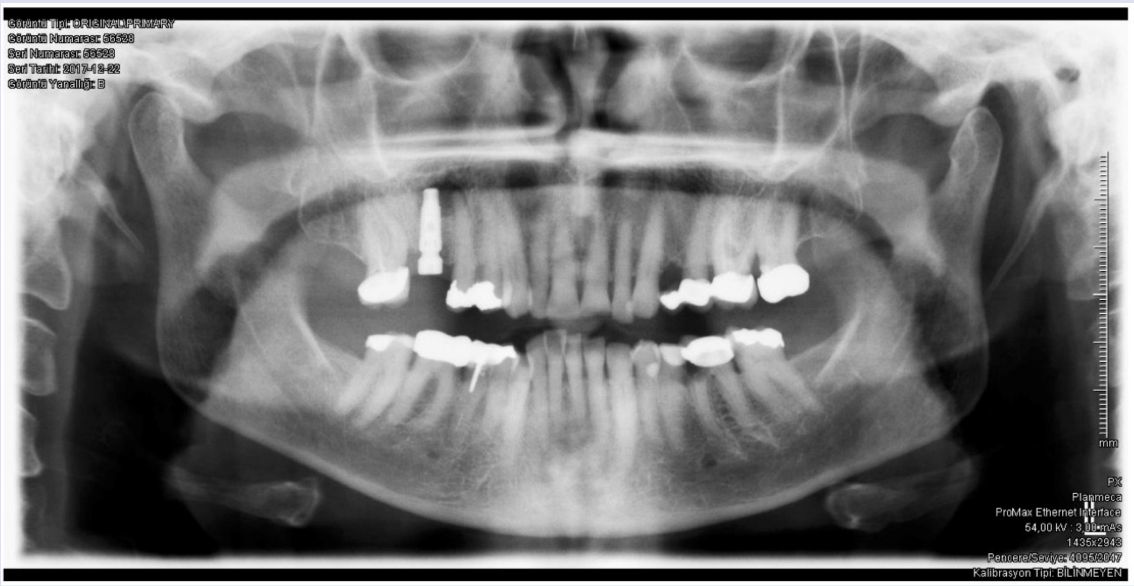

Figure 7. 7th month after implant surgery.

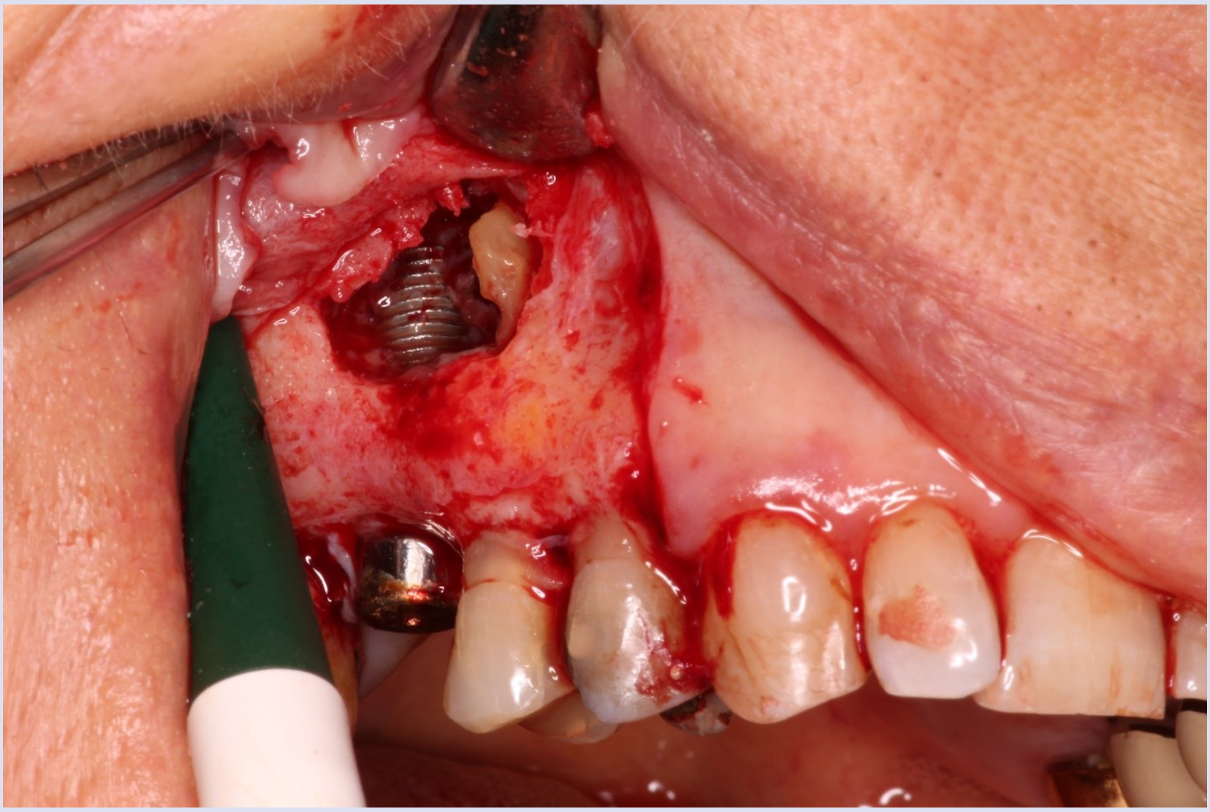

Figure 8.a. Opening the defect site.

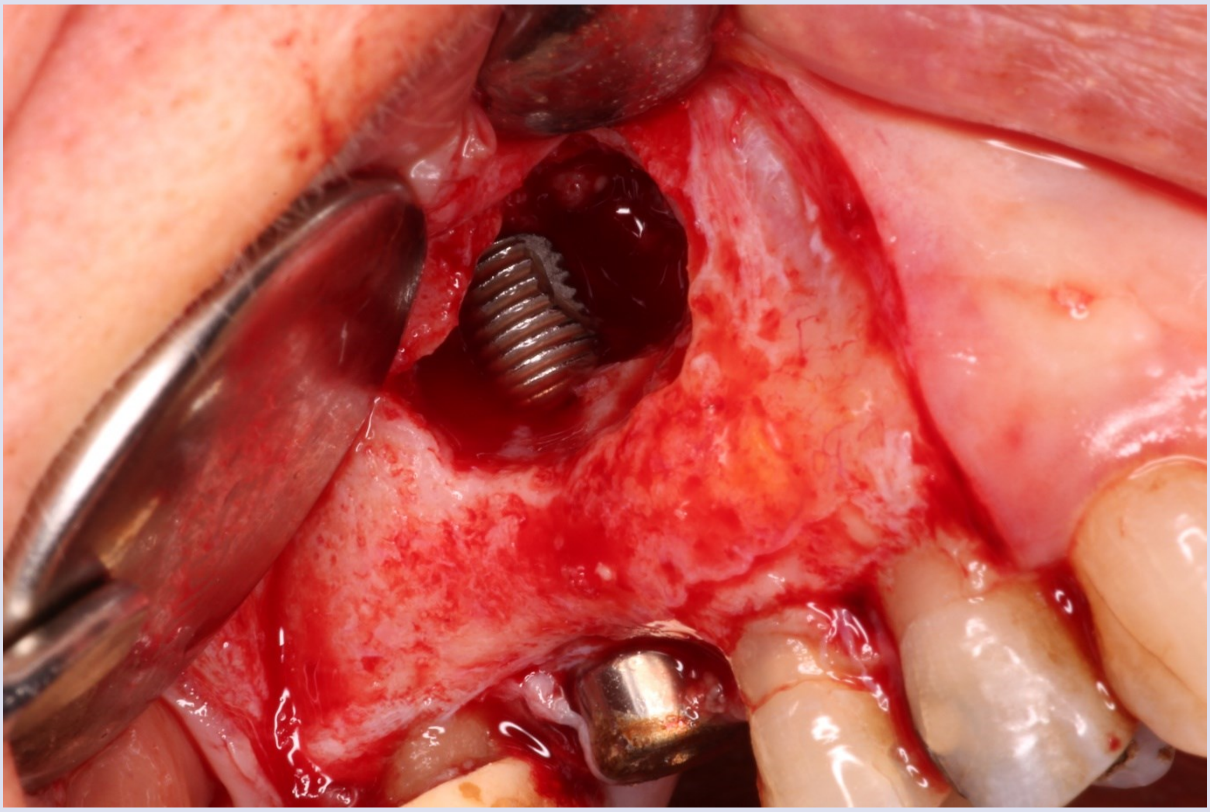

Figure 8.b. Removal of granulation tissues and apical resection.

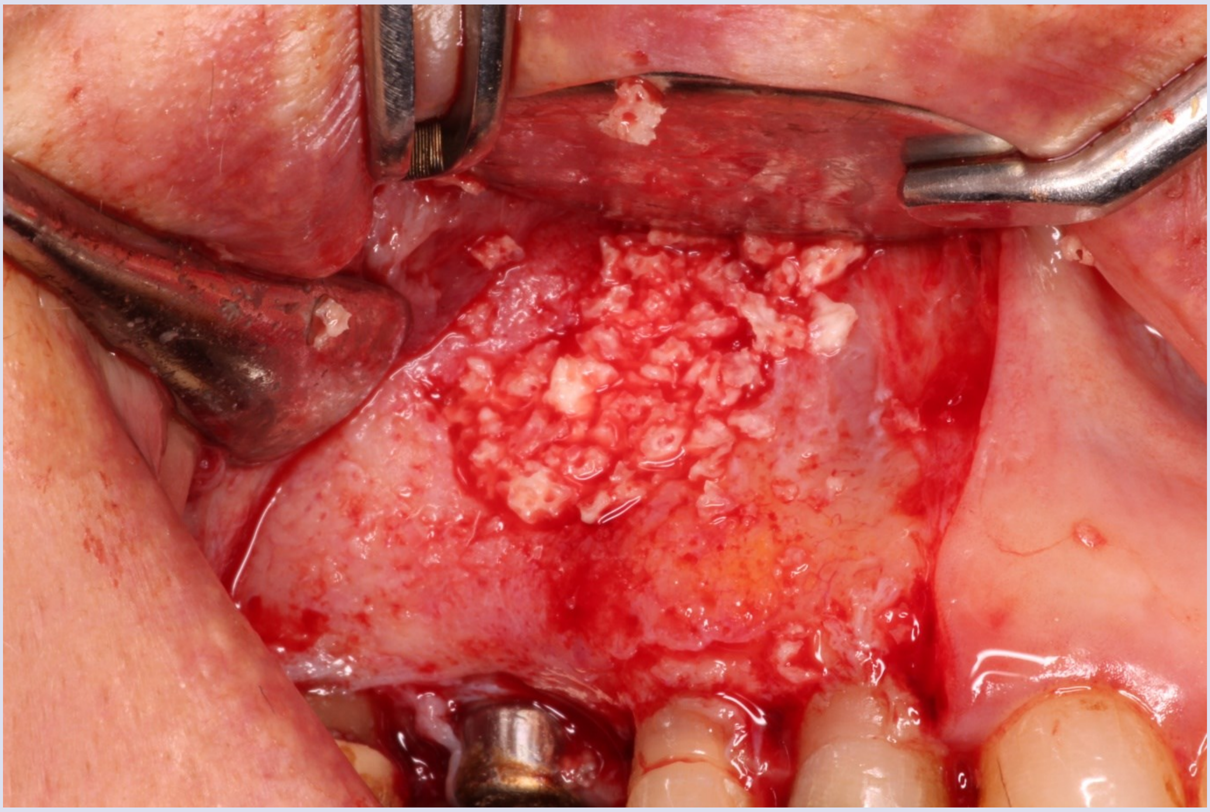

Figure 8.c. Filling the defect area with bone graft.

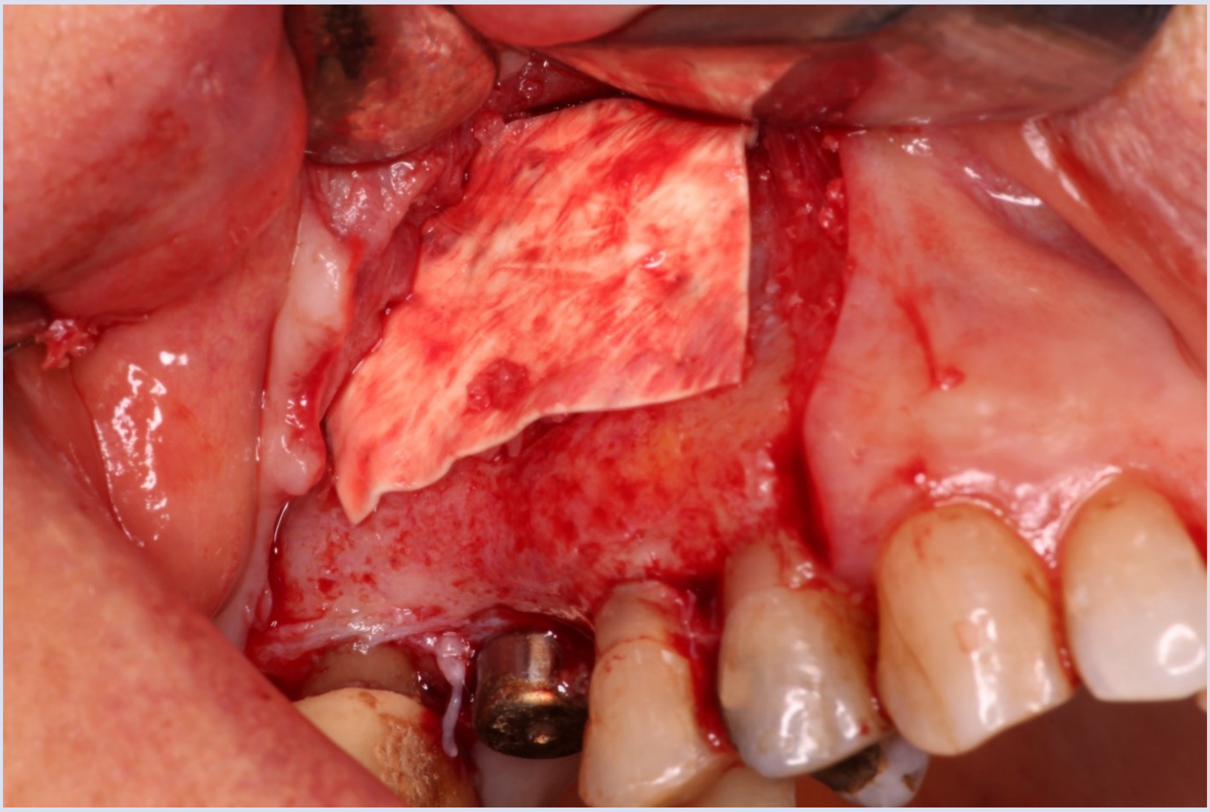

Figure 8.d. Covering the area with a collagen membrane.

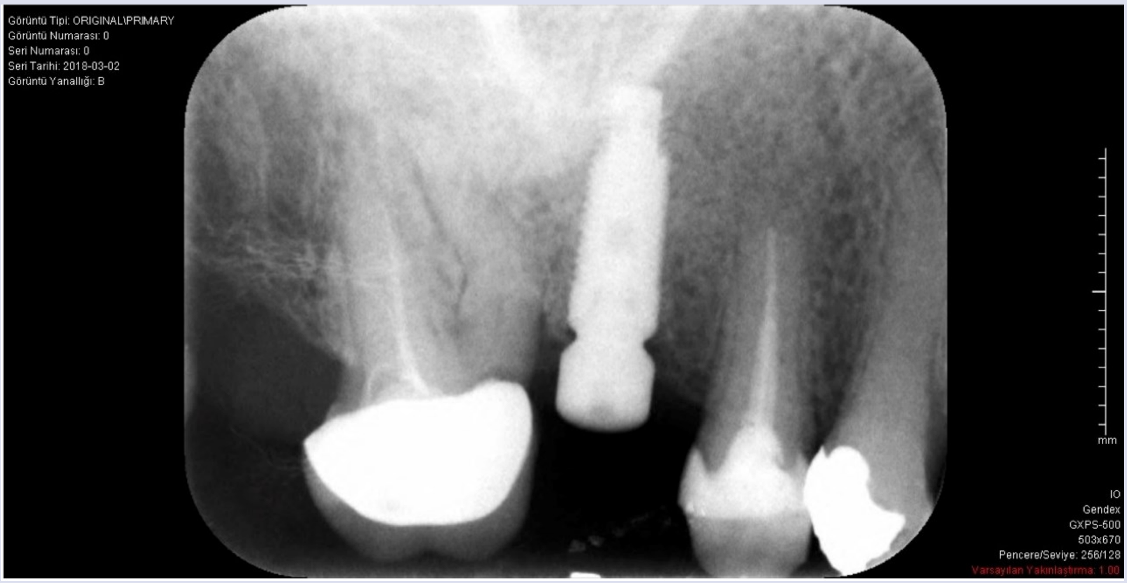

Figure 9. One year after surgery.

Figure 10. 7 years after surgery.

Discussion

Peri-apical lesions associated with implants are infrequent, with an incidence rate of roughly 10 occurrences per 3,800 implants, as observed by Reiser and Nevins.[28] These lesions may manifest 7 to 15 days post-surgery[33] or several months to years following implant placement.[35]

Palma-Carrió et al.[36] conducted a literature study on the factors of early dental implant failure. Their findings revealed that implant failure prior to loading ranged from 1.2% to 3%, but failure subsequent to loading varied from 0% to 1.8%. The study identified insufficient bone quantity and quality, smoking, and implant placement in posterior regions as statistically significant factors linked to early implant failure. However, only a few studies identified related risk factors.

In the first case presented, the peri-apical lesion occurred 16 days after immediate implant placement. When the preoperative CBCT images were examined, no anatomical conditions such as alveolar bone insufficiency, concavity, or abnormal bone density were found to prevent immediate implant placement. We hypothesise that the lesion may have been caused by implant bed contamination in the apical region during the procedure or the patient's continued smoking habit despite all warnings. In the second case, the lesion occurred 7 months later due to an endodontic problem in the adjacent tooth.

Several therapeutic alternatives have been presented in the literature. Waasdrop and Reynolds[37] proposed that asymptomatic peri-apical lesions associated with implants can be treated with antibiotic medication, circumventing the need for surgical intervention. Several published case series have indicated that antibiotic therapy alone was insufficient for controlling symptomatic or active lesions, necessitating surgical intervention.[32,38] Likewise, Romanos et al.[39] concluded in their review that therapy employing antibiotics alone is ineffective. At times, a small peri-apical radiolucency is incidentally detected during routine radiographic examinations. If the patient has no symptoms and the lesion is minor in size, treatment is generally unnecessary. Excessive preparation of the implant bed is often the main cause, and routine radiographic monitoring is advised instead. If the size of the radiolucency increases at any of the controls or if the patient develops symptoms, surgical treatment is required.[27]

Exposing the implant apex, debriding the defect site, detoxifying the implant surface, and promoting bone regeneration are recommended by most authors.[10] The only agreed point in the applied treatment options is the elimination of infection.[18] However, accurate diagnosis in the early stages of lesions provides early treatment and prevents implant failure.[27] Zhou et al.[40] treated six cases of IPL with surgical debridement and found successful outcomes when the lesions developed soon after implant placement. Ayangco and Sheridan[41] used tetracycline for one minute to disinfect the area after performing peri-apical surgery and irrigating the implant. Similar studies have evaluated the use of various agents, such as chlorhexidine or tetracycline, for decontamination of the implant surface in addition to curettage of the area. But there was insufficient evidence for the efficacy of any of such agents in IPL treatment.[27] Surgical debridement of the peri-apical lesion and implant surface debridement was applied, to the first patient who presented with pain and swelling 16 days after implant surgery. The area was irrigated with sterile serum physiologic solution. Due to the small size of the defect area and economic constraints, no graft or barrier membrane was applied. After 6 months of follow-up, bone filling was observed in the defect area. The results of this case indicate that surgical treatment of the area and removal of granulation tissue without the use of regenerative material is a successful treatment option in IPL cases.

In the literature, bone graft materials with or without barrier membranes have also been used in the treatment of IPL cases to ensure complete bone regeneration of the defect.[11,42] Bretz et al.[43] successfully treated an IPL case using a demineralised bone graft and an absorbable collagen membrane barrier, in addition to peri-apical surgery, curettage and chlorhexidine irrigation. Kutlu et al.[44] reported a case in which surgical debridement combined with guided bone regeneration was attempted for treating an IPL; however, the lesion did not heal. In the second case, granulation tissues were excised in the defect area, and the area was treated with regenerative procedures. Post-treatment controls showed that the area was filled with bones, and the condition remained stable during the 7-year follow-up period. The guided bone regeneration methodology is emphasised in the literature to restore the lost tissue and/or bone after controlling the infection. Nevertheless, the results from current studies do not clarify if genuine regeneration was accomplished or only repair occurred, as there is a lack of histological information from successfully treated IPL patients.

Although different treatment procedures were applied, implant surface debridement was performed following the removal of granulation tissue in both cases. Various instruments, such as curettes, ultrasonic devices, and air-abrasive systems, have been proposed for this purpose. Among them, air-abrasive systems have been shown to provide more effective debridement.[45] However, titanium curettes were preferred in both cases due to their widespread use and availability in clinical practice.

In contrast to the discussed treatment methods, Sussman advocates for the immediate removal of the implant to avert osteomyelitis and irreparable bone loss.[46] Similarly, Scarano et al.[23] and Piatelli et al.[47,48] recommended implant removal resulting in pain relief. Reiser and Nevins[28] and Oh et al.[49] proposed that, based on the extent of the lesion or the level of implant mobility, the infected tissue should be resected through apical resection of the implant or by complete removal of the implant. For a successful implant apicoectomy, it is essential to maintain sufficient length post-resection and to keep the implant unloaded for a minimum of six months; this treatment is linked to elevated success rates.[32,35,50] In both cases, implants were not removed because osseointegration was achieved, and no implant resection was performed to preserve the surface area in contact with the bone.

Conclucions

IPL is classified as acute and subacute according to the stages of evolution. If implant stability is preserved during the acute and subacute stages, the appropriate treatment option is implant peri-apical surgery.[27] The objective of surgery extends beyond merely halting the infection. The surgical approach should involve exposing the defect as conservatively as possible while ensuring adequate access, debriding the defect, detoxifying the implant surface, and performing guided bone regeneration if re-osseointegration is available.[39] However, if implant mobility is present in the subacute stage, implant's removal is necessary.[27] Although the reviewed articles report a high survival rate following IPL treatment, the number of cases documented in the literature remains limited. Moreover, other variables may have affected post-treatment results, including the type and implant surface dimensions and the defect chronicity in the referenced studies. Additional standardised research and data are essential to develop more reliable methods in the prevention and treatment of implants with IPL.

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