

Etiology and treatment of periapical lesions around dental implants

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During the last 30 years, the osseointegration principle has been proven by solid experimental and clinical data. This has led to increased popularity of the use of endosseous implants for the replacement of missing teeth and a patient's oral rehabilitation (1–3). However, their widespread use in recent years has resulted in different types of complications. Even with the most stringent rules for sterility (48), optimal surgical planning and careful patient selection/preparation, still some implants fail to integrate. The implant periapical lesion is a complication that may occur following implant placement, and case reports have suggested that such lesions are possible causes for early failure of an endosseous implant (6, 10, 20). The implant periapical lesion is also termed 'apical peri-implantitis' or 'retrograde peri-implantitis' (32). It is defined as a clinically symptomatic periapical lesion (and therefore diagnosed radiologically as a radiolucency on an intra-oral radiograph) that normally develops shortly after placement of the implant. The coronal portion of the implant often achieves a normal bone-to-implant interface (25, 32). The first cases were described by McAllister et al. in 1992 (26) and soon thereafter, in 1993, by Sussman & Moss (41). The purpose of this review is to give an overview of the literature on the etiology of these lesions and their prevalence and diagnosis, in order to understand the pathology and to suggest strategies for prevention and treatment.

Etiology

Different etiological factors play an important role in the development and emergence of a periapical implant lesion. 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 (26, 34). 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 (26, 34). 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. This can eventually compromise the primary stability of the dental implant. Uncontrolled thermal injury can result in the development of fibrous tissue, interpositioned at the implant–bone interface, compromising the long-term prognosis of the implant.

An 'active' periapical implant lesion can be caused by bacterial contamination (49) during insertion or by premature loading leading to bone microfractures before an adequate bone-to-implant interface has been established. 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. Furthermore, retrograde peri-implantitis should not be mistaken for nonintegration. When, during placement, the apex of an implant touches the tooth and/or when the implant is

inserted in an active endodontic lesion from an adjacent tooth, the implant may even exfoliate. Sussman & Moss (41) and Sussman (46) described two basic pathways of periapical implant pathology: type 1, the implant-to-tooth pathway; and type 2, the tooth-to-implant pathway. In the type 1 pathway, the implant-to-tooth lesion will develop when implant insertion results in tooth devitalization (as a result of direct trauma or indirect damage). This may occur when the implant is placed at an insufficient distance from a neighboring natural tooth or by overheating the bone during osteotomy preparation. When the osteotomy causes direct trauma to the apex of a natural tooth, it might destroy the blood supply to the pulp. The resulting periapical tooth lesion will contaminate the implant (11, 41, 44, 45). In the type 2 pathway, the lesion will occur quite shortly after implant insertion when an adjacent tooth develops a periapical pathology (caries involvement, external root resorption (16, 43), reactivation of a previously existing apical lesion or the removal of an endodontic seal (42)).

The majority of authors consider an endodontic pathology of the natural tooth at the site of the implant (or an adjacent tooth) to be the most likely cause for periapical implant pathology (33, 42). Ayangco & Sheridan (6) published a series of case reports in which oral implants were placed in sites where previous tooth root apical surgery failure had occurred. They reported that, even with thorough curettage of the sockets and a prolonged healing time before implant placement, bacteria have the capacity to remain in the bone and may cause lesions on the subsequent inserted implants. Brisman et al. (12) reported that even asymptomatic endodontically treated teeth with a normal periapical radiographic appearance could be the cause of an implant failure. They also suggested that microorganisms may persist, even though the endodontic treatment is considered radiographically successful, because of inadequate obturation or an incomplete seal.

Lefever et al. examined the periapical status of the extracted tooth at the implant site and the adjacent teeth before implant placement (24). The periapical status of the tooth at the implant site and the adjacent teeth before extraction was explored and identified as: no endodontic treatment and no periapical lesion; a periapical lesion at the root combined with or without an endodontic treatment; and an endodontic treatment without clear signs of a periapical lesion. If the extracted tooth showed no signs of a periapical lesion and had no endodontic treatment, the incidence of apical pathology was 2.1%. On the other hand, if an endodontic treatment or a periapi-

cal lesion at the apex of the tooth was present at the moment of extraction, a periapical lesion could be found around the implant in 8.2% and 13.6% of the cases, respectively. For teeth extracted without endodontic pathology, the adjacent teeth might have an effect on the future implant. If the adjacent teeth, on either the mesial or the distal side, showed no signs of pathology or did not receive endodontic treatment, only 1.2% of the implants presented with a periapical lesion. When an endodontic treatment had been performed previously, but no signs of periapical pathology could be detected, no periapical lesions around the implants could be found. However, if there were signs of periapical pathology at the neighboring teeth, 25% of the implants also showed a periapical lesion (24).

When the proper protocols are followed it has been shown that immediate implant placement into fresh extraction sockets is a valuable treatment strategy (15, 19, 22). The most clinically predictable method of addressing the immediate placement of implants in a fresh extraction socket containing apical pathology is obtaining access in conjunction with thorough debridement of all pathologic tissues. Adjunctive antibiotic therapy, both local and systemic, is highly recommended. Before the debridement of infected apical tissues, bacterial cultures should be obtained to inform the clinician if a specific antibiotic therapy is required. However, immediate implant placement in infected sites remains a topic of discussion. Several authors have considered immediate implant placement in infected sites as a contraindication (8, 9, 39, 47) because these sites may compromise an uneventful osseointegration (32) and may result in the development of an implant periapical lesion (6, 33). Alsaadi et al. (4) reported a greater tendency for implant failure in sites with apical pathology.

Crespi et al. (17) placed 15 implants immediately in periapical infected sites and 15 in noninfected sites. At the 12-month follow-up they recorded no difference in integration, hard and soft tissue conditions at both types of sites. Lindeboom et al. (25) placed 50 implants in chronic periapical infected sites. Twenty-five were placed immediately after extraction and 25 were placed after a mean healing period of 3 months. They reported a survival rate of 92% for the immediately placed implants compared with 100% in the control group. Furthermore, mean implant stability quotient values, gingival esthetics, radiographic bone loss and microbiological characteristics were not significantly different. They concluded that immediate placement of implants into chronically infected periapical sites may be a valuable treatment option. Fu-

gazzotto (21) conducted a retrospective analysis of 418 immediately placed implants in sites showing periapical pathology. The cumulative survival rates in this study were similar to those for immediately placed implants in sites showing no periapical pathology. In a recent study by Jung et al. (23), the immediate placement of implants in sites with periapical pathology was compared with immediate placement in healthy sites. The implants were followed during a 5-year period. They concluded that the replacement of teeth exhibiting periapical pathologies with implants placed immediately after tooth extraction can be a successful treatment modality with no disadvantages in clinical, esthetic and radiologic parameters compared with immediate placement of implants in healthy sockets.

Even though most authors consider a microbiological factor important in the pathogenesis of an active periapical implant lesion, convincing data remain very scarce. Romanos et al. (36) conducted a histologic investigation of 32 implants with periapical infection. Bacteria were found in only one case. Chan et al. (13) reported the presence of *Eikenella corrodens* in a surgically treated periapical lesion. Lefever et al. (24) took a microbial sample of 21 periapical implant lesions at the moment of treatment. These samples were analysed for *Enterococcus* species, *Aggregatibacter actinomycetemcomitans*, *Campylobacter rectus*, *Fusobacterium nucleatum*, *Prevotella intermedia* and *Porphyromonas gingivalis*. Also, the total counts of aerobic and anaerobic bacteria were analyzed. Bacteria were found in nearly all sites, but only at a concentration of $\geq \log 4$ in nine of 21 sites. The proportion of anaerobic species was always higher than that of aerobic species. *Porphyromonas gingivalis* and *P. intermedia* were detected in reasonable concentrations at six and four sites, respectively. The other specific species (*A. actinomycetemcomitans*, *C. rectus*, *F. nucleatum* and *Enterococcus* species) never reached the threshold level for identification. Further factors related to implant periapical pathology are: the presence of residual root fragments or foreign bodies (31, 34); placement of an oral implant in the proximity of an infected maxillary sinus (38); placement of the implant in the nasal cavity (40); and excessive tightening of the implant during insertion causing compression of the bone (38), although there is no way of ascertaining the degree of compression at the apex of the implant, even if insertion torques are higher than normal. The etiopathogenesis of an active periapical implant lesion remains controversial and it is believed to have a multifactorial pathogenesis (31, 37).

Diagnosis

The diagnosis of an implant periapical lesion is based on both clinical and radiological findings. As mentioned above, these lesions are classified into two groups: the inactive and the active forms. The inactive lesions are asymptomatic and are radiologically found as a result of the presence of radiolucency around the apex of the implant. These lesions do not need further treatment, although they should be followed radiologically on a regular basis, as an increase in size of the radiolucency may indicate activation and the need for further treatment. Active lesions are frequently (but not necessarily) clinically symptomatic. Clinical findings may comprise constant and intense pain (persisting even after analgesic treatment) (38), inflammation (27), dull percussion, the presence of a fistulous tract (6, 31) and the presence of mobility. If pain is present, this will not increase after implant percussion because the bone-implant interface is direct. Because there is no pressure to create a fistulous tract, and purulent materials can emerge through the still not fully consolidated interface between implant and bone, this clinical finding is not always present (28). According to Peñarrocha-Diago et al. (30), the diagnosis must include determination of the evolution stage of the lesion in order to apply the best treatment strategy. The authors divide the evolution of the periapical implant lesion into three parts:

- Part 1. The nonsuppurated acute periapical implant lesion: an acute inflammatory infiltrate is detectable and clinically characterized by the presence of acute spontaneous and localized pain that does not increase with percussion. The mucosa can be swollen and sometimes painful. Percussion of the implant will produce a tympanic sound. Radiographically, there are no changes in bone density around the apex of the implant.
- Part 2. The suppurated acute periapical implant lesion: a purulent collection is formed around the apex of the implant. This collection will result in bone resorption as it searches for a pathway for drainage. When this pathway has been established, the next stage is reached. Clinical signs are comparable with the nonsuppurated stage. Radiographically, however, a radiolucency can be detected around the apex of the implant.
- Part 3. The suppurated-fistulized periapical implant lesion: when the bone-to-implant junction is established in the coronal part a fistulous tract can develop from the apex of the implant

through the cortex of the buccal plate. If the coronal junction is not established, the peri-implant bone will also be destroyed in a coronal direction and eventually this will lead to implant loss. Clinical signs are various. Radiographically, bone resorption around the implant can be seen.

Care should be taken because two-dimensional periapical radiographs do not always show the actual size of an intrabony defect. Only when the junctional area is involved, can these kinds of defects be identified. Therefore, it might be possible that some periapical pathologies are not recognized on two-dimensional radiographs, which is a limiting factor in the diagnosis (48). Cone beam computed tomography can be used to overcome this limitation.

Prevalance

The prevalence of periapical implant pathology is fortunately rather low. Quirynen et al. (33) reported a prevalence of periapical implant pathology of 1.6% in the maxilla and 2.7% in the mandible in a retrospective study on 539 implants.

On a study of 3800 implants, Reiser & Nevins (34) found a prevalence of periapical implant pathology of 0.3%. According to a recent study by Lefever et al. (24) a periapical lesion around the implant can be detected in 8.2% of implants, which increases up to 13.6% if an endodontic treatment or a periapical lesion at the apex of a previously extracted tooth is present. For periapical pathology at the adjacent teeth, the percentage rises to 25%.

Treatment

Penarrocha-Diago et al. (30) and Zhou et al. (51) consider the correct and an early diagnosis of periapical implant lesions as a prerequisite for the prevention of implant failure. As periapical lesions around dental implants are considered to have a multifactorial etiology, there is no consensus regarding the therapy. A search of the literature leads to predominantly case reports of possible treatment options.

In some case reports, nonsurgical treatments are discussed. Chang et al. (14) 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. Waasdrop & Reynolds (50) also treat-

ed 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 (18, 34). So the question arises of whether the healing of periapical implant lesions in the previously mentioned studies was caused by treatment with the prescribed drugs or whether these lesions were inactive.

In order to prevent osteomyelitis, and because retaining the implant can lead to further and irreversible bone loss, some authors advised an early explantation of the infected implant(s) (5, 35, 38, 40, 45). However, most authors agree that the apex of the implant should be surgically exposed. How the therapy should be continued after exposure remains a topic of discussion. Reiser & Nevins (34) and Oh et al. (27) propose elimination of the infection and an implant apical resection or implant removal, depending on the extent of the infection and the stability of the implant. Zhou et al. (51) treated six implants in six patients, showing periapical pathology through trepanation and curettage (without resection of the apical part and the use of a bone-substitute material). The lesions were copiously irrigated with saline and chlorhexidine solution and the residual bone defects were further treated with tetracycline paste. Radiographically, the lesion disappeared and the implants were normally loaded after 3 months. The irrigation agents used most often for decontamination of the implant surface are saline solution (6, 29, 33), chlorhexidine solution (5, 13, 29) or tetracycline pastes (29). Whether any of these agents are efficient in decontaminating the implant surface remains questionable.

Some authors report on the use of bone substitutes, with or without the use of membranes, to achieve a complete resolution of the bony defect. Quirynen et al. (33) performed treatment on 10 cases with periapical implant pathology (out of a total of 426 solitary implants). The protocol for the treatment (Fig. 1) 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

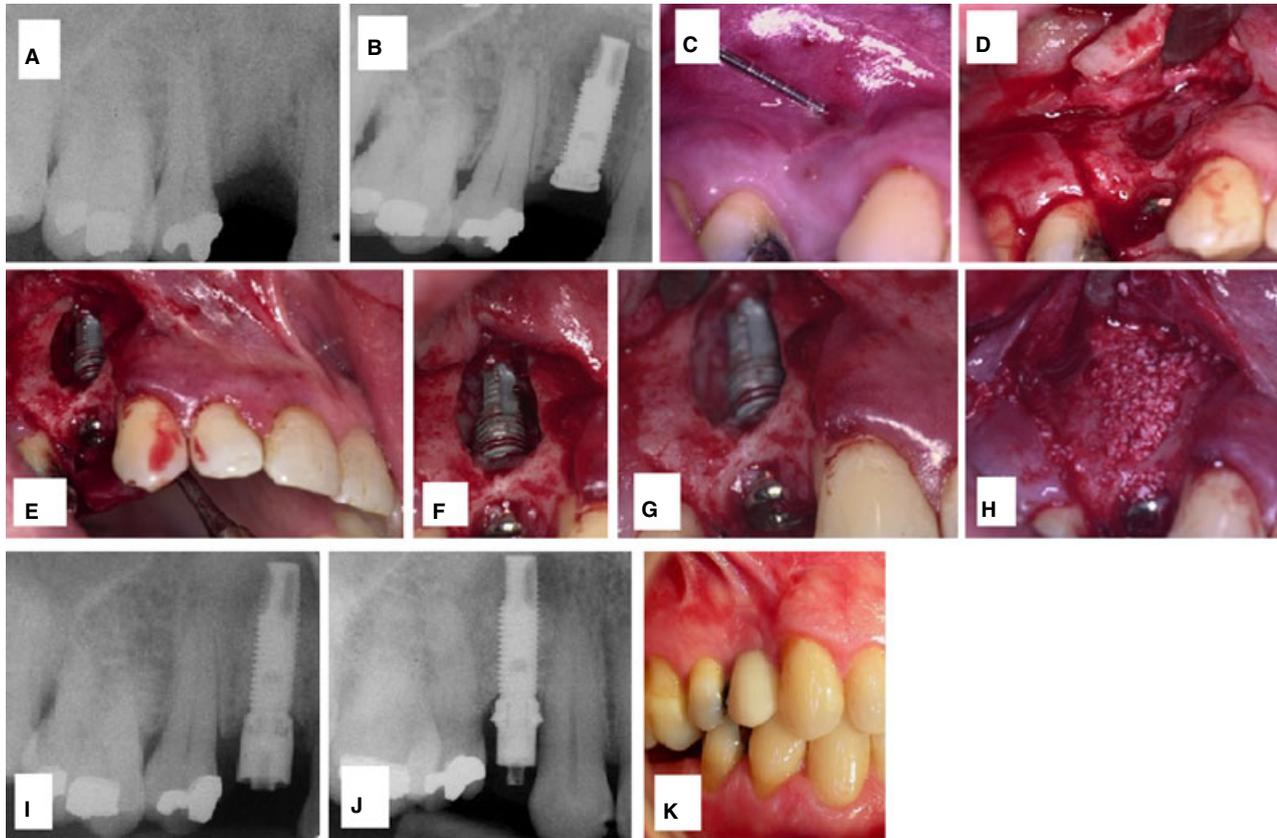


Fig. 1. (A) intra-oral periapical radiograph, 3 months postextraction. (B) Occurrence of a periapical implant lesion, 3 months after installation. (C) Presence of a fistulous tract. (D) Elevation of a full-thickness mucoperiosteal flap. (E–G) Defect inspection, curettage and irrigation with

sterile saline. (H) Bone substitute placement and guided bone regeneration. (I) intra-oral periapical radiograph, 4 months post-treatment and abutment connection. (J,K) Situation 1 year postloading.

all granulation tissue is sufficient to arrest the progression of bone destruction. Furthermore, implants in which only the coronal part is osseointegrated can successfully resist occlusal load for years, at least in the single-tooth replacement condition. Bretz et al. (10) surgically treated one case of periapical implant pathology via the elevation of a full-thickness flap, curettage of the apical lesion, irrigation with chlorhexidine gluconate, placement of demineralized freeze-dried bone and coverage of the site with an resorbable collagen membrane. The lesion was resolved and the prosthesis was still in function after 17 months of follow-up. Lefever et al. (24) retrospectively analysed 59 implants with periapical lesions. Different treatment options were chosen: explantation of the affected implant (17/59); curettage of the defect and application of a bone substitute (14/59); curettage and administration of systemic antibiotics (10/59); curettage only (11/59); no treatment (two of 59); systemic antibiotics without curettage (two of 59); curettage with the usage of a barrier membrane without application of a bone substitute (two of 59); and curettage and application of autogenous bone

chips (one of 59). Of the 42 nonexplanted implants, nine were lost during follow-up, all during the first 4 years of loading. The cumulative survival rate for an implant showing a periapical lesion was 46.0%. When the explanted implants were excluded, the cumulative survival rate reached 73.2% after 10 years. The authors conclude that a clear-cut selection of 'best treatment strategy' could not be identified. Balshi et al. (7) suggested apical resection of the affected implants. They used this approach on 39 cases. After flap elevation, they used a high-speed drill to create a bony window that was slightly larger than the lesion itself. The bony defect was thoroughly debrided and irrigated with a saline/tetracycline solution (Fig. 2). In 72% of the cases a deproteinized bovine bone mineral was used to graft the bony defect. In 38% a resorbable collagen membrane was used to cover the surgical site and to fulfil the guided bone-regeneration principle. A total of 39 implants in 35 patients were treated using this intra-oral apical resection procedure. Thirty-eight (97.4%) of the 39 implants remained stable and continued in function with no further complication up to 5 years. This suggests that

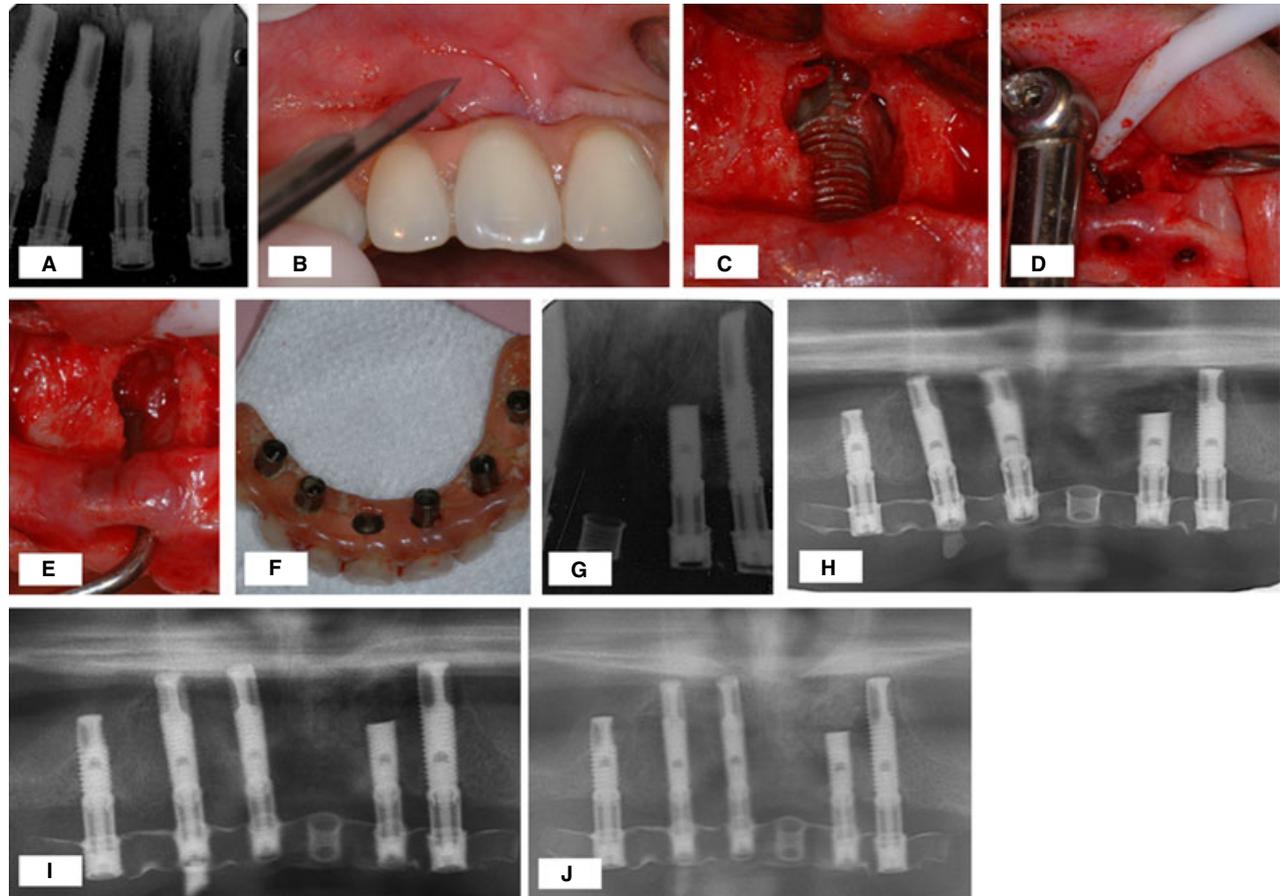


Fig. 2. (A) intra-oral periapical radiograph, 3 months after placement and immediate loading. (B–E) Removal of implant 21 and apicoectomy of implant 23. (F) Postoperative adjustment of the prosthesis. (G) intra-oral periapical radiograph, postoperatively. (H–J) Part of the panoramic radiograph, 2 (H), 4 (I) and 6 (J) years after removal and apicoectomy.

an apical resection procedure is preferable to a guided bone regeneration procedure.

Conclusion

Different factors have been proposed to play a role in the development and emergence of a periapical implant lesion. To date, there is no consensus about the exact etiology. Several factors might act together, pointing to a multicausality model. Diagnosis of an implant periapical lesion should be based on both clinical and radiological findings and, in order to apply the best treatment strategy, the evolution of the lesion should be taken into account. However, the treatment of this type of lesion is still empiric. Data, primarily derived from case reports, seem to indicate that the removal of all granulation tissue is a first step to arrest the progression of the bone destruction. The removal of the apical part of the implant seems a valuable treatment strategy.

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Abstract

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Abstract

The widespread use of oral implants in recent years has resulted in various types of complications. One of those complications is the periapical implant lesion. Different factors have been proposed to play a role in the development and emergence of a periapical implant lesion. To date, there is no consensus on the etiology and therefore periapical lesions around dental implants are considered to have a multifactorial etiology. The diagnosis of an implant periapical lesion should be based on both clinical and radiological findings. Additionally, in order to apply the best treatment strategy the evolution of the lesion should be taken into account. The treatment of this kind of lesion, however, is still empiric. Data, primarily from case reports, seem to indicate that the removal of all granulation tissue is a first step to arrest the progression of the bone destruction. The removal of the apical part of the implant seems a valuable treatment strategy.