Management of Apical Bone Loss Around a Mandibular Implant: A Case Report

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Various terms, etiologies, and treatment strategies have been suggested in conjunction with bone loss limited only to the apical portion of an implant that remains otherwise well osseointegrated. Proposed etiologic factors include bone overheating, microbial involvement of adjacent teeth, pre-existing bone infection, and overload. However, the mandible and maxilla seem to have different predispositions in response to these causative agents. Treatment protocols for peri-implant infection have included minimally invasive approaches such as granulation tissue removal and detoxification of the implant surface, as well as more aggressive measures. This case report demonstrates the achievement of osseous healing and reosseointegration in a patient who presented with presented apical bone loss and signs of infection around a mandibular implant. Reosseointegration was achieved following an intraoral apicoectomy-like approach, ie, removal of the infected nonintegrated portion of the implant, and meticulous debridement of the granulation tissue. A literature review of 13 relevant published studies was conducted. The current understandings regarding the etiology and treatment strategies for management of apical bone loss around dental implants are summarized and presented. (Case Report) INT J ORAL MAXILLOFAC IMPLANTS 2006;21:439–444

Key words: *apical bone loss, apicoectomy, bone-implant interface, dental implants, implant periapical lesion, osseointegration failure*

In general, bone loss around an implant has been recognized as a complication that can follow implant treatment.[1,2] While the first case in the literature demonstrating isolated apical bone loss was described by McAllister and colleagues[3] in 1992, it was Reiser and Nevins[4] in 1995 who first defined bone loss limited to the apical segment of an otherwise osseointegrated implant as an "implant periapical lesion" and further described the rationale for such an occurrence and possible treatment options. Sussman[5] further described periapical implant pathology and proposed 2 patterns of bone loss apical to implants. However, this report was limited to implants placed in partially edentulous jaws adjacent to natural teeth with a history of periapical dental pathology.

While the term "implant periapical lesion" appears often in the literature,[6–10] other terms for the same phenomenon such as "apical peri-implantitis,"[11] "retrograde peri-implantitis"[12–14] "abscess around the apex of an implant"[15,16] and "implant demonstrating periapical radiolucencies"[3] have also been identified in Medline searches of the English-language literature.

Reiser and Nevins[4] reported on 10 implant periapical lesions (9 infected and 1 asymptomatic) in a study sample of approximately 3,800 placed implants, suggesting a prevalence of 0.26%. This is the only value for prevalence of implant periapical lesions reported in the literature. Although the incidence of implants with apical bone loss is still unknown, the authors' literature search found 23 case reports in 13 studies. This suggests that they occur more frequently than initially thought.[4,9]

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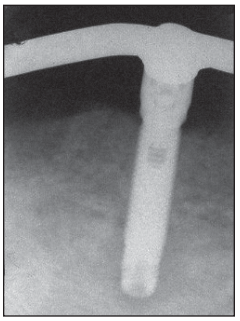

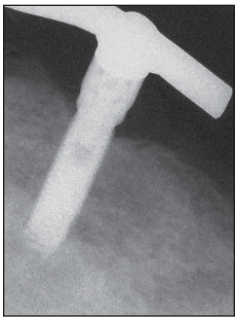

Fig 1 Periapical radiograph of right mandibular implant preoperatively, showing radiolucency around the apical third of the implant.

Fig 2 Postoperative radiograph 2 years following resection of the apical part of the implant.

Many etiologic factors have been suggested in previous studies.[4,7–9] However, the exact mechanism of bone loss in the apical area of an implant is still not well understood. It has not been possible to determine whether related lesions are composed of healthy tissue or created by the destruction of new tissue. It is also possible that such lesions may result from activation of a pre-existing condition.[4,6] The etiology is likely to be multifactorial.[17]

While observation and monitoring appears to be the preferred management option for small inactive lesions,[4] various treatment modalities have been suggested for infected lesions of larger diameter. Detoxification of the implant surface[3,12,13] and/or surgical treatment (an implant apicoectomy-type procedure following an extraoral[15] or an intraoral approach and placement of either a bone substitute with membrane coverage[4,9] or autogenous bone chips[18] in the bone defect) have been described.

The clinical management of apical bone loss around a mandibular implant using an intraoral apicoectomy-like surgical approach alone is presented. The results of a critical review of the literature on suggested etiologic factors and management options are also presented.

CASE REPORT

A 56-year-old male patient underwent stage-1 implant surgery at the Eastman Dental Hospital (London, UK) for the placement of implants to support an overdenture. Most mandibular teeth had been lost secondary to periodontal disease. The only remaining mandibular teeth were the left second premolar and first molar, which were to be extracted at implant placement. A panoramic radiograph showed no preexisting bone pathology. Two 3.75 × 18-mm Brånemark Mk III implants (Nobel Biocare, Göteborg, Sweden) were placed in the anterior interforaminal region of the mandible. A nonsubmerged protocol was followed, and two 3-mm healing abutments were connected to the implants before suturing. The patient was advised to keep his mandibular denture out for 2 weeks. The early postoperative period was uneventful.

Standard transmucosal abutments were attached at stage-2 surgery after 4 months. Following a standard prosthetic protocol, a mandibular denture supported by a gold bar with a small distal cantilever was inserted 9 months after implant placement. The unusual delay was caused by the patient's inability to attend the prosthetic appointments scheduled.

Six months after seating of the mandibular denture, the patient attended an emergency clinic complaining of discomfort around the right implant. He reported the initiation of pain 1 month after placement of the definitive prosthesis. On examination following removal of the gold bar, the right implant was found to be immobile. However, the soft tissues in the apical area appeared erythematous and slightly tender to palpation. The mucosa around the implant neck appeared healthy, and the probing depth was normal. A periapical radiograph showed a small radiolucent area around the apical third of the right implant (Fig 1). Marginal bone loss was stable at the first thread, which is consistent with previous studies on Brånemark System dental implants. Metronidazole was prescribed, and it was decided to explore the periapical lesion with resection of the apical portion of the implant.

The procedure was carried out under local anesthesia. A buccal incision exposed the area in the right mandible. No bone fenestration was found. A bony window was created over the apical area of the implant until the titanium implant could be seen. There was granulation tissue around the apical 4 mm of the implant, which was debrided. Under profuse sterile saline irrigation, the nonintegrated portion of the implant (4 mm) was trimmed using a tungsten carbide fissure bur. Hemostasis was achieved, and the wound was sutured to obtain primary closure. The patient was advised to avoid denture wear for 1 week and was prescribed metronidazole (400 mg 3 times a day for 7 days) and a chlorhexidine gluconate 0.12% mouthwash. No complaints were reported when the patient was examined 1 week later, and the tissues were found to be healing satisfactorily.

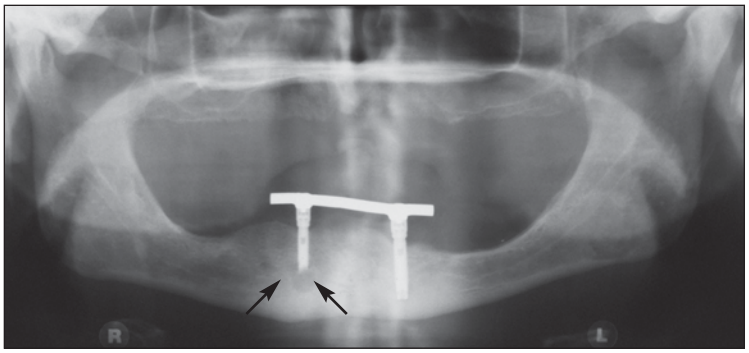

Fig 3 Osseointegrated normal and "apicected" implants rigidly connected with a gold bar. Two years later, the bone remodeling process is still ongoing *(arrows)* in the apical area of the right implant.

The patient was followed for 2 years during which time the implant and the surrounding tissue remained asymptomatic. There were no signs of adverse tissue reaction (Figs 2 and 3). There was no tenderness on palpation in the area, and the prosthesis has been stable and has functioned satisfactorily in the postoperative period.

DISCUSSION

An electronic literature search of English-language publications using "apical bone loss" and "dental implants" as key words identified 19 studies. Six studies in which periapical lesions from neighboring teeth and/or retained root tips had spread to involve the dental implants were excluded.[5,10,14,19–21] The remaining 13 studies[3,4,6–9,11–13,15,16,18,22] provided information on 23 cases of implants with periapical bone loss. It should be noted that although Reiser and Nevins[4] had reported on 10 implants with apical bone loss, details for only 5 of the implants were available in their study. However, data on implant site distribution were available for all 10 implants reported in that study[4] and are shown in parentheses in Table 1.

Maxillary implants were most frequently affected, with a ratio of almost 3:1. There was site variation in both jaws; however, predominant sites were the maxillary central incisor and first premolar areas. Interestingly, there were no reports of apical bone loss in molar sites or for completely edentulous maxillae. Occurrence of infected implant periapical lesions[4] and of radicular cysts of dental origin[23] has been reported more frequently in the maxilla.

Data available on implant length indicated that 14 of 23 implants were at least 12 mm long (one[22] of 12 mm, six[4,9,11–13] of 13 mm, five[7,9,13,16] of 15 mm and two[4,18] of 18 mm). The same implant length (≥ 12 mm) was estimated for the remaining 9 implants. This approximate implant length was clearly indicated by the radiographs, although numerical details were not available in the relevant studies.

Table 1 Distribution of Implant Sites with Apical Bone Loss

Jaw Distribution	No. of Implants
Maxilla	20 (6)
Central	5 (0)
Lateral	4 (0)
Canine	1 (1)
First premolar	9 (5)
Second premolar	1 (0)
Mandible	8 (4)
Incisor	3 (2)
Canine	2 (0)
First premolar	3 (2)

Data for 10 implant periapical lesions reported in a previous study[4] and included in the current evaluation are shown in parentheses.

Little information was available regarding the magnitude, direction, and timing of implant loading. The standard 2-stage protocol was almost always followed, with the implants receiving the definitive prostheses after appropriate healing times (range, 10 weeks to 9 months). While 9 implants were never loaded, 2 implant sites received provisional restorations immediately, 1 implant was helping support a fixed detachable prosthesis already, and no data were available for the rest of the implants.

Etiology

Although little is known on the etiopathogenesis[24] of early failures in achieving osseointegration, they should be viewed as a lack of osteogenic response in relation to endogenous factors (impaired healing) and/or exogenous factors (excessive trauma, infection, premature loading). Various etiologic factors for apical bone loss around implants have been suggested and are listed elsewhere.[14]

Microbial involvement either from remaining natural teeth adjacent to the implant[5,10,19–21] or preexisting bacterial contamination in the jaw bone[3,4,9,13] seem to be predominant factors of apical

bone loss around implants in partially edentulous maxillae. Bone overheating[25,26] caused by excessive force and insufficient cooling of drills might also result in necrosis of mesenchymal cells in the area[27] and in apical bone loss, particularly around implants placed in dense cortical bone in the anterior mandible.

A certain length is a common characteristic in these cases; the implant in the present study was 18 mm long before resection, and all the implants with apical bone loss identified in the literature were at least 12 mm. The longer the implant is, the higher the risk of overheating dense cortical bone. Local blood supply of this type of bone is poor in its deeper levels.[2,28] It has been also suggested that inactive implant periapical lesions, which resemble scars, could also be the end result of heat-induced aseptic bone necrosis[3,4,6] following bone overheating and/or implant placement.

Premature loading[3,4,15] during healing and/or overload[7] at the implant-bone interface have been considered responsible for inducing fibrous tissue encapsulation around the apical portion of the implant. However, it has been widely accepted that the initial breakdown of the implant-bone interface (late failure) starts in the coronal part of the implant, where loading forces are concentrated.[24] This process seems to occur independent of surgical approach (submerged versus nonsubmerged).[29]

Current knowledge[30,31] associated with cylindric threaded implants does not support bone loss limited to the apical portion of an implant that remains otherwise well osseointegrated. The authors are not aware of any study with documented apical bone loss around threaded implants related to overloading without significant concurrent marginal bone loss. Concomitantly, if unfavorable biomechanical distribution of occlusal loads was the rationale for bone loss around the implant apex, one would expect synchronous marginal bone loss around the implant neck as well. However, marginal bone loss was minimal and within the accepted range for the 18-mm Brånemark System implant used in the case presented, as well as for the rest of the implants with apical bone loss described in the literature.

Management

Elimination of the risk factors associated with implant periapical lesions can be achieved by using the proper surgical technique and adhering strictly to the surgical protocol.[2] Careful patient assessment and treatment planning are also necessary, especially when implants are placed adjacent to natural teeth with a history of pulpal, periradicular, and uncontrolled periodontal disease.

Reiser and Nevins[4] classified implant periapical lesions either as inactive or active, depending on the absence or presence of symptoms of infection, respectively. They hypothesized that residual bone cavities that remain after the placement of shorter implants in overdrilled osteotomy sites may repair with dense connective tissue instead of bone. They suggested that observing and monitoring seems the preferred management option for small inactive lesions.[4] Malo and coworkers[32] have shown radiographic resolution and healing of inactive periapical lesions around immediately loaded implants 3 months after initial presentation following a rigorous maintenance program and temporary prosthesis removal.

Surgical intervention is indicated only when *(1)* bone loss is limited to the apical area, *(2)* the implant remains osseointegrated, and *(3)* the implant is of sufficient length to allow removal of its apical portion while retaining support of a dental prosthesis.[4] Elimination of the source of infection by removal of the granulation tissue and cleaning/ removing the exposed apical implant part is essential to prevent further compromise of osseointegration while promoting bone regeneration and osseous healing/reosseointegration in the radiolucent area. In cases where elimination of the infection cannot be achieved, the implant should be removed.[4,6,7,8,14,16,22]

There are a variety of treatment alternatives, all of which have been shown to demonstrate radiographic resolution of the apical lesion but not necessarily reosseointegration. Many studies have described either implant surface detoxification by chemical means—eg, tetracycline paste,[13] tetracycline hydrochloride,[3] citric acid with ultrasonic instrumentation (particularly for hydroxyapatite-coated implants[3]), and chlorhexidine gluconate[12]—or placement of a calcium hydroxide paste in the bone defect instead.[11]

The achievement of complete resolution of the apical radiolucency by detoxification alone caused researchers to question the need for grafting and the use of barrier membranes.[13,24] However, it should be emphasized that most of the aforementioned implants had been placed in significantly resorbed maxillae with less-than-ideal bone around the implant body at primary surgery. Consequently, a guided bone regeneration technique[3,4,9,12] in the detoxified area was considered mandatory to restore the deficient bone volume.

Although partial[13] or complete resolution of periapical radiolucencies and elimination of symptoms was the end result, the quality of healing and the success of reosseointegration achieved with these treatment methods can also be questioned. Jalbout

and Tarnow[9] described a type of healing that included a fibrous soft tissue band between the exposed apical part of the implant and the bone substitute material following debridement of the granulation tissue and coverage of the exposed implant threads with a bone substitute material and a membrane only. They admitted that reossointegration may not have been achieved because of the difficulty of completely eliminating bacterial endotoxins from the implant surface.[9] It is likely that other cases in which conservative treatment without resection of the apical implant portion[3,11–13] was applied healed similarly. In such a case, however, the residual implant length would offer reduced apical bone anchorage for the implant, possibly resulting in less optimal support to the dental prosthesis. Therefore, it is likely that the initial treatment plan and/or superstructure design would need to be modified according to biomechanical principles.[33]

More aggressive treatment with an apicoectomy-like surgical approach has been reported in other studies. However, the treatment described in the present case can be performed through either an intraoral or an extraoral approach,[15] with synchronous placement in the apical bone defect of either autogenous bone chips[18] or a bone substitute and coverage with a membrane.[4,9] Balshi and associates[15] followed an extraoral surgical approach to treat an abscess formation and bone resorption in the incisor area of the inferior border of the mandible associated with a mandibular implant. In the present case, however, the inferior cortex of the mandible was intact, with no abscess or fistula formation; thus, an intraoral apicoectomy-like surgical procedure was followed. The patient remained asymptomatic, and the postoperative radiograph, taken 2 years after surgery, demonstrated complete osseous healing and re-established osseointegration.

CONCLUSION

Apical bone loss around implants is not common and may be avoided by a minimally traumatic surgical technique. Variations in bone density must be taken into account. Careful patient assessment and judicious treatment planning help to avoid placing implants in sites susceptible to microbial contamination from adjacent sites. Optimal treatment for symptomatic implants with apical bone loss necessitates surgical intervention and removal of the source of infection with an implant apicoectomy-like approach.

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