Implant Periapical Lesions: Etiology and Treatment Options

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Implant failures due to apical pathology are conditions that have not been extensively studied nor reported in the literature. The implant periapical lesion (IPAL) has different symptoms, and several etiologies have been proposed in the literature. This article reviews cases of IPAL reported in peer-reviewed journals and presents possible treatment options. Analysis of the data collected was performed based on diagnosis, cause of extraction of the natural tooth, location, period of implant placement, implant surface, and treatment approach. Even the data presented in this review are based on few reported cases the etiology of these lesions seems to be multifactorial or with an unknown origin. Contamination of the implant surface, bone overheating during surgery, excessive torquing of the implant, poor bone quality, perforation or thinning of the cortical bone, premature or excessive load over the fixture, fracture of the bone inside the hollow portion of the hollow implant, and an implant placement in an infected maxillary sinus have been discussed. In general, areas around endodontically compromised teeth should be carefully analyzed prior to implant placement to prevent implant failures.

Key Words: endodontic lesion, implant periapical lesions

INTRODUCTION

Endosseous oral implants, have demonstrated high survival rates with different treatment protocols, implant surfaces, implant design geometries, and diameters. When failures have been documented, possible causes have been proposed.

Implant failures are typically characterized by clinical and radiographic signs such as pain, paresthesia, mobility, peri-implant radiolucency, and excessive loss of alveolar bone. Such failures are traditionally categorized as failures due to infection (ie, peri-implantitis or retrograde peri-implantitis) or failures due to trauma (ie, excessive overloading or implant fracture). When mobility is present, the implant is considered a failure. However, when mobility is absent and there are signs of periapical pathology, such as bleeding, suppuration, deep probing depths, and evidence of osseous loss, the implant may be classified as ailing. Many different strategies are described in the literature to treat ailing implants. Most of these thera-
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Prognostic procedures are performed on implants with crestal (marginal) bone loss and crestal radiolucency. However, apical implant pathology is also a condition that may be indicative of a failing or ailing implant.

Although implant periapical radiolucencies have been reported, there is little information as to why such lesions occur and what the best treatment option is.

The implant periapical lesion (IPAL), also referred to as “abcess formation around the apex of an implant” or as “retrograde peri-implantitis” was first described in 1992 by McAllister et al. Since then, IPAL has been defined as an infection located at the apex of an implant. In 1995 the prevalence of such lesions was estimated to be approximately 0.26%. A more recent review article reported a 1.86% incidence with machined and textured surface implants.

Implant periapical lesions can either be inactive or active. They are considered as inactive when the radiographic findings are not associated with clinical symptoms (Figure 1). A periapical scar of dense collagen is usually seen when the drilling depth during osteotomy preparation exceeded the length of the implant placed or when the apex of the implant is placed near an existing scar (Figure 2). These situations should be periodically reevaluated. If they remain stable, no therapy is needed. In contrast, an IPAL can be categorized as active when there is an increase in radiolucent size, usually accompanied by symptoms (Figure 1). Although IPALs are often found around the apex of an implant, they may spread coronally and/or laterally (Figures 2 through 8). Therefore, an aggressive therapy is usually advised. Even with the diagnostic aids available today, it is still not possible to determine whether these apical lesions are healthy scar tissue, new tissue destruction, or a reactivation of preexisting pathology. The symptoms reported and radiographic evaluation are the main tools available for IPAL detection. Several etiologies have been proposed in the literature. Today, the etiology of these lesions is considered multifaceted.

The purpose of this article was to review all of the cases of IPAL reported in peer-reviewed journals up to December 2007 and to present possible treatment options.

**Materials and Methods**

Medline was searched on the words “periapical,” “dental implant,” and “lesion” alone and in combination within articles published between April 1990 and December 2007. Forty-eight articles were found.

Articles consisting of case reports, retrospective studies, and literature reviews were included in the present paper. Reports of IPALs directly associated with endodontic pathology that included an apical “implantitis” as an extension of a periradicular lesion on an adjacent tooth, or that resulted from the insertion of the implant, which resulted in adjacent tooth devitalization were considered as endo-implant or implant-endodontic infections and were excluded from the present analysis. Cases that involved root canal therapy or apicoectomy on the adjacent teeth do not require a surgical approach on the implant site. Therefore, only reported IPALs in which there were no evidenced periapical pathology at the time of implant placement, the adjacent teeth were vital or asymptomatic, and there was no invasion of the adjacent tooth periodontal ligament space, were included in this review.

Twelve cases were found in the literature, in which the etiology of the IPAL was multifactorial, thus fitting the previous criteria, and therefore were included in this review (Table).

Data were analyzed and included cause of extraction of the natural tooth, location, healing time, type of implant surface, diag-
nostic sign or symptom present, treatment approach, available histologic evaluation, and implant survival and failure rates.

**Results**

Analysis of the data collected (Table) is summarized below.

**Diagnostic**

Radiographic evidence of IPAL was found in all cases.

The signs/symptoms encountered were: fistulous tract (21/32, 65.6%), pain (14/32, 43.8%), and swelling (11/32, 34.4%).

The signs/symptoms of IPAL started from 6 days after implant placement up to...
Cause of Extraction of the Natural Tooth

Twenty-two of the 32 cases (68.8%) were on previous endodontically involved areas, and only 3 cases were related to advanced periodontal disease. For 4 of the implants the cause of extraction was not specified.

Location

Twenty-five of the 32 IPALs (78%) were found associated with maxillary implants.

Of the IPALs included in this literature review, one (3.1%) was a lower second molar, 4 (12.5%) were canines, 9 (28.13%) were incisors, and 16 of the 32 IPALs (50%) were found in the premolar areas.

Period of Implant Placement

Healing time from tooth extraction to implant placement varied considerably, from time of immediate placement until years after extraction.

Implant Surface

Implant surfaces studied were mainly machined (21/32). One implant had a HA-coated surface, 2 implants had acid-etched surfaces (Osseotite, 3i, Palm Beach Garden, Fla), and 7 implants had an oxidized titanium surfaces (TiUnite, Nobel Biocare, Yorba Linda, Calif).

Treatment Approach

In none of the cases reported was antimicrobial therapy alone considered an effective method of treating the IPALs.

All cases were accessed surgically with a mucoperiosteal flap elevation. A crestal incision was the preferred flap design (22/32, 68.8%), followed by the semilunar incision (6/32, 18.8%). One case report utilized an extraoral incision. Complete debridement of the lesion was attempted in 25 of the 32 cases with only 2 of these reporting implant failure (2/25, 8%). In one case there was no debridement of the implant surface, although the bony

18 months after loading. Twenty-three of the 32 cases were detected before the stage II procedure (71.9%), 6 before or at implant loading (18.8%), and 3 post loading (9.4%).

FIGURES 6–8. FIGURE 6. Clinical view of a fistula remaining after the adjacent tooth was removed. FIGURE 7. Clinical view after the implant was removed showing bone defect remaining after the implant was removed. FIGURE 8. Apical half of the implant body had little or no apical bone remaining. Because of the extent of the involvement, an apicoectomy was not recommended as the treatment of choice.
defect was addressed. The implant was still present 11 months following this therapy. In 2 cases no debridement\(^1\) was performed because the explorative flaps did not reveal perforation of the cortical bone. Survival of these implants was reported, but the follow-up duration was not specified. In the remaining cases without debridement (4), the implants were removed during the explorative opening.\(^{23,25,33,35}\)

In 3 cases apicoectomy (Figure 2) of the implant was performed to gain access to the defect walls and allow a complete debridement.\(^{16,32,34}\) One failure was reported after implant apicoectomy. This approach was also recommended in other articles,\(^{18,19,33}\) although the procedure was not performed in the cases cited by those authors.

Surface detoxification was attempted on 11 implants (34.4%) using tetracycline (8), chlorhexidine (1), povidone-iodine (1), citric acid (1), or calcium hydroxide (1). Only 1 failure was reported after the use of tetracycline.\(^{34}\) No failures were reported with the other implant surface conditioners.

Guided bone regeneration (GBR) was performed in 13 of the 32 cases. In 6 cases demineralized freeze-dried bone allograft (DFDBA) was used, 5 cases were treated using particulate bovine bone (Bio-Oss); in 1 case demineralized bone matrix (DynaGraft) was used and in another bioactive glass (PerioGlas). When particulate bone substitutes were used, 7 of the 11 cases were not covered with an occlusive membrane. No implant failures were reported with any of the GBR technique treated cases.

Exclusive guided tissue regeneration (GTR) was performed in 2 cases.

The use of GBR or GTR techniques for the treatment of IPALs was not recommended in one report.\(^{19}\)

**Histologic Evaluation**

In a total of 32 reported implants retrieved and histologically evaluated, bacteria were detected in 1 study, while 3 other studies showed aseptic bone necrosis. No other histologic data were available in the literature.

**Implant Survival and Failure**

Twenty-four of the 32 implants (75%) diagnosed with an IPAL survived after treatment with follow-up times varying from 4 months to 7 years.

In a total of the 32 implants diagnosed with IPAL were lost (21.9%) but only one failed after therapy. Therefore, the survival after IPAL diagnosis and treatment was 96.2% (25 out of 26).

It should be noted that in one of the case reports included in the present review,\(^{22}\) five implants with IPALs were described in a single patient but 4 of them were in close proximity to each other. This finding may be considered as a cluster effect.

**DISCUSSION**

The data presented in this review should be evaluated in light of the few reported cases. Statistics of survival rates might vary if the number of reported cases was larger.

The articles found in the literature show that factors can cause IPALs. The etiology of these lesions has been referred to as a multifactorial\(^{26}\) or with an unknown or inconclusive origin.\(^{32}\) The etiology, which has been described in the literature, included the following:

- **Contamination of the implant surface.** Contamination can be caused by the manufacturer or contamination of the fixture by the clinician,\(^{24,32,33,36}\) adjacent tooth endodontic infection,\(^{18,24,27,28,31,32}\) invasion of the adjacent tooth periodontal ligament during implant placement causing adjacent tooth endodontic infection, and subsequent IPAL\(^{28,31}\) and residual periapical lesion of the extracted natural teeth (granulomas, residual cysts, root remnants, and foreign body reac-
Most of the cases reported occurred in previous endodontically compromised areas. Although a microbial component is highly probable, only one of the implants analyzed histologically reported the presence of bacteria on the IPAL area. Therefore, an exclusive microbiologic etiology cannot be accepted as the only reason for such pathology.

Bone overheating during surgery. This could result in necrosis of the surrounding bone, and a subsequent sequestrum formation around the apex of the implant might occur.

Excessive torquing of the implant. Trauma during surgery with excessive in-depth placement of the implant causing compression of the bone fragments could cause ischemia, necrosis, and bone sequestration. Several histologic studies reveal trabecular microfractures with circulation impairment in compressed areas with subsequent necrosis. One of the articles reviewed rejected this hypothesis citing success found in osteotome procedures, in which bone compression is deliberately achieved with successful results.

Poor bone quality. This occurs where there is a lack of sufficient osteoprogenitor cells to colonize the apical area. Most of the IPALs reported were in maxillary locations, where the bone quality tends to be poor.

Perforation or thinning of the cortical bone. A small cortical surgically undetected perforation or excessive thinning from the medullary side of the cortex on the apical

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<th>Implant Type</th>
<th>Diagnosis of IPAL</th>
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†M indicates; HA, hydroxyapatite; and TiU, TiUnite; IPAL, impact periapical lesion; SI, Stage I; SII, Stage II; L, after load; F, fistula; P, pain; S, suppuration; X, X-ray radiolucency; GBR, guided bone regeneration; GTR, guided tissue regeneration; CLHX, chlorhexidine digluconate; DFBA, demineralized freeze dried bone allograft; FDDMA, freeze-dried demineralized allograft.
The area of the osteotomy could serve as a path of least resistance to infection progression.\textsuperscript{20,35} According to Scarano et al.\textsuperscript{24} if the cortical bone becomes thinner than 0.5 mm, natural bone resorption could cause a dehiscence leading to infection of the soft tissues.

**Premature or excessive load over the fixture.** This would result in bone microfractures around the implant.\textsuperscript{16,18,24} This etiology may be considered either when the IPALS appear after implant loading (small number of cases reported in the literature) or when the patient is supporting a removable prosthesis, which could cause a traumatic lesion.\textsuperscript{41} This hypothesis, however, does not explain the apical location of these lesions, since occlusal overloading is described in the literature as a situation that leads to crestal bone loss and bone microfractures at and near the bone-implant interface.\textsuperscript{42} Based on the literature, most of the IPALS were found before stage II procedures. Therefore, excessive loading probably may not be a primary risk factor.

**Fracture of the bone inside the hollow portion of hollow implants.** The rationale for this hypothesis is that the bone fracture would cause a vascular impairment and consequent aseptic bone necrosis in the hollow part of this type of implant.\textsuperscript{25} Since this type of implant is not routinely used any longer, this hypothesis will not be further discussed in the present paper.

Other etiologic factors proposed in the articles reviewed include: *implant placement in an infected maxillary sinus*, which could cause a spread of the maxillary sinus infection onto the implant surface,\textsuperscript{24} *absence of primary stability*,\textsuperscript{26} and *reduced healing ability of the host*.\textsuperscript{26} None of these etiologies were reviewed because no data were available in the literature to allow an adequate analysis of these possible factors.
According to the articles reviewed in the present report, the postextraction healing time appeared to be irrelevant in terms of preventing IPALs. However, several articles proposed a postponed implant placement after an endodontic failure that leads to extraction of the natural tooth in order to allow a more predictable bone healing.\(^{31}\)

In most of the cases, IPALs were associated with machined surface implants. One case reviewed was associated with one HA-coated implant,\(^{22}\) which was treated successfully and one reported an IPAL around TiUnite surfaced implants.\(^{22}\) The authors\(^{22}\) concluded that although the textured implant surface (specifically the TiUnite) was more sensitive to infection and bacterial invasion, the treatment of the IPAL was predictable.

All cases reported in the current review presented with periapical implant radiolucencies, which were accompanied by fistulous tract formation, pain, or swelling as the most frequent clinical findings. Therefore, a fistula formation in the apical part of the implant should be considered an important sign for the diagnosis of an IPAL.\(^{22,24}\)

Several therapeutic alternatives were presented in the literature. Most authors recommended the exposure of the implant apex, degranulation of the defect, detoxification of the implant surface, and regeneration of the lost bone.\(^{22}\) However, no consensus could be found concerning the treatment of such cases except that they should be approached surgically. Several methods were proposed to detoxify the implant surface. However, a distinction has to be made between HA-coated implants and machined titanium surfaces. For HA-coated implants, research supports the use of citric acid for 30–60 seconds.\(^{22,26}\) If the hydroxyapatite looks pitted, blotchy or resorbed, it should be removed down to the underlying titanium and then it should be treated as a pure titanium surface.\(^{22,26}\) For implants with machined titanium surfaces, surface treatment with tetracycline paste,\(^{19,22,24}\) 10% povidone-iodine solution,\(^ {16}\) 0.12% chlorhexidine digluco- 

On the other hand, when a laser is used, it can help in removing the infected tissue and promoting bone regeneration.\(^{30}\) Studies have shown that lasers can enhance the bone healing process, reduce inflammation, and improve the overall healing outcomes.\(^{31}\) The use of lasers in conjunction with other treatments can further enhance the success rate of IPAL treatment.

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around the implant surface, detoxification of the contaminated fixture surface should be attempted to not compromise that and allow fibroblast migration and attachment. But this hypothesis remains opened to discussion since no histologic evaluation of the wound healing was attempted in any study of IPALs to date. In the present literature review, only 2 implants were lost when debridement of the defect was performed in immobile implants with IPALs.21,34

More documented cases including histologic data from successfully treated cases and clinical studies comparing different protocols and their outcomes to determine an effective treatment protocol for implants with IPALs are necessary.

CONCLUSIONS

From the literature reviewed in this report, areas with endodontically compromised teeth might interfere with implant success; therefore, a careful analysis should be made prior to the implant placement regardless of the healing time after the extraction of the natural tooth. The IPALs were mainly found around implants in the maxilla and have a higher prevalence in the premolar area. When radiographic evidence of periapical lesions accompanied by symptoms such as pain, swelling, and fistula formation are present, these cases should be treated aggressively.

Lacking a consensus, the etiology of these lesions are preexisting bone pathology, contamination of the fixture before placement, surgical trauma with thinning, or perforation of the cortical bone during stage I surgery and a poor bone quality. Most of the articles in the literature suggest a microbial involvement, but an exclusive antimicrobial therapy was not considered in any article as effective treatment for the IPAL.

Surgical approach should include the exposure of the defect as conservatively as possible but with adequate access, debridement of the defect, detoxification of the implant surface, and a GBR if re-osseointegration is the goal of the surgery and not only arrest of the infection.

Although the high survival rate after therapy was reported in the articles reviewed, it should be stressed that there have been a small number of cases reported in the literature. Moreover, many variables existed in the studies cited including the type of implant surface, size, and chronicity of the defect, which might have influenced the results following treatment. More standardized studies and data are needed to more predictably prevent and treat implants with IPALs.

ABBREVIATIONS

CLXH: chlorhexidine digluconate  
DFDBA: demineralized freeze-dried bone allograft  
FDDMA: freeze-dried demineralized allograft  
GBR: guided bone regeneration  
GTR: guided tissue regeneration  
HA: hydroxyapatite  
IPAL: impact periapical lesion

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