Diagnosis and treatment of a large periapical implant lesion associated with adjacent natural tooth: A case report

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A possible cause for dental implant failure is the periapical implant lesion (PIL). In this case report we describe an apical periodontitis on a tooth adjacent to a dental implant that may have communicated with the apical region of the dental implant, and causing retrograde peri-implantitis. To our knowledge this is the first report demonstrating the concomitant successful treatment of the periapical implant pathology and the adjacent natural tooth without the removal of the implant. The presence of large bony defect at the apical region of the natural tooth and the implant, resulting in a sinus tract and a deep periodontal pocket, was also confirmed with computerized tomography.

The treatment procedure included root canal treatment followed by the debridement of the apical bone lesion, and guided bone regeneration. An uneventful healing with acceptable esthetic was observed.


Osseointegration is the healing of bone around dental implants so that the direct anchorage of the implant is then maintained allowing functional loading without the growth of fibrous tissue at the bone–implant interface. 1 Although a high dental implant success rate has been reported, 2,3 several reports demonstrated dental implant failures. 4-8 The etiology and mechanism of implant failure are multifactorial, and periapical implant lesion (PIL; implant periapical pathology or retrograde peri-implantitis) has also been reported as one possible cause for dental implant failures. 7,14

PIL often occurs by overloading and excessive tightening of the dental implant, bone overheating during surgical procedure, fenestration of the vestibular alveolar bone, presence of pre-existing bone pathology, contamination of the dental implant surface, pre-existing microbial pathology, or poor bone quality. 7,8,13-18 PIL was previously classified into two groups according to the activity of infection, including inactive (non-infected) and active forms. 9,12 No treatment is normally required for the inactive form, however; periodic recalls to monitor the lesion has been recommended. Surgical treatment was advised including dental implant removal, apical resection for the dental implant, or guided tissue regeneration for the active form of PIL. 5,9,11,12,19 In addition to the classification of the activity of PIL two basic pathways were also reported for the formation of implant periapical lesions, including implant to tooth and tooth-to-implant pathway. 11

Implant-to-tooth lesion occurs when the insertion of the dental implant results in tooth devitalization; tooth-to-implant lesion occurs when the periapical lesion from a nearby devitalized tooth encroaches upon the implant and contaminates it. 11 The present case report demonstrates a dental implant located at premaxillar region with a large periapical lesion, which was most likely started by the adjacent natural tooth. The diagnosis and treatment of the PIL will be described and, in addition, a detailed discussion about implant periapical pathology will be provided to review possible treatment options.

CASE REPORT
Dental history and diagnosis

A 25-year-old Caucasian male patient with no contraindication to dental treatment presented to the Department of Oral and Maxillofacial Surgery with a dull, persistent pain at his
right premaxillary area that tended to increase in severity and could no longer be controlled with analgesics. He reported gingival bleeding and pus drainage in the right maxillary incisor region. The pus formation and gingival bleeding had been of variable intensity during some time. The patient lost tooth #7 in February 1999 due to a traumatic injury which required the extraction. Complete bone healing was observed one year after extraction (February 2000) (Fig. 1) at which time a screw-shaped titanium dental implant was placed into the extracted area. The implant was restored by a full porcelain crown six months after dental implant surgery. The patient also reported that his tooth #8 was restored with a composite restoration one year (September 2001) after implant prosthodontic rehabilitation completed.

Although he had his composite restoration completed, he reported sensitivity in tooth #8 that continued. He further reported that the dentist he attended prescribed analgesics to reduce his pain. A couple of months after using the prescribed analgesics, his complaint about tooth #8 started again and he was examined in our department in March 2005. There was no periodontal disease or caries lesions. Tooth #8 was diagnosed as non-vital and sensitive to apical palpation. The tooth had a palatal composite restoration. Tooth #8 had no increased periodontal pocketing. However, a 7-mm probing depth with pus formation was found in the vestibular region of the dental implant located at tooth #7. A periapical fistula was also diagnosed in the apical region of the dental implant (Fig. 2). Radiographic evaluation showed a large periapical radiolucency (Fig. 3) involving both the apical regions of the dental implant and the central incisor (teeth #8 and #7 location). Computerized tomography (CT) was used to clarify the relationship between the dental implant and tooth #8 (Figs. 4, A–D).

Treatment protocol and follow-up

Tooth #8 was diagnosed as the potential etiologic factor for the formation of the large apical lesion involving the apical region of the dental implant located at tooth #7. The first step of the treatment plan was to complete the root canal therapy of tooth #8. The canal was instrumented using step-back technique until an apical preparation site #50 was achieved. During the root canal treatment, 2.5% NaOCl irrigation solution was administrated between instruments. The root canal was filled with gutta-percha and AH26 (De Trey, Konstanz, Germany) using lateral condensation technique.

Following the endodontic treatment, a surgical treatment phase was initiated. A full thickness flap was reflected by a sulcular incision followed by vertical releasing incisions...
located at the distal of the tooth #6 and the distal of the tooth #9. Complete loss of a labial cortical plate and a large periapical defect was observed (Fig. 5). The deepest point of the lesion was 10 mm, and the width of the lesion was 8 × 10 mm. Debridement of the tissues at the defect site was followed by the irrigation with sterile saline solution. Scaling and root planing was performed on the root surface of #8 and apicoectomy was done (Fig. 6). A root-end filling of glass ionomer material (Medicem, Promedica, Neumünster, Germany) was placed.\textsuperscript{23} The body of the dental implant (the labial side of the implant) was contaminated, however; the dental implant appeared intact, therefore implant detoxification was not performed. Guided bone regeneration\textsuperscript{20,21}—including commercially available resorbable grafting material, calcium sulfate (Fortoss Vital, synthetic tri-calcium phosphate in a hydroxyl sulphate matrix, Biocomposites, Wilmington, NC)—was packed into the bony defect (Fig. 7). A resorbable membrane was placed over the graft material (Biomend 15 × 20, resorbable collagen membrane, Zimmer Dental, Carlsbad, CA) (Fig. 8) and the wound closure was ob-
tained with 3-0 silk sutures. Following surgery, the patient was given a cold compress extra-orally to minimize swelling and bleeding. He was prescribed 0.2% chlorhexidine gluconate and instructed to rinse gently twice daily for 3 weeks. He was also prescribed antibiotics (amoxicillin 500 mg, four times daily) for 10 days. During this time, tooth brushing was discontinued. Ten days after surgery, the sutures were removed and an uneventful healing was observed. Patient was seen at 3 and 6 weeks, and 6 months. These follow-up visits included routine intra-oral examinations and professional plaque control. Six months after surgical procedure there were no symptoms of pain, inflammation, or discomfort. Radiological evaluation demonstrated an uneventful healing at the grafted surgical site, and almost the entire bone substitute was resorbed six months after surgery (Fig. 9). Patient was completely satisfied with the results of the treatment, and he also reported highly successful esthetic outcome.

**DISCUSSION**

Several etiologic factors for PIL have been reported, including the contamination of the implant, bone overheating during surgical procedure, excessive tightening of the dental implant, presence of existing microbial pathology, overloading of the dental implant, and fenestration of vestibular alveolar bone. Among these factors, although it is highly speculative, the expansion of the apical pathology located at the natural tooth adjacent to dental implant may have contributed as one of the etiologic factors. Based on the previous classification, the
present case report describes an ‘active’ and ‘tooth-to-implant’ classified form of PIL. We may suggest that when the adjacent natural tooth developed periapical pathology by a possible operative damage or a profound caries lesion to the dental pulp, the apical peri-implant pathology occurred at the apical region of the dental implant. The periapical lesion at the natural tooth may have communicated with the apical region of the dental implant followed by the contamination of the implant body, then resorbed the surrounding osseointegrated alveolar bone that may have resulted with the drainage of the apical lesion through the apical fistula located at the apical region of the dental implant and also through the periodontal pocket, which may be evaluated as retrograde peri-implantitis.

It has been suggested that dental implants cannot be bacterially challenged during the osseointegration period, and that an endodontic lesion may easily spread through the bone marrow and contaminate an adjacent implant, which may result in the loss of a dental implant.16,26 It was previously reported that PIL developed five months after surgery, where the implant was removed and histologic results demonstrated that vascular impairment, poor bone quality or fracture may be the etiology of the implant failure.10 Sussman11 reported that PILs occurred without any fistula formation due to the infection of adjacent natural teeth that may have contaminated the apical region of the implant, and although the adjacent natural teeth were successfully endodontically treated, dental implants were removed. Scarano and colleagues13 demonstrated that PIL was evaluated at a screw shaped mandibular dental implant without any fistula formation, which was diagnosed in a CT radiography but they could not specifically report the etiologic factor for the formation of PIL, and the dental treatment was resulted with the removal of the implant. Ayangco and Sheridan27 presented that retrograde peri-implantitis may be occurred due to implants replacing teeth with histories of failed endodontic and/or apicoectomy procedures. Furthermore, Flanagan14 stated that a PIL located at a posterior maxillary implant was diagnosed with an apical fistula formation two weeks after implant surgery, where they discussed several possible candidates for PIL formation as previously mentioned at introduction part. They also introduced that a successful healing was achieved after performing a regenerative surgery (calcium hydroxide paste) performed at the apical region of the implant.14 A recent report also demonstrated that a mandibular implant was lost due to PIL that developed three months after implant surgery, where Oh and colleagues12 reported that PIL may have occurred due to a furcation involvement at the adjacent natural tooth that may have contaminated the apical region of the adjacent dental implant. The authors further suggested that the dental implant was removed and another implant was placed into the area following the debridement of the contaminated region.12 Compared to these clinical reports, the present case referred for treatment five years after implant placement, where he was given surgical treatment including guided bone regeneration without the loss or the removal of the dental implant.

The inactive form of PIL should be periodically monitored and does not need any surgical treatment. However, if the lesion is active (pain and/or pus formation), it should be surgically removed, endodontic lesions should be debrided, and an antibiotic therapy should be performed to achieve a successful eradication of bacterial contamination.7,8,11,28 Surgical procedures including the removal or the resection of the contaminated dental implant was previously suggested to avoid a possible osteomyelitis,9,10 and surgical removal of the dental implants should be performed either the implants were mobile or not.10-13 On the contrary, based on the recent knowledge if the dental implant has a stable osseointegration and the periapical lesion does not risk the adjacent regions, it was suggested that the removal of the implant should be avoided, and the complete debridement of the lesion should be performed.12 Although a large bony defect was diagnosed in dental CT scan and in surgical operation, the dental implant was found stable (osseointegrated) after debridement. Therefore, the implant was not removed and the bony defect was filled with regenerative materials to achieve bone formation at the apical region and body of the implant, and also at the apical part of the central incisor.

Clinical symptoms including suppuration or fistula formation should be counted as the diagnostic features of active form of PIL, where a surgical treatment should be performed to avoid the contamination of dental implant that may result with the loss of implant.12 Seltzer29 reported that the histologic evaluation of periapical chronic lesions was found in one-half of the specimens obtained from 14 endodontically treated teeth, where the sampling was performed at 6 to 30 months following endodontic treatment. Therefore, Seltzer29 and Brisman30 concluded that the practitioners should be aware of that the periapical lesions could still serve as a bacterial reservoir following endodontic treatment although radiographic evaluations demonstrate the resolution of periapical lesions. Based on previous reports, the practitioner should not take the possible risk, where the contamination of the dental implant may still occur following endodontic treatment.12,28,31 Thus, it was suggested that endodontic treatment should be completed with the surgical debridement of the contaminated area.28 Although it was reported that a successful treatment outcome could be achieved after the debridement of the pathology,12,28 McAllister and colleagues19...
Presented that the debridement of the defects could still include bacteria, which may cause another pathology following treatment. Thus, it was recommended that antibiotic treatment may be continued following surgical debridement.19 In the present case, following endodontic treatment, the apical surgery including apical resection of the adjacent tooth and the debridement of the bony defect was performed, where amoxicillin was also prescribed immediately after surgical therapy.

Previous reports about PIL demonstrated that the dental implants placed were infected in a short while (in a couple of weeks or months), in which the PILs were related with pre-existing microbial pathology or surgical mistakes.10-14,32 Interestingly, the present case demonstrated that the PIL was formed five years after the implant surgery, which could be evaluated as a long period of time compared to previous reports.10-14,32 Based on the clinical and radiographic evaluations, the chronological patient history, and the formation of apical pathology in a long period of time, one can suggest that the periapical implant pathology could be originated from the apical pathology of the adjacent natural tooth in the present case. After the apical inflammatory lesion expanded to the apical region of the implant, an apical fistula may have formed. The apical fistula may have served as a drainage pathway for the contaminated dental implant and the adjacent natural tooth.

Moreover, obstructed canals, extruded root filling material, failed root canal treatment, and perforations during root canal treatment were attributed as the indications for periradicular surgery.20,22,33 However, von Arx20 presented that patient’s finances, treatment time, and psychological issues should also be counted as the possible indications for periradicular surgery. Periradicular curettage, apicoectomy, and root resection can be used in periapical surgery to achieve a successful outcome.20,22 It was shown that healing outcome in periapical surgery was also related to the condition of the buccal alveolar plate.21 Following apical resective surgery, epithelial down-growth along the buccal root surface was considered as an important etiology preventing successful healing.20 As well as resective surgical procedures, regenerative techniques was also introduced to achieve a successful bony regeneration.20

Therefore, we performed periradicular curettage, followed by the resection of the root apex and then augmented the bony defect with resorbable bone substitute and a collagen membrane to achieve guided bone regeneration. Guided bone regeneration was performed to maintain the stabilization of the dental implant and to avoid the possible formation of the epithelial down-growth through the coronal site of the surgical area.20 It was also reported that complete healing may take as many as 2 or more years in some cases following a root canal therapy.34 Further, even successful eradication of the root canal infection was performed; active functioning of host repair mechanisms was continued even after 1 year of the treatment.34 Furthermore, microorganisms are located in privileged and pivotal positions within the root canal containing necrotic pulp. The microorganisms located at the apical site of the root canal may expand and the inflamed periapical tissues will be surrounded by the polymorphonuclear leucocytes (PMNL).35 A balance between the aggression of the lesion including degranulation of the supporting alveolar bone, and the defense of the host results with the development of a chronic inflammatory disease.35 A process of alveolar bone resorption takes place during the expansion of the apical inflammatory lesion if the etiologic factor is not eradicated. PMNLs, T lymphocytes, B lymphocytes, plasma cells, natural killer cells, macrophages, and osteoclasts play active roles during the inflammatory process that may result with the loss and the expansion of alveolar bone resorption.20,21,34,35

The mineralized matrix of the alveolar bone of the tooth apex is degraded by multinucleated cells, and several proteases are also released into the lacunar space that excavate the alveolar bone tissue.35 By performing a successful root canal therapy, the inflammatory lesion is replaced by granulation tissue during the healing process.34 However, if a non-responding periapical lesion is concerned, the golden standard treatment method is attributed as periradicular surgery.34,36 Moreover, if the lesion was inducing pain and pus formation, it was suggested that the lesion should be surgically removed and apical lesions should also be debrided to achieve a successful bacterial eradication.7,8,11,28 When the possible colonization of the microorganisms following root canal treatment at the apical area20,29,30,36 and the equilibrium including resorption and apposition process following root canal therapy were taken into consideration,35 the present case was treated with apicoectomy to avoid the possible colonization of microorganisms around the apical area, and the possible formation of a secondary periapical lesion.20,29,30,34 It is clear that renewal of a dental implant takes higher costs to the patients. Thus, we did not want to take the risk of a secondary periapical lesion formation, which may cause the degranulation of the bone substitutes followed by the loss of the dental implant; in this case, an increased treatment cost and a prolonged treatment time should also be considered.

Although reports were presented about apical implant pathologies that generally resulted with dental implant removal or loss,10-14,27,32 to the authors’ knowledge, this is the first report demonstrating the successful treatment, with guided bone regeneration, of periapical implant pathology possibly caused by an adjacent tooth.
In conclusion, a necrotic natural tooth with a periapical inflammation may have had a significant role in infecting the neighboring dental implant through an apical pathway. The periapical lesion drained through a sinus tract. After completed endodontic treatment of an offending tooth, a stable dental implant with a PIL may be treated with the debridement of the apical lesion followed by guided bone regeneration. Six months follow-up resulted in an uneventful healing without the removal or the loss of the dental implant, where the patient reported highly successful esthetic outcome, and almost the entire bone substitute was resorbed radiologically. A detailed diagnosis, systematic treatment plan, and appropriate treatment procedures would minimize the occurrence of dental implant periapical lesions. The dental practitioner should not only carefully evaluate the surgical area but also examine and follow-up the adjacent teeth and/or alveolar bone to achieve a successful long-term outcome.

REFERENCES


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