



Case Report

A successful Treatment Outcome of Endodontic – Implant Pathosis

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Abstract: Dental implants are the norm of today's dentistry and are the answer for replacement of both missing tooth/teeth and edentulism. A factor of concern during treatment planning is the pulpal and peri – radicular condition of teeth adjacent to the implant site. Peri – radicular pathosis may initiate implant periapical lesions (IPL) which subsequently jeopardize the dental implant. IPL is the infectious – inflammatory process of the tissues surrounding the dental implant apex interface. This case report presents a case history related to IPL, which was caused by a pre-existing endodontic lesion from the adjacent tooth. Diagnosis is achieved by studying the presence of signs and symptoms such as pain, swelling, suppuration or fistula and radiographic examination. A diagnostic classification is proposed to establish the stage of the lesion, and determine the best treatment option accordingly. The aim was to report the success of treating IPL which was caused by the presence of radicular cyst from neighbouring infected tooth.

Key words: Implant periapical lesion, endodontic – implant pathosis, dental implant.

Introduction

The degree of dental implant stability achieved during healing relies on how well osseointegration has taken place and it seems to be crucial for this osseointegration to be maintained during functional loading. This observation may reflect the fact that the stability of implants is determined by the biomechanical properties of the surrounding bone and the implant interface. Bone regeneration is a host – driven response to heal fractures or defects (as a result of extraction or localised disease) to return the site to its original healthy state.

Buccal bone resorption which exposes dental implant threads may occur after implant surgery or it could be caused by pathological bony resorption from expanding tumour or cystic lesion adjacent to the implant. The main indications for covering exposed threads are to increase implant stability, avoid subsequent soft-tissue problems, and enhance the esthetic appearance¹. For that purpose, the use of different methods and materials has been advocated.

The aim of this study was to report a successful treatment outcome of salvaging an implant fixture which became compromised by contamination from an adjacent tooth that developed periapical pathology subsequent to implant placement. This report documented a case of endodontic-implant pathology where a favourable treatment outcome was

achieved after immediate bone grafting procedure post enucleation of maxillary cystic lesion in a single sitting.

Case Report

A 51-year old man with no relevant medical history reported to the Oral and Maxillofacial Surgery of University Malaya with pain and discomfort since 2 months previously in the zone of left maxillary incisors. On general assessment, patient was moderately built and nourished. Extraorally face was symmetrical with no obvious swelling. Further history revealed that he previously had a tooth implant inserted to replace his upper left central incisor (tooth 21) in 2013, however the dentist noticed a small radiolucency at the periapical region of the adjacent neighbouring tooth (22). The referring dentist placed a temporary acrylic crown on 21 implant fixture and completed endodontic treatment on tooth 22 after an electrical pulp test showed result of non – vitality. There was no complaint or issue regarding the dental implant at that time.

During subsequent 2 months review, radiographic examination prescribed by the dentist revealed that devitalized tooth 22's root – end inflammatory process proceeded to communicate with the surface of the 21 implant fixture. Aspiration was performed and a clear straw – colored fluid

was collected in the 10 cc syringe confirming the presence of cystic lesion around the vicinity of the devitalized tooth 22. A localised infection of bone secondary to endodontic – implant pathology was suspected.

A cone beam computer tomograph (CBCT) scan was prescribed for the following reasons: (1) to confirm the diagnosis; (2) to determine the seriousness of the infection and the evaluate the extent of the anatomical structures involved; (3) to plan the surgical procedure and (4) for medicolegal reasons. The CBCT showed a well circumscribed roundish radiolucent lesion arising from the periapical of devitalized tooth 22 measuring approximately 7.35(A) x 9.81 (W) x 9.80 (H) mm in its greatest dimension (Figure 1). The lesion did not cross the midline but there was evidence of palatal bone thinning noticed from the scan. Our clinical diagnosis at this stage was persistent radicular cyst of devitalized tooth 22.

Total enucleation of the cystic lesion with apicoectomy and retrograde filling with MTA, surgical debridement for exposed threads of 21 implant fixture and immediate placement of EthOss® synthetic bone graft was used without membrane to pack the bony cavity under local anesthesia was performed

(Figure 2, 3, 4 and 5). 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 1 week. He was also prescribed antibiotics (amoxicillin 500 mg, three times daily) for 5 days.



Figure 3. Cystic lesion was carefully curettaged and enucleated.

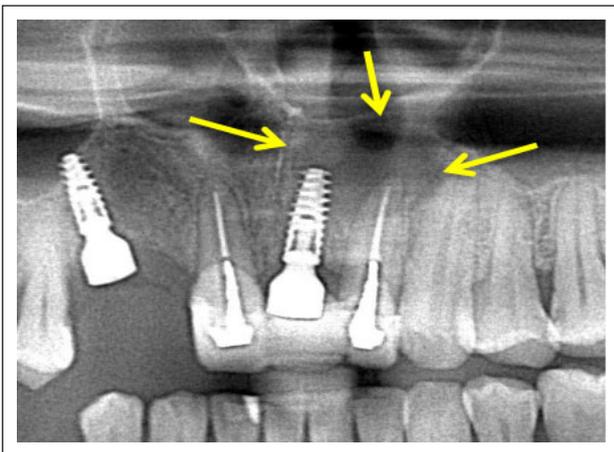


Figure 1. Dental implant located at premaxilla region with a large periapical lesion (arrows), which was most likely started by the adjacent devitalised tooth.



Figure 4. Surgical debridement using metal micro – brush on the exposed implant threads to remove particulate and debris attached to it. Apicoectomy along with retrograde MTA filling was done on tooth 22.



Figure 2. Surgery was performed under local anesthesia (2% Mepivacaine with 1:100,000 adrenalin), raising a full thickness flap in the affected zone.



Figure 5. EthOss® Synthetic bone graft placement without membrane. Closure of the flap with Vicryl 4/0.



Figure 6. Completely healed soft tissue after 1 week post surgery.

Dental pantomogram (DPT) taken at 1 month post surgery showed no gapping present between the implant threads interface and augmented bone graft. Almost 90% of the implants threads are covered with the bone grafts. After 4 weeks, the architecture of the gingival ridge was preserved, and clinical observation revealed excellent soft tissue healing without loss of attached gingiva. Healing was unremarkable and patient had no complaint thereafter (Figure 6). Following osseointegration of the implant with the bone, permanent ceramic crown prosthesis will be given to the patient.

Discussion

The increasing popularity of implants has led to a considerable increase in the incidence of implant periapical lesions (IPL). IPL is the infectious – inflammatory process of the tissues surrounding the dental implant apex interface². The pathogenesis of IPL – mainly failure of osseointegration has been described to occur through two pathways: classical and retrograde². Classical pathogenesis (from the soft tissue apically to the bone) is associated with bacterial plaque-induced inflammatory changes in the soft tissues surrounding dental implants, which can lead to progressive destruction of the peri-implant tissue with resulting bone loss, and ultimately to implant loss. Meanwhile, retrograde pathogenesis (bone to soft tissues) is associated with bone loss occurring at the bone crest as a result of micro-fractures caused by overloading, premature load, or lateral forces related to occlusal or restorative factors. Nevertheless, in some clinical situations, both classical and retrograde pathways may overlap, making it difficult to establish with precision whether infectious or occlusal overload etiologic factors are responsible².

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 factors³. Radicular cysts are the most common type of cyst found in the jaw, and are classified as inflammatory cysts preceded by a chronic periapical granuloma and stimulation of cell rests of Malassez found in the periodontal membrane⁴.

In recent years, theories of osmosis-related bone loss are receding into the background and being replaced by newer

molecular biological concepts to describe cyst growths. A study done by Meghji and co-workers in 1989 concluded that the secretion of IL – 1 from cyst wall cells is a possible cause of osteoclastic bone resorption⁴. Lindeboom et al. recently presented histologic evidence demonstrating that breakdown around natural teeth is not the same as that around implants. The study concluded that the inflammatory infiltrate was in bone around the implant. This type of inflammation closely resembles osteomyelitis. The article further reported that the lesion was osteomyelitis and not periodontitis. They further stated that, since the implant had no periodontal ligament around it, treatment would probably result in failure. Therefore, the best course of action would be to remove the implant fixture⁵.

In our case, although the adjacent natural tooth which had a periapical lesion was devitalized and root canal treated, the expansion of the apical pathology was still proceeding. One plausible explanation for this pathogenesis is that nutrient canals and marrow spaces histologically exist, and that they drain inflammation from the supragingival tissues. Therefore, it is possible that contamination from the endodontically involved tooth that developed post-operatively spread to the implant fixture via these spaces. Ayangco and Sheridan described 3 cases of IPL in patients with a history of endodontic treatment and failed apicoectomy before implant placement⁶. According to these authors, despite curettage, socket cleaning, and a long waiting time, bacteria would have persisted in the bone, causing implant periapical alterations as a result⁶.

In any event, odontogenic cysts are well established at the time of their diagnosis because of their origin within the bones of the jaws. 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 implant². Seltzer through his study proved that the periapical lesions could still serve as a bacterial reservoir following endodontic treatment although radiographic evaluations demonstrate the resolution of periapical lesions⁷. Thus, it was suggested that endodontic treatment should be completed with the surgical debridement of the contaminated area⁷.

Another study done by Tözüm *et al.* mentioned that there is no clinical protocol for the management of an IPL². They proposed therapies for an IPL, which are similar to those for peri-implantitis, including the following: 1) non-surgical treatment via systemic antibiotics, 2) resective treatments including debridement along with detoxification of the implant surface using a chemical agent (chlorhexidine gel, stannous fluoride, tetracycline hydrochloride, hydrogen peroxide, citric acid, polymyxin B, or chloramine T), and intraoral apicoectomy of the implant apex, 3) regenerative treatments including debridement, detoxification of the implant surface, intraoral apicoectomy of implant apex, and guided bone regeneration, and 4) removal of the infected implant². Besides these, a study done by Chang and co-workers proposed a non-surgical way of IPL therapy⁸. They successfully treated IPL with medical methods by prescribing systemic medications (prednisolone, augmentin and mefenamic acid) and reported a complete resolution of IPL without any signs of recurrence at 2-year follow-up⁸.

The treatment done in our case study was in accordance with the therapy proposed by Tözüm *et al.* with slight modifications². We performed a thorough curettage and enucleation of the cystic lesion followed by apicoectomy of the devitalized natural tooth and surgical debridement of exposed implant threads using special metal micro-brush bur. Then, guided bone regeneration (synthetic) was placed to maintain the stability of the dental implant and to avoid the possible formation of the epithelial down – growth through the coronal site of the surgical area. Successful osseointegration is achieved by bone growing almost completely to contact the implant.

The reported case suggests some considerations. IPL is a preventable disease. The combination of careful evaluation of planned implant sites for potential contaminants, careful surgical technique, and meticulous sterilization techniques may limit the incidence of infected IPLs. There are few studies with small sample, without homogeneity of criteria for diagnosing the disease and without design of scientific evidence. Currently etiology lacks consensus. The early diagnosis of IPL during the osseointegration phase and early treatment, will lead to a higher survival rate of implants treated, hence preventing the need for implant extraction⁹.

Conclusion

In conclusion, a necrotic natural tooth with a periapical inflammation may have had a significant role in infecting the neighbouring dental implant through an apical pathway. We propose apicoectomy with retrograde filling of the devitalized tooth and implant surgical debridement procedure in the case of radicular cystic lesions, while implant extraction is reserved for those situations in which the entire bone surface in contact with the implant is affected, or primary implant fixation is lost. Although previous studies did mention that spontaneous bone regeneration could be obtained after enucleation of large odontogenic cysts without using bone grafts, we wanted to make sure the previously inserted implant lasts a lifetime even after functional loading forces is applied to it.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Declaration of Patient Consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

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