CASE REPORT

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Early Dental Implant Failure in Patient with Active Implant Periapical Lesions: Lesson Learnt from Two Case Reports

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ABSTRACT

Implant periapical lesion (IPL) is also known as retrograde peri-implantitis and as the name suggests, it involves inflammation surrounding the apical part of the dental implants. Previously, many studies have reported the event of IPL that further delays osseointegration, and some reported failure of implant placement due to this disease. In this article, we described two cases of early dental implant failure that was associated with active IPL and correlated the clinical and radiographical findings with the histopathological findings.

Keywords: *Apical peri-implantitis; early implant failure; implant periapical lesion; retrograde peri-implantitis*

INTRODUCTION

Endosseous dental implants have shown high survival rates of over 95% for a period of 10 years (Howe *et al.*, 2019). However, implant failure may occur, either due to technical, biological, or aesthetic complications (Pjetursson *et al.*, 2004; Romeo & Storelli, 2012). Among those factors, implant periapical lesion (IPL) could also be one of the causes of implant failure and it may interfere with the osseointegration process and progression of the lesion (Quirynen *et al.*, 2005; Kim *et al.*, 2015).

Active IPL or retrograde peri-implantitis has been defined as an infectious-inflammatory alteration surrounding an apical part of the implant, which is characterised by swelling, suppuration, fistula formation and alveolar bone loss (Esposito *et al.*, 1999). It develops shortly after surgical implant placement while normal bone to implant interface was achieved at the coronal portion of the implant (Quirynen *et al.*, 2003).

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The aetiopathogenesis of an active IPL is multifactorial. This condition may result from bacterial contamination during implant insertion, premature loading, or pre-existing inflammation with the presence of bacteria, inflammatory cells or cells remnants from a cyst and granuloma (Quirynen *et al.*, 2005). Thus, we aimed to report two early dental implant failure cases that were associated with active IPL and correlated with the histopathological findings.

CASE REPORT

The authors report two cases of patients who underwent surgical implant placement on consecutive days by different Periodontic Residents in the Periodontology Clinic, Faculty of Dentistry, Universiti Teknologi MARA (UiTM). Three dental implants were placed in the two patients. Both surgeries involved two stages of dental implant placement.

Case 1

A fit and healthy 51-year-old Malay lady attended the Periodontology Clinic, requesting for an implant-supported crown for missing upper left premolars teeth. Periapical radiograph of retained root tooth 24 showed no pathological abnormality (Fig. 1A). Minimally traumatic extraction of retained root of tooth 24 was performed 10 months prior to implant placement. The extraction of tooth 24 was performed with no post-operative complications, and no active complaints reported by the patient. Patient had extraction of tooth 25 due to caries approximately four years ago in 2014. Pre-operatively, the edentulous area of tooth 24 and tooth 25 showed no pathological signs and symptoms both clinically and radiographically (Fig. 1B). Cone-beam computed tomography (CBCT) with the radiographic stent revealed no pathological deformities and adequate bone width and height (Fig. 1C). During the surgical procedure, two dental implants were placed in the edentulous area of tooth 24 and tooth 25 under local anaesthesia, following the surgical principles and guidelines from the manufacturer's instructions under aseptic procedure (Fig. 1D). The implants used were self-tapping screw design and presented with basic cylindrical shape with apical conical taper. After raising the full mucoperiosteal flap, a 3 mm surgical length round bur was used to create an initial depression in the crestal bone. Marking drilling was performed followed by pilot drilling to determine implant length and axial alignment. This initial step was guided by using a customised surgical stent. A depth gauge was inserted into the area and intraoral periapical radiograph was taken to check the preparation depth and axial alignment. The pilot drill hole was extended by using extension drill and further extension drilling was performed depending on planned implant diameter. During this time, the extension drills were kept at even lower speed, below 60 rpm. For each step, copious irrigation was performed as external cooling using sterile, physiological saline solution. Two bone level implants with 3.4 mm width and 9.5 mm length were placed. The implants were placed with a final torque 25 N/cm^2 by a torque measuring wrench with a good primary stability parallel with the roots of the adjacent teeth. The flap was repositioned and sutured. Periodontal dressing (Coepak™) was placed. Patient received analgesic for 5 days (Ibuprofen 400 mg three times per day when needed).

Periodontal dressing and sutures were removed after one week. Patient was reviewed weekly after the surgery with no apparent symptoms. In the third week of review, she complained of dull throbbing pain at the surgical site and consumed 1000 mg paracetamol when needed. She stated that the pain started after the second week of review with on and off swelling at the site of complaint. Clinical examination revealed swelling on the buccal of implant 24 with mobility grade 3 (Figs. 1E and 1F). Almost full exposure of the cover screw was noted approximately 3×4 mm in diameter. Vitality test using electric pulp test (EPT)

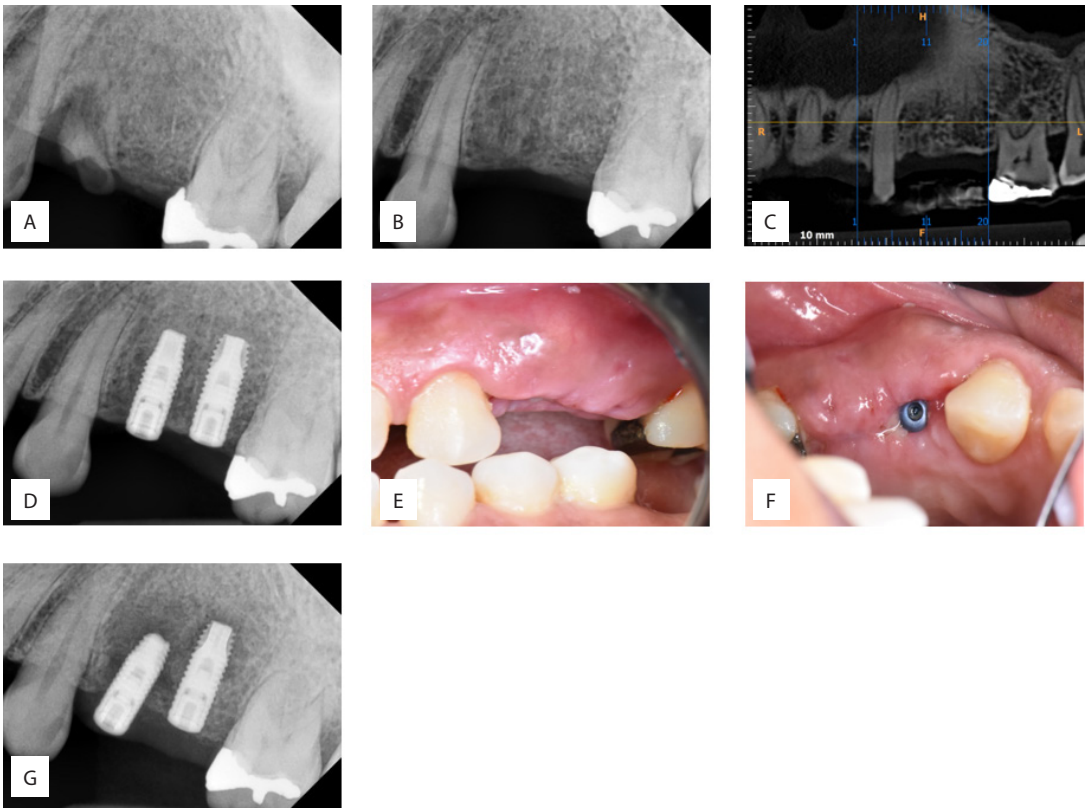

Fig. 1 (A) Periapical radiograph of retained root tooth 24 and edentulous region of tooth 25 shows no pathological periapical lesion; (B) Periapical radiograph of edentulous region of tooth 24 and 25 shows no abnormalities at 10 months after extraction of retained root tooth 24; (C) CBCT of radiographic stent shows no pathological radiolucency; (D) Periapical radiograph of implant 24 and 25 after placement shows no abnormal radiolucency; (E) Clinical (buccal) view at three weeks shows ill-defined swelling on buccal attached gingiva of tooth 24 edentulous region; (F) Clinical (occlusal) view at three weeks shows almost full exposure of cover screw on implant 24 with pus on the distal; (G) Periapical radiograph of implants 24 and 25 shows periapical radiolucency noted on implant 24 extending to mesial of implant 25 6×5 mm in diameter.

revealed that the adjacent tooth 23 and tooth 26 responded to stimuli. A periapical radiograph revealed poorly defined periapical radiolucency in the periapical regions of both implants 24 and 25 (Fig. 1G). Following discussions with the patient and periodontist, it was decided to explant both implants due to implant mobility and failure to achieve osseointegration.

After raising the flap, both implants were carefully removed with dental forceps, and the site was meticulously debrided with copious saline irrigation. The soft tissue and bone tissue specimens from the socket, were removed and sent to the laboratory for histological examination. The microscopic findings revealed a soft tissue lined by non-keratinised stratified squamous epithelium supported by fibrous connective tissue wall (Fig. 2). A diagnosis of peri-implantitis cyst was given based on the clinico-correlation. A review after a week showed uneventful healing of the site with no sign and symptom. The patient was reviewed at four months and no pathological findings were seen clinically and radiographically. Following the removal of implants, the patient was re-evaluated and a new treatment plan was discussed with patient. However, patient decided to replace the edentulous area at tooth 24 and tooth 25 with a fixed porcelain fused metal (PFM) bridge between teeth 23 and 26.

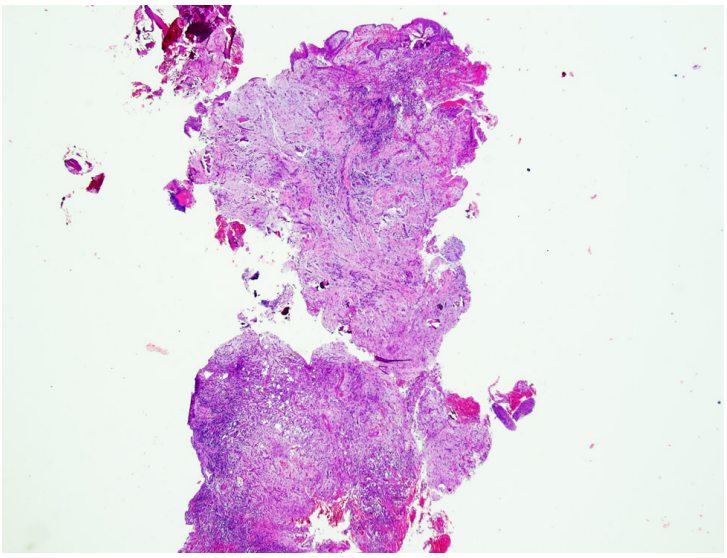

Fig. 2 Photomicrograph showing soft tissue lined by non-keratinised stratified squamous epithelium. The fibrous connective tissue wall composed of lymphocytes, plasma cells, neutrophils and macrophages (hematoxylin and eosin staining, 4×).

Case 2

A 63-year-old Malay gentleman was referred to the Periodontal Clinic for treatment of periodontitis. He was diagnosed with hypercholesterolemia and was prescribed with rosuvastatin calcium. The patient denied any smoking habit, consuming alcohol or other parafunctional habits. Periodontal examination was performed and patient was diagnosed with localised periodontitis Stage III Grade A (Tonetti *et al.*, 2018). He also complained of gum bleeding during tooth brushing and history of gum swelling on the upper right posterior tooth. He claimed that the tooth had endodontic treatment in 2010 and the porcelain fused metal (PFM) crown was issued in 2018.

Clinical examination revealed a PFM crown on tooth 15, with sinus at buccal attached gingiva of tooth 15. Deep periodontal pocket depth (PPD) of 6 mm was noted on distopalatal of tooth 15. The intraoral periapical radiograph of tooth 15 revealed crown with post, with incomplete root canal filling about 5 mm from the radiographic apex and periapical radiolucency (Fig. 3A). The clinical and radiographic findings suggested combined periodontal-endodontic lesion (Herrera *et al.*, 2018) and previously treated, asymptomatic apical periodontitis (AAE, 2009). The prognosis of tooth 15 was irrational to treat following consultation with an endodontist. The decision of irrational to treat is agreeable following the pretherapeutic single tooth prognosis by Lang & Lindhe (2015). Hence, a plan was devised to extract the tooth and replaced it with a single implant. Initial phase therapy was performed consisted of extraction of tooth 15, customised oral hygiene instruction, scaling and root debridement of teeth with PPD ≥ 5 mm. Periodontal review after eight weeks showed that the periodontal status of the patient was stable.

Four months following periodontal review, pre-operative CBCT of edentulous area of tooth 15 showed no pathological lesion (Fig. 3B). One implant was placed into the edentulous area of tooth 15 under local anaesthesia, by following the implant surgical principles and manufacturer's instructions under aseptic procedure. The type of implant used was the same as in Case 1. Implant placement protocol and post-operative analgesic were also the same as previously mentioned in Case 1.

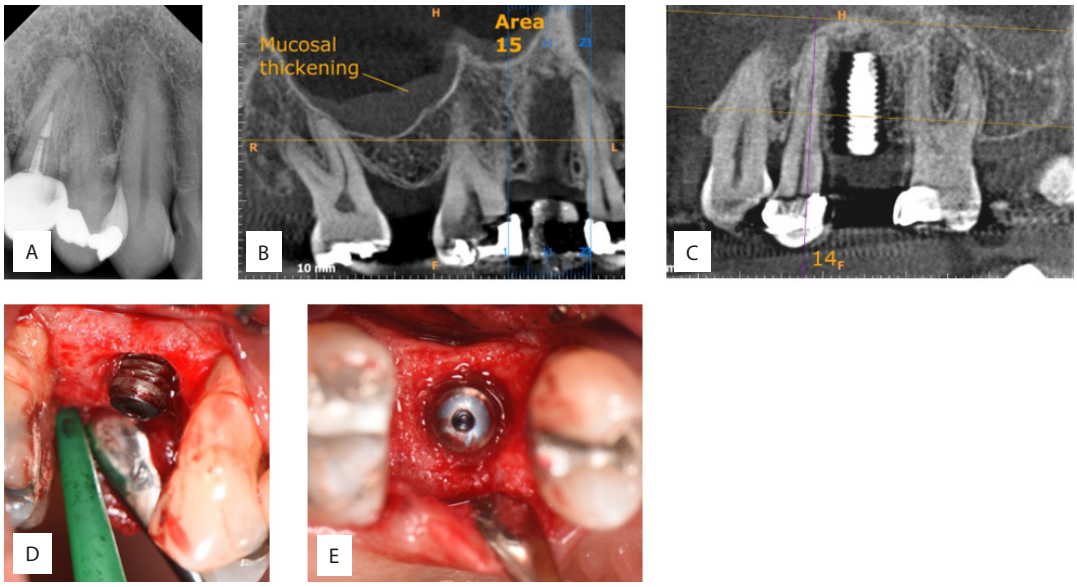

Fig. 3 (A) Periapical radiograph of tooth 15 with inadequate root filling with periapical radiolucency; (B) CBCT of edentulous tooth 15 region at four months after extraction before implant placement; (C) CBCT of implant 15 and tooth 14 showing an osteolytic region with ill-defined margin originating from disto-apical surface of tooth 14, extending to the mesial surface of implant replacing tooth 15; (D) Buccal view shows presence of gap surrounding 15 implant after flap full-thickness flap elevation and one third of implant length is exposed; (E) Occlusal view shows a gap between the implant surface and surrounding alveolar bone.

One week after implant placement, the patient complained of slight pain at the surgical area which was relieved by analgesic given. During this time, the periodontal dressing was removed, and the surgical area was cleaned with chlorhexidine mouthwash 0.12%. Suture removal was also performed.

The patient returned to the clinic one month later with a periodontal abscess on tooth 15 edentulous area. The patient claimed that the swelling increased in size but was not associated with pain. Vitality testing using EPT on tooth 14 revealed that the tooth responds to stimuli. The CBCT of implant 15 revealed persistent osteolytic region with ill-defined margin originating from disto-apical surface of tooth 14, extending to the mesial surface of implant replacing tooth 15 (Fig. 3C). The initial treatment plan included surgical debridement of the dental implant. Upon examination, bone crater presented at mesial and buccal of the implant. The implant was presented with mobility grade 2. Therefore, explantation of implant was decided due to the failure to attain osseointegration. Following soft and hard tissue curettage, the implant was removed with dental forceps. The specimens collected from the socket were sent to the laboratory for histopathology analysis. The histopathological findings of biopsied tissue showed fibrous connective tissue with diffuse infiltration of chronic inflammatory cells consisting of lymphocytes, plasma cells and macrophages (Fig. 4).

A week after explantation, no symptoms were noted, and uneventful healing achieved in both patients. Both patients were given antibiotics (Amoxicillin 500 mg three times a day and metronidazole 400 mg three times a day) for five days post-explantation. Patients were reviewed at four months and no pathological findings were seen clinically or radiographically. In Case 2, the patient decided to proceed with new implant placement to replace failed implant 15 after the area healed.

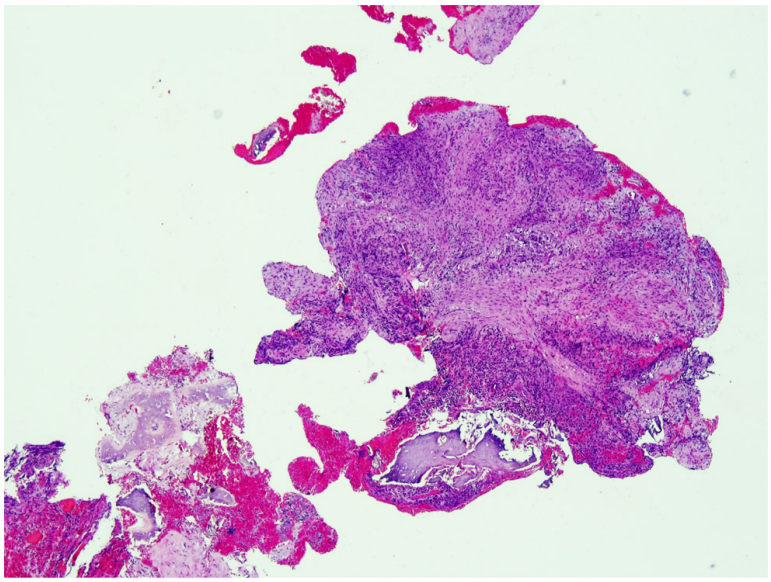

Fig. 4 Photomicrograph showing fibrous tissue having diffuse chronic inflammatory cells composed of lymphocytes, plasma cells and macrophages. No evidence of epithelial lining (hematoxylin and eosin staining, 4×).

DISCUSSION

A strict application of implant placement protocols, advancement and improved implant materials and surfaces do not necessarily guarantee a successful outcome. Although the incidence of failed dental implants is low, the clinician should consider various risk factors to achieve a successful implant placement (Alsaadi *et al.*, 2008; Sakka *et al.*, 2012). In our cases, the incidence of implant failure was observed during the early period of implant placement. Although it is a rare complication, previous studies have discussed the early shortcomings of dental implants due to endodontic pathological causes (Peñarrocha-Diago *et al.*, 2009; Romanos *et al.*, 2011; Qu *et al.*, 2014; Ramanauskaite & Machiulskiene, 2020).

IPL can be categorised into active and inactive lesions according to the activity of the infection (Reiser & Nevins, 1995). Peñarrocha-Diago *et al.*, (2009) distinguished the IPL into acute and chronic phases. Many predisposing conditions may lead to the active lesion such as infection, overheating, implant surface characteristic, poor bone quality at implant site, microfracture, foreign bodies, epithelial rest of Malassez, penetration of bone into nasal cavity and condition of the patients (Qu *et al.*, 2014). As for the infections, IPL may arise from the pre-existing infection of the implant site (Ayangco & Sheridan, 2001; Quirynen *et al.*, 2005), adjacent teeth (Scarano *et al.*, 2000; Chaffee *et al.*, 2001; Quirynen *et al.*, 2005), infected maxillary sinus (Reiser & Nevins 1995), or contamination during the surgery (Peñarrocha-Diago *et al.*, 2009). The pre-existing infection of the implant site may occur due to extraction of endodontically failed tooth (Sisli & Pektas, 2020; Di Murro *et al.*, 2021) or from residual lesions (Di Murro *et al.*, 2021). It was postulated that the bacteria from failed endodontic tooth and reactivation of residual lesion by drilling during implant placement were causing the subsequent bacteria colonisation of the implant apex (Di Murro *et al.*, 2021). Prior periapical lesion was also reported to be associated with an increased risk of early implant failure (Sisli & Pektas, 2020).

In the present case study, both patients developed signs and symptoms of inflammation at approximately three to four weeks after the implant placement. These clinical findings are similar with other previous reports (Dahlin *et al.*, 2009; Peñarrocha-Diago *et al.*, 2009; Kim *et al.*, 2015). In both cases, the symptoms of pain kept increasing until the third week and patient came with swelling and pus on the buccal aspect. Further radiological findings became apparent on the third and fourth week revealing periapical radiolucency in the periapical regions of both implants 24 and 25 in Case 1. Whereas in Case 2, periapical radiolucency was noted at the disto-apical surface of tooth 14, extending to the mesial surface of periapical of implant replacing 15. However, tooth 14 was still responsive to the electric pulp test. Thus, based on these clinical and radiological findings, we could justify the symptoms were of acute inflammatory origins based on the early symptoms (Dahlin *et al.*, 2009; Qu *et al.*, 2014; Kim *et al.*, 2015).

Few studies had investigated the histological findings of peri-apical tissues of endodontically treated teeth in animal (Rowe & Binnie, 1974), cadaver (Green *et al.*, 1997) and human (Seltzer, 1999). The studies demonstrated that although radiographs may indicate the absence of periapical radiolucency, the apex of endodontically treated tooth often exhibits histological signs of inflammation or persisting microorganisms. Rowe & Binnie (1974) reported that 61 of 129 teeth that showed no radiographic abnormality had histological apical inflammatory response. Similarly, Green *et al.* (1997) reported that there were 5 of 19 specimens showed signs of inflammation histologically, although all endodontically treated teeth showed no periapical radiolucency. Seltzer (1999) investigated the radiographic and histological findings by removing small block sections of the root apex and periapical tissues from 14 endodontically treated teeth at 12-, 18- and 30 months during periapical surgery. Histological evidence of periapical chronic inflammatory lesions was found in at least half of the specimens. Based on these findings, the deposition of bacterial colonisation with by-product endotoxins and immune response of inflammatory cells infiltrates could be presented microscopically even in the successful clinically endodontic-treated tooth. Thus, bacterial colonisation on the surfaces with the host immune response might be responsible for the early implant surface contamination that led to the implant failure in the Case 2.

In a case series, Ayangco & Sheridan (2001) reported that even after thorough debridement and irrigation of extraction sockets of failed endodontic teeth and after sufficient healing time post extraction, the bacteria would have persisted in the bone and led to the initiation of retrograde peri-implantitis (Ayangco & Sheridan, 2001). This would be the possible consequences seen in Case 2 whereby the implant was placed to replace a failed endodontically treated tooth. Difficulty to get sterile bony condition and total removal of contaminants after extraction could be the cause of re-infection after implant placement. The histological evaluation of biopsied sampling from this case was consistent with a chronic inflammatory reaction, suggesting peri-implantitis. However, there was no conspicuous evidence of bacterial infection present. In order to classify the specific type of bacterial infection, a further microbiological examination would be required if clinically indicated.

Furthermore, initiation of IPL has been suggested to be contributed by pre-existing pathologies, such as granulomas or residual cysts (Quirynen *et al.*, 2005). In Case 1, the patient had retained root of tooth 24 that might be associated with infected intracanal and apical periodontitis before the extraction commenced. The histological examination of this case revealed a cystic lesion lined by nonkeratinising epithelium with the supporting inflamed fibrous tissue. After correlation with the clinical and radiological findings, the histological features were

suggestive of a peri-implantitis-induced inflammatory cyst. Although it is rare, this could be postulated due to reactivation of remnants of epithelial rest of Malassez after initiation by prolonged inflammation which is commonly observed in other inflammatory cysts such as radicular cyst and lateral periodontal cyst.

A recent systematic review by Marshall *et al.* (2019) had assessed the histopathological and microbiological endodontic complications associated with retrograde peri-implantitis. Thirty out of 259 implants from one retrospective case-control study (Lefever *et al.*, 2013) and 5 case reports (Chaffee *et al.*, 2001; Chan *et al.*, 2011; Qu *et al.*, 2014; Shaffer *et al.*, 1998; Tseng *et al.*, 2005) had developed retrograde peri-implantitis. Among the reported aetiologies were associated with apical radiolucencies related to the adjacent tooth, existing infection at extraction site associated with failed endodontic treatment, apical periodontitis or remaining infected roots. Their findings indicate that irrespective of either the infection is originated from endodontic or periodontal, the commensal or pathogenic microbiota of the oral cavity may be involved in peri-implantitis. They highlighted the important of strict sampling procedures to avoid potential contamination during biopsy procedures. Thus, they recommended for further investigation to assess the effects of implants and the quality and quantity of microbiota in the development of retrograde peri-implantitis (Marshall *et al.*, 2019).

Poor bone quality that contributes to the scarcity of osteoprogenitor cells is another potential factor related to IPL as this condition may interfere with the formation of mineralised tissues around the implant (Scarano *et al.*, 2000). A review article by Romanos *et al.* (2011), included evaluation of 12 case reports that involved IPL without periapical pathology at the time of implant placement, vital or asymptomatic adjacent teeth, and no invasion of the adjacent tooth periodontal ligament space. Additionally, out of the 32 IPL reported, 25 were associated with maxillary implants (Romanos *et al.*, 2011). This could be related to our cases whereby both implants were placed in the maxillary arch. In addition, mandibular implants were also placed in both patients before maxillary implant placement, and the mandibular implants were survived. Therefore, placement of implants in maxillary arch may have influence on the formation of IPL in our patients.

Based on the correlation between the radiological and histological findings from these two case reports, it was necessary to exercise caution when interpreting radiographic diagnostics. The key point is to integrate all the knowledge, thorough history, and clinical examination of the patient prior to implant placement. Hence, recognition of potential periapical pathology at the implant site will be increased. Marshall *et al.* (2019) also emphasised on the need for sufficient local interventions and endodontic treatment of adjacent teeth before implant placement for implant maintenance. Although there is a trend suggesting an increased risk when placing implants in relation to an endodontic pathology the overall implant maintenance is not affected as long as adequate management is provided. However, there is a risk of implant placement in the presence of extra-radicular infection or inflammation without clinician awareness.

Additionally, early implant failures may occur during osteotomy preparation such as bone overheating, lack of primary stability due to overpreparation of the implant site and implant contamination. Early implant failure is characterised by implant mobility and loss of bone-to-implant contact. In this case, the implant can be removed by rotating the implant counter-clockwise with forceps, driver or a counter-torque ratchet (Froum *et al.*, 2011). However, in some cases, implant removal may be difficult and necessitate an invasive procedure. Various methods of implant removal can be found in the literatures such as by using tooth extraction set, trephine burs, piezo

surgery, laser surgery, counter-torque ratchet technique and electrosurgery (Solderer *et al.*, 2019). When implant removal is required, the appropriate, minimally invasive implant removal technique must be considered. As the implants were mobile in our cases, they were only removed with dental forceps.

However, not every implant with active IPL must be extracted. Sarmast *et al.* (2016) proposed a management guideline for retrograde peri-implantitis based on recommendations and successful treatment in 20 case reports. The clinician must consider whether the implant is symptomatic or asymptomatic, as well as whether the pulp status of the adjacent teeth is vital or necrotic. If the implant is symptomatic, and the adjacent teeth are vital, surgical debridement of the implant, with or without guided bone regeneration (GBR) should be performed. If the lesion does not resolve, an implant apicoectomy and GBR should be performed. The decision for implant removal needs to be evaluated during follow up visit. However, if the adjacent teeth is non-vital, root canal treatment should be performed. If the lesion persists, a tooth apicoectomy as well as surgical debridement of the implant with or without GBR should be performed. In our cases, both patients developed symptoms and an endodontic examination of adjacent teeth revealed vital pulp status. In Case 2, initial treatment plan was surgical debridement. However, upon intrasurgical examination, the implants were found to be mobile and had failed to achieve osseointegration in both cases. As a result, the implants were removed. Another systematic review by Blaya-Tárraga *et al.* (2017) recommended that implant periapical surgery should be performed in the acute and subacute stages if there is no evidence of implant stability loss. Contrarily, in the subacute stage associated with implant mobility, the implant must be removed.

Following the implant removal, another implant replacement at the same site is possible after bone healing, but a second attempt at implant placement should be approached with caution. Because a defect will be created following implant explantation, the primary stability of the replacement implant must be taken into account. Implant replacement success can be increased by improving implantation site with bone augmentation and using larger implants with improved surfaces (Alsaadi *et al.*, 2006). Other factors that may improve the outcome of reimplantation include meticulous removal of granulation tissues on the failed implant site (Grossmann & Levin, 2007).

Chrcanovic *et al.* (2017) reported a 73% survival rate for implant placed at the same site after implant failure, compared to a 94% survival rate for traditionally placed implant in the original cohort. The findings are consistent with those of another study by Grossmann & Levin (2007). In contrast, Wang *et al.* (2015) found a high cumulative survival rate of 94.4% after a mean follow up of 69.5 months in sites with initial early implant failure. According to the authors, implant replacement at the same site is possible after an adequate bone healing period.

CONCLUSION

The aetiology of implant failure caused by IPL has not been revealed yet. Pre-existed bacterial pathogenicity at implant site either by previous extracted tooth or the adjacent tooth associated with periapical disease may contribute to the implant failure. Therefore, it is essential to properly manage and treat the possible infection source prior to implant placement. Recognising IPL in the early stage before jeopardising the stable implant and surrounding bone structure will allow proper management depending on the type of infection from either active or inactive form. Further studies would be necessary to determine a definitive aetiology in order to provide a well-defined therapeutic approach for IPL.

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