Implant Periapical Lesions: Potential Etiology and Treatment

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ABSTRACT

임플란트 치근단 병소 : 잠재적 원인과 치료

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임플란트 실패의 원인 중 하나인 임플란트 치근단 병소는 많은 임상가에게 고민을 안겨 주어왔다. 지금까지 임플란트 치근단 병소의 병인론에 대하여는 별로 많이 알려져 있지 않으나, 일반적으로 다원인성으로 고려된다. 여덟가지 원인적 요소가 이 종설에서 제안되고 토의된다. 여기에는 기존의 세균성 병소의 존재, 임플란트의 오염, 수술중 골의 과열, 임플란트의 과도한 조임, 구강 전정골(vestibular bone)의 천공, 임플란트의 조기부하 및 과도한 부하, 임플란트 식립부의 불량한 골질, 임플란트 사이 또는 임플란트와 자연치 사이의 부족한 공간 등이 포함된다. 문헌을 기초로 볼 때 기존 세균성 병소의 존재 및 골의 과열과 같은 외과적 손상이 임플란트 치근단 병소의 가장 주요한 원인인 것으로 사료된다. 임플란트 치근단부의 절제, 항생제 투여, 유도 골재생술(GBR) 등의 다양한 복합 처치가 임플란트 치근단 병소의 처치를 위해 제안되어 왔다. 미래에 이 임플란트 치근단 병소의 실제 원인을 밝히는 연구가 필요하다.

Key Words : Implant periapical lesion, implant failure, microbial involvement, surgical trauma, detoxification, regenerative procedure

INTRODUCTION

Endosseous dental implants have been utilized as a successful treatment modality for the restoration of missing dentition since the introduction of the concept of osseointegration. Recently, success rates of dental implants have dramatically increased with aids of better understanding of bone response and improvement in loading concepts.[1-5] Nevertheless, there have been significant numbers

of reports that have demonstrated implant failure. The exact mechanism of implant failure has not been clarified due to the fact that various factors are attributed to the failure of dental implants, either directly or indirectly.

Up to date, an implant periapical lesion, one of possible causes of implant failure, has not been sufficiently discussed. According to *Resier* and *Nevins*, the implant periapical lesion is described as a periapical lesion which is located at the apex of a dental implant.[6] In addition, *Esposito* and his coworkers described that the implant periapical lesion is often found around long implants placed in dense bone, and the coronal portion of the implant is supported by normal bone in intimate contact with a stable implant.[7] *Misch* defined the implant periapical lesion, also termed a retrograde peri-implantitis, as a retrograde implant failure possibly due to bone microfractures caused by premature implant loading, overloading, other trauma, or occlusal factors.[8] This implant failure is characterized by periapical radiographic bone loss without, at least initially, notable gingival inflammation.

Implant periapical lesions can be classified into the inactive form and infected form.[6] The inactive form appears similar to periapical scar without characteristic clinical symptoms, resulting from a residual bone cavity created by placing an implant shorter than the prepared drill site. Although the inactive form is not considered a clinical challenge, it should be examined radiologically on periodic visits. On the other hand, the infected form occurs when an implants apex is placed in proximity to an existing infection or when a contaminated implant is placed. Bone necrosis caused by overheating and other trauma may also be considered to be an etiologic factor for the infected form. Two pathologic pathways, from implant to tooth and from tooth to implant, were described regarding the infected periapical lesion for treatment strategy.[9] It can be speculated that in both types, the resultant periapical pathology contaminates the fixture and inhibits osseointegration of the implant during healing period.

Although the incidence of implant periapical lesions is unknown, frequency of their occurrence appears to be low. In 1995, *Reiser* and *Nevins* reported 10 periapically infected implants among 3,800 implants examined.[6] That is the only report evaluating the incidence of implant periapical lesion up to date. However, the increased use of dental implants has resulted in increasing number of implant periapical lesions, as demonstrated by recent case reports in the last few years. The purposes of this paper are to address possible etiologies of the implant periapical lesions and to discuss potential treatments based on currently available literatures.

I. ETIOLOGY OF IMPLANT PERIAPICAL LESION

Various etiologic factors including microbial involvement and surgical trauma have been suggested by several authors (Table 1).[6, 10-12] Although most case reports showed the occurrence of implant periapical lesions attributed to microbial contamination from an endodontic lesion of an adjacent tooth or surgical trauma such as overheating, some case reports have not clearly revealed the etiology of the implant periapical lesion.

1. Presence of pre-existing microbial pathology

It was suggested that dental implants do not possess the ability to withstand any bacterial

Table 1. Etiologic factors of implant periapical lesions

- Presence of pre-existing microbial pathology
- Contamination of implant
- Bone overheating during implant placement (surgical trauma)
- Excessive tightening of the implant with compression of bone chips
- Premature loading and/or overloading of implant
- Fenestration of vestibular bone
- Poor quality of bone site
- Inadequate space between implants or between implant and tooth

challenge during the first stage of osseointegration, and that an endodontic pathology can travel through marrow spaces and contaminate an adjacent implant fixture.[13] This may lead to loss of the implant or potential osteomyelitis.[14,15]

Unfortunately, it is very difficult to distinguish an infected form from an inactive form. It was shown that even if resolution of periapical lesion was observed on radiograph, microorganisms might persist indefinitely.[16] In fact, it was demonstrated that 26% of specimens examined had histologic signs of inflammation despite normal radiographic findings.[17] Furthermore, histologic evidence of periapical chronic inflammatory lesions was found in at least one-half of the specimens obtained from 14 teeth that were endodontically treated 6 to 30 months prior to the sampling.[18]

Another situation that may cause the implant periapical lesion is immediate placement of an implant into a socket of a previously infected tooth (endodontically or periodontally). *McAllister et al.* claimed that even with vigorous debridement, bacteria remaining in the socket can initiate infection.[10] On the other hand, *Novaes* and *Novaes* stated that the placement of an implant into a socket with a chronic lesion does not necessarily result in failure if certain precautions, including pre-and postoperative care as well as meticulous debridement of the alveolus, are taken.[19] Locating an implant in or near a cystic cavity does not necessarily subject the implant to immediate failure, but the area may be jeopardized from the expansion of the cyst.[20] However, it has been speculated that complete removal of etiologic factors with careful and thorough debridement of the socket, in addition to the use of antibiotics, can reduce or eliminate changes resulting from bacterial contamination.

2. Contamination of implant

The implant can be contaminated due to manufacturer errors, by the operator, from non-titanium instruments, or by intraoral microorganisms. A contaminated implant surface possibly could lead to early osseodisintegration.[20]

Interestingly, autoclaving a contaminated implant will bake the bacteria onto the implant surface. Consequently, when the implant is placed in the body, it becomes almost impossible for phagocytic cells to clean off this material. This may result in implant failure because it prevents close adaptation between the implant and bone.[20] *Linkow* claimed that the implant surface should be cleaned by a radiofrequency glow discharge unit or a plasma cleaner.[21] Dental implants can be also contaminated via metal transfer from non-titanium instruments. *Weiss* stressed that all instruments that contact implants should be titanium-tipped to prevent potential metal contamination.[22] Another plausible factor that contaminates implant surface is glove powder, which acts as a film over the implant body if contact occurs.[20] Bacterial contamination on an implant surface may be possibly derived from plaque contamination while the implant is being inserted.[19] Therefore, careful plaque control should be performed before implant placement.

In a study, it was reported that implants placed under surgically "clean" conditions had the same success rate statistically as those placed under sterile conditions.[23] Although the absolute sterility is not required, or not feasible, during implant placement, a surgical condition which can prevent an implant site from contaminating through external environment needs to be maintained.

3. Bone overheating during implant placement (surgical trauma)

Minimal temperature elevation during surgical drilling of the bone is the key to the atraumatic surgical technique.[24] *Eriksson* and *Albrektsson* reported bone cell death when a temperature of 40 ℃ was applied for 7minutes or when a temperature of 47℃ was applied for 1 minute to the bone.[25] They concluded that bone density was a far greater indicator of drilling temperature than depth of the osteotomy. If bone cells are damaged due to surgical trauma, a connective tissue interface is formed between the implant and the viable bone, thus leading to a failure of osseointegration.[26]

A number of factors could be associated with bone overheating, which include factors related to the operator, manufacturer, recipient site, and patient.[27] During implant site preparation, heat generation is most likely influenced by factors related to the operator, such as pressure applied to surgical drills, drilling speed and time, and continuity of drilling, and manufacturer-related factors including sharpness of cutting tool and cooling system. In addition, bone density, the thickness of cortical bone and drilling depth could affect bone overheating.

Overheating of the apical portion of an implant bed is critical for occurrence of the implant periapical lesion. In particular, when a long implant is placed, special consideration should be employed to prevent overheating because coolants might not reach the apical portion of the bone. Some controversy exists on cooling systems, internal or external cooling system, with regard to the efficacy of overheating prevention.

4. Excessive tightening the implant with compression of bone chips

It was claimed that an excessive tightening of the implant compresses bone chips produced during bone site preparation, possibly resulting in subsequent bone ischemia and necrosis, and formation of a sequestrum.[11] *Misch* mentioned that the surgeon should not overtighten the implant in its final position since it may cause microfractures and compromise the entire implant thread-to-head profile and interface development.[28] He further claimed that the implant should not be threaded to the full depth of osteotomy with a hand rachet when thick porous bone exists, because excessive initial strains may form at the interface with even one extra rotation, causing microfractures. In the area of thick cortical bone, it was recommended to unthread one-half turn in final position to ensure that there is no residual stress along the bone-implant interface. The placement of self-tapping implants in dense bone has demonstrated a significantly higher degree of hard tissue trauma and is therefore not recommended.[29]

5. Premature loading and/or overloading of implant

Around natural teeth, periodontal mechano-receptors tune occlusal forces through the central nervous system via feedback control, consequently preventing occlusal overload.[30] However, the periodontal mechanoreceptors are lacking around

dental implants.[31]

According to *Branemark* 's protocol, a stress-free healing period of 3 to 6 months is required for osseointegration to occur.[32] *Misch* stated that at 16 weeks, the surrounding bone is only 70 % mineralized, and there is still woven bone, an unorganized, immature form of mineralization, which cannot withstand full-scale stresses.[28] It has been speculated that, once a biomechanical demand has exceeded the load-bearing ability of the bone, microfracture of the bone at the implant interface can occur. If bone fatigue and micro-damage accumulate at a slow rate, the normal bone turn-over is able to repair them. On the other hand, if microdamages accumulate faster than they can repair, a fatigue fracture at the bone-implant interface may result.[33]

Premature and/or overloading may tend to result in crestal bone loss or failure of osseointegration[34] rather than occurrence of the implant periapical lesion. Also, there has not been any report that micromovement in an immediately loaded implant causes the implant periapical lesion.[12] Hence, premature and/or overloading cannot be considered a major cause of the implant periapical lesion.

6. Fenestration of vestibular bone

Fenestration of vestibular bone is characterized by a perforation in the facial or lingual alveolar cortical plate which does not communicate with the crestal marginal bone. Implants placed in sites with bony concavities (i.e. canine fossa, submandibular, and sublingual areas) have sometimes been associated with fenestration defects. Fenestration defects can induce dehescience defects if the remaining marginal bone isthmus is subsequently lost.[35] *Piattelli et al.* speculated that if the cortical bone is thinner than 0.5 mm, a cortical bone dehiscence may develop due to insufficient bone remodeling capacity of the area, increasing chances of soft tissue infection.[11] This possibly causes an implant periapical lesion.

7. Poor quality of the bone site

Increased implant failure rates have been reported in the bone with poor quality.[36] The scarcity of osteoprogenitor cells due to poor bone quality at the surgical site can produce, most probably, a negative influence on the formation of mineralized tissues around the implant.[11] Although systemic diseases which are related to impaired bone remodeling such as osteoporosis, irradiation therapy and bone diseases may not be contraindicated, strict attention is necessary to minimize the risk of implant failure.

Regarding the implant periapical lesion, poor bone quality does not explain the occurrence of the lesion in the anterior mandible,[12] and there have been no reports showing a direct link between the occurrence of the implant periapical lesion and poor bone quality.

8. Inadequate space between implants or between implant and tooth

In general, it has been believed that there should be a certain bone thickness between neighboring implants and between implant and tooth to allow sufficient blood supply and consequently proper bone remodeling on those areas. *Tarnow et al.* found less radiographic crestal bone loss on implants with more than 3 mm of a distance between implants, as compared to implants with a distance of 3 mm or less (0.45 mm versus 1.04 mm).[37] *Askary et al.* stated that highly dense bone requires more space between implants, as

compared to cancellous bone, in order to avoid overheating with subsequent death of bone cells.[20] Although literature has revealed the importance of a certain distance between implants and/or between the implant and tooth, its consequence is not limited to the implant periapical region.

II. POTENTIAL TREATMENT

The treatment strategy for implants with periapical lesions is dependent upon the etiology individualized. Although stable asymptomatic inactive forms would not require any specific treatment, periodic monitoring of the lesion, especially with radiographic assessment, should be followed. On the other hand, infected lesions around stable implants may need to be treated aggressively with a combination of antibiotics and surgical therapy. However, the treatment of failing implants is still based mainly on empirical consideration, which is often derived from in vitro findings, in vivo animal research, or anecdotal case reports performed on a trial-and-error basis.

Surgical approaches for the treatment of implant periapical lesions have been advocated by several authors. *Sussman* stated that an infected implant should be immediately removed to prevent an osteomyelitis since retention of an implant with an osteitis can lead to irreversible bone loss.[9] In addition, it was also recommended that a mobile implant must be removed immediately.[6]

Resection of infected implant apices (implant apicoectomy) may be considered as a treatment modality for the treatment of the implant periapical lesion if the lesion is localized to the apex. It would be possible to eliminate the implant periapical infection and salvage the implant if : (1) the implant is stable (osseointegrated) ; (2) the infection remains apically compartmentalized; and (3) the implant is of a sufficient length to allow for removal of its apical portion without jeopardizing stability. When implant apicoectomy is performed, the implant body should be horizontally sectioned at the most coronal aspect of exposed threads in the apical region, removing the apical end of the implant. It appears to be unnecessary to prophylactically remove or resect an implant if the periapical area is small and inactive.[6]

A combined surgical therapy has been suggested for the treatment of the implant periapical lesion, including surgical exposure, degranulation of the defect, detoxification of the implant surface and regenerative procedures.[10] Generally, a full thickness flap, which provides good visibility for pathoses, is applied for surgical exposure of an infected implant. However, a semilunar flap, which can preserve the marginal tissue and avoid anatomic vital structures, may also be applied.[12] Complete debridement of the lesion is the most critical factor for the treatment of implant periapical lesions. The apex of implant can be sectioned if it prevents thorough and complete debridement.[6, 12] *Balsh et al.* proposed an extraoral approach for surgical access, preserving the implant and integrity of final prosthesis for the treatment of an abscess around the apex of a mandibular implant.[38] Although the extraoral approach can provide good access to an infected site, its application is limited due to resultant external scar.

Guided bone regeneration (GBR) can be used for the restoration of hard tissue lost due to the periapical lesion. In an experimental histological study in dogs, evidence of "re-osseointegration" was demonstrated by use of GBR.[39] In the study, abundant lamellar bone formation was histologically observed beneath barrier membranes,

2 months after GBR procedures to treat peri-implant defects induced by plaque accumulation.

Detoxification of the implant surface with a chemotherapeutic agent is often concomitantly used with GBR in the treatment of a failing implant due to the periapical lesion. Chemotherapeutic agents, including chlorhexidine gluconate, stannous fluoride, tetracycline hydrochloride, hydrogen peroxide, citric acid, polymycin B, and chloramines T have been used for the detoxification of the surface of failing implants.[40] It has been suggested that " infected" surfaces of HA-coated implants should be cleaned with citric acid (pH 1) for 30 seconds to 1 minute.[41] There have also been studies proposing chemotherapeutic detoxification for the treatment of failing implants, by use of chloramines T solution[42] and tetracycline.[10, 40, 43, 44] Chlorhexidine gluconate may be of a greater benefit for the detoxification of implant surfaces since it has antimicrobial efficacy as well as substantivity. However, well-controlled clinical investigation has been lacking with regard to the effectiveness of these detoxifixation agents.

GBR can be facilitated by an adjunctive use of various osteoinductive and osteoconductive materials, such as FDBA,[6, 10] DFDBA,[45, 46] particulate bovine bone[12] and demineralized bone matrix,[12] in the restoration of implant periapical lesions. For the choice of membrane types, an absorbable membrane such as collagen membrane may be preferred since the site does not need to be reentered.[12]

In cases of suppurative peri-implant infection, the use of specific systemic antibiotics against anaerobic microorganisms, such as metronidazole, is often recommended.[47] It has been proposed that the administration of a combination of amoxycillin and metronidazole for 10 days would be beneficial for the treatment of peri-implant infection.[48] However, antibiotic administration alone is unlikely to be successful because of the difficulties in eradicating bacterial colonies from the implant periapical lesion.[49] After surgical treatment of the periapical lesion, a non-steroidal anti-inflammatory agent in combination with antibiotic therapy can be employed to the patient to further surpress the microbiota not removed by clinical procedures, and to reduce inflammation and enhance the host response.[50]

CONCLUSION

Little is known on the etiopathogenesis of the implant periapical lesion. It is apparent that the implant periapical lesion has a mutifactorial background, including presence of pre-existing microbial pathology, contamination of implant, bone overheating during implant surgery, excessive tightening of the implant with compression of bone chips, premature loading and/or overloading of implant, fenestration of vestibular bone, poor bone quality of implant site, and inadequate space between implants or between implant and tooth. Based upon currently available literature, microbial involvement of pre-existing pathoses and surgical trauma such as bone overheating may be the most likely causes of the implant periapical lesion.

Appropriate endodontic therapy and removal of potential sources should be performed prior to implant placement to prevent implant periapical lesions. If healing of the lesion could not be achieved by conventional endodontic therapy, apical surgery or extraction should be considered. In addition, meticulous removal of potential infection sources such as granulation tissue, root

fragments, foreign bodies, and periodontal or periapical infection in the proposed implant site is critical for implant success, especially when an immediate implant is to be placed in a fresh extraction wound. To prevent overheating during implant osteotomy in dense bone, recommended surgical protocol, including an effective cooling system and incremental drilling, should be applied.

For the treatment of the implant periapical lesion, various combined therapies can be utilized, including surgical exposure, degranulation of the defect, detoxification of the implant surface with chemotherapeutic agents, antibiotic usage, and GBR. In addition, it is necessary to differentiate an infected lesion from an inactive form before surgical approach. The optimal implant therapy would be to minimize the occurrence of implant periapical lesions by careful diagnosis and treatment planning.

There has been little evidence on the mechanism of the occurrence of implant periapical lesion; in addition, causality of etiologic factors of the implant periapical lesion remains unclear. Therefore, randomized well-controlled studies are needed to unveil the true mechanism of the implant periapical lesion.

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