치의과학박사 학위논문

Effects of adjacent periodontitis on osseointegrated implants

인접한 치주염이 골유착된 임플란트에 미치는 영향

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서울대학교 대학원

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2023년 7월

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Abstract

Purpose: This study aimed to investigate whether newly induced periodontitis or apical periodontitis on the adjacent teeth affects osseointegrated dental implants in a beagle dog model.

Materials and Methods: The mandibular second and fourth premolars on both sides of three beagles were extracted. Two months after extraction, four SLA (sandblasted with large grit and acid-etched) implant fixtures, with an average surface roughness of Ra 2.0-3.0 μm, were placed at the bone level in the edentulous area, two on each side. Six weeks after implant surgery, healing abutments were connected. After sufficient osseointegration, plaque control was performed in the control group, while periodontitis and apical periodontitis were induced in the experimental groups on adjacent teeth. The beagles were euthanized for histological analyses five months after induction of experimental periodontitis. Statistical analyses were performed using the Kruskal–Wallis test with Bonferroni correction to compare the three groups.

Results: The implants in the control and apical periodontitis groups were well-maintained, while those in the periodontitis group

showed clinical signs of inflammation with bone resorption. The bone-to-implant contact (BIC) values in the periodontitis group (mesial, 42.46% ± 28.82%; distal, 46.56% ± 33.20%) were lower than those in the control group (mesial, 65.49% ± 10.50%; distal, 74.82% ± 7.91%) and apical periodontitis group (mesial, 67.97% ± 9.35%; distal, 75.26% ± 9.86%) but the difference was not statistically significant ($P > 0.05$). Similarly, the bone area values (BA) in the periodontitis group (mesial, 29.77% ± 29.94%; distal, 17.84% ± 33.23%) were lower compared to those in the control group (mesial, 65.89 ± 24.49; distal, 66.95% ± 4.33%) and the apical periodontitis group (mesial, 69.31% ± 21.40%; distal, 69.20% ± 12.76%), but there was no statistically significant difference ($P > 0.05$). The distance between the implant shoulder and the first BIC was significantly greater in the periodontitis group (distal, 4.14 mm ± 3.12 mm) than in the control group (distal, 0.06 mm ± 0.13 mm) ($P < 0.05$).

Conclusion: Unlike apical periodontitis, the presence of periodontitis in adjacent teeth can pose a risk to dental implants, potentially resulting in peri-implantitis. Within the limitations of this study, periodontal care is necessary due to the effects of periodontitis in adjacent teeth on osseointegrated implants.

Keyword: Apical periodontitis, Dental implant, Osseointegration, Periodontitis, Periodontal maintenance

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I. Introduction

Osseointegration of dental implants results in stable anchorage with direct bone-to-implant contact [1]. The concept of osseointegration was first proposed by Brånemark et al., who observed firm anchorage of intra-osseous titanium implants in the rehabilitation of masticatory function in dogs [2]. Although the long-term survival rate of implants has been reported to be over 95% [3, 4], a number of factors can cause implant failure. These failures are classified into early and late failures depending on whether they occur before or after the development of osseointegration [5]. Early implant failures occur before or at abutment connection because of inadequate osseointegration due to interference in the healing process [6]. The main reason for such failures is the predominance of fibrous tissue formation between the implant surface and surrounding bone over osseointegration [7-11]. According to a prospective multicenter study on dental implants in partially edentulous patients, early failures were clustered in patients with high dental plaque and gingivitis indices [12]. Among the various etiologic factors of early failure, microorganisms are a common cause of failure of osseointegration [12-14]. Infections and inflammatory process adjacent to an integrating implant may

interfere with osseointegration [14]. For late implant failures, the most common etiologic factor is peri-implantitis [15], and implants with peri-implantitis reveal microbiota encompassing pathogens associated with periodontitis, which includes members of the red complex species (*Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythia*) and orange complex species (*Fusobacterium sp.* and *Prevotella intermedia*) [16].

Several previous studies have reported that patients with a history of periodontitis may show more implant loss due to greater marginal bone loss and peri-implantitis than patients without a history of periodontitis [17-19]. Levin et al. reported a prospective cohort study that revealed an 8-fold higher incidence of late implant failure in patients with severe periodontitis compared to periodontally healthy patients [20]. Furthermore, we have previously shown that untreated experimental periodontitis was correlated with compromised osseointegration of dental implants in a beagle dog model. Higher rate of early implant failure was observed in the experimental groups with induced experimental periodontitis compared to the control groups receiving oral hygiene care [21]. Conversely, the occurrence of periodontitis after achieving osseointegration of a dental implant may also impact implant survival. However, to the best of our knowledge, this study

is the first experimental study on this topic.

Furthermore, the existence of periapical pathology and history of endodontic infections may compromise successful osseointegration of dental implants [22]. Several studies have reported impaired osseointegration in the periapical region of dental implants adjacent to teeth with periapical lesions [23–25], and the term “retrograde peri–implantitis”, is commonly used to refer the lesion in the periapical region of dental implant [26–28]. Retrograde peri–implantitis is defined as a symptomatic lesion presenting with progressive bone loss at the apex of the implants with intact peri–implant bone in the coronal portion, which is considered as a distinct condition from peri–implantitis [26]. Even though multiple etiological factors such as excess heat during osteotomy [25], the presence of a pre–existing microbial pathology [29], and residual lesions of extracted teeth [27] have been suggested, periapical lesions on neighboring teeth have been frequently mentioned [23]. While endodontic infection in adjacent teeth is considered the most common potential etiology regarding retrograde peri–implantitis [23], our understanding of its etiology remains incomplete [24]. Additionally, there is a limited availability of experimental studies addressing this topic.

Therefore, this study attempted to investigate whether

induced periodontitis or apical periodontitis of adjacent teeth affects osseointegrated dental implants in a beagle dog model.

II. Materials and Methods

One control group and two experimental groups, namely, the periodontitis and apical periodontitis groups, were defined based on the presence of experimental periodontitis or apical periodontitis. One beagle dog was randomly assigned to each group.

1. Animals

This study was approved by the KNOTUS Institutional Animal Care and Use Committee of Incheon, Republic of Korea (KNOTUS IACUC 21-KE-1015). The animal experiments were performed in accordance with the principles of the 3Rs (Replacement, Reduction, and Refinement) and the Animal Research: Reporting of In Vivo Experiments (ARRIVE) guidelines. Three male beagles aged 1–2 years and weighing 11–12 kg were used for the study. The beagle dogs were fed appropriately under standard laboratory conditions with ad libitum access to water and housed individually at an ambient temperature of 23 ℃ ± 3 ℃ and relative humidity of 55% ± 15%. The entire surgical procedure was performed under general anesthesia induced using intravenous alfaxalone 3 mg/kg (Alfaxan; Jurox, Kansas City, USA) and maintained with isoflurane 1% to 3% (Terrell; Kyongbo

Pharmaceutical, Ansan, Republic of Korea). Local anesthesia at the surgical sites was induced by injecting 2% lidocaine hydrochloride with 1:100,000 epinephrine.

2. Surgical procedures

The experimental schedule and the surgical procedure are presented in Figs. 1 and 2, respectively. Four implants were assigned to each of the three groups (control group, n=4; periodontitis group, n=4; apical periodontitis group, n=4).

The mandibular second (2P2) and fourth premolars (4P4) on both the left and right sides were extracted (Fig. 2a). The teeth were cut in a buccolingual direction in the furcation region using a high-speed handpiece with a diamond point bur. The roots were extracted individually to minimize damage to the alveolar bone. Eight weeks after extraction, an incision was made in the midcrestal area in the edentulous sites on both the left and right sides, and full-thickness flaps were raised. Four implants, with two fixtures on each side, were placed in the edentulous area of each beagle dog, specifically in the middle between the mandibular first premolar (1P1) and the mandibular third premolar (3P3), and in the middle between the mandibular third premolar (3P3) and the mandibular first molar (1M1) (Fig. 2b). The implants used for this study were

internal-type bone-level implants (TSIII; Osstem Implant Co., Seoul, Korea) with a diameter of 3.5 mm and length of 8.5 mm. The surface of the implants was sandblasted with large grit and acid-etched (SLA), with an average surface roughness of Ra 2.0-3.0 μm. The cover screws were connected to the fixtures and the flaps were sutured with 5-0 nylon (Ethilon; Ethicon, Cornelia, USA). After 8 weeks of healing, full-thickness flaps were raised, and the cover screws were disconnected. Healing abutments with a diameter of 4.0 mm and a length of 3.0 mm were connected to the fixtures (Fig. 2c). All implants in the three groups showed successful osseointegration, and no early implant failure was observed.

3. Induction of experimental periodontitis and apical periodontitis

Ten weeks after implant placement, experimental periodontitis and apical periodontitis were induced in the experimental groups and oral hygiene care was provided to the control group. In the control group, oral hygiene care with scaling and plaque control procedures was performed monthly, and healthy periodontal conditions were confirmed clinically (Fig. 2d) and

radiographically (Fig. 3a). Experimental periodontitis was induced using gingival retraction cords to allow plaque to accumulate on teeth. According to an experimental study conducted by Lindhe et al., it was observed that periodontitis could gradually develop from a healthy periodontium simply by allowing plaque to accumulate on the teeth of beagle dogs [30]. In addition, gingival retraction cords were soaked in a suspension of *Porphyromonas gingivalis* (*P. gingivalis*, ATCC 33277) [31]. *P. gingivalis*, an anaerobic, gram-negative, nonmotile rod, is significantly associated with chronic periodontitis [32]. *P. gingivalis* produces virulence factors, including lipopolysaccharide (LPS), fimbriae, outer membrane proteins, and metabolic end products, which contribute to its pathogenicity and disease progression in chronic periodontitis [33]. Gingival retraction cords were ligatured at the cervical area of the mandibular first premolar (1P1), third premolar (3P3), and first molar (1M1), and packed into the gingival pocket. The condition of the retraction cords was routinely checked, and *P. gingivalis* was applied monthly. After approximately 20 weeks, experimental periodontitis was confirmed on the basis of clinical signs of gingival inflammation (Fig. 2e) and radiographs showing alveolar bone loss (Fig. 3b).

Using methods of previous studies on periapical lesion

model [34, 35], experimental apical periodontitis was induced at 1P1, 3P3, and 1M1 on both sides of the mandible. Balto et al. reported periapical bone resorption in a mouse model through surgical pulp exposure, followed by bacterial infection with a combination of four pulpal pathogens: *Fusobacterium nucleatum*, *Prevotella intermedia*, *Peptostreptococcus micros*, and *Streptococcus intermedius* [34]. Using a similar method, Oseko et al. examined the formation of periapical lesions by infecting surgically exposed pulp with *P. gingivalis* and *P. intermedia* [35]. For this study, the pulp was exposed using a carbide round bur, and a suspension of *P. gingivalis* was injected into the pulp and sealed with a temporary restorative material (Caviton; GC, Tokyo, Japan) (Fig. 2f). The procedure was performed monthly until the periapical lesion could be clearly verified on periapical radiographs (Fig. 3c). The beagles were euthanized 20 weeks after the induction of experimental periodontitis and apical periodontitis.

4. Histologic examination and histometric analysis

The mandibles of the beagles were retrieved and placed in 10% neutral buffered formalin. Tissue blocks, each containing the implant and surrounding soft and hard tissues, were prepared using a diamond saw (Exakt; Kulzer, Germany). Ground sectioning was

performed according to previously described methods [36]. The specimens were dehydrated in increasing concentrations of ethanol and embedded in acrylic resin (Technovit 7200 VLC resin; Kulzer, Germany). Each block was sectioned mesiodistally parallel to the implant axis. Two sections were obtained near the center of the implant, and each section was reduced to approximately 50 μm by microgrinding. One was stained with Masson–Goldner's trichrome and the other with hematoxylin and eosin. Digital images of the sections were obtained using a digital slide scanner (Panoramic 250 Flash III; 3DHistech, Hungary). Histological and histomorphometric analyses were performed using image analysis software (CaseViewer; 3DHistech, Hungary and Image-Pro Plus; Media Cybernetics, USA). The following data were obtained from the mesial and distal sides of each implant: 1) the percentage of bone-to-implant contact (BIC) from the first BIC (fBIC) at the coronal part of the implant to the bottom (Fig. 4a); 2) the percentage of bone area (BA) surrounding the implant in the region of the coronal 3.0 mm (Fig. 4b); and 3) the distance between the implant shoulder (IS) and fBIC (IS-fBIC) (Fig. 4c).

5. Statistical analysis

Mean values and standard deviations were calculated for

each group. A normal distribution could not be assumed because of the sample size. The Kruskal–Wallis test, a nonparametric method, was performed to analyze the overall difference among the three groups, with Bonferroni correction to identify specific group pairs that have significant differences while controlling the Type I errors during multiple comparisons. The significance level was set at a p-value of less than 0.05. Statistical analyses were performed using SPSS version 25.0 (IBM Software, Armonk, NY, USA).

III. Results

1. Clinical findings

In the control and the apical periodontitis groups, all implant sites showed uneventful healing without significant inflammation (Fig. 2d and 2f). However, in the periodontitis group, the implants showed signs of inflammation with gingival swelling, redness, and pus discharge on the peri-implant mucosa (Fig. 2e). At the time of euthanasia, one of the implants in the periodontitis group showed complete loss of the surrounding bone and had spontaneously fallen out.

2. Histologic findings

Direct contact of the bone with the implants was observed in all groups (Figure 5). Typical trabecular bone patterns surrounding the implants were observed in all groups, and osteocytes were embedded in the lacunae. No marked inflammatory cells were detected in the control (Figure 5a) and apical periodontitis (Figure 5c) groups. Furthermore, the marginal bone was intact for most of the implants in the control (Figure 5a) and apical periodontitis (Figure 5c) groups. However, the periodontitis group showed

prominent marginal bone loss and infiltrated connective tissue (Figure 5b, 5d) as well as complete loss of the surrounding bone near the failed implant.

3. Histometric analyses

The BICs in the periodontitis group (mesial, 42.46% ± 28.82%; distal, 46.56% ± 33.20%) were lower than those in the control group (mesial, 65.49% ± 10.50%; distal, 74.82% ± 7.91%) and apical periodontitis group (mesial, 67.97% ± 9.35%; distal, 75.26% ± 9.86%), although the differences were not statistically significant (Table 1). Likewise, the BAs in the periodontitis group (mesial, 29.77% ± 29.94%; distal, 17.84% ± 33.23%) were lower than those in the control group (mesial, 65.89 ± 24.49; distal, 66.95% ± 4.33%) and the apical periodontitis group (mesial, 69.31% ± 21.40%; distal, 69.20% ± 12.76%); however, the differences were not statistically significant. The distal IS–fBIC value in the periodontitis group (distal, 4.14 mm ± 3.12 mm) was significantly higher than that in the control group (distal, 0.06 mm ± 0.13 mm) ($P < 0.05$). The BIC and BA of the failed implant were both considered 0%, while its IS–fBIC was counted as 8.5 mm for the statistical analyses.

IV. Discussion

In the present study, the osseointegrated implants adjacent to teeth with experimentally induced periodontitis showed progressive bone loss along with inflammation on the peri-implant mucosa, while apical periodontitis did not have any notable effect on the peri-implant supporting bone during the experimental period. The average IS-fBIC values in the periodontitis group were the highest, and the distal IS-fBIC value in the periodontitis group was significantly higher than that in the control group. In the histological assessments, one of the osseointegrated implants in the periodontitis group failed due to progressive loss of the supporting bone. These results indicate that periodontitis induced in adjacent teeth can further affect the implant, resulting in peri-implantitis.

One possible explanation for these findings could be the transmission of periodontopathic microbiota from teeth adjacent to implants. Based on their findings, Quirynen et al. proposed that the gingival crevices surrounding the remaining teeth in partially edentulous patients serve as "reservoirs" for the colonization of newly placed abutments on implants. This conclusion is supported by the lack of significant differences in the distribution of bacterial morphotypes around natural teeth and titanium implants observed in

their intra-subject comparison [37]. In other study investigating the correlation between periodontopathic bacteria in periodontal pockets and implant sulcus within individuals using polymerase chain reaction (PCR) detection, the authors reported a statistically significant correlation in the colonization of *P. gingivalis* and *A. actinomycetemcomitans (A.a)* between the periodontal pockets and the implant sulcus [38]. Another study reported that the colonization of periodontopathic bacteria such as *A.a*, *F. nucleatum*, *Prevotella intermedia*, *P. gingivalis*, and *Treponema denticola* at the implant sulcus was associated with the microbiota in the gingival crevice of the adjacent teeth instead of the contralateral and occluding teeth [39]. Our results showing that peri-implantitis was induced at implants adjacent to the teeth with periodontitis but not in those in the control group is in line with the findings of previous studies regarding the transmission of microorganisms.

Regarding a history of periodontitis as a risk factor for peri-implantitis, Lindhe & Meyle recommended informing patients about the elevated risk of peri-implantitis, addressing periodontal disease before implant placement, and providing appropriate periodontal care [40]. The results of this experimental study support the notion. Stacchi et al. conducted a systematic review, to evaluate whether the history of periodontitis increases the risk of peri-implantitis

[41]. The study included three prospective studies with more than three years of follow-up, comparing patients with periodontal disease to periodontally healthy individuals [19, 42, 43]. Both implant-based and patient-based meta-analyses showed a significantly higher risk in patients with periodontitis, but more long-term prospective studies with large subjects were needed due to limited evidence [41].

This study aimed to investigate whether the presence of periodontal or endodontic infection in adjacent teeth can affect osseointegrated dental implants, and the results from the periodontitis groups supported the hypothesis. Additionally, prior findings from our research group, as reported by Lee et al., provided evidence of a significant correlation between untreated experimental periodontitis and compromised osseointegration of dental implants in a beagle dog model. Among the 12 implants in the experimental group with induced periodontitis, four implants (three from the immediate implant placement group and one from the delayed implant placement group) failed to achieve osseointegration after placement, while no failures were observed in the control group where plaque control procedures were implemented [21]. These findings suggest that the presence of microbial infection caused by periodontitis can pose a risk to dental implants both

before and after osseointegration.

On the other hand, implants immediately placed after teeth extraction with periodontal or endodontic infections demonstrate a high survival rate when appropriate clinical procedures, such as meticulous cleaning and socket debridement, are implemented [22]. In a retrospective study by Bell et al., involving 922 implants, dental implants placed immediately into extraction sites with chronic periapical infections after curettage and irrigation of the periapical lesions had a success rate of 97.5%, while implants placed in sockets without signs of periapical infections exhibited a success rate of 98.7%, but the difference in success rates between the two groups was not statistically significant [44]. Crespi et al. conducted a prospective 4-year study to compare the outcomes of immediate loading of dental implants placed immediately in teeth extraction sites, with and without chronic periodontal lesions. Prior to dental implantation, antibiotic administration, meticulous cleaning, and alveolar debridement procedures were performed. At the 48-month follow-up, the survival rates of 197 implants placed in periodontally infected sites and 78 implants placed in non-infected sites were 98.9% and 100%, respectively, with no statistically significant difference observed between the two groups [45].

The lack of significant difference between the periapical

periodontitis group and the control group contradicts the findings of several studies that suggest periapical lesions on adjacent teeth of retrograde peri-implantitis as a potential etiological factor. Sarmast et al. reviewed 20 case reports, which involved a total of 95 dental implants affected by retrograde peri-implantitis. According to the authors, the most frequently suggested possible etiology was an endodontic infection in the adjacent tooth [23]. In a retrospective analysis reported by Lefever et al., the prevalence of retrograde peri-implantitis in implants without endodontic pathology in adjacent teeth was found to be 1%. However, when endodontic pathology was present in adjacent teeth, the percentage increased to 25%, with an odds ratio of 8.0 [46]. Another retrospective study reported a prevalence of 7.8% with implants placed adjacent to endodontically treated teeth and higher incidence of retrograde peri-implantitis in cases where the distances between implants and adjacent teeth were shorter (<2 mm), suggesting that the presence of pre-existing inflammation in adjacent teeth could potentially lead to retrograde peri-implantitis In other words, the study suggests that maintaining a minimum space of 2 mm between the implant and adjacent tooth is recommended to decrease the occurrence of retrograde peri-implantitis [47]. However, in this present experiment, the distance between periapical periodontitis and

neighboring implant was not taken into consideration, and this could be a possible reason why the results of the study showed no correlation between periapical lesions and retrograde peri-implantitis. Regrettably, the underlying mechanisms were not investigated in this study, and we hope to address this topic in future studies.

Additionally, a small sample size was allocated for each group, with only four implants per beagle in each group. Low statistical power resulting from insufficient sample sizes can compromise the reliability of statistical analysis, making it difficult to interpret the results accurately [48]. Therefore, it is important to consider the limitations imposed by the sample size and approach the interpretation of results with caution.

V. Conclusion

Experimentally induced periodontitis in adjacent teeth significantly affects osseointegrated implants, resulting in progressive bone loss along with inflammation of the peri-implant mucosa. On the other hand, experimentally induced apical periodontitis in adjacent teeth had no effect on osseointegrated implants during the experimental period. Within the limitations of this study, proper periodontal care is necessary due to the effects of periodontitis in adjacent teeth on osseointegrated implants.

VI. Table and figures

Table 1. BIC, BA, and IS−fBIC values

	BIC (%)	BA (%)	IS-fBIC (mm)
Control			
Mesial	65.49±10.50	65.89±24.49	0.08±0.09
Distal	74.82±7.91	66.95±4.33	0.06±0.13*
Periodontitis			
Mesial	42.46±28.82	29.77±29.94	3.50±3.57
Distal	46.56±33.20	17.84±33.23	4.14±3.12*
Apical periodontitis			
Mesial	67.97±9.35	69.31±21.40	0.21±0.17
Distal	75.26±9.86	69.20±12.76	0.18±0.18

BIC, Bone−to−Implant Contact from the first BIC (fBIC) at coronal part of implant to bottom

BA, Bone Area surrounding implant in the region of the coronal 3.0 mm

IS−fBIC, Distance between Implant Shoulder (IS) to the fBIC

* P−value less than 0.05 by post hoc comparison using Kruskal−Wallis test with Bonferroni correction

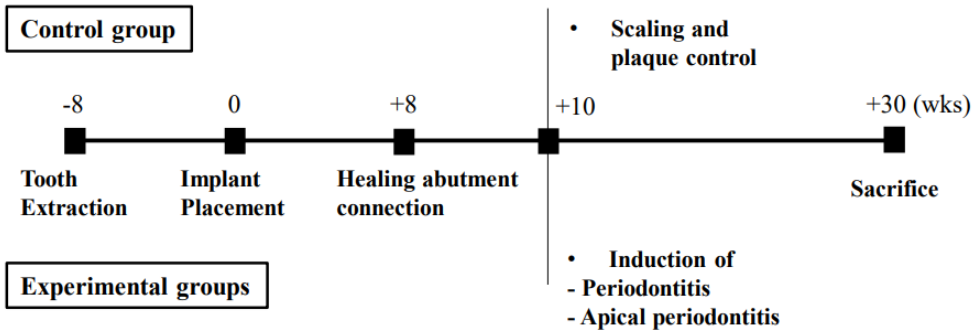


Figure 1. Experimental procedure

Implants were placed eight weeks after tooth extraction in all groups. Healing abutments were connected at 8 weeks after implant placement in all groups. After two weeks of healing, abutment connection, scaling, and plaque control were performed monthly in the control group, and experimental periodontitis and apical periodontitis were induced in groups 2 and 3, respectively. The induction procedures were conducted monthly until the lesion could be clearly verified on periapical radiographs. All beagles were euthanized 20 weeks after the induction of periodontitis.

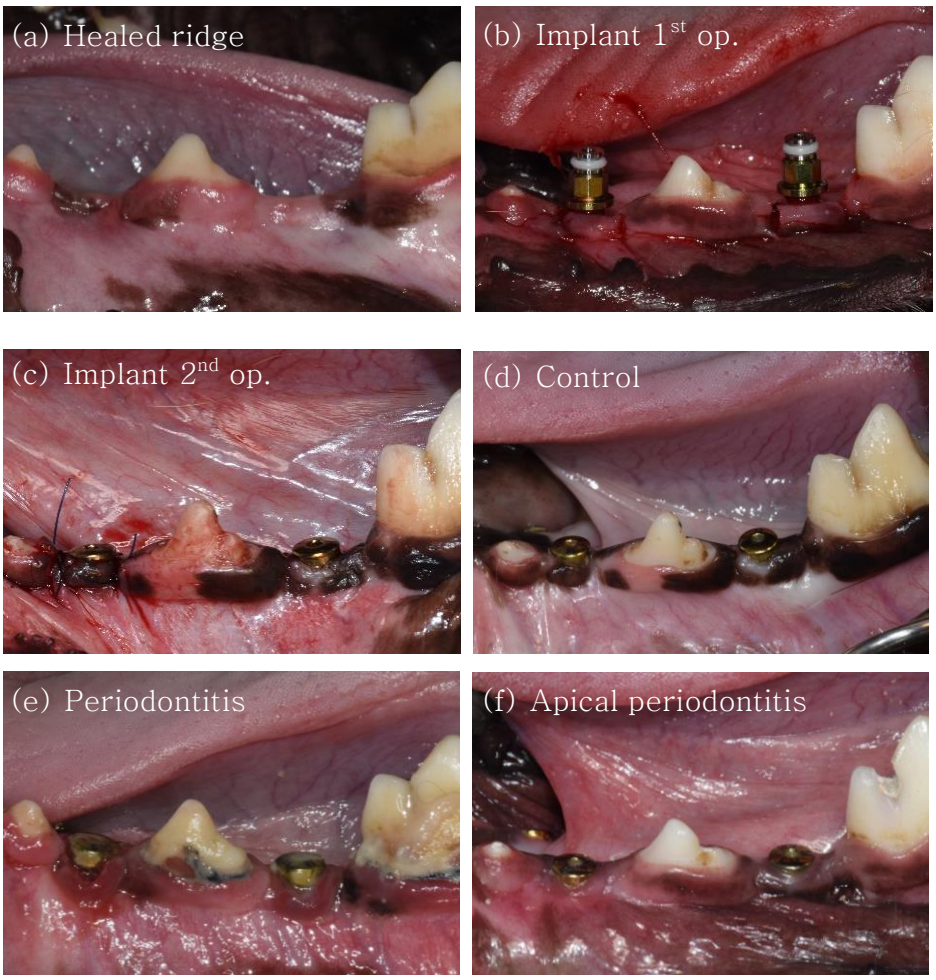


Figure 2. Clinical photos of the experimental procedure

(a) At 8 weeks after tooth extraction, the ridge had healed well. (b) The first implant operation to place the fixtures was performed in the healed ridge. (c) At 6 weeks after implant placement, the second implant operation to connect healing abutments was performed. (d–f) At 2 weeks after healing abutment connection, scaling and plaque control were performed monthly in the control group, and experimental periodontitis and apical periodontitis were induced in the experimental groups. The induction procedures were

conducted monthly until the lesion could be clearly identified on periapical radiographs. At 20 weeks after induction of the experimental conditions, the control (d) and apical periodontitis (f) groups showed a favorable gingival condition, but the periodontitis group showed gingival inflammation (e).

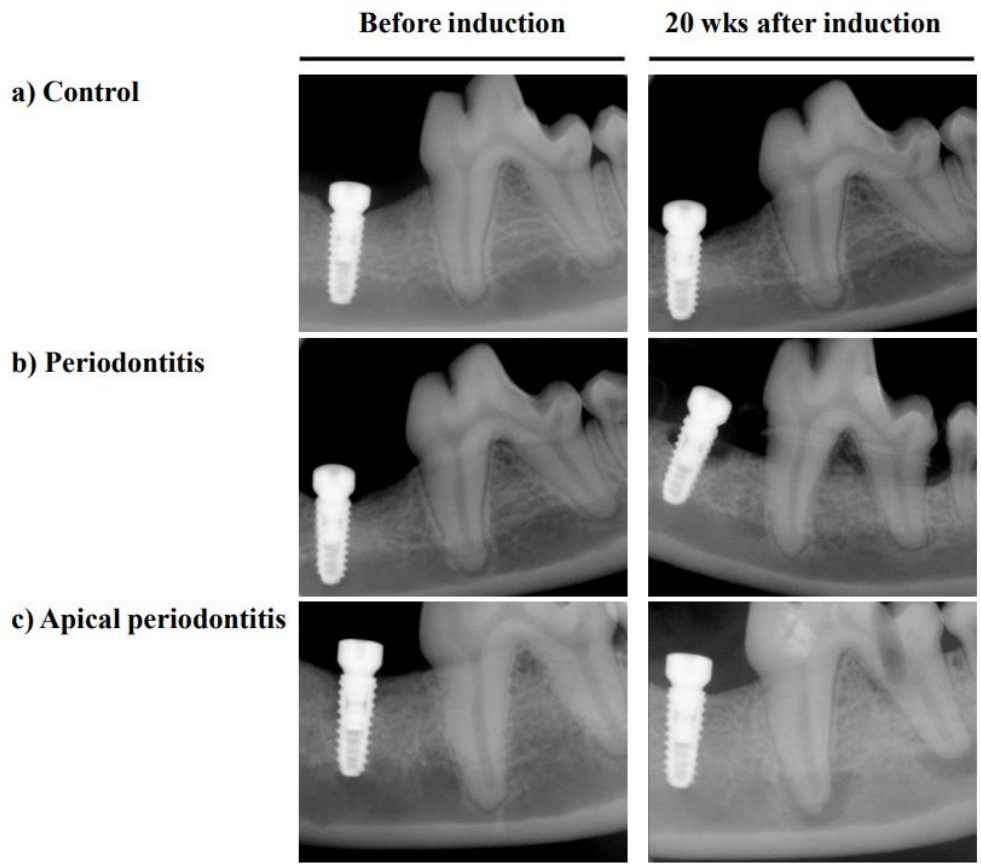
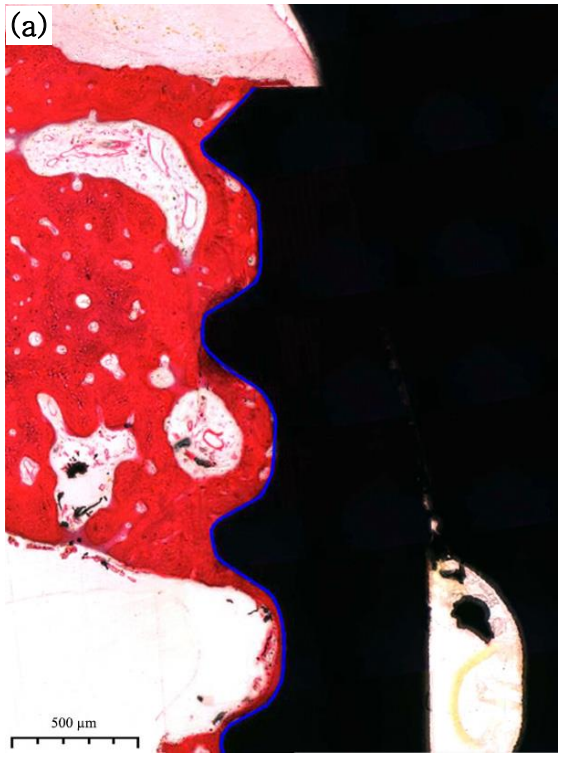
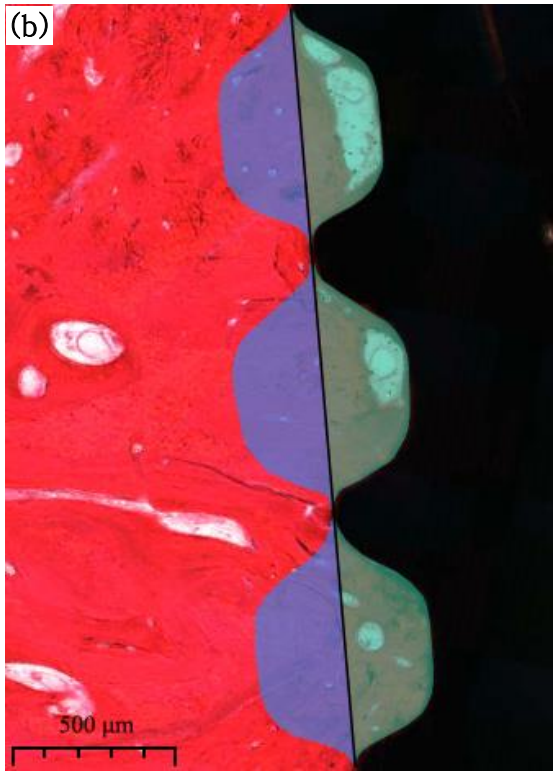


Figure 3. Radiographs obtained before and after induction of experimental periodontitis and apical periodontitis

The left column shows the radiographs obtained before the induction of periodontitis, and the right column shows the radiographs obtained 20 weeks after induction. (a) Control, (b) periodontitis, and (c) apical periodontitis


(a)
500 μm


(b)
500 μm

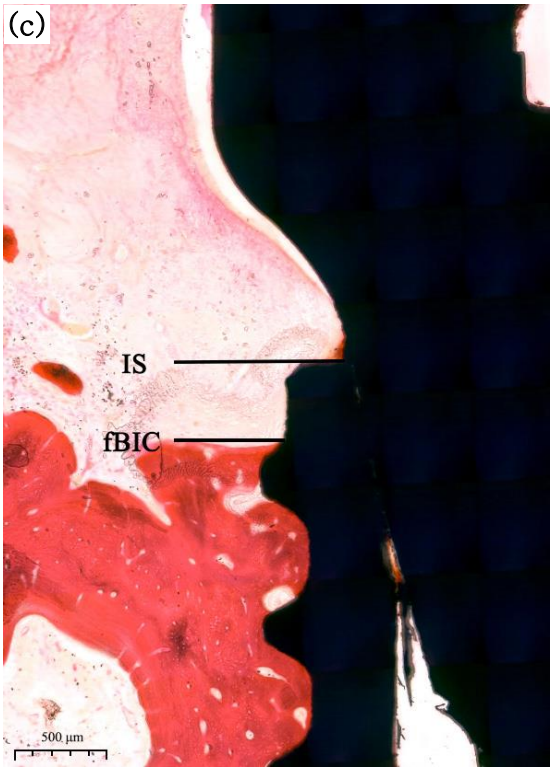
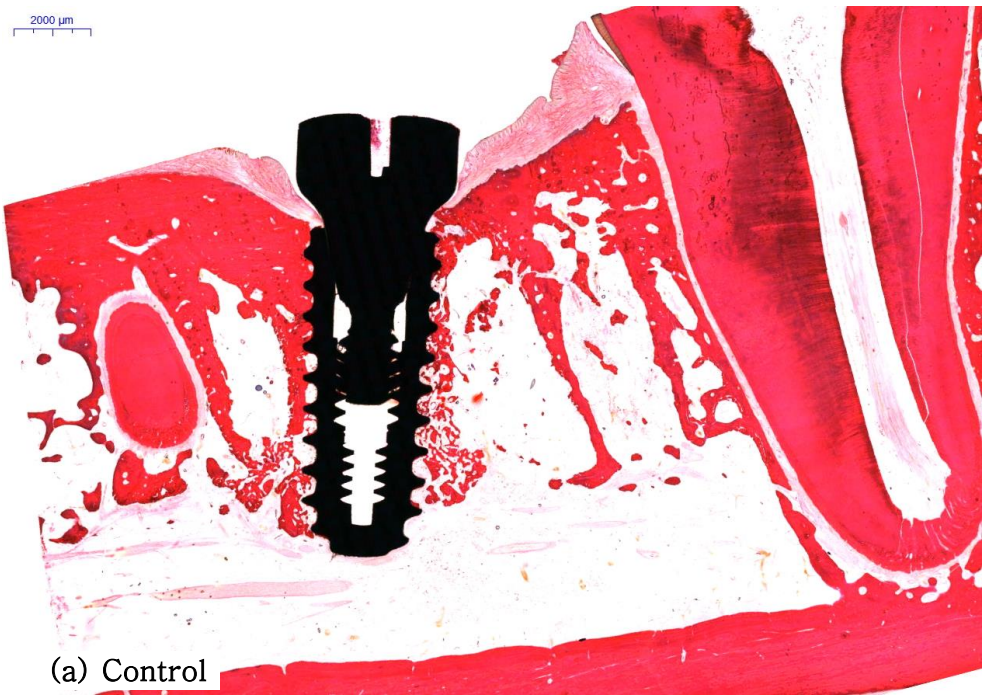
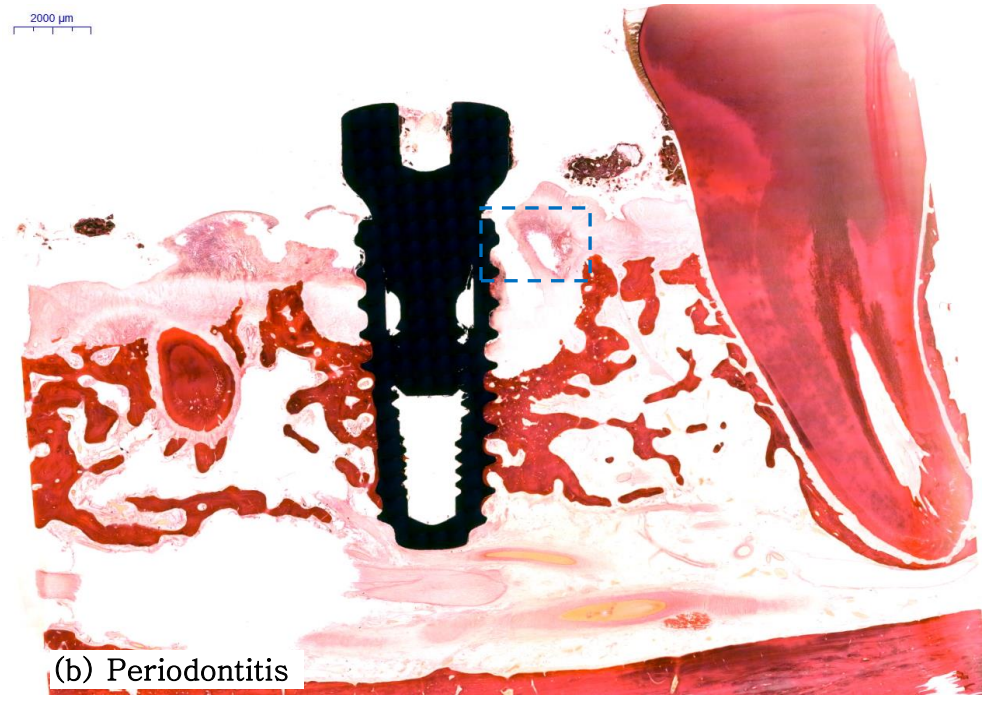


Figure 4. Methods for histometric analysis

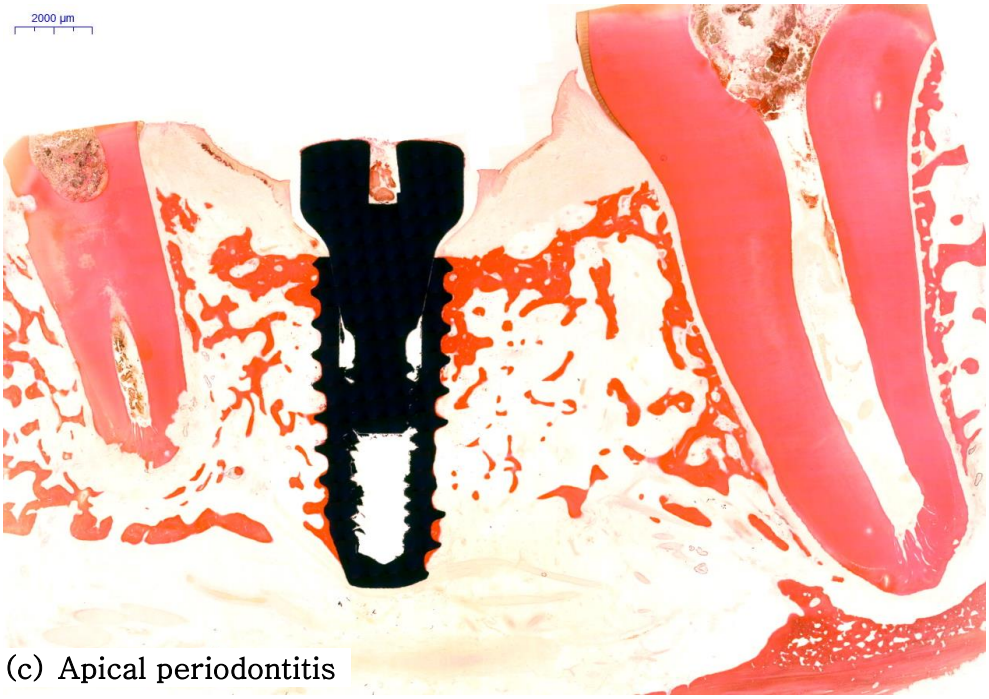
(a) The length of the bone-to-implant contact (BIC) indicated by a blue line in the figure was measured from the mesial and distal sides of the implant surface, and the percentage of BIC was calculated from these lengths. (b) The area within the implant threads and the reproduced mirror area were chosen as the area of interest for analyzing bone area (BA). (c) Implant shoulder (IS) and first bone-to-implant contact (fBIC) measurements were used to evaluate marginal bone loss.



(a) Control



(b) Periodontitis



(c) Apical periodontitis

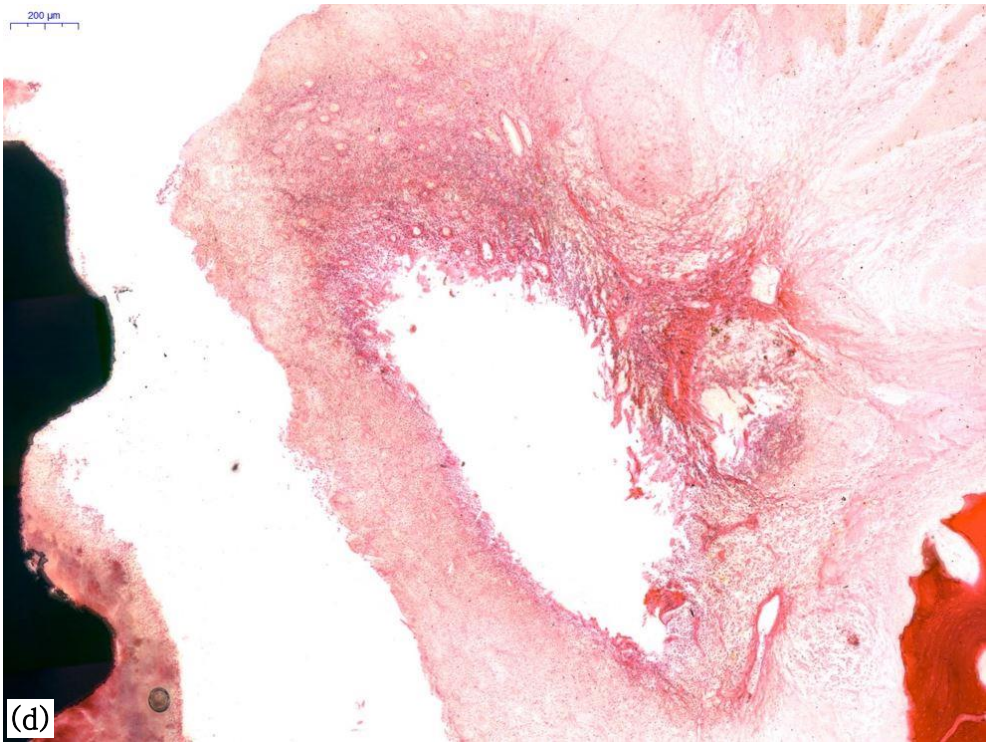


Figure 5. Histologic images of all groups

(a) Control, (b) periodontitis, (c) apical periodontitis.

Scale bar = 2.0 mm. Direct bone-implant contact, along with typical trabecular bone patterns surrounding the implants, was observed in all groups. (a) Control group. No marked inflammatory cells were detected, and the marginal bone was intact for the implant in the control group. (b) Periodontitis group. Periodontal lesion was observed in the mesial area of 1M1. Prominent marginal bone loss and infiltrated connective tissue was seen in around the implant in the periodontitis group (c) Apical periodontitis group. Periapical lesions were observed in the periapical area of 3P3 and 1M1. The bone-implant contact surrounding the implant remained intact. (d) An enlarged image of the area outlined by the dashed box in (b). Infiltrated connective tissue was observed around the implant in the periodontitis group.

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국문초록

인접한 치주염이 골유착된 치과용 임플란트에 미치는 영향

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1. 목 적

본 연구는 인접치아에 새롭게 진행되는 치주염 및 치근단 치주염이 이미 골유착이 완성된 임플란트에 미치는 영향을 비글견 모델에서 방사선학적 및 조직학적으로 평가하고자 하였다.

2. 방 법

3 마리의 수컷 비글견을 대상으로 하악 제2, 4 소구치를 발치하고 2개월 후 좌우 무치악부위에 4개의 SLA 표면처리한 고정체를 식립하고 cover screw를 체결하였다. 임플란트 수술 6주 후 치유 지대주를 연결하고 골유착을 위한 충분한 시간을 부여한 후에 치주염군 (1 마리)를 대상으로 하악 제1, 3소구치 및 제1대구치에 치주염을 유발하였다. 치근단 치주염군 (1 마리)를 대상으로 하악 제1, 3소구치 및 제1대구치에 치

근단 병소를 유발하였다. 대조군 (1마리)에서는 매달 스케일링 및 치면 활택술을 시행하여 염증이 유발되지 않게 하였다. 실험적 치주염 유발 5 개월 후에 방사선학적 및 조직학적 분석을 위해 희생하였다.

3. 결 과

대조군과 치근단 치주염군의 임플란트는 잘 유지되었지만, 치주염군의 임플란트는 염증의 임상적 증상과 골흡수가 나타났다. 치주염군의 골-임플란트 접촉율(BIC)과 골 면적(BA) 값은 다른 군들보다 낮은 수치를 보였다. 임플란트 숄더(IS)와 첫 번째 골-임플란트 접촉점(fBIC) 사이의 거리(IS-fBIC)는 치주염군에서 대조군보다 유의하게 크게 나타났다.

4. 결 론

인접한 치아에 유발된 치근단 치주염과 달리 인접치의 치주염은 골유착이 완성된 임플란트에 유의미한 영향을 미쳐, 임플란트 주위 점막의 염증과 함께 진행성 치조골 흡수를 유발할 수 있다. 따라서 임플란트의 장기적인 성공을 위해서는 이미 골유착이 완료된 후에도 인접치아의 치주건강을 유지 관리하는 것이 중요하다.

주요어: 치근단 치주염, 치과용 임플란트, 골유착, 치주염, 치주관리

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