





Does unresolved peri-implant dehiscence defect affect the progression of peri-implantitis?:

A preclinical canine model experiment

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Does unresolved peri-implant dehiscence defect affect the progression of peri-implantitis?: A preclinical canine model experiment

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나지영 올림



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Abstract

Does unresolved peri-implant dehiscence defect affect the progression of peri-implantitis?: A preclinical canine model experiment

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(Directed by Professor Ui-Won Jung, D.D.S., M.S.D., PhD.)

Objective: To investigate the progression of pathologic marginal bone loss induced by peri-implantitis in the implant having a dehiscence defect only covered by the mucosa.

Materials and Methods: In each of 6 male Mongrel dogs, 4 dental implants were placed in the posterior maxilla of both sides (2 implants for each). Based on the group allocation, each implant was randomly assigned to one of the following 4 groups to decide whether



buccal dehiscence defect was prepared and whether silk ligation was applied at 8 weeks post implant placement for peri-implantitis induction: UC (no defect without ligation); UD (defect without ligation); LC (no defect with ligation); and LD (defect with ligation) groups. Eight weeks after the disease induction, radiographic and histologic analyses were conducted, and the outcomes were statistically compared among the groups (p < 0.05).

Results: Micro-CT evaluation showed that the area of exposed implant was smallest in the UC group (p < 0.0083). Histologically, both the distances from the implant platform to the first bone-to-implant contact point and to the bone crest were significantly longer in the LD group (p < 0.0083). In the UD group, there was a sign of spontaneous bone fill from the base of the defect for 8 weeks after implant placement.

Conclusions: In the implant with unresolved dehiscence defect, it was more prone to experience progressive pathologic marginal bone loss when peri-implantitis was induced, however, when it was not exposed to the dental plaque, the defect could be filled by spontaneous bone gain.

Keywords: Animal experiment; Dental implant; Peri-implantitis; Dehiscence defect; Micro-computed tomography; Histology



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I. INTRODUCTION

Dental implant placed in an edentulous ridge needs to be surrounded by vital bone in order to achieve circumferential osseointegration and keep the stability of peri-implant mucosa (Monje et al., 2023), and it is widely agreed that at least over 1.5 mm of buccal



bone thickness is needed for keeping both the hard tissue and soft tissue around the implant in a healthy state (Jensen et al., 2023; Merheb et al., 2014; Monje et al., 2019). For this reason, when placing an implant to an atrophied edentulous ridge based on a prostheticdriven planning, bone augmentation surgeries have become popular which would possibly increase the horizontal ridge dimension (Chiapasco and Casentini, 2018; Hämmerle and Karring, 1998). Guided bone regeneration (GBR), one of the most commonly performed bone-augmenting procedures, has shown an excellent and stable long-term outcome in terms of resolving peri-implant dehiscence defect (Benic and Hämmerle, 2014; Chiapasco and Casentini, 2018).

Despite the effort of clinicians, the outcome of GBR may not be successful, so that the defect can be left which eventually causes several threads of the implant fixture to be uncovered (Benic et al., 2022; Schwarz et al., 2012). Even if the defect is not resolved, it has been revealed that the peri-implant dehiscence defects smaller than 5 mm in height left to spontaneous healing without any grafting resulted in no significant difference in the aspect of implant stability compared to the grafted ones (Jung et al., 2017; Palmer et al., 1994; Rasmusson et al., 1997; Waller et al., 2020). Moreover, a recently published pilot clinical study reported that clinical parameters, such as probing depth (PD) and bleeding on probing (BOP), of the dehiscence defect sites which received connective tissue graft instead of GBR were similar compared to the sites that received GBR (Zuercher et al., 2023).

Nonetheless, it is still widely agreed that the unresolved peri-implant bone defect may deteriorate the stability of soft and hard tissues, potentially leading to biologic complications (Monje et al., 2016; Monje et al., 2023a). Given the fact to date that the orientation of collagen fibers of peri-implant mucosa never gets inserted to the fixture surface in a perpendicular manner but only appears to be parallel to the implant (Ivanovski and Lee, 2018; Song et al., 2023), it could be easily assumed that leaving the peri-implant bone defect unregenerated and letting it just covered by the mucosa would make the peri-implant tissue more susceptible to forming a pathologic pocket which serves as a niche for



the bacteria and eventually accelerate plaque-induced marginal bone loss once it is initiated. The evidence, however, which supports this hypothesis is still scarce.

Thus, the present preclinical research using a mongrel dog experimental model aimed to evaluate the progression of peri-implantitis-induced marginal bone loss in the implant having a dehiscence defect only covered by the mucosa compared to that in the implant completely supported by the intact vital bone tissue.



II. MATERIALS AND METHODS

1. Ethical statement

The experimental protocol and the management of experimental animals were reviewed and approved by the Animal Care and Use Committee of Yonsei Medical Center, Seoul, South Korea (Approval No. 2019-0265). The outcomes of the present research were reported according to the ARRIVE (Animal Research: Reporting of In Vivo Experiment) guidelines (Kilkenny et al., 2010).

2. Experimental animals and their housing and husbandry

A total of 6 male Mongrel dogs, weighing 25 - 30 kg and aged 12 - 15 months, were prepared. The animals were housed under a proper and standardized laboratory condition at a room temperature of 15 - 20 °C and humidity of over 30%.

3. Study design and group allocation

Based on the split-mouth design and random allocation (Song et al., 2021a; Song et al., 2021b), one side of upper premolar site was assigned unligated groups, while the other side was assigned ligated groups, and within each side, control group without any defect and defect group with a buccal dehiscence defect were randomly allocated, resulting in following 4 groups (Fig. 1):

• Unligated control (UC) group: an implant surrounded by intact bone tissue without peri-implantitis induction



- Unligated defect (UD) group: an implant having a buccal dehiscence defect (3 mm in apico-coronal height and 3 mm in mesiodistal width) without periimplantitis induction
- Ligated control (LC) group: an implant surrounded by intact bone tissue where peri-implantitis was induced
- Ligated defect (LD) group: an implant having a buccal dehiscence defect (3 mm in apico-coronal height and 3 mm in mesiodistal width) where peri-implantitis was induced

4. Experimental protocol

The entire research protocol is summarized in Figure 2.

4.1 Extraction and implant placement

Both the extraction and implant surgery were performed under general anesthesia with intravenous injection of alfaxalone (2 mg/kg, Alfaxan, Jurox, Rutherford, Australia) and inhalation of isoflurane (2%, Ifran, Hana Pharm, Seoul, South Korea) followed by local anesthesia (2% lidocaine UCL with epinephrine 1:100,000, Huons, Seongnam, South Korea).

The maxillary second and third premolars of both sides were extracted after hemisection, and at 12 weeks post extraction, implants were placed along with or without the defect formation according to the random group allocation. After the elevation of mucoperiosteal flaps, 2 sequential osteotomies which allowed 6 mm of inter-fixture distance were carried out in each side. All sites had buccal bone thickness of 2 mm, and for the UD- and LD-group sites, buccal dehiscence defects of 3 mm in corono-apical height and 3 mm in mesio-distal width were prepared by a highspeed diamond bur. Then sandblasted, large-grit and acid-etched (SLA)-surfaced dental implants (3.6 * 10 mm,



Superline III, Dentium, Suwon, South Korea) were placed, ensuring the top of fixture platform to match the bone crest level, and machined-surfaced healing abutments (4.0 * 3.5 mm, Dentium) were applied, followed by primary closure of the flaps using a 4-0 resorbable suture material (Monosyn 4-0 Glyconate Monofilament; B. Braun, Melsungen, Germany). After every surgical procedure (extraction and implant placement), antibiotics (enrofloxacin 5 mg/kg; Baytril, Bayer Korea) and analgesics (ketorolac tromethamine 0.5 mg/kg; Keromin, Hana Pharm) were given once daily for 5 days. The suture materials were removed after a week.

4.2 Induction of peri-implantitis

After 8 weeks of healing period to achieve osseointegration, silk ligatures (2-0 Gingipak®, Camarillo, CA, USA) were applied and tied around the healing abutments of the LD and LC groups and inside the mucosal sulcus for 6 weeks in order to accumulate dental plaque and induce peri-implant inflammation (Fickl et al., 2015; Yoon et al., 2020a; Yoon et al., 2020b), while the healing abutments of the UD and UC groups did not receive any ligation. Once every 2 weeks during this peri-implantitis-induction period, peri-apical radiographs were taken to visually confirm whether the circumferential bone loss of over 3 mm occurred only in the disease-induced (LD and LC) groups, and clinical examinations were also carried out to ensure whether mucosal redness and swelling along with BOP appeared only in the LD and LC groups. After the 6 weeks of active breakdown, the silk materials were removed from the healing abutments of the LD and LC groups to allow spontaneous disease progression for the next 2 weeks, and clinical and radiographic evaluations were kept in all 4 groups during this disease-progression period.

4.3 Euthanasia of the experimental animals



Sixteen weeks after the implant placement, the experimental animals were euthanized by intravenous administration of 15 mg/kg of zolazepam combined with tiletamine (Zoletil 50, Virbac Animal Health, Carros, France) and 4 mg/kg of xylazine (Rompun, Bayer Healthcare LLC, Leverkusen, Germany), followed by intravenous injection of potassium chloride. The maxilla including the soft and hard tissues were dissected and fixed in 10% neutral-buffered formalin for 10 days for further radiographic and histologic analyses.

5. Micro-computed tomographic analysis

The obtained samples first went through micro-computed tomography scanning (micro-CT; Skyscan 1173, SkyScan, Aartselaar, Belgium) under the voxel size of 50 μ m voxel size, acceleration voltage of 130 kV and beam current of 60 μ A. The scanned images were processed in DICOM format and three-dimensionally (3D) reconstructed by a computer software (OnDemand3d; Cybermed, Seoul, South Korea) using the thresholding parameter of 40–255 range for bone level and 100–255 range for implant level. For the quantitative measurement of exposed fixture area (mm²), the surface area of the fixture portion which was not covered by bone tissue at buccal was measured from the 3D reconstructed image using a computer software (Photoshop 2023, Adobe, San Jose, CA, USA) (Fig. 3a).

6. Descriptive histology and histomorphometric analysis

After the fixation, the samples containing the implants were dehydrated with ethanol solutions and embedded in resin for ground section to achieve undecalcified specimens. Among the bucco-lingual sections, the most central sections were chosen to be stained by Goldner's trichrome. Digital images of histologic specimens were qualitatively observed with a computer software (CaseViewer; 3DHISTECH, Budapest, Hungary). For the



histomorphometric analysis, a vertical reference line was drawn corresponding to the long axis of the implant, and 6 horizontal reference lines which were perpendicular to the vertical reference line were defined at 1 mm intervals from the implant shoulder level to the level 5 mm below the shoulder. Then following parameters were quantitatively measured by an experienced examiner who was blinded to the group allocation (J.Y.N) using the computer software (Photoshop 2023, Adobe) (Fig. 3b):

- Vertical dimensions of the buccal side:
 - fBIC-IS (mm): a distance between the first bone-to-implant contact point and implant shoulder
 - fBIC-M (mm): a distance between the first bone-to-implant contact point and peri-implant mucosal margin
 - BC-IS (mm): a distance between the bone crest margin and implant shoulder
 - BC-M (mm): a distance between the bone crest margin and peri-implant mucosal margin
 - IS-M (mm): a distance between the implant shoulder and peri-implant mucosal margin
- Horizontal thicknesses of the peri-implant buccal bone (HT_mm): mean value of what measured at 1 mm intervals along the 6 different horizontal levels from the implant shoulder (HT 0 to 5)

7. Statistical analysis

The achieved data (mean \pm standard deviation) were statistically analyzed by a computer software (SPSS version 23, IBM, Armonk, NY, USA). Kruskal-Wallis test was performed for intergroup comparison followed by post hoc Mann-Whitney U test under Bonferroni modification if necessary. Statistical significance was set p < 0.05.

III. RESULTS

1. Clinical findings

All experimental animals demonstrated neither systemic nor local complication, and all of the implants survived during the experimental period. Due to the ligation which induced plaque accumulation, the LC and LD groups presented clearer signs of periimplant mucosal inflammation, i.e. mucosal redness and swelling, compared to the mucosa of UC and UD groups where any inflammation sign was not observed. Between the LC and LD group, the mucosal inflammation of the LD-group implant was severer than that of the LC group.

2. Micro-computed tomographic analysis

The exposed implant fixture surface not covered by the bone was significantly smaller in the UC group $(1.01 \pm 0.61 \text{ mm}^2)$ compared to the rest of the 3 groups (p < 0.0083). The areas measured in the UD ($4.62 \pm 1.18 \text{ mm}^2$), LC ($7.24 \pm 4.39 \text{ mm}^2$) and LD ($9.16 \pm 3.63 \text{ mm}^2$) groups were not significantly different when compared to each other. The radiographic measurement is summarized in Table 1.

3. Descriptive histology

In the histology, a definite difference in the appearance was found between the unligated (UC and UD) and ligated (LC and LD) groups. In the LC and LD group, pathologic bone resorption induced intrabony bone loss on the buccal side, whereas it was not seen in the UC and UD groups of which the bone tissue surrounding the implant fixture appeared to be in a mature state with a lamellar structure. The peri-implant buccal bone of



the UC group was completely preserved without any inflammatory sign, and in the UD group, bone even grew coronally along the implant surface spontaneously resulting in reducing the height of the dehiscence defect. The mucosa surrounding the healing abutment of the LC and LD groups presented long junctional epithelium penetrating apically along the healing abutment surface with elongated rete pegs, while the mucosa of the UC and UD groups did not show such an appearance (Fig. 4).

4. Histomorphometric analysis

The results from the histomorphometric analysis are summarized in Table 2.

4.1 Vertical dimensions of the buccal side

The fBIC-IS was longest in the LD group $(2.15 \pm 0.37 \text{ mm})$, while it was shortest in the UC group $(0.58 \pm 0.28 \text{ mm})$. Statistical significance in the post hoc comparison was found only between the UC and LC $(1.31 \pm 0.67 \text{ mm})$ groups, between the UC and LD groups, and between the LC and LD groups (p < 0.0083).

Similarly, the BC-IS of the LD group $(1.90 \pm 0.15 \text{ mm})$ was the longest, whereas that of the UC group $(0.17 \pm 0.23 \text{ mm})$ was the shortest. The post hoc comparison revealed that significant difference was only observed between the UC and LC $(1.17 \pm 0.66 \text{ mm})$ groups and between the UC and LD groups (p < 0.0083). The fBIC-M and BC-M were also longest in the LD group but shortest in the UC group, but the differences among the groups were not statistically significant. The IS-M was comparable among the groups.

4.2 Horizontal thicknesses of the peri-implant buccal bone

At the implant shoulder level, there was no buccal bone observed in the UD, LC and LD groups, and the HT_0 was measurable only in the UC group (0.16 ± 0.36 mm). Still, the HT_1 of the LD group was not measurable, but those of the other 3 groups were able



to be measured, and the HT_1 of the UC group $(1.05 \pm 0.44 \text{ mm})$ which was the thickest was significantly thicker when compared to the LC (0.20 ± 0.27) and LD $(0.00 \pm 0.00 \text{ mm})$ each (p < 0.0083).

The HT_2 was thinnest in the LD group $(0.31 \pm 0.24 \text{ mm})$, and it was thickest in the UC group $(1.26 \pm 0.32 \text{ mm})$, however, statistical significance was only seen in the differences between the UC and LD groups and the LC $(1.03 \pm 0.27 \text{ mm})$ and LD groups (p < 0.0083). Rest of the measuring parameters (HT_3, 4 and 5) were comparable among the groups.



IV. DISCUSSION

The present animal study aimed to investigate whether the pre-existing unresolved dehiscence defect affects the progression of pathologic loss of peri-implant marginal bone when the implant suffers from plaque-induced peri-implant inflammation. Compared to the implant completely surrounded by vital bone tissue, the implant having unresolved dehiscence defect was found to be more vulnerable to pathologic marginal bone resorption when exposed to the plaque-accumulating environment. On the contrary, when there was no silk ligation and so the peri-healing abutment was kept cleaner, spontaneous bone gain in the coronal direction was found at the base of the pre-existing dehiscence defect.

It is widely known that highly polished and smooth titanium surface is advantageous over the rough surface with complex topography in terms of retaining bacterial biofilm, and it explains why the soft tissue collar part of tissue-level implants or the abutments applied over bone-level implants which are commercially available used to have a machined and smooth surface (Werner et al., 2009). Despite the contribution of surface roughness which increases the hydrophilicity and so significantly contributes to successful osseointegration (Abrahamsson et al., 2004; Song et al., 2021b), once the rough-surfaced fixture threads at coronal are not osseointegrated and just covered by mucosa, this part would serve as a reservoir for dental plaque and may deteriorate mucosal inflammation (Monje et al., 2023a). This supports the finding that the mucosal redness and swelling was more prominent in the LD group than in the other three groups in the present experiment, especially compared to the LC-group mucosa where a slightly more inflamed state was seen compared to the unligated groups (UC and UD groups), of which the mucosa presented a healthy state.

Whether the plaque accumulation was accelerated and whether the unresolved dehiscence defect was present seemed to have significantly affected the marginal bone



status during the follow-up period after the implantation. Based on the micro-CT images, there was an increase of the thread exposed area in the LD group, while in the UC group, there was almost no change in the exposed area with a negligible amount of increase possibly due to the physiologic bone remodeling (Coli and Jemt, 2021). Compared to the UC group, the exposed fixture area of the LC group was larger which could be interpreted by the influence of plaque-induced bone resorption (Yoon et al., 2020a; Yoon et al., 2020b), and interestingly, in case of the UD group, the area even decreased when compared to the size of the dehiscence defect prepared along with the implant placement.

Similar findings were observed in the histology. In the groups which were ligated (LC and LD groups), an intrabony defect was observed near the fixture implying active bone destruction. The only difference between the two groups was where the destruction started from, the platform level in the LC group whereas the base of the pre-existing dehiscence defect, since plaque-inducted inflammatory starts from the coronal, considering the current assumption that peri-implant mucositis precedes peri-implantitis (Jepsen et al., 2015; Schwarz et al., 2018). What was surprising was the amount of fixture exposure in the LD group. On the day of implant placement, the height of dehiscence defect artificially made was 3 mm, however, in the histologic images after 16 weeks, both the fBIC-IS and BC-IS were less than 3 mm, approximately 2 mm. This implies that during the 8 weeks prior to the silk ligation, spontaneous bone gain and osseointegration in the coronal direction had occurred before the implant got contaminated by the dental plaque. In the same vein, the defect resolution was also partially found in the UD group in a coronal manner, approximately 1.7 mm in mean.

This phenomenon of spontaneous bone fill and new bone attachment to the implant surface in the dehiscence defect was previously reported. In a series of previous canine experiments, spontaneous bone gain was promoted in an acute-type buccal dehiscence defect which was left untreated when the surface of implant fixture was hydrophilic (Schwarz et al., 2007; Schwarz et al., 2008), and higher the hydrophilicity was, more the bone regeneration occurred (Schwarz et al., 2008). Just like the forementioned studies, the



present experiment used SLA-surfaced implant which was hydrophilic, and on its buccal side, the acute-type defect was made. It might be therefore obvious that the extent of fixture exposure became smaller, not only in the UD group but also in the LD group.

The initial thickness of the buccal bone around the implant is thought to have significantly influenced the histologic outcome. Previously, several studies had reported various opinions concerning the critical amount of buccal bone thickness for ensuring the peri-implant soft and hard tissue health (Monje et al., 2019; Spray et al., 2000), and a recently published systematic review has concluded that when the thickness of peri-implant buccal bone is thinner than 2 mm, there is more chance to compromise the integrity of peri-implant tissue (Monje et al., 2023a). Moreover, a recent preclinical canine model research has shown that more vertical bone loss occurred when the buccal bone was thinner than 1.5 mm compared to when it was at least 1.5 mm (Monje et al., 2019).

In the present experiment, regardless of the group allocation, the implants were surrounded by the buccal bone of 2 mm in thickness. This amount seems to be sufficient for the UC-group implant to have an optimized health of peri-implant soft and hard tissue. Even though in the UC group, the horizontal bone thickness at the platform level was less than 0.2 mm in mean, and fBIC-IS was also measurable (0.58 mm in mean), horizontal bone dimension around the UC-group implant was kept 1.05 - 1.70 mm in mean from the level of 1 mm below the fixture platform. The marginal bone resorption occurred in the UC group is thought to be acceptable because of physiologic bone remodeling after implant placement causing marginal bone loss (Adell et al., 1985; Adell et al., 1981; Coli and Jemt, 2021). It has been thought successful for an implant to have a spontaneous marginal bone resorption of 1.5 - 2 mm during the first year following implantation in terms of preventing a progressive bone loss over time (Albrektsson et al., 1986; Galindo-Moreno et al., 2015; Misch et al., 2008; Papaspyridakos et al., 2012).

The sufficient amount of initial buccal bone thickness seems to have also contributed to the spontaneous bone regeneration observed in the UD group throughout the entire experimental period and in the LD group 8 weeks before the disease induction. The



thickness of 2 mm at the defect base made the buccal dehiscence defect have a fully contained configuration which would have optimized regenerating environment by promoting the blood supply and the recruitment of pristine bone-origin osteogenic factors as well as maintaining blood clot within the bony housing (Sculean et al., 2019). Furthermore, in case of the UD group where the peri-implant tissue was not contaminated, this favorable bone-regenerating environment might have allowed the bone attached to the implant surface to get matured to achieve a lamellated structure during the 16 weeks of postsurgical period, similar to the histologic appearance seen in a previous animal study (Song et al., 2021b).



V. CONCLUSION

Within the limitation of the present preclinical study, the implant having an unresolved dehiscence defect would be more susceptible to experiencing progressive pathologic marginal bone loss when it is exposed to the dental plaque. On the other hand, when the environment of peri-implant tissue is kept uncontaminated and clean, there is a chance for the unresolved defect to be spontaneously regenerated, if the defect possesses a contained configuration.



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TABLES

Table 1. Radiographic measurement of the surface area of exposed implant portion (mean \pm standard deviation).

Group	Surface area (mm ²)	
UC	$1.01 \pm 0.61*$	
UD	7.24 ± 4.39	
LC	4.62 ± 1.18	
LD	9.16 ± 3.63	

* (bold): Statistically significant difference was found when compared to the other 3 groups (p < 0.0083).



Table 2. Histomorphometric analysis (mean ± standard deviation).

Group	fBIC-IS	fBIC-M	BC-IS	BC-M	IS-M
UC	$0.58 \pm 0.28^{*,+}$	3.52 ± 0.67	$0.17 \pm 0.23^{*,+}$	3.10 ± 0.48	2.94 ± 0.45
UD	1.31 ± 0.67	4.59 ± 0.82	1.17 ± 0.66	4.45 ± 0.96	3.28 ± 0.47
LC	$1.36 \pm 0.29^{*,++}$	4.17 ± 0.90	$1.15\pm0.44^*$	3.95 ± 0.87	2.81 ± 1.07
LD	$2.15 \pm 0.37^{+,++}$	5.03 ± 0.88	$1.90 \pm 0.15^+$	4.66 ± 0.67	2.70 ± 0.77

(a) Vertical measurements (mm)

* (bold): Statistically significant difference was found when the UC and LC groups were compared to each other (p < 0.0083).

+ (bold): Statistically significant difference was found when the UC and LD groups were compared to each other (p < 0.0083).

++ (bold): Statistically significant difference was found when the LC and LD groups were compared to each other (p < 0.0083).

Group	HT_0	HT_1	HT_2	HT_3	HT_4	IS-M
UC	0.16 ± 0.36	$1.05 \pm 0.44^{*,+}$	$1.26\pm0.32^+$	1.46 ± 0.54	1.60 ± 0.51	1.70 ± 0.56
UD	0.00 ± 0.00	0.26 ± 0.59	0.57 ± 0.57	0.91 ± 0.68	1.28 ± 0.91	1.63 ± 1.33
LC	0.00 ± 0.00	$\textbf{0.20} \pm \textbf{0.22}^{*}$	$1.03 \pm 0.27^{++}$	1.73 ± 0.52	2.17 ± 0.69	2.35 ± 0.96
LD	0.00 ± 0.00	$0.00 \pm 0.00^+$	$0.31 \pm 0.24^{+,++}$	0.71 ± 0.28	0.98 ± 0.28	1.44 ± 0.29

(b) Horizontal measurements (mm)

* (bold): Statistically significant difference was found when the UC and LC groups were compared to each other (p < 0.0083).

+ (bold): Statistically significant difference was found when the UC and LD groups were compared to each other (p < 0.0083).



++ (bold): Statistically significant difference was found when the LC and LD groups were compared to each other (p < 0.0083).



FIGURES



Figure 1. Schematic description of group assignment.

Based on the group allocation, each implant was randomly assigned to one of the following 4 groups: (1) Unligated control (UC) group: an implant surrounded by intact bone tissue without peri-implantitis induction, (2) Unligated defect (UD) group: an implant having a buccal dehiscence defect (3 mm in apico-coronal height and 3 mm in mesiodistal width) without peri-implantitis induction, (3) Ligated control (LC) group: an implant surrounded by intact bone tissue where peri-implantitis was induced, (4) Ligated defect (LD) group: an implant having a buccal dehiscence defect (3 mm in apico-coronal height and 3 mm in mesiodistal width) without peri-implantities was induced, (4) Ligated defect (LD) group: an implant having a buccal dehiscence defect (3 mm in apico-coronal height and 3 mm in mesiodistal width) where peri-implantities was induced.





Figure 2. Schematic timetable of experimental protocol with clinical

photographs.





Figure 3. Radiographic and histomorphometric measurements.

(a) In each group, the surface area (mm²) of exposed implant fixture portion (purple-dotted line) was measured. (b) Implant shoulder (IS), first bone-to-implant contact point (fBIC), bone crest (BC) and mucosal margin (M) were represented by red, green, orange, and blue triangles, respectively. Vertical reference line (white-dotted line) was defined parallel to the long axis of the implant fixture, and horizontal reference lines (red-, green-, orange-, and blue-dotted lines) were drawn perpendicular to the vertical reference line, passing the triangle in a same color. All linear measurements were conducted parallel to the vertical reference line. The linear distances (mm) between the green and red reference lines (fBIC-IS), between the green and blue reference lines (BC-IS), between the orange and blue reference lines (BC-M), and between the red and blue reference lines (IS-M) were estimated.





Figure 4. Representative histology.

Pathologic bone loss causing intrabony defect was found in the LC and LD group (red arrows), whereas the UC and UD group did not present such an appearance, of which the bone tissue attached to and surrounding the fixture surface was in a mature state with a lamellar structure (yellow asterisks). Only the mucosa of the LC and LD group presented long junctional epithelium on the healing abutment surface (blue arrows) and elongated rete pegs at the epithelium (green arrows).



국문요약

임플란트 주위 열개형 골 결손부가

임플란트 주위염의 진행에 영향을 미치는가?:

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나 지 영

임플란트 주위 열개형 골 결손부가 연조직과 경조직의 안정성을 저하시켜 잠재적 으로 생물학적 합병증을 유발할 수 있다는 사실은 널리 알려져 있다. 이에 따라. 임플 란트 주위 열개형 골 결손부가 존재할 경우, 박테리아의 틈새 역할을 하는 치주낭을 형성하기 쉬운 환경이 만들어질 가능성이 높아지고, 결국 플라그에 의해 유발되는 진 행성 변연골 소실을 가속화할 것이라는 가설을 수립하였다.

따라서 본 연구는 임플란트 주위 지지골에 의해 잘 보존되어 있는 임플란트와 비 교하여 임플란트 주위 점막으로만 덮인 열개형 골 결손부가 임플란트 주위염으로 인



한 병리학적 변연골 소실의 진행에 미치는 영향을 조직학적, 방사선학적으로 평가하고 자 한다.

총 6마리의 성견을 대상으로 양측 상악 제2소구치와 제3소구치를 발치하고 12주의 치유기간을 거친 후, 양측에 4개의 임플란트를 식립 하였다. 각 임플란트는 협측부위에 열개형 골 결손부의 형성 여부와 임플란트 식립 8주후 결찰의 적용 여부를 다음 4개의 군에 무작위로 배정하였다: 1) UC군(열개형 골 결손부가 없고, 결찰을 적용하지 않은 군). 2) UD군(열개형 골 결손부가 있고 결찰을 적용하지 않은 군), 3) LC군(열개형 골 결손부 가 없고, 결찰을 적용한 군), 4) LD군(열개형 골 결손부가 있고, 결찰을 적용한 군). 결찰 에 의한 질병 유발 후 8주의 기간을 거친 뒤, 실험 동물을 희생하여 방사선학적 및 조직 학적 분석을 시행하였고, 각 실험군 간의 결과를 통계적으로 비교하였다(*p* <0.05).

연구결과 노출된 임플란트 표면적(mm²)은 다른 세 군에 비해 UC군에서 유의하게 가장 작았다(*p* <0.0083). 또한 임플란트 플랫폼에서부터 첫 번째 골-임플란트 접촉점과 치조골 상방까지의 거리 모두 LD군에서 유의하게 더 길었다(*p* <0.0083). UD군에서 결손 기저부로부터 자발적인 골 재생이 일어나 열개형 골 결손부의 높이가 감소하였다.

결론적으로, 임플란트 주위 열개형 골 결손부는 임플란트 주위염이 유발되는 상황에 서 진행성 병리학적 변연골 소실을 경험하기 쉽고, 플라그에 노출되지 않은 상황에서는 자발적인 골 재생으로 치유될 수 있다.

핵심되는 말: 동물 실험; 치과 임플란트; 임플란트 주위염; 열개형 골 결손부; 미세단 층촬영; 조직학