Part 3 - Implants and clinical dentistry

Chapter 1 Implants and periodontology

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I. Basic concept for implant

In the 1960s, the Branemark *et al.* developed osseointegrated implants that function by gaining support from the bone tissues[1]. Schroeder *et al.* later found that that the implants had no ligament tissues support unlike those of the natural tooth, and that the implant and the bone were in direct binding with one another[2].

The structures of surrounding tissues of implant and natural tooth include: 1. epithelial attachment, 2. connective fiber tissues and its adherent form, and 3. periodontium. The significant differences that lie between the surrounding structures of implants and natural teeth are with the latter two factors (2 and 3). The structures of the epithelial attachment (1) for both implant and tooth are similar, however, the epithelial tissues around the implant develop as gingival stratified squamous epithelium in the wound recovery process. The epithelium of the implant is therefore fundamentally different from those of natural teeth in terms of developmental studies, and its ability to function as a barrier is reduced. It is also believed that epithelial layer surrounding implant has fewer barriers function than that of natural teeth. Many researches regarding roles of implants and soft tissue adhesion in phylaxis have been conducted. By understanding the differences between the surrounding tissues of implants and those of natural teeth it should enable the selection of maintenance methods that are most appropriate, and to avoid inflammation in the surrounding tissues.

A. Structure of periodontal tissue on natural teeth (attachment forms of teeth and ginvivae)

The gingivae consist of mucosal tissues that lie over the alveolar bone and the tooth cervix. The mucosal tissues consist of three cell layers, epithelial, connective tissue and tunica propria. The characteristic features of this region are the presence of junctional epithelium and oral sulcular epithelium.

The surface of the oral sulcular epithelium is keratinized, and covers the sulcus that lies between the enamel and the free gingiva. The basal cells of the junctional epithelium that lies beneath this are under constant regeneration. Both basal cells and the cells in the layer above are organized so that the long axis is parallel to the plane of the tooth.

Connective tissue forms the greater part of the gingivae, and other components are collagen fibers (approx. 60%), fibroblasts (approx 5%), and vessels and nerves (approx 35%). Other cell types include mast cells, macrophages, and inflammatory cells (including heterophilic leucocytes, lymphocytes and plasma cells). Fiber components are made up primarily of collagen fibers, reticular fibers (in tissues that lie next to the basal membrane), oxytalan fibers (rare in the gingivae), and elastic fibers (distributed

around the blood vessel sites). With regards to collagen fibers, most of these are in bundles and are arranged in a fixed direction. The gingival fibers can be classified into: a circular group (orientated so as to encircle the free gingiva), a dentogingival group (spread out in a fan-form in that the fibers extend from the root in the cementum at the suprabony pocket, to the buccal and lingual sides or in the direction of free gingiva), a tooth-periosteal group (run from the root in the cementum, past alveolar bone to the side of the root apex, in the direction of the junctional epithelium), and a transseptal group (run in the cementum at the suprabony pocket along the side of the tooth) (Fig. 3-1-2).

There are two types of gingival attachments to the teeth, epithelial attachments and connective tissue attachments. From the alveolar bone to the crown, they are organized in the order of connective tissues, and epithelial attachments, and finally reaching the gingival sulcus. Each is 1 mm wide, a distance known as the "biological width" and is a standard that must be taken into account when fabricating the attachments [3].
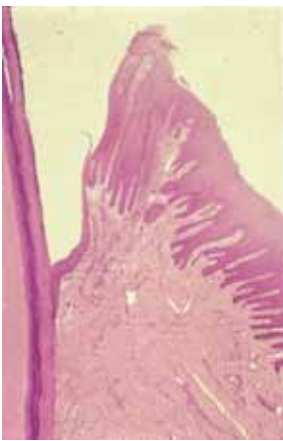

Fig. 3-1-1
Junctional epithelium and oral sulcular epithelium exists in the region of the teeth and gingiva

Fig. 3-1-2
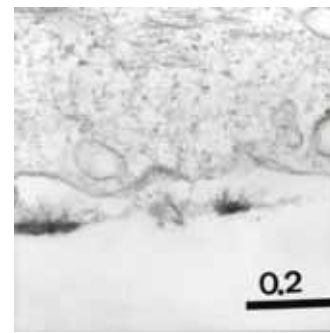Majority of the collagen fibers are bundled and run in one direction

Fig. 3-1-3 Epithelial attachment is an attachment of the epithelial cells with the teeth. It is attached to the teeth via internal basal lamina and hemidesmosome

B. Structure surrounding the implant body

After implant surgery, mucoadhesion (pierced mucoadhesion) occurs to the implant body protecting the connective tissues and bone tissues from intraoral substances such as bacteria. The soft tissues structures surrounding titanium implant abutments have been determined by conducting clinical studies in human and animal experiments. The research by Berglundh in 1991 conducted on dogs, which has become the basis for the series of experiments that followed, to compare the biological difference between the gingivae of natural teeth and the surrounding tissues of the implant body [7]. There were histological similarities between the two. Epithelium of natural tooth gingivae are highly keratinized and stretch to the junctional epithelium, the main fiber within the subepithelial connective tissues spread in a fan-shaped form towards the soft and hard tissues surrounding the periodontal

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membrane through the cementum of the tooth root. The mucous membrane around the implant is also covered by highly keratinized epithelium, which is connected to epithelial barrier facing the abutment. This epithelial barrier corresponds to the junctional epithelium of natural tooth. The length is roughly 2 mm long and is attached to the surface of implant through hemidesmosome[8]. However, since implant material is fundamentally a foreign material, the epithelium around the implant is thought to be more prone to invasion by foreign substances and bacteria than around the natural teeth[9].

The collagen fibrils are present within the 1 to 1.5 mm layer of connective tissues that reside in between the epithelial barrier and alveolar crest. This structure attaches to the plane of the teeth, referred to as connective tissue adhesion. The difference in the connective tissue attachment between the implant surroundings and the natural teeth were investigated by Berglundh *et al*[7].

The cementum exists on the rhizoplane of a natural tooth. The collagen fibers forms bundles on the interface between the tooth plane and gingivae, and tooth plane and alveolar bone, and run in a lateral direction to the coronal, and to the root apex. Contrarily, the collagen fibers run from the periosteum of the alveolar crest, in parallel to the implant surface, or in straight line in bundled form (Fig. 3-1-4-a). The forms of attachment were analyzed with various implant types by Abrahamasson *et al*, but a similar pierced mucosal attachment form as mentioned above were found [10].

An analysis of connective tissues constituents in the implant body attachment structures were conducted by Moon *et al* on dogs. The results showed two types of attachments, where the first did not consist of any blood vessels with presence of fibroblast that in aligned parallel to the vertical axis of the implant body (collagen 67%, blood vessel/nervous structure 0.3%, fibroblast 32%). The second type was found to exist external to the former type, that consisted of fewer fibroblast but with higher constitution of collagen fibers and vascular nerve structures (collagen 85%, vasculature 3%, fibroblast 11%).

The investigation into the biological width of the pierced mucosal attachment was also conducted on dogs[12]. In this experiment, after implanting into both right and left sides of the jaw, the thickness of the right mucosa was decreased to less than 2 mm. The results showed that the pierced mucosal attachment to be constructed from 2 mm barrier epithelium and a layer of connective tissue adhesion that was 1 to 1.5 mm thick. In the portion of the mucosa on the right, in which the thickness was decreased, bone resorption was shown to result in the surrounding ridges of the fixture, and had consequently established a thickness of more than 3 mm.

An experiment was conducted to investigate the pocket depth around the implant and the natural tooth on beagle dogs[13]. The probe with 0.5 mm diameter tip was inserted with 0.5 N pressure into clinically healthy tissues surrounding the implant. The surrounding tissues of implant were pressurized and became laterally displaced with the probing. The tip of the probe was inserted to the interface of the connective tissues and the abutment thus indicting that it was positioned further in than the tip of the barrier epithelium (Fig. 3-1-4-b). The results showed that the probes became in close contact with the alveolar crest indicating that the soft tissue attachment with the implant surface to be weaker than those of the natural teeth. It thus suggested the need to reduce the probing pressure when applying to examine the implant attachment structures, and in exertion of excess force, there is a risk of mechanically loss of the attachment between the soft tissues and the implant surface.
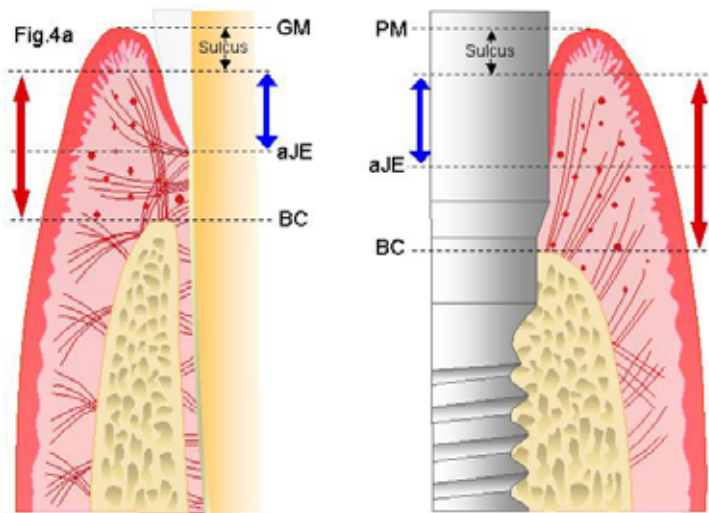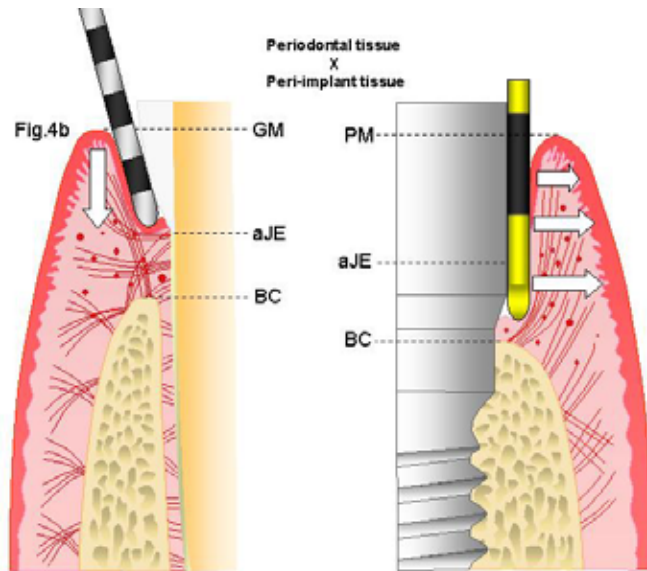
Fig.3-1-4-a

Fig.3-1-4-b

Fig. 3-1-4-a,b

The collagen fibrils in the structures surrounding the implant differ from those of the natural teeth, in that it starts from the periosteum of the alveolar crest then runs in parallel to the implant surface or lined in a straight line in a bundle form (a). In probing the implant surroundings, the probe tip reaches further in than the barrier epithelium (b).

II. Its association to periodontal disease

It has been reported that periodontitis and peri-implantitis have many disease states in common. Peri-implantitis is defined as a loss of the supporting bone caused by inflammation of the tissues surrounding the osseointergrated implant (Fig. 3-1-5-a, b, c). On the other hand, the fact that peri-implant mucositis occurs due to accumulation of plaque around the implant and similarly to gingivitis, a reversible inflammation of the soft tissue surrounding implant to occur became evident. The

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host responses at the early stages against bacteria are also identical. Peri-implant mucositis also has common features with periodontitis besides the loss of supporting alveolar bone. Both are induced by periodontal disease bacteria, and in a similar manner that gingivitis does not always progress to periodontitis, mucositis does not always progress to periateritis. The distinction however exists between the two. For periodontitis, the space between the gingivae (existence of bacterial flora) and bone are separated by the healthy peradentium fibers thus preventing the bacterial infection from directly affecting the bone. In per-implantitis, however, the infection can directly affect the alveolar bone, resulting in a lesion.

The causative microorganisms for the peri-implantitis such as, *porphyromonas gingivalis*, *Prevotella intermedia, Fuso-bacterium spp.,* and *Treponema denticola* have been found to be the same as those of the periodontal disease[17),18)]. The causative agent for the periodontal disease present in the periodontal pocket has been found to easily spread to the tissues surrounding the implant within the oral cavity. The detection rates of the causative agents for periodontal disease were determined using DNA probes. In investigating the subgingival periodontal pockets located adjacent to the natural teeth or the implants, the presence of another unique organism was found known as *Tannerella forsythia* (*Bacteroides forsythus*). This finding indicated that the periodontal pocket serves as a reservoir for microorganisms to readily spread throughout the mouth, emphasizing the importance of maintaining healthy periodontal tissues for prevention of the spread of pathogens for implant treatments[19)].

Monbelli *et al.* investigated the presence of Gram-positive and Gram-negative bacteria in the samples collected from the depth of the periodontal pockets and those surrounding the implant of a patient who was treated for periodontal disease. The analysis of the detection rate of both these bacteria around the implants, after three or six months, resulted in a positive correlation for both natural teeth and implant [20)].

The differences in the flora of the periodontal pockets of the teeth and implant surroudings at the varying depth within an oral cavity, from the shallow end to the mid-section, were analyzed by Quirynen *et al.* using checkerboard DNA-DNA hybridization and real-time PCR. Gram-positive cocci and bacilli, gram-negative bacilli (including periodontal bacteria) and the family of treponema were tested [21)]. Within two weeks of the abutment placement, the detection rate for each of the classes including periodontal disease bacteria, became the same in the area surrounding implant and in the periodontal pocket, indicating that the bacterial flora to be established in both structures.

The resistance of the tissues to inflammation resulting from the accumulation of plaque is evidently much lower with the implant than that of natural teeth, with consideration to the nature of the structures surrounding the implant[22)]. It is therefore essential to treat the periodontal disease of the remaining teeth, before the osseointegrated-implant treatment to avoid onset of peri-implantitis at the maintenance stages[19)].
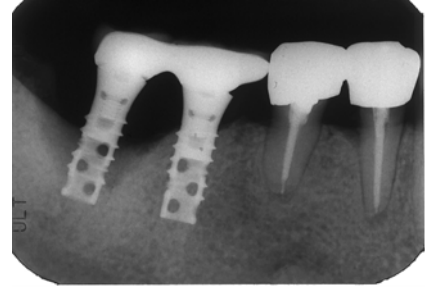
Fig.3-1-5-a   Fig.3-1-5-b   Fig.3-1-5-c

Fig. 3-1-5-a,b,c

Implantitis is an inflammatory condition arising in the tissues surrounding the implant, giving rise to the loss of the supporting bone.

A. Evaluation criteria for the outcome of implant treatment

Periodontitis is the primary cause of tooth loss; therefore there have been significant number of patients, affected with periodontitis, who have undergone implant treatment. This has enabled many clinical examples to be achieved, and leading to the conclusion of the importance of treating the periodontitis before the implant treatment. In treating the periodontitis it does not indicate the change in the sensitivity to the pathogens, and whether this will have any consequences on future onsets of implantits; or on the frequency of implant loss is in need of clarification. In other words, the correlation between the peridontitis in the past and the prognosis of implant should be the priority.

A systematic review to investigate of the correlation was conducted by Van der Weijiden *et al.* in 2005. Here, the results of implant treatment to periodontis-affected patients and non-peridontitis patients were compared. The results indicated of the differences in both, the alveolar bone supporting the implant; and in the frequency of implants lost to be the most significant[23].

In 2008, Ong *et al.* also published a systematic review that studied whether periodontal disease in the past affected the outcomes of implant treatment[24], as shown below:

1. Survival rate of implant

Cumulative "survival rate" indicates the existence of implant in the mouth during the observation period; and "unsuccessful rate" indicates the time of losing implant from the time of operation and classify the number of implant loss.

2. Success rate of implant

The standard for the success of implant had not been defined, therefore Albrektsson *et al.* Buser *et al.* and Karoussis *et al.* made "standard of success" in the cases where the symptoms listed below could not be detected:

Restlessness. discomfort such as pain, peri-implantitis around implants, acceleration of transparency of the bone surrounding implant, existence of pockets with sizes over 5 mm, and more than 0.2 mm progress in mesial or distal perpendicular bone defect after a year[25],[26],[27].

3. State of bone around implant

4. Incidence rate of peri-implantitis

Karoussis *et al.* defined peri-implatitis as a disease state in which a pocket of size more than 5 mm; presence of bleeding on probing (BOP); and bone resorption to be detected with X-ray radiography[28].

1. Implant survival rate in treated periodontitis compared with non-periodontits patients (Fig. 3-1-6)

The investigations into the implant survival rate in the vast number of studies have employed various evaluation standards thus making it impossible to draw conclusion from these comparable studies. For example, the period to be defined from the time of planting the implant or the time that the occlusional force was applied; or whether each implant or each patient was evaluated as a unit. The unit for evaluation by Karoussis *et al.*, Evian *et al.*, and Roos-Jansåker *et al.*, used of the time of planting the implant, and Watson *et al.*and Hardt *et al.* used the point at which biting force is applied as the standard. Concerning the evaluating unit, Watson *et al.*, Hardt *et al.,* and Karoussis *et al.*, used each implant, whereas Evian *et al.* and Ross-Jansåker *et al.* used each patient[28),29),30),31)].

Every other reports except that of Watson *et al.* showed a good correlation with the survival rate of implant for patients without a history of peridontitis compared with those treated for periodontitis. Evian *et al.* and Ross-Jansåker *et al.* also studied numerous cases for a long duration of time and concluded that there was a significant difference between the two patient types. However, it was later found that their observations were conditional on periodontal states of the patients[29),30)].

Fig. 3-1-6
Implant survival in treated periodontitis compared with non-perodontitis patients

2. Implant success rate in treated periodontitis compared with non-periodontitis patients (Fig. 3-1-7)

Similarly, the studies on the success rate of implants varied with their defining factors for evaluation such as the time of treatment, either from the time of planting the implant or the time that the occlusional force was applied; or whether to evaluate each implant or each patient as a unit. Thereby it was impossible to evaluate these results as comparable studies. The unit for evaluation by Rosenberg *et al.* and Mengel *et al.*, used the time of planting the implant, and Watson *et al.* used the point at which biting force was applied, Brocard *et al.*, used 6 months after the wound healing as the standard, whereas Karoussis *et al.* defined the standard as one year, respectively[28),31),32,33)].

Rosenberg *et al.* evaluated the failure examples by sorting them into two types. These were distinguishing by, firstly failure of osseointegration, and secondly, where peri-implantitis resulted[32)].

Overall, except for the evaluation by Watson's group, all of the other groups reported differences in the success rate to exist between the two patient groups. The patients without a past history of periodontitis had higher success rate than the treated patients. Furthermore, the cohort studies conducted by Karoussis *et al.* showed a significant decrease in the success rate of the patients with past history[28)]. With

regards to the other observations, their significance have not yet been verified.

Fig. 3-1-7
Implant success in treated periodontitis compared with non-periodontitis patients

3. The state of bone around implants in treated periodontitis compared with non- periodontits patients. (Fig. 3-1-8)

There have been five volumes of researches reported on the changes in the states of the bones surrounding implants with X-ray radiography. In all of these studies, the extent of bone resorption in patients without a history of periodontitis was found to be less than the treated group.

A significant difference was reported by Hardt *et al.*, as an exception. However, since there was no mentioning of the of the periodontitis treatment that was used, we cannot exclude the possibility that the progression of periodontal disease could have caused the bone resorption. The publication by Haggi *et al.* showed p = 0.058, a borderline, but they have since come to a conclusion that in case of patient with aggressive periodontitis, they are at increased risk of alveolar bone resorption compared with patients who have not been affected by chronic periodontits or periodontits[36]. Other researchers have not conducted statistical processing[28] to see if there are significant differences in bone resorption[33] between the two patient groups.

Fig. 3-1-8
Bone level change around implants in treated periodontitis compared with non-periodontitis patients

4. Peri-implantitis around implants in treated periodontitis compared with non-periodontits patients.

Three volumes of thesis[28),32),37)] indicated the reduction in the frequency of peri-implantitis incidence with implants in unaffected patient compared with periodontitis treated individuals. Regarding the two reports published by Karoussis *et al.* and Ross- Jansáker *et al.*, a significant difference was found between the two patient groupds. Both researches showed that the patient with a history of periodontitis to have higher incidence of peri-implantitis and to show low success rate[28)].

Fig. 3-1-9

Peri-implantitis around implants in trated periodontits compared with non-periodontitis patients

B. Clinical notes.

Patients who received periodontal treatment are more likely to encounter problems in the area surrounding the implant, such as loss of implant, bone resorption and inflammation of tissues around the implant. It is therefore necessary to inform and convince the patients before the implant treatment of the likeliness of contracting peridontitis later in the course.

The periodontal treatment should be conducted before starting the implant treatment, and by gaining a comprehensive view of the state and the conditions of the surrounding tissues, early detection and treatment should be possible, preventing the onset of periodontitis and peri-implantitis.

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