Behavior of peri-implant soft tissue in the interface with titanium: A literature review

Gabriela Farias de **MELO***
Iris **DURÃES****
Emilena Maria Castor Xisto **LIMA*****

Abstract

The soft tissue's adhesion to titanium, biologically capable to preserve and protect the peri-implant structures contributes to peri-implant mucosa's stability and maintenance of pink esthetics. Thus, the object of this study was, through a literature review, to describe the interaction between the titanium used in the prosthetic and implant components and the soft tissue to the maintenance of the stability and health of peri-implant tissues. It was concluded that the peri-implant soft tissue's stability is one of the criteria of success to rehabilitation with implants, once the establishment of an intimate relation between the soft tissue and the titanium of the implant, as well as the prosthetic components, promote a protective barrier to penetration of bacteria and its metabolic products, favoring the implant's long-term performance.

Keywords: Dental implants. Titanium. Oral mucosa.

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* Specialist in Prosthodontics, Bahian School of Medicine and Public Health.
** MSc student in Clinical Dentistry, Bahian School of Medicine and Public Health.
*** Doctor in Clinical Dentistry, Piracicaba School of Dentistry / Campinas State University. Adjunct Professor, Bahian School of Medicine and Public Health; School of Dentistry of Federal University of Bahia.

Contact address

Iris Durães Costa
Praça Marcone 59 – apto 502B, Pituba
CEP: 41.680-360 - Salvador/BA – Brazil
E-mail: iris.duraes@hotmail.com

Introduction

The treatment with dental implants is an established option in terms of functionality and durability.[25] However, taking the treatment as a whole, its success is defined not only by establishing and keeping the osseointegration, but also by the stability in peri-implant tissues (contour, color, texture), by the presence of keratinized tissue, interdental papilla, gingival contour in harmony with the adjacent teeth, as well as an adequate sealing between the soft tissue and the titanium, consisting in the creation of peri-implant soft tissue's contours that cannot be readily distinguished from natural adjacent tooth.[14]

Some authors[1-4] highlighted the importance of epithelial and connective tissue insertion around the implant to keep the osseointegration. The success of long-term osseointegrated implants depends on the adherence of the epithelium and of the connective tissue to the titanium surface, protecting the osseous tissue against microorganism from the oral cavity, and this adherence depends on the topography and chemical composition of the biomaterial.[9]

Titanium — commercially pure (c.p.) or in alloys — is usually recognized as bioinert, i.e., neither it releases any harmful substance nor it provokes any adverse reactions on the tissue.

Its biocompatibility is related by its capacity to form oxide layers on the surface while in contact with oxygen, hence it is widely used in orthopedic and dental surgeries.[21]

In terms of technology, there was a significant evolution in the manufacturing of implants, prosthetic components and its designs, due to the improvement of materials used and to the treatment applied to its surface. In this development context new parameters were adopted. Besides the functional requirement, the esthetics quality, once discredited, has become highly requested, increasing the expectations from the patients.[25]

Therefore, keeping the gingival health, the esthetics and stability is a prime challenge and special attention must be given to soft tissue and its behavior in face of the diversity of materials used on implant's prosthetic rehabilitations. Such concern begins in the treatment planning phase, ongoing the surgery stages throughout the installation of provisional and definitive prosthesis.

The insertion of peri-implant mucosa around commercially pure titanium implants was studied in different animals as well as in humans.[3-6] However, more studies are needed to understand the connection between artificial materials and living tissues, which type of material allows a better tissue response and what kind of surface is preferred by soft tissue or bone cells. This knowledge would help in the predictability of the response from bone or soft tissues when implants are inserted.[11]

Researches in implantology focus mainly in osseointegration, with only a few studies on the integration between soft tissues and implants. Thereby, the objective of this paper was, through a literature review, to clarify the biological behavior of peri-implant soft tissue contacting the titanium.

Literature review

Biological distance

Histological and radiographic observations suggest that there is a constant distance between hard and soft tissues and the implant, extending apically to the implant/abutment interface, similar to the dentogingival tissue, histologically named biological distance.[20]

After implants insertion, there is a lapse of time for bone remodeling that establishes a space to the insertion of a junctional epithelium. The components of this junctional epithelium have a composition similar to the periodontium after non-surgical treatment of periodontitis. Thus, it is established the biological peri-implant space (Fig 1), which has similar dimensions to the biological periodontal space (Fig 2), around 3 mm. Mucosa insertion around the titanium implants was of 3-4 mm high and included two portions: An epithelial component which is around 2 mm and a conjunctive one around 1-2 mm high.[1]

Hermann et al[17] presented a study, using dogs, with the objective of describing the changes that occur on the depth of the sulcus, junctional epithelium length and contact area of conjunctive tissue. This study showed that the biological dimension, which is the combination of soft tissues, did not change in any of the three evaluation periods, however, significant changes were observed in every tissue compartment (depth of the sulcus, junctional epithelium and conjunctive tissue). The depth of the sulcus and the dimension of the contacting conjunctive tissue decreased while the length of junctional epithelium increased. The fact that the total dimension of the biological distance remains the same after the healing process, suggests that the non-submerged one-piece implants allow a physiological stability of peri-implant tissues.

As well as in natural dentition, the biological tissue answers to the implant insertion through formation of periosteum, connective and epithelial tissue, creating a soft tissue strip which keeps the integrity of periodontium. The biological distance determines a minimum dimension of peri-implant mucosa that protects the junctional epithelium and the conjunctive tissue, keeping a sealing around the implant which provides protection against biological and mechanical agents.[24]

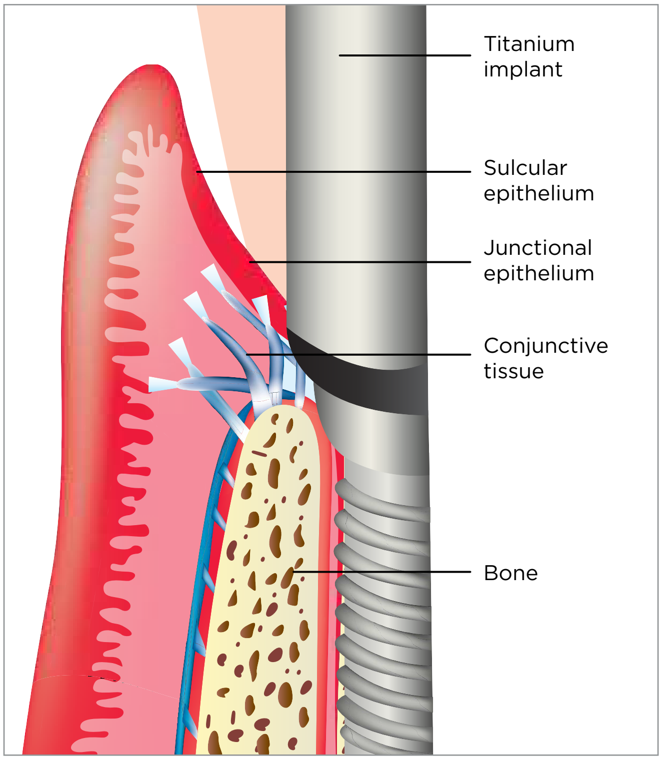

Figure 1 - Insertion apparatus of peri-implantar tissues.[26]

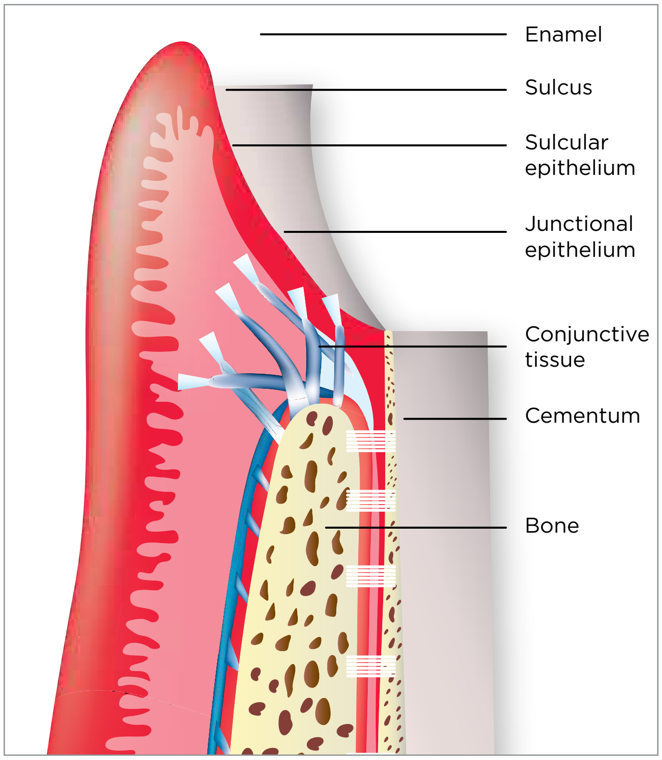

Figure 2 - Insertion apparatus of periodontal tissues.[26]

Epithelial tissue

The characteristics of transmucosal junctional epithelium and the conjunctive insertion in the implant are established during the healing of the implant installation surgery. In this context, it is believed that the epithelium plays an essential role on wound healing, covering all the connective tissue, which is sectioned during the surgery. Thus, the epithelial cells located on the edge of the mucosa, produced by the implant installation, are encoded to divide and migrate through the wounded part until the epithelium restoration. The epithelial cells have the ability to merge to the implant surface and establish a barrier that has common characteristics with the junctional epithelium of the tooth.[29]

Because of the capability of the epithelium to proliferate and move over surfaces, the epithelium found on the edge of the surgical incision crosses the fibrin clot/granulation tissue bridge, which is formed right after the implant installation. Before reaching the implant surface, the epithelium moves 2 mm in coronoapical direction, yielding the junctional epithelium. This migration is interrupted when it finds a connective tissue organized with fibroblasts and collagen fibers adhered to the implant surface.[29]

In the implant site, the apical portion of the junctional epithelium is consistently separated from the alveolar bone by a non-inflammatory, collagen-rich, but cell-poor zone. This zone size is about 1-1.5 mm, it is continual to the junctional epithelium and it establishes an implant-mucosa adherence which sizes 3-4 mm. On the collagen-rich zone, the fibers invest on the marginal bone in a somewhat parallel course to the implant surface.

Adhesion mechanisms

Some studies[1,9,14] demonstrated that the mucosa portion in contact with the titanium abutment surface may be divided in two different zones: A marginal zone which lodges a junctional epithelium and an apical zone, that is composed by a fiber-rich conjunctive tissue.

The laminin is an important component of the basal membrane and seems to be an adhesive stabilizer factor of the hemidesmosome, retarding its cellular motility.[22]

Regis and Duarte,[25] in 2007, as well as Buser et al,[9] in 1992, suggested that the connective tissue on the interface zone is similar to a cicatricial tissue (scarce in cells and vascular structures, but rich in collagen fibers) firmly united to the implant surface.

Hormia et al[18] performed a study using cell culture to investigate the adherence and growth of fibroblasts and epithelial cells on titanium surface. The study suggests that the titanium surface topography influences the behavior of such cells, favoring an apical epithelialization under pathological conditions. These two types of cells show distinct preferences for different molecules and potentiates the insertion of gingival cells in different implant surfaces.[19]

In an experiment made by Lauer et al,[19] in 2001, it was found that gingival keratinocytes have adhesion in three different types of titanium surfaces that were studied: Glossy polished; sandblasted; and plasma-sprayed. Through migration and proliferation, these cells covered the three surfaces. The biggest extension was observed on the polished surfaces and the smallest on the plasma-sprayed ones. This study showed that keratinocytes have more attraction for polished than to rough surfaces. However, it could not be confirmed greater adhesion force on polished surfaces when compared to treated surfaces.

Berglundh et al[8] held a study in 2007 using dogs with the objective of verifying the progression of the peri-implantitis around the implants. They observed, through radiographic and histological exams, that the disease progression is more favorable on implants that have a more rough surface than on implants that present more polished surfaces.

Connective tissue

In a natural teeth, the dentogingival collagen fibers are firmly inserted into the cementum and into the bone, oriented perpendicularly or obliquely to the tooth surface, posing as a barrier to epithelial migration, thus preventing a bacterial invasion.[13]

The peri-implant biological components are similar to the dentogingival complex in its constitution and in the formation of biological distance. Both tissues react to the presence of plaque with an increase of leukocyte migration through the junctional epithelium and with the establishment of an infiltrate of inflammatory cells in the connective tissue.[4] However, it is important to highlight some differences, such as the collagen fiber disposition and its absence on the titanium surface, which allows the formation of a system more vulnerable to bacterial invasion and mechanical aggressions while compared to the tooth.

The absence of cementum in the implants promote a parallel orientation of the fibers of supracrestal soft tissue in relation to the implant.[3,9] This peculiar arrangement offers less mechanical resistance when compared to periodontal ligaments, being able to affect the prognosis of dental implants, often being observed gingival recession, gingival pocket and bone resorption.[9]

Gargiulo, Wentz and Orban[13] named as connective tissue attachment the region located between the apical portion of the junctional epithelium and the bone crest. Such denomination seems to be appropriate, because there are still contradictions concerning the real adhesion existing between the tissue and the implant and/or the prosthetic component.

Vascular supplement

The vascularization between the periodontium and the peri-implant happens in a distinct way. Studies about the vascular topography of periodontal and peri-implant tissues observed that the gingiva and the supracrestal connective tissue adjacent to the tooth were supplied by blood vessels originated on the periodontal ligament, while the peri-implant mucosa was vascularized by blood vessels originated on the periosteum next to the implant. In both situations, the blood vessels build a characteristic vascular plexus sided to the junctional epithelium.[4]

In teeth, the supracrestal connective tissue showed a rich vascularization, while in a similar region in the implant few or no blood vessels were found.[12] The scarcity of blood vessels next to the implant surface supports the statement of Buser et al,[9] that the peri-implant soft tissue would have a reduced capacity of defense against exogenous irritations, for instance, biofilm.

Defense mechanism against aggression

In teeth, the presence of periodontal pocket, evidenced by increased probing depth, is due to periodontal disease or bone loss.[6] However, in implants it is risky to associate the dental sulcus depth and this disease, as there are reports of stable and rigid implants with probing depth varying from 2 mm to 6 mm.[12]

When an external agent invades the biological depth, the epithelium responds to it by migrating to a place far from the harmful agent, in an attempt to isolate it and create a defensive distance that may assure the peri-implant integrity. This leads to resorption, thus assuring the reestablishment of biological distances.[6,7,24]

Abrahamsson et al[1] demonstrated the existence of an infiltrate of inflammatory cells which is bigger on the interface between the implant and the ceramics abutments when compared to the titanium ones. They also suggested that this fact could be explained by the existence of a larger microgap between implant and the ceramics abutment. Opposing to this statement, Yüzügüllü and Mhemet[33] observed the existence of a bigger microgap on the implant/titanium interface when compared to the interface between the implant and alumina or zirconium.

Keratinized mucosa

The presence of an adequate strip of keratinized peri-implant mucosa with good thickness conditions offer functional and esthetic benefits to rehabilitations. The main advantages are a greater facility in conditioning the peri-implant tissues and molding, lower occurrence of recessions with abutment exposure, greater facility in peri-implant biofilm control, lower mucosa sensibility during oral hygiene procedures and better protection against infection in peri-implant tissues.[27]

Warrer et al[31] verified that the absence of keratinized mucosa increases the susceptibility and tissue destruction in implant sites. However, the presence of keratinized mucosa was observed with lower frequency in the peri-implant pocket when compared to implants surrounded by alveolar mucosa.

Though, the indispensable need of keratinized mucosa to peri-implant health is controversial. From a clinical point of view, there are no evidences that the absence of keratinized mucosa harms the longevity of implant-supported rehabilitations when oral hygiene is satisfactory. The implants can survive even with a lack of such tissue. This can be observed in patients who received protocol-type prosthesis and were accompanied on the long term, showing fixation stability.[27]

Vertical and horizontal distances and the papilla formation

The proximal bone linked to the adjacent teeth is a precondition to the presence of papilla. The bone presence does not necessarily guarantee that the papilla fill the interproximal space. Nevertheless, in 80% of the cases the spontaneous filling may occur if the design of the prosthesis is taken into account and if the proximal bone is present.[23]

Tarnow et al[30] evaluated the vertical distance from the crest of bone to the height of the interproximal papilla between adjacent implants, independently from the contact point location. A total of 136 interproximal papilla heights were examined in 33 patients. The average papillar tissue height found between two adjacent implants was 3.4 mm, with wide diameter between 1 and 7 mm. The most frequent probing depths were: 2 mm, in 16.9% of the cases; 3 mm, in 35.3% of the cases; and 4 mm, in 35.7% of the cases (totaling 90% of the total). They concluded that caution must be taken when installing two adjacent implants in esthetic areas, as in most of the cases one can expect only 2 mm, 3 mm or 4 mm of soft tissue to cover the inter-implants bone crest.

Grunder,[15] in 2004, related that the ideal inter-implants distance to form papilla would be 4 mm, believing that a 3 mm distance, as Tarnow et al[30] related in their study, wouldn't be sufficient.

Characteristics of the titanium surfaces

The quality of the surfaces has maximum importance to the establishment of a proper relation between the implant and the tissues, which refers to the surface structure as well as its chemical and biological properties. Progress in knowledge regarding these biological effects can provide a better response on the implant interaction with the tissues and its clinical performance.[11]

The implant surface rugosity can be obtained through the manufacturing process or through subsequent treatments involving machining, particle blasting, titanium plasma spray, chemical or electrochemical attack, and a combination of these procedures.[32]

The geometric dimensions of the surface microstructure influence the cellular adhesion, morphology, orientation, proliferation, differentiation, and the production of local factors. The effect of surface topography in cellular adhesion varies according to the type of cell. Human fibroblasts have more adhesion in electropolished surfaces than in treated surfaces. Contrarily, osteoblasts demonstrate more adhesion in rough surfaces than in flat ones.[32]

Until the early 90's the cervical portion of the most of dental implants used to have a machined-surface of Brånemark System® type. The intention was to prevent plaque accumulation when the implants were exposed to the oral environment, which could cause severe problems like peri-implantitis. However, nowadays it is believed that the microgrooves in the neck of the implants can promote improvements to the epithelial tissue adaptation on the long term, though more studies are necessary to confirm the real benefits of this implant structure modification.[25]

Çomut et al[10] observed, on the one hand, an effective formation of mucosa insertion in c.p. titanium surfaces and in hydroxyapatite-revested titanium, with the fibers organized in a parallel orientation in all samples. On the other hand, the insertion of human gingival fibroblasts into c.p. titanium proved to be significantly bigger than in hydroxyapatite, porous and nonporous.[16]

The cells adhesion and its proliferation in a biomaterial depend, among other factors, on the surface wettability which, in its turn, is affected by the roughness of the biomaterial. Implants with moderate surface roughness have clinical advantages when compared to implants with a too-flat or too-rough surface.[2]

Lauer et al,[19] in their turn, observed that after a 6-day period, 28.1% of the titanium flat surface was covered, and after a 12-day period this covered surface had increased to 61.3%. In contrast, in 6 days, the cultivated gingival keratinocytes had expanded less on a sandblasted surface (11.3%) and a plasma-sprayed titanium surface (11.1%). Nonetheless, the adhesion did not seem to be as good as on the two other surfaces examined, though the layers of gingival epithelial cells covered an extensive area of the titanium flat surface.

In spite of the consensus around the importance of the formation of an effective barrier between the peri-implant soft tissue and the titanium to keep the stability of the gingival margin, more studies are necessary about the mechanisms of such adhesion, as well as other elucidations about the design, surface topography and material composition.

Bone loss and stability of gingival margin

The peri-implant mucosa represents a type of cicatricial tissue created by a surgical intervention without insertion of supracrestal fiber on the cementum; and the peri-implant bone level constitutes a base for the supracrestal soft tissue. In the period between the implant insertion and the abutment connection, a significant quantity of buccal bone loss was reported in a bone thickness < 1.8 mm, measured right after the mounting of the implant. This can negatively influence the topography of the soft tissue and the esthetical result of the restoration.[28]

Repeated installation and removal of the abutment can provoke an aggression to the soft issue, which will respond with mucosa recession. In an attempt to reestablish the biological distance needed to promote a sealing of the soft tissue around the implant head, it happens a bone crest resorption, aiming at creating space for the three main marginal structures: The sulcus epithelium, the junctional epithelium and the area of connective junction.[5] This phenomenon was demonstrated in another study,[1] with monthly changes of the abutment, leading the tissue to a loss of insertion. During the cicatrization process, there is a reinsertion of the peri-implant mucosa with the formation of two zones: Junctional epithelium (2 mm) and connective tissue (1 mm).

Likewise, when the phenotype of the peri-implant mucosa is thin (2 mm or less), bone resorption is observed, sustaining the theory that a minimum soft tissue thickness is required (approximately 3 mm) to reestablish a biological distance.[5]

Hermann et al[17] verified that the bone crest level around the implant screws was located around 1.5 mm to 2 mm below the implant-abutment junction (IAJ). Radiographically, it could be verified that this bone resorption usually reaches the first screw of the implant. One of the theories developed to explain such phenomenon states that a bone remodelling happens due to an inflammation located on the soft tissue of the implant-abutment interface, in an attempt to obtain space for the establishment of a mucosa barrier around the implant head.

One of the difficulties to restore the papilla between two adjacent implants is that the biological width around the implants is re-established apically to the implant-abutment connection. Allied to it, in esthetic areas, the implant is installed approximately 4 mm apical to the height of the soft tissue in adjacent teeth. Another factor that contributes to this difficulty is the difference in the location of the biological space. Between the platform of two implants, this space is subcrestally located, while in normal teeth it is supracrestally located.[30]

On Implantology, the difficulties are increased while attempting to solve the problems that occur in soft tissues after the implant insertion. Prospects are not the same, for example, when it is done a coating in titanium structures of implant, as it happens in several coating techniques for radicular recessions in natural teeth.

Conclusion

The stability of peri-implant soft tissues is one of the success criteria in implant rehabilitations, once the establishment of an intimate relation between the soft tissue and the titanium of the implant, as well as the prosthetic components, promote a protective barrier against the bacterial penetration and its metabolic products, thus influencing the long-term performance of the implants.

Besides, this interaction between the peri-implant soft tissue and the titanium piece depend of some factors that must be taken into consideration, such as: The chemical composition of the material, the surface topography and the periodontal phenotype of the patient.

For this reason, it seems to be evident and justifiable the need of an anticipated intervention in the implant mounting, aiming at the improvement in the quality of peri-implant soft tissues.

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