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“Comprehensive Dentistry in the International Community”

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Changes of Periodontal Tissue in Periodontitis

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Abstract

Chronic periodontitis is an inflammatory condition of teeth-supporting tissues, which cause the loss of tooth attachment, alveolar bone damage, and tooth mobility. The disease process results in clinical and histopathologic changes of periodontal tissues. The purpose of this paper is to observe the successful healing of periodontal therapy. Periodontal disease progression depends on the presence of bacterial plaque. Its accumulation causes histopathological of healthy gingival tissue turns into initial lesion, in which then proceed to early lesion and further to established lesion. In early lesion, junctional epithelium changes in the coronal area; meanwhile, in established lesion the proliferation lateral arises along with the depth of gingival sulcus accompanied by the formation of gingival pockets. In periodontitis, epithelial proliferation develop towards apical and periodontal pocket. Periodontal therapy aims to establish periodontal tissue healing. Classification of periodontal healing are reattachment and regeneration of the epithelium, as well as of the connective tissue.

Keywords: periodontitis, periodontal therapy.

INTRODUCTION

Chronic periodontitis is an infectious disease with an inflammatory condition of teeth-supporting tissues, causing the loss of tooth attachment to the supporting tissue and alveolar bone damage as well as tooth mobility.¹

Initiation and progression of periodontal disease depends on the presence of bacterial plaque. The accumulation of bacterial plaque resulting in healthy gingival tissue provides histopathology illustration on the initial
lesion of which then converts to early lesion and further to established lesion. In initial or early lesion there are changes on the junctional epithelium in the coronal area. While in established lesion, the proliferation lateral arises caused from junctional epithelium, along with the depth of gingival sulcus, occur with the formation of gingival pockets (pseudopocket). The proliferation epithelium in periodontitis occurs towards apical in which form pocket periodontal.

Gingivitis in adults is usually in the category of established lesions, which is known with a quite high intensity. Initial and early lesions are precursors of established lesions. In adults initial and early lesions may persist for several days. Meanwhile, in children, early lesions may persist for some time and clinically observed. Established lesions in adults can last for several years, might also be in a decade without progress towards periodontitis. The disease process of gingivitis to periodontitis can be caused by the change of pathogenic potential of plaque. Periodontitis might manifest clinically in a variety of forms.¹

Gingivitis is an inflammatory process without the presence of attachment loss. Gingivitis is caused by plaque, gingival changes can be found in the hormonal milieu during the time of menstruation, systemic disease, and side effects of drugs. If accompanied by alveolar bone destruction due to inflammation of the periodontal tissues, thus it is called periodontitis.

The relationship between gingivitis and periodontitis is a complex relationship. Plaque accumulation most likely causes gingivitis, however this is not always refer to a progress towards periodontitis. Periodontal tissue destruction and the pocket establishment are unpredicted. When there is damage in periodontal tissues and pockets formed, it will result in the loss of attachment.

Periodontal pocket is deep depression of the gingival sulcus pathologically and is the entrance of bacteria and an indicator of periodontal disease. In healthy gingival composed of connective tissue and epithelial is divided into three areas: oral epithelium, sulcular epithelium and junctional epithelium of which acts as a barrier against bacteria that cause periodontal disease.
Healthy gingiva has a clinical picture of color pink in color, stippling, with a tapered gingival margin and a scalloped outline; it would not bleed when probing with light pressure.

The main goal of periodontal therapy is the healing process development in periodontal tissues. The healing process is affected by epithelial tissues. Four types of classification of the possibility of this remedial process are epithelial reattachment, epithelial regeneration, connective tissue reattachment, connective tissue regeneration.

LITERATURE REVIEW

Pocket depth can be increased for two reasons, namely the movement of the gingival margin to direction of coronal due to the swelling of the gingival sulcus or the sulcus becomes deepen. Inflamed periodontal pocket lesions are initiated by bacteria found in plaque biofilm layer, which is then followed by damage to the surrounding of connective tissue. Mechanisms that affect the loss of collagen in the connective tissue are enzymes produced by healthy cells as well as inflammatory cells and collagen fibers phagocytosis by fibroblasts. As a result of loss of collagen from the apical to the junctional epithelium, therefore the epithelial cells will migrate towards the apical and coronal portion of the junctional epithelium will be separated from the root surface of the teeth along with the increased number of cells that endure inflammation. As part of the inflammatory process, gingival also experience intumesences in line with the increased number of cells and serum elements, hence gingival margin looks swollen toward coronal direction. Epithelial layers on the pockets will lose its integrity, therefore leukocytes and inflammatory products will be released into the pocket and then bumps epithelium (rete pegs) will spread out from the pocket layer to the area of connective tissues.

The content within the pocket has an effect on the root surface of the tooth. Lipopolysaccharide (endotoxin) which is derived from organisms in biofilm penetrates to the cementum, which will damage the epithelial attachment to the tooth root surface. Endotoxin disposal, cleaning calculus and biofilm layers are included in the rationale of root planning.
Pathogenesis Periodontitis

It is uncertain that when gingivitis condition ends, the symptoms of periodontitis begin. Some researchers believe that periodontitis will commence as soon as gingivitis start to develop and is experiencing loss of epithelial attachment. Nevertheless, further clinical studies have not shown if and when those transitions occur. In periodontitis disease, pathogenesis feature is the widespread inflammatory properties in the area of epithelial attachment and the formation of periodontal pockets. The following two mechanisms describe the initial deployment of infection:

1. Bacteria and their products will damage the lining liaison between sulcular epithelium and the tooth surface and cause the release of junctional epithelium attachment.
2. Bacterial products will hamper the normal growth and sulcular epithelium attachment, resulting in damage to the part.

In the same case, along with the development of inflammation thus sulcular epithelium is thickened and will begin to infiltrate into the underlying connective tissue.

The pocket becomes deepen due to the destruction of collagen fibers in gingival connective tissue because of the presence of enzyme collagenase, which is produced by a variety of bacterial plaque and host cells which are inflamed. Formation of periodontal pockets and the deepen process occurs when junctional epithelium extends toward the apical of which is then detached from the base of the crown of the tooth / cervical area. Bacterial plaque will release a chemotactic substance which increases the flow of neutrophils into the gingival sulcus. The reaction between neutrophils against bacteria will produce suppuration (pus or exudate) which is often seen in the progression of periodontal disease.

Periodontitis begins with the migration of junctional epithelium towards apical direction and loss of alveolar bone crest. Bone is an active tissue that is constantly undergoing a process of formation and resorption, thus it is difficult to ascertain whether or not the bone loss caused by periodontitis. At the time of bone resorption exceeds apposition process / its formation, then the reduction in bone composition will occur. The loss of the alveolar
bone crest due to the inflammatory process is called periodontal tissue loss. Most cases of bone loss which are seen in periodontal disease are more commonly caused by local inflammation compared to systemic change.

By the time symptoms of periodontal disease begin to appear, then either plasma cells or lymphocytes will appear in the periodontal tissues. Plasma cells are very important in antigen-antibody reaction that activates the flow to reproduce inflammatory cells into the periodontal tissues. These cells will also produce active molecules which cause severe damage to the collagen fibers in the periodontal connective tissue. Lymphocytes itself when are stimulated by bacteria found in plaque biofilm layer, it will release lymphokines of which refer to another type of active protein. Lymphokines provide a wide range of effects on inflammatory systems, including producing chemical factors that will enable the osteoclasts to do its job that is to resorpt bone.

Other products from the interaction of bacterial plaque with inflamma-
tory cells include prostaglandins, which also stimulates bone resorption process. In combination with other factors such as complement activation, then it will cause damage to the bone and the development of periodontitis symptoms. Interestingly, by trying to understand the mechanism of bone resorption course, it will provide guidance on new methods on treating periodontal disease. For instance, nonsteroidal anti-inflammatory drugs would inhibit prostaglandins, while recent studies suggest that those drugs also seem to inhibit bone breakdown that is generally occurred in periodontitis. However, this process will result in various degrees of variations in pocket depth, tissue inflammation, bone destruction, tooth root furcation opening section, and tooth mobility which are common in periodontitis.

For several years it has been believed that periodontal disease is constantly experiencing infectious growth phase which has the characteristics of nonspecific pathogenesis. As the understanding of specific plaque hypothesis increases, then the concept of periodontal disease is imperfectly start to appear. This means that periodontal disease has a number of activities which have been followed by periodic healing process. During the active phase, bone and other periodontal tissue experience
damage and pockets are getting deepen. Active period is often associated with bleeding tissue and provide an opportunity for researchers to perform diagnostic tests. Whereas during the passive period, the disease course will remain static and pockets are not getting deepen.

**Periodontal Healing**

The main goal of this therapy is to eliminate the current causative factor, and the increased in periodontal tissue healing. Various kinds of research which have been conducted in recent years have addressed the question of the possibility of healing in the form of re-attachment and regeneration of epithelial and connective tissue. (Figure 1).³

![Figure 1. Periodontal pocket a) Before treatment, b) After treatment](image-url)

- Pocket
- Junctional epithelium
- Destroyed periodontal ligament (PDL)
- Inflammatory Infiltrate
- Level of PDL attachment
- ▽ Boundary of treated root surface
- SC Scaler tip
- CT Connective tissue
- OE Oral epithelium
Healing that occurs in periodontal tissues comprises of epithelial regeneration, connective tissue repair (new attachment), connective tissue regeneration / periodontal and healing failure causing apoptosis or cell death. In the regeneration of epithelial, healing which is formed comes from formations of long junctional epithelium that is growing toward the apical between root surface and gingival connective tissue. In the repair of connective tissue, the collagen fibers grow parallel to the root surface that has undergone periodontal disease. Periodontal regeneration is a healing that comes from the formation of cementum, periodontal ligament, alveolar bone, and gingiva (Figure 2).³

![Diagram of Healing Types](image)

Figure 2. Type of Healing c) long junctional epithelium, d) periodontal regeneration
DISCUSSION

There is a correlation between the clinical and histopathologic representation of gingivitis. Early infiltration of PMN which appears in healthy gingiva occurs as a host response to the number of existing plaques. Some types of plaque composed of non-pathogenic microorganisms, such as Gram positive.

Along with the increase in plaque accumulation, the clinically detectable inflammation is increased in order to comply with the increased number of inflammatory cells infiltration. The inflammatory cells consist of lymphocytes B and some types of leukocytes. The increased infiltration causes large numbers of PMNs headed to the junctional epithelium. If the inflammatory process is continuing, junctional epithelium will change its role into pocket epithelium (barrier). ¹

Gingival Epithelium

Gingival epithelium consists of stratified layers of squamous epithelium, with its cell types namely keratinocytes (the primary) and nonkeratinosit. Epithelial cells play an active role in the innate host defense by interactively responding bacteria, which means epithelium participates actively in responding to infection, the host-cell signaling in further reaction, and in integrating the innate and adaptive immune response. Epithelial cells could respond to bacteria by enhancing proliferation, changes in cell signaling events, changes in differentiation and cell death, and changes in homeostasis tissues.

The main role of the gingival epithelium is to protect the deeper structures, particularly at the time of the selective exchange with the oral environment. This is achieved by proliferation and differentiation of keratinocytes. Keratinocyte proliferation occurs by mitosis in the basal layer and diminishes in the suprabasal layers, where some fraction of cells remains as proliferative compartments while larger numbers of cells begin to migrate more to the surface. Each oral epithelium experiences continuous renewal. Its thickness is maintained by a balance between the formation of new cells in the basal and spinous layers and the release of old cells on
the surface. Mitotic activity takes place in 24-hour period continuously. The mitotic rate is higher in the nonkeratinized area and increases in gum inflammation, with no significant gender differences.

Differentiation involves keratinization process, which consists of a series of biochemical and morphological events that occur in cells as they migrate from the basal layer (Figure 3).

![Diagram of epithelial layers](image)

Figure 3. Diagram that shows the cells of various stratified layers of squamous epithelial as seen by electron microscopy. (Modified from Weinstock J: Dalam Ham AW: Histology, ed 7, Philadelphia, 1974, Lippincott).

**Types of Gingival Epithelium**

Components of the gingival epithelium illustrate morphological variations of each area that reflects the network adaptation to the tooth and alveolar bone. Three different regions can be defined based on the perspective of the morphological and functional of gingival epithelium are oral, sulcular epithelium and junctional epithelium.
Oral Epithelium (outer)

Outermost or oral epithelium covers the summit area and the outer surface of the marginal gingiva and the surface of the attached gingiva. The epithelial thickness is approximately 0.2 to 0.3 mm. Oral epithelium consists of four layers: the stratum basale (basal layer), stratum spinosum (prickle cell layer), stratum granulosum (granular layer) and stratum corneum (keratin layer). It also contains different shapes, such as parakeratinized or keratinized or various combinations (Figure 4). Gingival keratinization level decreases with age and the onset of menopause but is not necessarily related to the phases of the menstrual cycle. Keratinization of oral mucosa varies in different areas in the following orders: palate (most keratin), gingival, ventral tongue, and cheek (least keratin).  

![Image of oral epithelium with labels](image)

Figure 4. Variations in the gingival epithelium. A, keratin. B, Nonkeratinized. C, Parakeratinized. Corneum layer (H), granular layer (G), the prickle cell layer (P), the basal cell layer (Ba), evenly surfaced cells (S), parakeratotic layer (Pk).

Sulcular Epithelium

Sulcular Epithelium is an epithelium that covers gingival sulcus with the type of stratified squamous epithelium without thinned rete pegs and extends from the boundary of the coronal junctional epithelium to the top of gingival margin. Gingival sulcus has the shape of 'V' with a deep space or groove between the teeth and gums that surround the tip of the crown which has just erupted. The sulcus comprises a shallow gap which is the culmination of junctional epithelium attachment and is bounded by the teeth on one side and sulcular epithelium on the other side. The coronal expansion of the gingival sulcus is gingival margin.
As with other nonkeratinized epithelium, epithelial sulculer has no stratum granulosum and stratum corneum and cytokeartins. Sulcular epithelium has a crucial role because it can act as a semipermeable membrane against harmful bacterial products that get into the gingival and tissue fluid from gingival crevice into the sulcus. Unlike the junctional epithelium, however, sulcular epithelium is not much infiltrated by polymorphonuclear neutrophil leukocytes (PMN), and appears to be less permeable.¹

Junctional Epithelium

Junctional epithelium consists of a webbing of stratified nonkeratinized squamous epithelium and is formed by the confluence between oral epithelium and reduced enamel epithelium during eruption. Junctional epithelium only continues to survive on perfect tooth eruption. Junctional epithelium is composed of 3 to 4 thick layers in early life, but the number of layers increases along with age, up to 10 or even 20 layers. These cells can be grouped into two strataums: basal layer facing connective tissue and suprabasal layer extends to the surface of the tooth. Long junctional epithelium is ranged from 0.25 to 1.35 mm.

Junctional epithelium is a structure that continues to renew itself, with mitotic activity that occurs in all cell layers. Epithelial cells that regenerate move toward the surface of the tooth and along the direction of the coronal to the gingival sulcus, where they are separated (Figure 5). The migrating cells provide continuous attachment to the tooth surface.

In sum, junctional epithelium shows some unique structural and functional features that contribute to prevent pathogenic bacterial flora colonizes on the subgingival tooth surfaces. Firstly, junctional epithelium is firmly attached to the tooth surface, forming epithelial barrier against plaque bacteria. Second, it allows gingival fluid access, inflammatory cells, and host immunologic defense components to the gingival margin. Third, junctional epithelial cells shows rapid changes, in which contribute to the balance of host-parasite and rapid repair of damaged tissues. Furthermore, some research suggests that junctional epithelial cells have the same endocytic capacity which is similar with macrophages and neutrophils and
hence this activity may be a natural protector.  

Figure 5. Junctional epithelium in tooth eruption. Junctional epithelium (JE) is formed by the merger of the oral epithelium (OE) and the reduced enamel epithelium (REE). Afibrillar cementum (AC) is sometimes formed on the enamel after degeneration of the REE. The arrows indicate coronal movement of epithelial cells regeneration, which proliferate more rapidly than at OE JE. E, Enamel; C, root cementum. The same pattern of changes in the cells can also be seen on perfect tooth eruption. (Modified from Listgarten MA: J Can Dent Assoc 36:70, 1970).

CONCLUSION

Chronic periodontitis is an infectious disease with an inflammatory condition of teeth-supporting tissues, causing loss of tooth attachment to its supporting tissue and bone destruction as well as tooth mobility.

Periodontal pocket is a deep depression of the gingival sulcus pathologically and is the entrance of bacteria and an indicator of periodontal disease. In a healthy gingival composed of connective tissue and epithelial
is divided into three areas: oral epithelium, sulcular epithelium and junctional epithelium of which acts as a barrier against bacteria that cause periodontal disease.

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