Ultrastructural modifications at the level of marginal periodontium in the case of incorrect dental reconstruction

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Abstract
Our study focused on the evolution of the marginal periodontium inflammatory process caused by an incorrect dental reconstruction. Our research studied a control group and a group of patients having traumatic and inflammatory lesions in different stages of evolution. A pronounced rarefaction of the junction desmosome structures as well as an inflammatory process pointed out by the presence of macrophages, neutrophils, Langerhans' cells, and mastocytes. The presence of altered fibroblasts and collagen fibers in the electron microscopic sections of vascular lesions represents microscopic signs of the inflammation and support the theory of local immunoglobulin synthesis.

Keywords: marginal periodontium, incorrect dental reconstruction, electron microscopic sections.

Introduction
The morphology of the proximal dental surfaces as well as the architecture of the interdental contact point has an important role in maintaining the health of the marginal gingiva and especially in the interdental papilla area.

The normal form of the interdental papilla in the front region is pyramidal at the posterior side teeth; the spatial form of the interdental papilla was compared to the form of a tent, with a cavity on the superior edge [1, 2].

In the cases of dental reconstruction, which do not reproduce the same structure, the interdental papilla presents signs of acute or chronic inflammation, which permits the penetration of the microbial toxins through the lining epithelium.

The degree of finishing of the dental reconstruction has an important role in maintaining the integrity of the periodontal tissues. Hence, the epithelium of the channel clings to a fine polished reconstruction, while a rough surface favors small traumas of the epithelium, sometimes with the appearing of micro ulceration and inflammation [3].

There are inflammatory gingival lesions due to the chemical composition of the materials used in the reconstructions. The inflammatory lesions appear frequently around the acrylate or silicate reconstructions. These can be explained by the low pH of the silicate and the residual allymer freed by the acrylate.

With the metal and amalgam reconstructions, there are less inflammatory traumatic lesions.

The purpose of our study was to follow the evolution of the periodontium inflammatory process caused by incorrect dental reconstructions.

Material and Methods
We selected a control group and a group of patients (three) which presented subjective and objective clinical phenomena, consisting of inflammatory and traumatic lesions in different degrees of evolution.

The persons from the patient lot present the following clinical table: a red-violet gum, tumid, sometimes with micro ulcerations and frequent bleedings when touched. The fibromucous when palpated has a soft or tougher consistency depending of the presence or the absence of the extra-added inflammation. The gums of the control subject are not inflamed and the contact points not damaged.

The morphological analysis was made on the normal fibromucous gum of the teeth pulled out in orthodontic purpose. The patients surveyed did not suffer of general diseases such as: hyperglycemia, hypothyroidism, the Addison disease, agranulocytosis, atherosclerosis, hematological diseases, pregnancy, hypovitaminosis, cirrhosis, which influence the local metabolism or/and the individual biotype aspect of the periodontium tissues. The sampling of the pathological and normal tissues was made at persons between 20 and 30-year-old.
in order to be analyzed at the Electronic Transmission Microscope (ETM), type Jeol JEM 1010, in the Center of Electronic Microscopy of the “Babeș Bolyai” University, Cluj-Napoca.

**Results**

Our study illustrates electron-microscopic images of global aspect of the spiky layer in the normal gingival epithelium of the control subject (witness) and of the patients.

Figure 1 shows a section through normal gingival tissue. We can see the tight relationship between the cells, united by numerous desmosome junctions in the intercellular spaces. An epithelial cell is formed of nucleus and nucleolus, the cytoplasm rich in mitochondrion and ribosomes, rough endoplasmic reticule. The cellular components: the cytoplasm, the nucleus, the nucleolus are shown.

![Figure 1](image1)

**Figure 1 – Section through normal gingival tissue (a), nucleus (b), nucleolus (c), cytoplasm (d), intercellular spaces with numerous desmosomes. TEM, ×6000.**

Next image illustrates normal gingival chorion presenting a fibroblast surrounded by collagen fascicles. The fibroblast nucleus and cytoplasm are shown. The most numerous cells of the gingival chorion, are fibroblasts and are disposed around the blood vessels and between the fibrillar elements (Figure 2).

![Figure 2](image2)

**Figure 2 – Normal gingival chorion presenting a fibroblast surrounded by collagen fascicles (a), cytoplasm (b), nucleus (c), – transversal sectioned collagen fascicles. TEM, ×15 000.**

In Figure 3 is present a cell of the spiky layer from the inflamed and traumatized gum. We can see the desmosome junction alteration, their rarefaction and the intercellular space expansion.

![Figure 3](image3)

**Figure 3 – Cell of the spiky layer from the inflamed and traumatized gum (a), desmosomes with altered structure (b), dilated intercellular space. TEM, ×15 000.**

Next figure shows a global image of inflamed and traumatized gingival chorion. The collagen and fibrocytes alteration is evident due to the bacterial invasion, as well as lysis zones (Figure 4).

![Figure 4](image4)

**Figure 4 – Inflamed and traumatized gingival chorion (global image) (a), altered collagen (b), bacteria (c), lysis areas (d), altered (non-functional) fibrocytes. TEM, ×15 000.**

A spiky layer of the inflamed and traumatized gingival epithelium is shown in Figure 5. We can see the presence of a Langerhans’ cell among the cells of the spiky layer. The components of the Langerhans’ cell are shown: nucleus, nucleolus, and cytoplasm. At the electronic microscope they appear with a clear cytoplasm with characteristic granulations, without connection with the process of keratinization; they are cells without tonofilaments, without a desmosome system of uniting them to other cells.

![Figure 5](image5)

**Figure 5 – Cell of the spiky layer from the inflamed and traumatized gum. TEM, ×15 000.**

Figure 6 presents another global image of the inflamed and traumatized gingival chorion. The cell population is numerous, there are fibroblasts and fibrocytes with slightly altered structures; as well as the slightly alteration of the collagen. The presence of a high number of mastocytes is evident. Sanguine
capillary are shown. The mastocyte is a non-regular cell with a 5 to 30 µm diameter. The nucleus is small, spherical and centrally disposed in the cell.

**Figure 5** – Spiky layer of the inflamed and traumatized gingival epithelium (detail with Langerhans’ cell) (a), nucleus (b), nucleolus (c), cytoplasm (d), Langerhans’ cell. TEM, ×15,000.

Details regarding the structure of a mastocyte (the granules, the nucleus and the nucleolus) from the inflamed gingival chorion are shown in Figure 7.

In Figure 8, that illustrates an image from the inflamed and traumatized gingival chorion, we can see a proliferation of the peroxisome number. The membrane, the matrix and the nucleoid of a peroxisome are shown. The peroxisomes are small spherical organites, respectively vesicles with 0.5–1 µm diameter, which contain enzymes implicated in the oxidation of certain sub layers, especially the oxidation of very long chains of amino acids containing more than 18 C-atoms.

A detail of a macrophage with altered structure from inflamed and traumatized gingival chorion is presented in Figure 9. Cytoplasm vacuolations, fibrocytes and altered collagen are shown.

The last image (Figure 10) shows a zone from the inflamed and traumatized gingival chorion. It is evident the neutrophils infiltration among the altered structures of the chorion. An altered collagen area is shown.

**Figure 7** – Detail regarding the structure of a mastocyte from the inflamed gingival chorion (a), nucleus (b), nucleolus (c), granulations of mastocytes. TEM, ×15,000.

**Figure 6** – Inflamed and traumatized gingival chorion (global image) (a), fibroblasts (b), fibrocytes (c), slight alteration collagen (d), sanguine capillaries (e), mastocyte. TEM, ×2500.

**Figure 8** – Image from the inflamed and traumatized gingival chorion (detail with microbodies) (a), peroxisomes (b), nucleolus of peroxisome (c), matrix of peroxisome (d), membrane of peroxisome. TEM, ×20,000.

**Figure 9** – Inflamed and traumatized gingival chorion (detail of a macrophage) (a), altered fibrocyte (b), macrophage with altered structure (c), cytoplasm vacuolations (d), altered collagen. TEM, ×6000.
Discussion

In the case of the sampled gingival tissue from a control subject, global sections from the gingival tissue were examined at the electronic transmission microscope. The cells of the gingival epithelium are reunited at the surface through formations named desmosomes. A desmosome is the product of two adjacent cells, each of them participating with the half of the desmosome structure [1, 2, 4]. Desmosomes are intercellular bonding structures, formed of two attaching plates, pierced from one cell to another by intracellular formations/tonofilaments, which spread in the cell cytoplasm in a “brush” manner. The role of the tonofilaments is to synthesize the keratin [5–7].

Fibroblasts: participate at the synthesis of different types of collagen; they produce proteoglycans, glycoprotein and hence they have an important role in maintaining the integrity of the gingival chorion. The collagen fibers of the gingival chorion are the most numerous and appear organized in fascicles [5, 8–10].

In the case of the pathologic tissue, we examined on the microscope sections through the spiky layer of the inflamed and traumatized gingival epithelium, where we can see the pronounced rarefaction of the junction desmosome structures, the effect being the attenuation and loss of the cohesion between the cells. In a section through a cell of the spiky layer in the inflamed and traumatized gingival epithelium are shown alterations and rarefactions of the junction desmosome as well as the dilatation of the intercellular space. In this dilated intercellular space desmosomes are isolated and have a dilated structure. We saw rarefactions of the desmosomes in the passing area between the epithelium and the inflamed and traumatized gingival chorion, especially at the level of the basal layer. In ETM sections through the inflamed and traumatized gingival chorion it was evident the rarefactions of the cell population especially aged, nonfunctional fibrocytes, with a profound altered structure surrounded by collagen fascicles with partially altered structures. At the chorion level, because of the inflammation process there is a reduced number of fibroblasts, which have a low metabolic activity, fact that leads to a reduced collagen synthesis. In the inflamed and traumatized gingival chorion, there are fibroblasts, but in a reduced number, and which have a reduced quantity of cytoplasm. Among the altered collagen fascicles stand out the penetration of the bacteria resulting in cellular lysis areas. Also, the presence of bacteria stands out at the level of the sanguine capillaries [3, 11–13].

A particular element is the presence of some Langerhans’ cells. They come from the medullar alveolar bone and are multiplied in the gum. The functions of the Langerhans’ cells are related to the triggering of a specific immune tissue response. In this respect, they present the antigenic information to the T-lymphocytes that they activate. From this point of view, the Langerhans’ cells act as macrophages in the stimulation of the antimicrobial defense processes [6, 14, 15].

There is a numerous presence of cell population in the chorion, with fibroblasts and fibrocytes with slightly altered structures. The presence of some mastocytes stands out, in a higher number indicating the existence of the inflammatory process as well as the slight alteration of the collagen. Characteristic is the fact that in the cytoplasm there are many granulations of 0.3–0.5 µm diameter, with a rich content of bioactive principles of the chemical mediator type.

The main function of the mastocyte is the synthesis, the secretion and the stocking of the chemical mediators of the inflammatory response. The granulations of the mastocytes contain histamine, heparin, leukotrienes, serotonin and ECF-A or the chemotactic factor of the eosinophils. The mastocytes have also a role in the synthesis of the hyaluronic acid and of the acid mucopolysaccharides, contributing at the formation of the fundamental substance. They have a role in the anaphylactic reactions (type I hypersensitivity reactions) [11, 16, 17].

We can also observe in the inflamed and traumatized chorion a proliferation of the number of peroxisomes. Peroxisomes are different from lysosomes by their content of enzymes, entirely different. Peroxisomes contain at least three oxidases: D-amino acid oxidase, urate oxidase and catalase.

A distinctive element is represented in chorion by the presence of a macrophage with an altered structure, which denotes a reduced metabolic activity. The macrophages come from circulating monocytes. The monocytes circulate in the blood and in a following stage, they migrate in the conjunctive tissue where they become mature and become macrophages (histiocytes). The main functions of the macrophages are ingestion, respectively the particle phagocytosis and their digestion by the lysosomes.

In the structures of the collagen stands out the neutrophil infiltration among the altered structures as well as numerous erythrocytes flown out of their vessels due to the break of the walls of the sanguine capillaries. Neutrophils represent 60–70% from the total of circulating leucocytes. Their number rises in acute infections and reduces in the case of some blood diseases.

The condition of the periodontium is determined by
the immune system of the host and by a series of environmental factors such as: the quantity and the composition of the saliva, the tartar, incorrect prothetic reconstructions and traumatic occlusions [6, 18, 19].

The decrease of the capacity of defense of the body as well as the high virulence of the bacteria has an effect on the periodontium [12].

Our previous researches were meant to also evaluate the specific immune responses generated by incorrect dental reconstructions. In this respect, we measured IgA, IgM, and IgG.

The existence in the electron microscopic field of some vascular lesions, of the leucocytes, macrophages, mastocytes, desmosomes, fibroblasts and of the altered collagen fibers represent microscopic signs of the inflammation and sustain the hypothesis of the local immunoglobulin synthesis. Also, it constitutes a morphological proof of the possibility of an activation of NADPH-oxidase with composition of SRO (time of phagocytosis). Our researches plead in this respect.

Conclusions

Recovering of the contact points the degree of finishing the dental reconstructions as well as the material used for this, have an important role in maintain the health condition of the marginal gingival and especially in the interdental papilla area.

The anatomical particularities of the junction epithelium and the reducing (absence) of the keratinization explain its vulnerability character against the physical and biological aggressions;

During the healing process, we successfully used the chamomile fluid extract solution.

The clinical cases exposed show inflammatory and traumatic lesions in different cases of evolution.

The presence of the bacteria, the neutrophils, the macrophages in the ultramicroscopic sections of the gingival margin at the persons with incorrect dental reconstructions, compared to the control lot, constitutes a morphological proof of the possibility of an activation of the NADPH-oxidase with the formation of SRO (time of phagocytosis).

References


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