The ultra structure of healthy and periodontals diseased gums

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In this study healthy and periodontally diseased gums were ultrastructurally investigated. In periodontally diseased gingivas the frequency of desmosomes and tonofilaments were decreased and the volume of intercellular matrix was increased. Collagen fibril bundles were decreased. Microorganisms were observed in lamina propria causing the periodontal disease. [Turk J Med Res 1995, 13(3): 90-93]

Key Words: Periodontal disease, Electron microscopy

The tooth supporting periodontium is a complex structure that includes gingiva, periodontal membrane, alveolar bone and cement. The periodontitis or the periodontal disease, which is the result of the inflammation of periodontium, is the main reason for the tooth loss above the age of 40. Clinically, the gum diseases are divided into two groups: Gingivitis is the case if the disease is only in the gum, but If the disease covers more than one of the periodontal tissues, then, periodontitis is the case. Microorganisms in the teeth and gingival crevice cause a bacterial plate to form. The reaction rising from this plate progresses through the surrounding tissues and further to the alveolar bone, and together with prédisposai factors, It causes alveolar bone destruction (14,15,18).

Various effects or pathologies of the periodontal diseases on the gum surroundings have been examined by several investigators (3,6,8,9,11-14,16,17,19).

We aimed to examine the ultrastructure of gums of patients with periodontitis.

MATERIALS AND METHODS

Ten healthy and 10 periodontally diseased gum samples were taken from free and bound gums of the patients. The healthy gum materials were taken with a bistoury-as to be at a thickness of 1 mm-from the gingival crevices of the teeth whose depth as lower than 2 mm and which had to be extracted for orthodontical purposes. The diseased gums were also taken with a bistoury-as to be 1 mm-from the gingival crevices of the teeth which clinically showed periodontitis and which had to be extracted. The samples were determined with glutaraldehyde and osmium. After alcohol dehydration, inclusion was made with araldit CY 212. The cross sections were colored with lead citrate and uranll acetate and were then examined with a JEOL 100 SX electron microscope.

RESULTS

In the periodontal disease in our previous study, the epithelium and connective tissues, being observed at a light-microscopic level, were in the same way observed through small enlargements or semi-thick cross sections by the electron microscopic blocks, too (Figure 1). At ultrastructural level, the epithel cells or keratinocytes were at known structure in the healthy controle blocks. The cells were in general round, with large nucleus and less chromatin. The tonofilament bundle packages were observed almost everywhere in the cytoplasm and at the level of desmosomes. But the intercellular matrix between the keratinocytes in many cross sections was observed to be very narrow which was not expected and by making interdigitations, the cells mostly were attaching to each other with the desmosomes (Figure 2).

Besides the inflammatory cell infiltration in the epithelium in periodontally diseased gums, the pathol-

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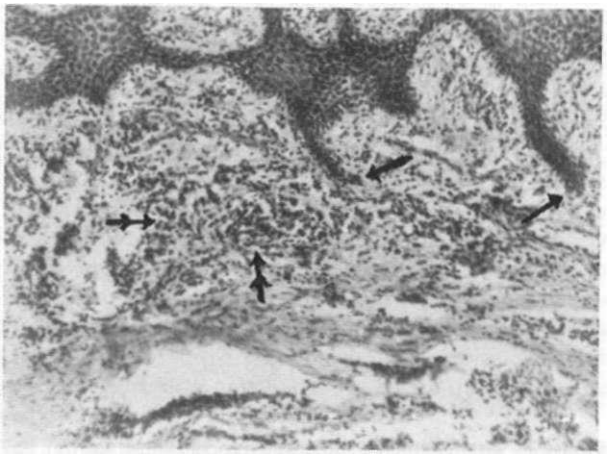

Figure 1. Periodontally diseased gum. Changed retepeg structures (single arrows) and the inflammatory cell infiltration in the connective tissue (double arrows) are to be seen. Hem.-eo, X100

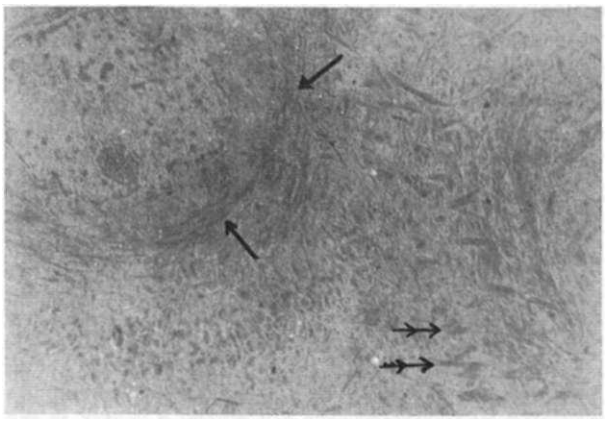

Figure 2. Healthy gum epithelial cell. The tonofilament bundles in the cytoplasm (single arrows) and desmosomes in the attachment regions of the cells (double arrows) are to be seen. X3000

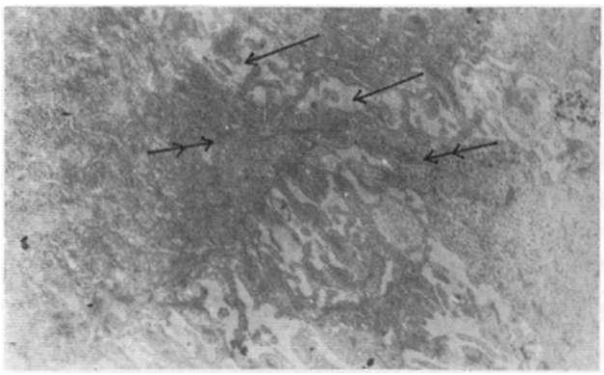

Figure 3. Periodontally diseased gum. The extended intercellular matrixes between the epithelial cells (single arrows) and the electron micrograph showing the decreasing tonofilament bundles (double arrows). X8000

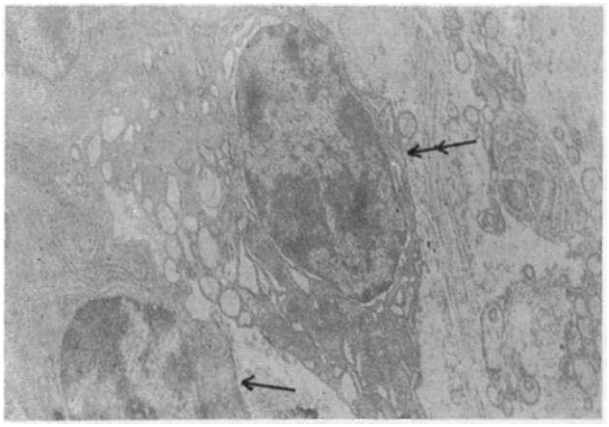

Figure 4. Cells of the immune system in periodontally diseased gums. Plasmocyte (single arrows) and macrophage (double arrows) are seen.X5000

ogy in epithelial cells was that the intercellular matrixes were extremely wide in almost every parts of the epithel.

It was often hard to determine the desmosomes in the mutual attachment regions of the cells. In the cytoplasma of the keratinocytes, the tonofilament bundles were very rare, too, or there were no bundles in some regions. Including desmosomes, the tonofilament bundles were less denser or uncertain with respect to healthy gums (Figure 3).

In the connective tissue under the epithelium, cells of the immun system were In majority. These cells were frequently forming epitheloid clusters side to side. Most of the cells in such regions were plasmocytes which could be easily fixed from the granular endoplasmic reticulum by their rich cytoplasmas and eccentric nuclei; or they were oval shortly extensional macrophages with large vacuole and heterogeneous granule; or they were polymorphonucleated, granular granulosites and heterochromatic nucleate lymphosites with less cytoplasm (Figure 4,5). Within this connective tissue, fibrocytes in their known structure and nerve fibers were frequently observed. But the collagen fibril bundles, which are expected to be observed especially near the nerve fibers or the fibrocytes, were quite rare. At most times, rare or dispersed short fibers were being observed (Figure 6).

In the periodontally diseased gum lamina propria, the most unusual thing was the observation of clusters which were sometimes huge, oval and covered by a thin granular membrane or by a shorter, again denser but a certain shell; or the clusters consisted of small, oval, dense and breadthwise cut structures and struc-

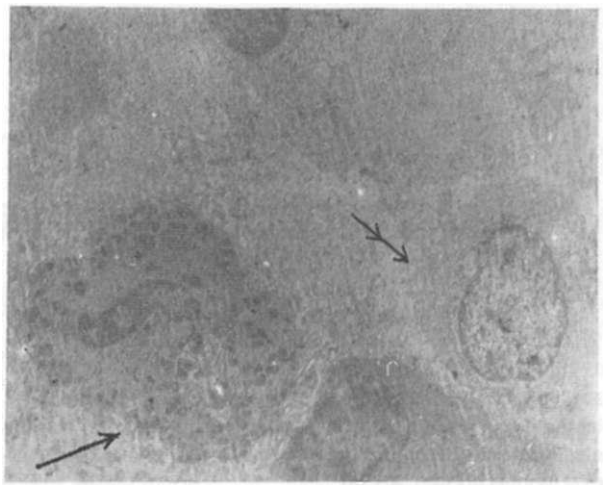

Figure 5. Defensive cells in the connective tissue of a diseased gum. The neutrophyl granulocyte (single arrows) and lymphocyte (double arrows) are to be seen. X3000

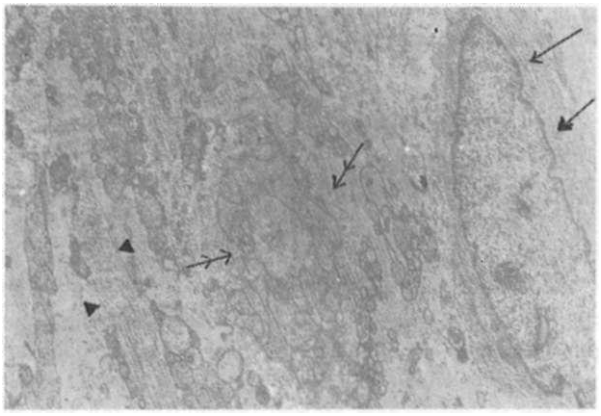

Figure 6. The connective tissue of a diseased gum. Fibrosite (single arrows), nerve fibres (double arrows) and the decreasing collagen fibres (arrowhead) are to be seen. X4000

tures of different diameters, again oval, with various densities of content. The diameter of these colonies was sometimes at the size to fill one mesh of the grid (Figure 7). The grid is a small cupper cage with thin cage, there are partitions whose number differ from 50 to 100. Each of these partitions are called "mesh".

DISCUSSION

In all kinds of gum diseases, inflammation is a general property except for pathological processes like atrophy, hyperplasy and neoplasy (14-16,19).

In our study, cells were observed which determine the inflammatory reaction both in the epithelium and in the connective tissue. These cells in the

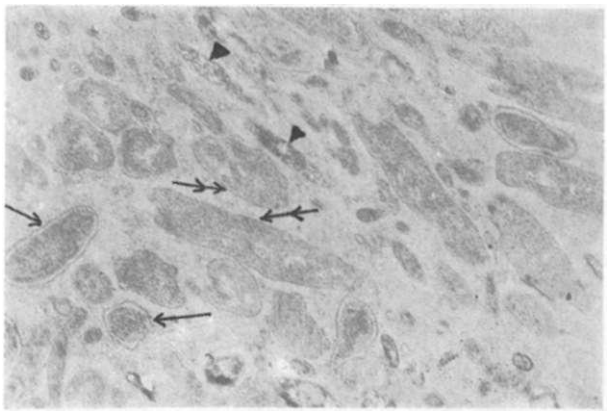

Figure 7. Gram negative and positive cocci (single arrows), bacillus (double arrows) and spiral organisms (arrowhead) are seen. X8000

epithelium had been infiltrated to the widened intercellular matrixes. The intercellular matrixes in the epithelium parts where these cells are not present were extremely broad with respect to the normal. The desmosomes were also few. The tonofilaments in the cytoplasm of the keratinocytes which take part in the attachment structure or which are observed in the cytoplasm were very rare with respect to healthy keratinocytes. The decrease in tonofilament and desmosomes must cause the attachment of the cells sometimes to be weak. In addition, a change or increase in the structure of the intercellular area in the inflammation region is a known event (7,10,11,13). In our opinion, the reason for the extension of the intercellular area between the keratinocytes in gum inflammation is the changing intercellular area besides the infiltrated inflammatory cells but mostly it is due to the connection structure of the keratinocytes which get weaker.

The main participant cells in the body defense are the connective tissue cells. For that reason, it is quite normal to see these cells in the connective tissue below the epithelium. We frequently have observed in our preparations the plasmocytes, lymphocytes, granulocytes and macrophages of the connective tissue in diseased gums. These cells, whose morphologies showed no certain structural change, have been determined by some investigators to be short-living (12,17). Like these inflammatory cells, the fibrocytes were plenty and in the expected structure, too. What was observable from the morphology in the connective tissue of the lamina propria was that, apart from the density of the pathologic cells, the intercellular area was also precise in diseased gums. Thus, the collagen fibres in the intercellular area were different than the contrôle group in terms of density and order. Through our preparations, we observed the collagen fibres to be short and their bundles to be thin

and few. Michelet et al have determined in their experimental gingivitis studies a collagen fibre loss together with an increase in the cell number and an increase in the extracellular matrix elements (11). The collagen fibre decrease may be result of the structure of the intercellular area (1,7,10,14,18).

The unusual structures to be observed in the lamina propria were the oval clusters with a dense content in the form of huge colonies. These extremely huge colonies may consist of microorganisms which are effective on the appearance of periodontal diseases. Just as, Newman, Thurre and their friends have determined gram negative and positive anaerobic cocci and bacillus together with spiral organisms in the gingival crevice (12,17).

The bacterial plate is believed to be the cause of the periodontal disease (5,6,9,17). The idea that gram negative bacteria and the endotoxins which emerge with the death of the spirochets directly cause a tissue necrosis has been very strong. The "hyaluronidase" and "chondroitinase" enzymes which rise from microorganisms cause the tissue permeability to increase by breaking the collagen fibres and the structure of the intermediary substance (5,9,13-15,17).

The decrease of the collagen fibers in diseased gums may result from the expected increase of the substantia fundamentalis or from the direct tissue necrosis of the endotoxins.

The periodontal disease has many agents like tartar, trauma, sistemic diseases, bacteria or mashroom infiltration. In each inflammation, there is a structural change especially in the connective tissue or the epithelium, and in the cellular and intercellular area. Lysis and necrosis may develop in changing tissues. Caused by bacterial infiltrations, reactions, which begin from the mechanical effect of the microorganisms on the tissue and process till the lysis and even the necrosis, may develop in addition (2,5,6,9,11-17). The tissue destruction in periodontitis cases with a bacterium plate formation must happen much earlier and in big sizes. For that reason, the removing of the plate of bacteria in such patients must be the primary aim of the therapy (11).

Sağlıklı ve periodontal hastalıklı dişetlerinin ultrastrüktürü

Çalışmamızda sağlıklı ve periodontal hastalıklı dişetleri ultrastrüktürel olarak kıyaslı incelendi. Periodontal hastalıklı dişetlerinde epitel hücrelerinin birbirine tutunma yapıları olan desmozomlar ve sitoplazmalarındakitonofilamanlar azaldı ve interselüler aralık genişledi. Kollagen lif demetleri seyrekleşti. Lamina propriada alışılmışın dışında periodontal hastalığın oluşumunda etkili olan mikroorganizmalar saptandı.
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