

# HISTOPATHOLOGICAL RESEARCH IN CHILDREN'S PERIODONTITIS WITH MALOCCLUSIONS

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## ABSTRACT

Marginal periodontal diseases do not bypass childhood and concerns pediatricians and orthodontic dentists due to the frequency of periodontitis in children and young people. Changes accompanying the presence of the permanent teeth are temporary and reflect different from gingival mucosa.

The sequence of permanent teeth and temporary, with their distinctive features, prints on the periodontium and on its regions, during childhood and adolescence, anatomical and structural features. In order to conduct a study on pathologically marginal periodontitis, we have taken pieces of the lining of the marginal gingiva, teeth and adhesions from patients with periodontitis orthodontics.

Dominant changes in gingival mucosal chronic marginal periodontitis are represented by lesions that can be determined by a tissular immunological reaction of tissue with delayed hypersensibilization. Besides the mechanisms of hypersensitivity reactions may occur and bacterial factors aggravating injuries

Keywords: periodontal, marginal periodontitis, Pediatric Dentistry, inflammation, histology

## INTRODUCTION

Marginal periodontal diseases do not bypass any childhood, while the pediatric dentistry and orthodontics concern for this area is fully justified, especially as, since 1938, Mc CALL stressed that "foundation of all adult periodontal diseases are in childhood" (quoted by BALLESTA, 1983), the prognosis is unfavorable periodontitis (Schulte, 1966).

Chronic marginal periodontitis, which we recognize today in various forms, in terms of basic lesions, early or advanced stage, appear in an increasing number of patients, regardless of age and geographical location of human communities investigated.

Children's and adolescents Periodontium is changing almost permanent, temporary and end between the exfoliation of temporary teeth at the end of the eruption of permanent . The sequence of permanent teeth and temporary, with their distinctive features, prints on the the periodontium and on its regions, during childhood and adolescence, anatomical and structural features.

Changes accompanying the presence of the permanent teeth are temporary ones reflect different from gingival mucosa in the three areas of his free marginal gingiva with gingival sulcus, attached gingiva or fixed one and the interdental gingiva or papilla.

Marginal gingivitis is characterized by the following clinical signs. Inflammatory reaction of gingival mucosa in children presents four classic signs

of inflammation namely tumor (swelling, swelling), rubor (vasodilation as the first stage of inflammatory reaction), calor (high local temperature due to an increased metabolism in the area) and dolor (discomfort, pain zonal) in marginal gingivitis in children is found in inflamed gingival mucosal bleeding plus the smallest touch, because ulceration of sulcular epithelium in the bottom of the bag and the tendency to hyperplasia of the epithelium lining around ulcers.

Frequency of marginal gingivitis in children is attributed to the following causal factors: poor dental buco- hygiene, the child up to 5 years that do not practice it properly, tooth eruption by intrusion mucosal inflammation promotes the oral phase, food rich in sugar soft, lingering teeth, wrong dental position, permanent tooth anomalies of structure during growth, labial and lingual phrenic are hypertrophied, job retention may be food, mouth breathing frequency, the presence of thrush, biting mouth, children still have no automaticity format mastic direct trauma or indirect in the game, proximal caries and its complications, faulty restorations, fillings overflowing, inadequate braces correctly.

Simply wearing an orthodontic appliance may result however well adapted to increased bacterial virulence gram-positive groups, especially gram-negative, leading to serious forms of marginal gingivitis. It is possible that pressure-traction forces exerted by braces on the teeth to be the main cause of transformation in a saprophytic microbial pathogenicity, as evidenced experimentally in rats (Firu, 1967)

Given the statistical literature that the incidence of periodontitis is about 1% at age 10 years and 10% at age 20 years, we conducted this study in order to highlight the histopathological features of aspects of the histological lesion different layers of the marginal periodontium in children.

A special attention is given by specialist doctors on the frequency of periodontitis in children and young people, and the study of histopathological changes from periodontal tissue can provide some new information in understanding and perhaps treatment of periodontal disease.

## MATERIALS AND METHODS

In order to conduct a study on pathologically marginal periodontitis, we have taken pieces of the lining of the marginal gingiva, teeth and adhesions from patients with periodontal orthodontic diseases.

The fragments were collected from 10 subjects between 14 and 16 years who had chronic marginal periodontitis and from two healthy subjects with morpho-functional gum integrity - the control group.

Biopsy material was prepared using the usual histopathological methods: fixing solutions of formalin 10%, inclusion in paraffin and cutting the blocks obtained at 5 microns.

For the most comprehensive evidence as histological and histochemical changes occurring gums affected by a process we call marginal periodontitis three specific stains: stain with haematoxylin and eosin, Van Gieson method and the PAS method. You specify the substrates that are highlighted by these stains and major stroke were driven to achieve them.

Staining with haematoxylin and eosin is the most important method of study used in pathology. It allowed us to highlight architectural and cellular structures at the level of free gum and at the level of the lesion. Are needed, in essence, the following steps: the first stage dewaxing, hydration, staining with hematoxylin second Harris, the third washing and rinsing, staining with eosin is the fourth and final stage of treatment with alcohol, dehydration, clarification, installation.

Van Gieson method: We used the technique of color to highlight existing collagen in gingival chorion. These fibers appeared colored in red, allowing us to study optimal process sclerosis. The most important times to go to achieve this technique are: hematoxylin staining, differentiation and steering, picrofuxinã staining, washing, dehydration and mounting.

PAS method (periodic acid Schiff) I used it to highlight the neutral mucopolysaccharides and glycogen, both in the gingival epithelium and chorion. These substrates, known as PAS positive were stained by this technique in red. The steps were as follows: dewaxing, hydration, oxidation with periodic acid, Schiff's reagent treatment (performed), sulfur water washing, staining with hematoxylin background.

## RESULTS

Reported histological aspects as topography, may be systematic changes in gingival epithelium and chorion.

Squamous epithelium at the gingival lesion I found in most cases, after staining with haematoxylin and eosin, the existence of a process of hyperplasia (Figure 1).

It is characterized by an uneven thickening of the epithelium, with long spurs that reach deep Chorionic interpapilla. Some papillae penetrate superficially constitutes the debut of a process of atrophy.

Parakeratosa was identified by the presence in the upper layers of a process of keratinization with persistence in it of nuclear waste in the stratum spinosum cells.

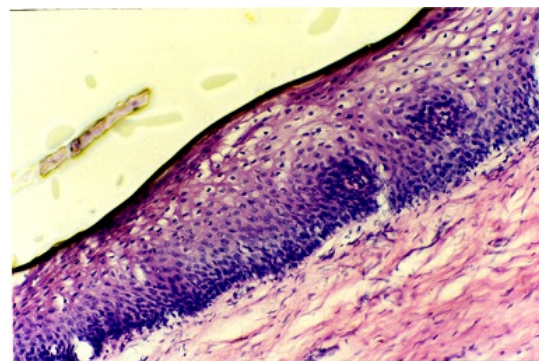


Figure 1

In cases characterized by a long evolution of the inflammatory process we found the presence of epithelial atrophy process (Figure 2)

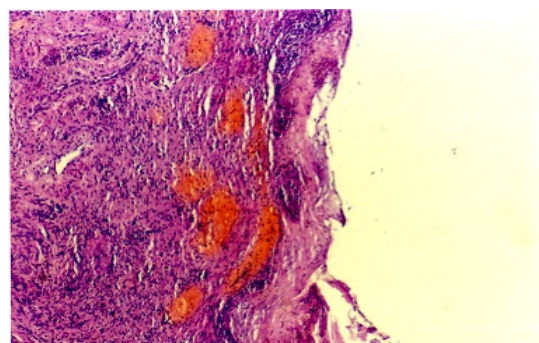


Figure 2

This layer is characterized by reduced, sometimes to extinction, appearing chorionic covered only two to four rows of flattened cells, in deepness they are penetrated by long epithelial spurs with strong edema, with increased cell dystrophies, this predominantly in clinical forms with obvious symptoms. Epithelium suffers constantly of a degree of edema, characterized by enlargement of intercellular spaces, especially in deeper layers, the represented cell spines appear more elongated junctions, taking a look of stellate cells, or compression (Figure 3).

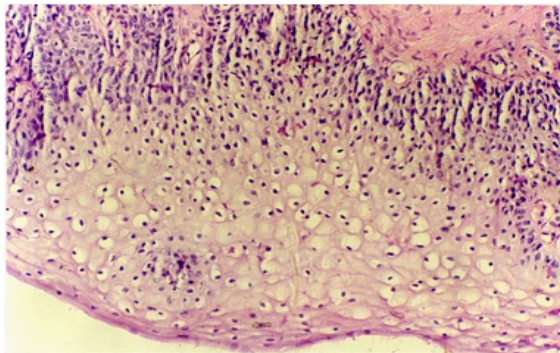


Figure 3

Malpighiene cells showed varying degrees and expressed distress picnoze cell nuclear intumescent granular or vacuolar degeneration. Almost all the cases studied were characterized by the presence of epithelial ulceration areas, single or multiple, of variable întidere.

These were microscopically as areas of epithelial disruption of continuity, with the denudation of the the chorion surface .

With PAS method we studied the expression of glycogen in the epithelium. To issues encountered in unaffected epithelium, where glycogen is absent or present in very small quantities in inflamed gum Shallow cell layers is evident a clear trend of storing this substance. The large amounts of glycogen were observed in cells adjacent to areas of ulceration occurred apparently intact epithelium or areas that tend to parakeratosis is moderate.

Emergence of large quantities of glycogen in the superficial epithelium appears to be subject to alterations of epithelium with minimal maintenance or moderate parakeratosis trend on which to graft an inflammatory reaction of acute myelogenous. In observed areas we see the persistence of parakeratotic glycogen in the superficial layers of epithelium, but in small quantities; it's presence is not constant throughout the superficial layers.

Chorion aspects are more characteristic than the epithelial and may be linked to them and in some aspects appearing as determinants of epithelial changes. The main fact to note, impressive by constant

inflammatory infiltrate is the presence of lymphoplasmocitar. Also consistently found a very pronounced edema and, in varying degrees, a process of diffuse fibrosis. As a special observation point out a particular behavior of the superficial chorion (chorionic papillary) than deeper one.

Chorionic surface of the gingiva with periodontitis has two characteristic features: the presence of massive edema and the absence or presence of a small amount of the inflammatory infiltrate. Fibrillar collagen elements (Van Gieson staining) appear fragmented and dissociated and fundamental substance is acromă (Figure 4).

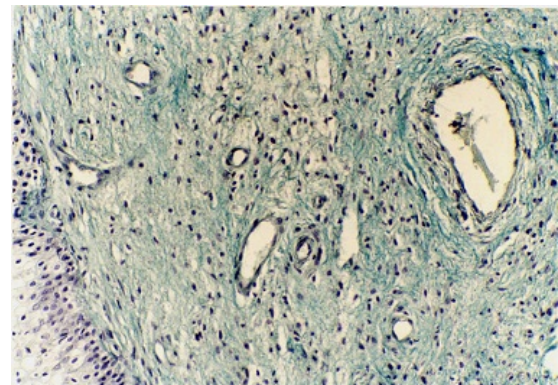


Figure 4

Besides fragmentation structures that can distinguish particles of collagen fibers in the intercellular substance granules or blocks still appear eosinophilic PAS-positive material, which could be a destruction of material from fibers (Figure 5).

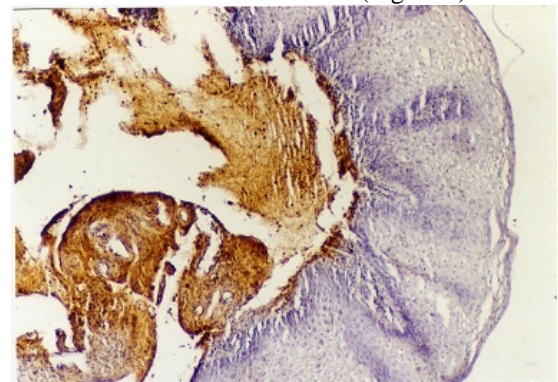


Figure 5

They may also be viewed fibrocin with a more hematocatic nucleus, and cytoplasm and PAS-May acidophilous increased positivity. Capillaries are apparently unchanged or structural alterations minimal, rarely congested. Thick walls seem to have changed, no PAS reaction shows no significant thickening of basal membranes. Where we have identified the presence of inflammatory infiltrate in the chorionic surface, it was mainly constituted less plamocite and lymphocytes (Figure 6).



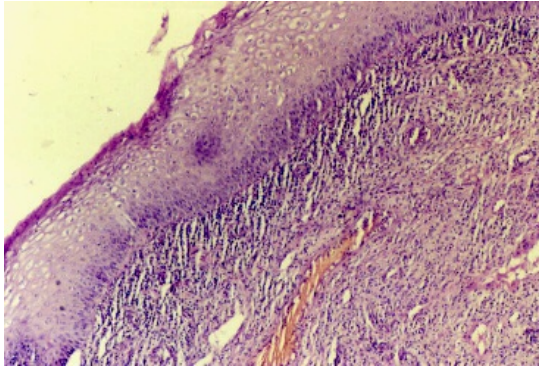


Figure 6

The first of these are arranged in cords, one or two rows parallel to the basal epithelial membrane. Granulocytes were inconsistent highlighted more clearly the neutrophils than eosinophils. Histiomacrophag's tissue are also poorly represented. Note that inflammatory cells are, however, consistently present in areas characterized by epithelial ulceration. Here we see the granulocyte elements. We noted the existence of areas with significant degradation of interstitial elements, connective cells, necrotic debris surrounded by fine. Chorionic deeply is, in turn, grafted two lesion types: the presence of a marked infiltration of the inflammatory process and diffuse fibrosis.

The inflammatory infiltrate is much better represented than in the chorionic surface is represented relatively equal proportions of plasma cells and lymphocytes. Granulocytes, neutrophils attach and acidophilous and macrophages are very few. We noted a tendency for aggregation of these elements predominantly perivascular inflammatory. Interstitial fibrosis of the deep chorion is present, with variable intensity in all cases investigated pathological. Van Gieson staining is evident in the disappearance of cellular elements characterized by the reduction fundamental substance and proliferation of fibrillar elements.

Blood vessels, unlike the chorionic surface, showed some changes. However, and here, vascular elements located outside the areas of inflammatory infiltration showed no detectable microscopic changes. Vessels included in the infiltration areas showed inflammatory changes with endothelial cells. Capillaries from areas of fibrosis appeared to be dilated with stasis phenomena, thin walled, with a tendency to break and sometimes with interstitial hemorrhage (Figure 7).

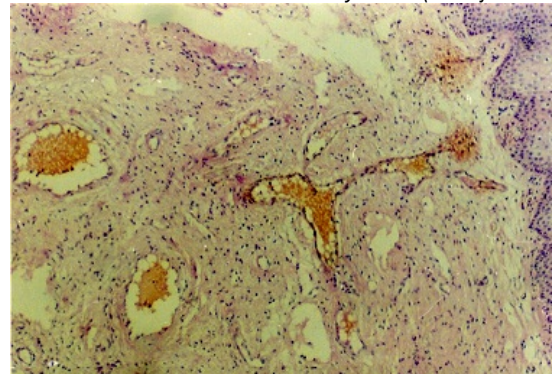


Figure 7

## DISCUSSION AND CONCLUSIONS

Gelfand et al. (1978) reported that the gingival mucosa intact and its changes during tooth eruption, exert a connective tissue induce epithelial specificity, influencing the expression of keratinization capacity. Our observations highlight this interrelation: the presence of an inflammatory reaction evolves to paracheratosis of the chorion's epithelium.

The association of paracheratosis, edema and dystrophic alterations are present in a relationship with a chorionic lympho-plasmocitary defensive reaction, the reaction occurs between cells and granulocytes, epithelia exhibit more pronounced tendency to necrosis and ulcers to develop the inflammatory infiltrate predominance of lympho-epithelial plasmocitary lesions shows that iparacheratosis is initially modified with hypertrophy and has the potential for the development of atrophy.

Can be considered epithelial alterations in the initial phase may be caused by emissions from the surface acting by diffusion, while the frequency of ulceration and necrosis identified the trend towards advanced to express the failure of the barrier epithelial altered microbial germs. Changing the nature of epithelial lesions and behavior is demonstrated in epithelial glycogen content, as shown above. Similar observations were made by Baba and Severineanu, who found a decreased glycogen content in the initial stages, its reappearance in the advanced.

The presence of plaque can influence the trophic epithelium hazards resulting from the metabolism of microorganisms (enzymes, endotoxins). Thus, bacterial hyaluronidase depolymerization causes intercellular substance intercellular space widening with imbibiția retention and edema. In the deep chorion, two structural alterations observed inflammatory infiltration and sclerosis, have an unparalleled development; sclerosis appears to be secondary, as a result of the inflammatory process by repairing mechanisms potentially retractable increased fibrosis.

Undoubtedly the most significant histopathological changes in the chorion, at the level of the marginal periodontitis, is the presence of inflammatory infiltrate. It has several features that indicate a specific way to attack gum inflammatory response: is more pronounced and more consistently in the early stages of the disease. In these phases infiltrate is diffuse without nodular aggregation tendency. When there is a tendency to separation barrier lymphocytic and fibrosis, it is to the deep, never towards the epithelium. In advanced forms of the disease may appear as nodular infiltrate, but no organization nodules characteristic of chronic inflammatory processes of separation. Immunocompetent elements of the infiltrated plasma of predominate cells are numerically lower lymphocyte. There is no obvious reaction histiocyte-macrophage.

All these features of immunological reaction advocates an infiltrate tissue, a local phenomenon of delayed hypersensitivity. Significant assumptions seem to support this: non-participation of macrophages in response to cellular components, suggesting macrophage inhibition, a phenomenon characteristic of this type of immunological reaction and vascular changes, which by their nature vasoplegic, producing destruction with bleeding walls remember vascular changes or reactions that occur in Arthus phenomenon.

Some particular aspects make us say that in addition to immunological phenomena reported in chronic marginal periodontitis work and some self-healing or aggravating factors. Among them we mention the possibility that in addition to diffuse in gingival tissue antigen and other harmful microbial products such as collagenase, which could be largely responsible for degradation of mesenchymal structures. It's possible involvement of hyaluronidase released by mast cells concentrated in the outbreak response

## CONCLUSIONS

Changes in gingival mucosal with chronic marginal periodontitis lesions are represented by lesions that can be determined by an immunological reaction of tissue with delayed hypersensibilization. In the lining of these changes it is a hypersensitivity to the factors reflected in the depolymerisation of the intercellular substance and edema, accompanied by dystrophic and dysplastic phenomena. Functional value is affected by the epithelium of parakeratosis' lesions, atrophy and ulceration Chorion papillary changes may be caused by penetration through damaged epithelium, the bacteria in dental plaque factors as hyaluronidase and collagenase.

In chorionic deeply coexist two types of changes: immunocompetent cell diffuse inflammatory reaction associated with the development of a process of sclerosis. Immunological tissue reaction character is

shown by the predominance of plasma cells in the inflammatory infiltrate, the antibody's precursor cells. Local hypersensitivity can occur through the appropriate antigens plaque germs. Besides the mechanisms of hypersensitivity reactions may occur and bacterial factors Trace character aggravating injuries (proteolytic enzymes, hyaluronidase).

I found particular aspects in patients with malocclusions.

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