Microscopic aspects of pulpal changes in patients with chronic marginal periodontitis

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Abstract
Chronic periodontitis is one of the most frequent and severe diseases involving the tooth. Untreated, they can lead to tooth loss. Our study involved 67 patients with chronic marginal periodontitis who underwent tooth extraction, of which 29 had moderate periodontal lesions and 38 severe periodontal lesions. The microscopic study of the dental pulp revealed significant changes in all patients. In patients with moderate periodontitis the pulp tissue was found to be the site of an enhanced process of collagenous fibrosis associated with a moderate inflammatory infiltrate, dystrophic mineralization, reduced blood vascularization and arteriolosclerosis. The dental pulp of patients with severe periodontitis showed an abundant chronic inflammatory infiltrate associated with pulpal necrosis, vascular congestion, microhemorrhages, dentin demineralization and odontoblast impairment.

Keywords: periodontitis, dental pulp, dystrophic mineralizations, fibrosis, inflammatory infiltrate.

Introduction
Chronic periodontitis are chronic bacterial infections that affect the marginal periodontium, resulting in an inflammatory process that progressively alters the supporting and anchoring mechanisms of the teeth within the alveolar socket, with irreversible alteration of their physiological substrate. If left untreated, the disease gradually develops and is currently considered the main cause for tooth loss [1].

Pulp damage in periodontal disease has been less studied. In recent years, several studies have emerged showing that untreated periodontal disease can affect dental pulp resulting in endodontic lesions. Therefore, the American Academy of Periodontology included in the classification of periodontal diseases a group of combined endodontic-periodontal lesions when occurring simultaneously in the same tooth, endodontic and periodontal lesions whose evolution and response to treatment are correlated with each other [2]. Simultaneous occurrence of both lesions suggests that there may be an interrelation between the pulp tissue and periodontium [3]. According to some authors [4], pulp lesions occur only when periodontitis affects the tooth apex. Other authors [5] believe that pulp lesions may also occur in cases where the inflammatory periodontal process does not affect the apical periodontium.

In this study, we aimed to analyze some histological changes occurring in the pulp tissue from patients with chronic periodontitis.

Materials and Methods
The study was performed on human tissue, that is odontium and periodontium from a total of 67 patients aged between 46 and 75 years, who presented to the dentist for pain, fetid halitosis, tooth mobility, chewing difficulty or esthetic problems. Of the 67 patients, 42 were male and 25 female.

Clinical examination revealed the presence of gingival bleeding, gingival edema, periodontal pockets, tooth mobility, gingival retraction and even tooth migration. Among the contributory factors of periodontitis, we recorded smoking (39 patients, 58%), diabetes (12 patients, 18%), poor oral hygiene (47 patients, 71%), recurrent gingivitis (33 patients, 49%). After evaluation of periodontal lesions in 29 patients, we found moderate periodontal lesions and severe periodontal lesions in 38 patients. All patients required dental extractions, followed by alveolar curettage.

For the microscopic study, the biological material collected, represented by teeth and periodontal fragments, immediately underwent fixation in 10% neutral formalin solution.

Periodontal tissues were fixed for 24 hours, while teeth were fixed for seven days. Then, the teeth were subjected to decalcification in 10% trichloroacetic acid solution for 30 days.

In order to speed up the decalcification process, tooth fragments within the decalcification solution were
placed each day for seven hours on a mechanical shaker. Following decalcification, the samples were washed in slow running tap water for 24 hours.

Longitudinal and transverse 2 mm thick sections from the decalcified teeth were obtained using a very sharp scalpel, which were then included in paraffin using the conventional histological technique.

Five-μm thick serial sections were cut using a rotary microtome (Microm HM350) equipped with a waterfall based section transfer system (STS, Microm). Sections were stained with Hematoxylin–Eosin or Goldner–Szeckeli’s trichrome.

Results

Our histological study on pulp changes in patients with chronic periodontitis allowed us to observe a variety of histological aspects that may correlate with the severity of periodontal lesions. Thus, in patients with moderate periodontal lesions, the dental pulp showed fibrotic changes, more or less extensive, associated with the presence of cells belonging to the immune system (Figure 1).

The presence of large amounts of collagen fibers in the dental pulp is due to the stimulation of local fibroblast activity. It is known that fibroblasts arise from mesenchymal cells have the ability to multiply rapidly when stimulated and to synthesize and secrete fibrous proteins of the extracellular matrix such as collagen, elastin and reticulin macromolecules, as well as glyco-protein complexes such as glycosaminoglycans, proteoglycans and structural glycoproteins that are part of the fundamental substance of connective tissue, regardless of their type.

Changes of dental pulp tissue were accompanied by vascular changes. Thus, in patients with moderate periodontal disease was the presence of mineral salt deposits, mainly calcium, as well-defined nodular structures (Figures 2 and 3). Of the 67 patients studied, we identified pulp calcification in only five cases. The shape and sizes of these “dystrophic mineralizations” varied from one patient to another. Some limestone deposits were seen in the periphery of the pulp, others were centrally placed.

The histological structure of these parties’ structures was represented by calcium salts arranged on non-fibrillary structures, probably non-collagenous proteins and proteoglycans, as these dystrophic mineralizations were friable during processing, and collagen fibers within the pulp were diverted or separated by such histopathological elements, aspect that fundamentally differentiates them from denticles.

Changes of dental pulp tissue were accompanied by vascular changes. Thus, in patients with moderate periodontitis we encountered a heavy vascular remodeling, with fewer blood vessels and arteriolosclerotic changes (Figure 4).

Patients with severe periodontitis, in whom periodontal impairment involved the apex, showed inflammatory changes within the pulp. In all 38 patients with severe periodontitis, we identified the presence of numerous mononuclear round lymphocytic-, plasma cell- and macrophage-type cells, and fewer granulocytic-type cells. Pulp necrosis was also noted, in which large spaces were occupied by cellular and fibrillar debris because of the inflammatory process (Figure 5).

Blood vessels showed congestive changes or discontinuities of the vascular wall, allowing extravasation of red blood cells, sometimes taking the appearance of microhemorrhages.

The chronic inflammatory process present in the pulp also resulted in alterations of odontoblasts and circumpulpar dentin. Thus, we identified areas where odontoblasts were absent due to cellular necrosis, and dentin showed a festooned appearance with multiple small areas of patchy demineralization (Figure 6).
Periodontitis is one of the most frequent and severe dental diseases. Studies in India show that 50% of individuals over 35 years show periodontal lesions and that 30% of extracted teeth after the age of 35 years are due to periodontal disease [6]. However, until now there are only few histological studies that evaluated the influence of periodontitis on pulp tissue.

In our study, patients with moderate periodontitis showed a sharp increase in the amount of collagen fibers in the dental pulp, which could be an expression of periodontal stimuli, because immune cells develop and release a series of biochemical mediators within the inflammation site which could stimulate the proliferation of pulp fibroblasts.

An increased amount of fibroblasts and collagen fibers in pulp tissue from patients with periodontitis was also observed by other authors [7], regardless of the severity of periodontal lesions.

Another possible cause for collagen fiber densification could be represented by vascular changes within the pulp, since the reduction of blood supply within a tissue creates local hypoxia and is followed by proliferation of fibroblasts, with increased synthesis of collagen fibers. In our study, the number of blood vessels in the dental pulp and especially arterioles was smaller, showing signs of arteriolosclerosis.

According to some authors [8], smokers with periodontitis have a lower vascular density in the superficial periodontium. We believe that periodontitis in smokers is associated with a reduced blood supply not only in the periodontium, but also within the pulp.

Several studies have shown that periodontitis is associated with vascular endothelial dysfunction, as well as systemic atherosclerosis [9, 10]. In addition to possible vascular endothelial dysfunction, which is at least partly responsible for changes in pulp tissue, one must take into account the changes induced by age, because advanced age alters the vasculature of all tissues, including pulp tissue.

In severe periodontitis we identified the presence of a chronic inflammation within the pulp, more or less intense, as well as pulp tissue necrosis, indicating the
propagation of the periodontal inflammatory process towards the pulp tissues. Several studies have shown that communication between the pulp and periodontium may occur not only via the apical foramen, but also via accessory canals [11, 12].

Other authors [13] have shown that in periodontal disease communications between the periodontium and dental pulp via dental canaliculi may develop. The same authors [13], using microbiology techniques, showed that in 83% of patients with periodontal disease fragments of root dentin contained bacteria, with dentin becoming a bacterial reservoir for re-colonizing periodontal tissues in case of incomplete treatment. In addition, they showed that in 59% of patients pulp tissue contained the same bacteria as the periodontium. This study demonstrated that periodontal infection could also spread via the dental canaliculi and not only through the apical foramen.

Using the same microbiology techniques (incubation of dentine fragments on anaerobic cultures), Giuliana G et al. [14] found that in patients with periodontitis the middle dentin was invaded in 53.8% of cases by pathogens present in the periodontium, such as Prevotella intermedia, Porphyromonas gingivalis, Fusobacterium nucleatum, Bacteroides forsythus, Peptostreptococcus micros and Streptococcus intermedius. Other authors [15–17] have also shown that the microflora within periodontal and pulpal lesions is generally similar.

Pulp tissue necrosis, blood vessel, odontoblast and dentine impairment observed by us may be due to pathogens as well as immune cell reaction that synthesise and release a variety of biochemical agents within the connective tissue, especially metalloproteinases capable of pulp tissue destruction [18].

Pulpal changes in patients with chronic periodontitis should be correlated with other systemic diseases such as diabetes mellitus [19], atherosclerosis [20], hypertension [21], immune dysfunction, etc. [22], which may contribute to alterations of pulp tissue.

For the dentist it is particularly important to determine whether or not periodontal lesions are associated with pulp damage, as well as the degree of impairment in order to apply the correct treatment. Sometimes it is clinically difficult to determine whether periodontal lesions are accompanied by endodontic lesions, which is why some authors recommend further investigations such as radiological examination of affected teeth, pulp vitality tests, microbiological analysis, measurement of periodontal pockets, evaluation of superficial periodontium and laboratory investigations [11, 23].

Like other authors, we also believe that currently there is little scientific evidence to clarify various aspects of the relationship between periodontal disease and endodontic changes [24].

☐ Conclusions

Chronic marginal periodontitis can cause multiple histological changes within pulp tissue, and pulp lesion severity may correlate with the severity of periodontitis.

References

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Received: December 15th, 2011

Accepted: October 20th, 2012