INFLUENCE OF PERIODONTAL THERAPY ON THE PATHOLOGY OF DENTAL PULP IN THE ENDO-PERIODONTAL SYNDROME

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Abstract

Aim: The study aims at evaluating the influence of deep periodontal pathology and of some etiological therapies (scaling and root planning) on radicular pulp, through clinical and paraclinical modifications. Materials and method: Clinical and paraclinical evaluations were performed on teeth with periodontal pathology subjected to various mechanical treatments, such as scaling, root planning and local drug treatment. Results and discussion: Scaling and planning are indispensable manoeuvres in the treatment of the periodontal disease, even if some intempestive or too frequently repeated techniques also remove part of the radicular cementum and of dentin, thus opening the dentinary tubules to the oral environment, while microbial colonization of the exposed dentin favors bacterial invasion, which might cause pulp inflammation. Normally, pulp vitality is not at risk, even if such therapeutical techniques may cause radicular dentinary hypersensitivity.

Conclusions: The results of the clinical and experimental studies performed indicate that correct scaling and root planning do not affect pulp vitality.

Keywords: endo-periodontal syndrome, scaling, root planning, dentinary hypersensitivity

INTRODUCTION

Formation of bacterial plaque on radicular surfaces as a result of some periodontal diseases may cause modifications at pulp level in the same way as the endodontic infections affect the periodontal tissue. Consequently, the bacterial products and the substances released by the inflammatory process in the periodontium may reach the pulp through lateral canals and the apical foramen or even through the dentinal tubes. Scaling and root planning are indispensable manoeuvres in the treatment of the periodontal disease, even if some intempestive or too frequently repeated techniques also remove not only the deposits occurring on the radicular surface but also part of the radicular cementum and dentin. In this way, the dentinal tubules will be exposed to the oral medium, while microbial colonization of the exposed dentin allows bacterial invasion, which might cause pulp inflammation. Normally, pulp vitality is not at risk, even if such therapeutical techniques may cause radicular dentinary hypersensitivity.

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Materials and method: Clinical and paraclinical evaluations were performed on teeth with periodontal pathology subjected to various mechanical treatments, such as scaling, root planning and local drug treatment.

Results: To maintain a healthy periodontal condition, scaling and root planning are being repeatedly applied, each time another part of dentin being removed, which weakens the dental structure and produces a higher amount of repair dentin in the pulp room.

Conclusions: The studies performed showed that the periodontal disease seldom affects the pulp. Other experimental studies indicate that scaling and root planning do not affect pulp vitality. Dentinal radicular hypersensitivity is a difficult to treat disease, creating serious discomfort.

1. Anatomo-pathological aspects of the periodontal disease effect upon the endodontic space

For the time being, no clear-cut relation could be established between the development of the periodontal disease and pulp affection. As a
matter of fact, even if the presence of necrosed pulp tissues was noticed in front of the lateral canals in the teeth with periodontal problems (1), the clinical studies performed did not confirm a direct relation between the periodontal disease and pulp modifications (2, 3). In a study performed by Bergenholtz and Lindhe (4), 70% of the examined teeth demonstrated no pulp affections, even if attachment losses occurred. The radicular surface exposed to experimental destruction of the periodontal tissue can show a few inflammatory cells, both in the presence or absence of repair dentin, being also present in the pulp zones from the vicinity of these areas. (fig. 1)

Such modifications are frequently associated to surface radicular resorption, suggesting that the dentinary tubules opened prior to irritation, may act as transmission ways (5).

The conclusion reached was that the cementum layer is extremely important in protecting the pulp against the actions of the bacterial plaque.

The absence of any correlation between the presence of the periodontal disease and the pulpar ones may depend on the number of communication ways – which is quite low. More than that, in the moment in which the dentin/pulp was exposed to bacterial attack, healing and repair phenomena occur, the remaining tissue being not affected (6).

In the study of Bergenholtz and Lindhe (4), the periodontal tissue was experimentally destroyed for a short period of time (5-7 months) while, in human teeth, such destruction occurs over a period of several years. The observation was made that the pulp of the periodontally affected teeth becomes fibrous, and some mineralization forms may occur, in parallel with a decrease in the number of blood vessels and nervous fibres.

It is understandable that tissular modifications represent the cummulated, even if a rather weak response of the pulp, whereas repeating the stimulus causes exposure of the dentinary tubules and of the lateral canals. The studies dedicated to such aspects showed that the periodontal disease seldomly affects the pulp, while the teeth with attachment losses maintain their functions, pulp destruction occurring only when the periodontal disease is in its terminal stage, a case in which the bacterial plaque reaches the apical foramen (7, 8). Apparently, as long as the blood vessels from the foramen level remain intact, the pulp is capable of resisting the irritating elements released by the periodontal lesion.

2. Influence of the periodontal etiological therapy (scaling and root-planning) upon the endodontic space

Scaling and planning are indispensable manoeuvres in the treatment of the periodontal disease, even if some intempestive or too frequently
repeated techniques also remove not only the deposits occurring on the radicular surface but also part of the radicular cementum and dentin. In this way, the dentinal tubules will be exposed to the oral medium, while microbial colonization of the exposed dentin favours bacterial invasion in the dentinal tubules (1), which might cause pulp inflammation (9, 10). Normally, pulp vitality is not at risk, even if such therapeutic techniques induce radicular dentinal hypersensitivity.

Figure 3 a, b evidences the clinical and radiographic aspects in a patient during the maintenance phase of the periodontal treatment. Even if the gingiva is in a very good condition, with no periodontal pockets, a considerable loss of dental substance may be observed at the cervical level. One of the incisives was later on fractured, without opening the pulp room, as due to the deposition of reaction dentin at the level of the pulp room.

Fig. 3 a, b Radicular exposure after intempestive scaling (a.clinical aspect, b.radiographic aspect)

To maintain a healthy periodontal condition, scaling and root planning are being repeatedly applied, each time another part of dentin being removed, which weakens the dental structure and produces a higher amount of repair dentin in the pulp room. In patients with maintenance therapy of 4-13 years, pulp necrosis hardly occurred (3%) and, if present, it is caused especially by caries or by the worsening of the periodontal disease and not by the periodontal treatment (10, 11). In very few cases, intempestive scaling may open the lateral canals, thus producing pulpite symptoms.

3. Influence of the periodontal etiological therapy on the occurrence of dentinal radicular hypersensitivity

The patients subjected to scaling and root planning may show increased sensitivity to tactile, thermal and osmotic stimuli (5, 9, 12). Usually, when present, such symptoms are manifested for about a week, after which they gradually disappear. Nevertheless, in certain situations, pain becomes chronic, continuing for months or even for years, most affected being the patients with periodontal surgical treatments.

Pain is called hypersensitivity, or dentinal sensitivity, cervical dentinal sensitivity or radicular sensitivity, which proves the confusion regarding its etiology (12, 13). The radicular surface becomes sensitive after the periodontal treatment, when the tubules which come into contact with the external medium get open, as well as a result of hydrodynamic forces. The stimuli lead to hydrodynamic modifications of the liquid from the dentinal tubules, thus inducing painful sensitivity (14). It is only this mechanism that can explain the sensitivity experimented immediately after the periodontal treatment and its persistence for a short period, nevertheless, not justifying its persistence for longer time periods.

The increased intensity of pain may be explained in several ways: 1) the smear-layer resulting from scaling and root planning dissolves away in a few days, so that hydraulic conductance at the level of the dentinal tubules increases (12) and the peripheral resistance of the liquids from dentin decreases. 2) the open dentinal tubules represent ways for the entrance of the bacterial elements from the oral cav-
ity into the dental pulp, thus inducing inflammatory phenomena (4). This is only a transitory phenomenon, observed until inflammation is over. An important element which increases radicular sensitivity after the treatment may be related to the peripheral sensibilization of the nociceptors, due to the released mediators of inflammation. In the teeth affected by hypersensitivity, several open dentary tubules may be noticed, while those without sensitivity show mineral deposits which obliterate the dentary tubules. (fig. 4. a, b, c, d)

Fig. 4. Electronic microscopic images of teeth with hypersensitivity and without sensitivity (Yoshiyama et al. 1989,1990).

The hydrodynamic mechanism of dentary pain will be inactivated. More than that, the diffusion potential of the bacterial elements towards the dental pulp is reduced. Microscopic observations showed that, in the teeth without radicular dentary sensitivity, the dentary tubules were not open, (14) whereas the radicular surfaces of the teeth with hypersensitivity had a large number of open dentary tubules.

Any treatment of radicular dentary hypersensitivity should be preceded by analysis of the causes having induced pain, special attention being paid to occlusal trauma.

Control of the bacterial plaque is essential for the prevention and treatment of dentary hypersensitivity. The clinical observation was made that the patients with a good hygiene have a tough, smooth and insensible radicular surface. Microscopically, dentin evidences mineral deposits which obturate the dentary tubules. However, when signs of radicular hypersensitivity occur, the patient cannot have a good hygiene. In such cases, desensitisation agents may be employed, as they temporarily obturate the dentary tubules, thus allowing a suitable hygiene.

In more severe cases, the possible treatments are pulpectomy and radicular obturation.

CONCLUSIONS

1. The studies performed showed that the periodontal disease seldom affects the pulp, the teeth with attachment loss preserving their functions, while pulp destruction appears only when the periodontal disease is in a terminal stage, which explains why the bacterial plaque reaches the apical foramen.

2. The results of the clinical and experimental studies indicate that scaling and root planning do not affect pulp vitality. Dentary radicular hypersensitivity is a difficult to treat disease, causing serious discomfort.

References


