CLINICAL, HISTOLOGICAL AND RADIOLOGICAL ASPECTS REGARDING THE INFLUENCE OF SOME EXTERNAL FACTORS ON THE PULP-DENTIN COMPLEX

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Abstract

The present study aimed at assessing – by clinical, histological and radiological investigations – the influence of some external factors on the pulp-dentin complex, and at providing a causal interpretation of the structural changes observed.

Materials and methods. Clinical and radiological exams were performed on 65 old patients with ages between 60-75, and also on 40 young patients with ages between 20-35, presenting different dental-periodontal pathologies. The pulp-dentin complex was submitted to a morphopathological examination, to highlight the structural changes observed at microscopic level. Fragments of dental pulp were immersed in a 4% formaldehyde solution with phosphate buffer 0.1 M., pH 7.2, for 12-14 hours, at a temperature of 4°C, and 3-5 µm thick slices were prepared. The slices were coloured with hematoxylin-eosine (HE), by the trichromic technique – Masson. Photographies were taken with a Zeiss microscope, with Kodak 200 ASA.

Results. Significant differences were observed, between the two groups of patients, as to the external factors that produce structural changes on pulp-dentin organ. In the group of young patients dental caries and coronal fillings prevailed, while the group of old patients was mostly associated with attrition and chronic marginal periodontitis. Out of the 40 young patients, 30 presented chronic dental caries (75%), while, among the 65 old patients, only 24 presented dental caries (36.9%). The percentages of coronary fillings between the two study groups were close, which could be considered as one of the causes producing changes in the pulp-dentin organ, following aggressive preparation of cavities, the action of materials used for the protection of pulp-dentin complex or of the materials used for coronal fillings.

Conclusions. Dental pulp has a remarkable ability to counteract the action of harmful factors, producing a mineral barrier and stimulating the reparatory processes. Changes in the endodontic space can be produced in both experimental groups, but more intensely and more frequently in the old patients. The endodontic space is modified, both physiologically and pathologically, including deposition of secondary or tertiary dentin (reactionary or reparative dentin), as well as pulp reactions, such as: inflammation, fibrosis, calcium degeneration or vacuolisis. At cell level, microscopic images showed a decrease of the odontoblasts number and sizes, as well as a reduced fibroblasts/fibrocytes ratios. These changes are associated with progressive vascular and nervous changes, that can be considered both their cause and effect. These pathological transformations are related with a more difficult preparation of the endodontic space and can be considered as having a major role in the failure of endodontic therapy.

Keywords: pulp-dentin complex, attrition, coronal filling, chronic marginal periodontitis, dental caries.

INTRODUCTION

The pulp-dentin complex suffers multiple influences of some external factors, more or less aggressive, provoking a pulp tissues reaction associated with various structural and functional changes. The normal reactions of the pulp-dentin complex, under the influence of thermal changes or of slow dental abrasion, are represented by deposition of secondary dentin.

Secondary dentin is the newly formed dentin during dental eruption, after its eruption and after the end of apexification process. Secondary dentin is formed continuously as a regular uniform layer around pulp cavity, at a very low rate. It has the chemical composition of an anorganic and organic material, similar with primary dentin, ye differentiated by some characteristics [1]. As to the pathological changes, they are produced under the influence of more aggressive factors, that interfere with the normal activity of the pulp-dentin complex, determining various reactions, that may cause the loss of pulp vitality. Among these factors, mention should be made of: treatments of dental caries, the action of restorative materials, attrition and chronic marginal periodontitis.

The reaction of pulp to these factors differs as to nature, intensity, action time, being expressed...
as deposition of tertiary dentine (reactionary dentin, or reparatory dentin) [2,3], or as mild/severe pulp inflammation. In cases with severe aggression, the function of destroying odontoblasts is assumed by the “odontoblast–like” cells, represented by undifferentiated mesenchimal cells or even by fibroblasts, that produce reparatory dentin [4, 5]. The deposition model and the dimensions of the new tertiary dentin area are correlated with the intensity and action period of the harmful/aggressive agent. When the intensity (nature) of the aggression factors is only moderate, the surviving odontoblasts can synthetize the reactionary dentin, with a structure similar to primary dentine [6].

Under the influence of the aggressive factors, pulp tissues suffer multiple structural changes, the most frequent causes being represented by acute or chronic inflammatory processes, associated with a series of characteristic pathological phenomena. These changes can induce necrosis, fibrosis, mineralising dystrophy, being associated with denticles or pulp calculus.

Dental pulp also tries to stop the evolution of the carious process through remineralisation of dental demineralised matrix and dentine sclerosis [7]. At dentin level, the presence of dentinal tubules, as well as a lower content of minerals and the cristalisation process, allow a more rapid evolution of dental caries [8]. When the high intensity of the aggressive factors is combined with the absence of therapeutical measures, the odontoblasts suffer destructive processes. Inside the pulp-dentine complex, a defensive layer is formed by the mobilisation of PMN, lymphocites, macrophages, plasmocites, along with induction of an immune response and formation of a fibrosis tissue barrier following a more increased activity of fibroblasts, with the final aim to isolate the inflammatory area by the healthy pulp tissue [9]. Pulp ageing is associated with a high degree of "physiological ageing", morphological and functional changes, and progressive deterioration [10]. The periodontal recessions associated to ageing or to marginal periodontal diseases, expose the cement-enamel jonction to the aggressive factors of the oral environment [11]. The attachment loss represents the sum of the two types of visible and invisible recession [12].

**MATERIALS AND METHOD**

Clinical and radiological exams were performed on 65 old patients with ages between 60-75 years, and also on 40 young patients with ages between 20-35 years, presenting different dental-periodontal pathologies. The pulp-dentin complex was submitted to morphopathological exam, to highlight the structural changes produced at microscopic level. The dental pulp fragments were immersed in a 4% formaldehyde solution with phosphate buffer 0.1 M., pH 7.2, for 12-14 hours, at 4°C. 3-5 µm thick slices were prepared.

The slices were coloured with hematoxylin-eosine (HE) and by the tricromic technique-Masson. The examination and photographs were performed using a Zeiss microscope, with Kodak 200 ASA.

**RESULTS AND DISCUSSION**

Between the two groups of patients significant differences were observed regarding the external factors that produce structural changes on the pulp-dentin organ. In the experimental group of young patients, dental caries and coronal fillings prevailed, while the group of old patients was mostly associated with attrition and chronic marginal periodontitis. Out of the 40 young patients, 30 presented chronic dental caries (75%) while, out of the 65 old patients, only 24 presented dental caries (36.9%). This aspect highlights the fact that young patients have a higher prevalence of dental carious disease with more rapid evolution to acute pulp inflammation. For old patients, the evolution of dental caries is slower, because of the continuous deposition of secondary dentin along pulp room walls. The percentages of coro-

nal fillings between the two groups were close, which could be a cause of the changes observed in the pulp-dentin organ, following aggressive preparation of cavities, the action of materials used for the protection of pulp-dentin complex or materials used for coronal fillings. Higher percents of pulp-dentin organ changes were found in old patients, presenting attrition or chronic marginal periodontitis, as a result of local inflammatory factors and accentuated hard tissues loss.
Dentin depositions (fig. 3) prevailing at the level of pulp horns, determine retraction of pulp room roof, sometimes followed by pulp room disappearance. Decrease of pulp room volume or even pulp room disappearance lead to more difficult endodontic treatments, especially for beginner dental practitioners.

For multiroot teeth, the furcation area is frequently associated with dentin deposition, having unfavourable effect on the access of root canals, as well as on the mechanico-chemical stage of the endodontic treatment, as evidenced by the radiographical image of a maxillary molar in an old patient. The external aggressions exerted along years, represented by deep proximal decay and an inflammatory periodontal process, were followed by modification of endodontic space anatomy (fig. 4).

Coronal fillings represent another factor associated with the modification of endodontic space anatomy. Aggressive instrumentation performed
for cavity preparation and filling, as well as coronal restorative materials represent harmful factors that can provoke the deposition of successive dentin layers, as shown by the radiographical image of 3.6. (fig. 5).

The next radiological image (fig. 6) allowed the identification of an endodontic space with significant radioopacity at 4.4., both in coronal third and apical third of root canal, which makes more difficult the mechanical preparation during the endodontic treatment. Tooth 4.3. also presents a pulp calculus in the coronal third of the root canal.

The next figure presents the histological aspects of a tooth with open ulcerative pulpitis associated with the presence of lymphocites, plasmocites, macrophages, dilated vessels, stasis, interstitial oedema (fig. 8).

In old patients, the modifications are more various and complex. The following figure plots the histological aspect of a pulp sample from a tooth with intense attrition and a chronic inflammatory process. The image shows an area with pulp fibrosis and inflammatory oedema (Fig. 9).

To highlight such pulp changes, pulp fragments were sampled and processed for microscopic examinations, from the teeth with severe pulp inflammations. In young patients, structural changes of pulp were localised only in areas associated with inflammations produced by deep dental caries or after aggressive prosthetic preparation of the dental structure. In the case of a tooth with deep distal-occlusal decay, microscopic image shows a discrete disorganisation of the odontoblastic layer, slight oedema of the extracellular matrix and the presence of star-shaped fibroblasts (fig. 7).

The next image shows a pulpal area presenting a vacuolisis degenerative process, with oval-shaped or polyhedral-shaped white adipocytes (fig. 10).
Another pulp reaction, seen at a tooth with chronic marginal periodontitis and attrition, is represented by the mineralisation areas appearing between the collagen fascicles. The odontoblastic layer is well-represented, while the rest of pulp contains more fibrocytes than fibroblasts (fig. 11).

**Fig. 11. Incipient pulp mineralisation**

**CONCLUSIONS**

Our research approached aspects regarding structural changes of the pulp-dentin complex, initiated by different external stimulus. To differentiate the effects of these stimuli, as to their nature, intensity and duration, two experimental groups, of young (20-35 years) and, respectively, old patients (60-75 years), were investigated by clinical, radiological and histological examinations.

We have focused on the pulp-dentin complex changes and on the defence pulp reactions, following the action of some external aggressive factors. The microscopic images showed the remarkable ability of pulp to oppose to the action of the harmful factors, producing mineral barriers and stimulating the repair process. The causes of these changes in the pulp-dentin complex were: dental caries, coronal fillings, chronic marginal periodontitis and attrition.

Modifications in the endodontic space can be produced in both groups, being however more intense and more frequently occurring in old patients. The nature and intensity of these reactions differ among patients and teeth, highlighting the influence of the individual, genetically transmitted features. The endodontic space, both physiological and pathological modifications, included deposition of secondary or tertiary dentin (reactionary dentin or reparative dentin), as well as pulp reactions, such as: inflammation, fibrosis, calcium degeneration or vacuolisation. At cellular level, the microscopic images showed a decrease of the odontoblasts number and size, as well as reduction of the fibroblasts/fibrocytes ratio. These changes are associated with progressive vascular and nervous modifications, that can be considered simultaneously a cause and an effect. These pathological transformations, related with a more difficult preparation of the endodontic space, can be considered a major cause in the failure of endodontic therapy.

**References**