World Journal of Medical Sciences 10 (2): 135-138, 2014
ISSN 1817-3055

DOI: 10.5829/idosi.wjms.2014.10.2.1139

Structural-morphological Changes in a Primary Teeth Tissue as Predisposing Causes to Dental Caries Development

[1]*Marina Anatolyevna Danilova,* [1]*Yulia Vadimovna Shevtsova,*
[2]*Elena Sergeevna Patlusova and* [1]*Natalia Alekseevna Machulina*

[1]The State Budget Educational Institution of Higher Professional Education
"Perm State Medical Academy Named after Academician Eugenie Anatoljevich Vagner"
of Health's Ministry of Russian Federation, Petropavlovskya Street, 26, Perm, 614 990 Russia
[2]Pathology Department of GBUZ PC Perm Regional Children's Clinical Hospital,
Bauman Street, 22, Perm, 614 066 Russia

Abstract: In the course of this research histological features of primary teeth during the change to permanent teeth have been searched. This research is based on in vitro analyse of 36 extracted intact primary molars. In this article there are morphological changes as a result of vital activity of a tooth related to all its structural components including enamel, dentine and cellular elements. Due to the resorption of primary teeth not only disturbances of local mineral metabolism have been found (enamel destruction with following extra attrition and under layers' exposure, hydropic degeneration, colliquation and pathological dentine formation), but also perversion of synthetic process of biogenic tooth matrix – fibrous elements (pre-dentin structure changes, alterant processes at an odontoblasts level). Indicated changes cause disorganization and deformation of structural constituents in temporary teeth hard tissues. Described features in histological structure of tested teeth have been regarded as factors reducing primary teeth resistance to environmental activities and cause pathologic process especially tooth caries.

Key words: Histology · Structural-morphological changes · Primary teeth · Enamel · Dentine

INTRODUCTION

Describing histological structure of primary teeth the majority of authors of the study base themselves upon comparison between temporary and permanent teeth [1, 2, 3, 4]. At the same time the description of primary teeth has mainly generalized character without regard to stage-by-stage approach of odontogenesis [5, 6, 7, 8].

Histological structure of primary teeth hard tissues differs according to a certain stage of a tooth life [9]. The most significant and strong changes are observed during a period of primary teeth involution. The processes during normal exfoliation time are of physiological nature but at the same time can cause a descend of its resistance to unfavorable factors especially caries [10, 11, 12].

MATERIALS AND METHODS

Histological research of features of primary teeth structure in the period of mixed dentition has been conducted. Thirty-six extracted (due to exfoliation time) intact primary molars have been taken for that purpose.

This material was fixated in 15 % formaline and then the teeth were deterged and rinsed in flow water. Our material was decalcified in 10% nitric acid during 4 weeks. After that the teeth were replaced into 5% potassium alum liquid for 24 hours and then were rinsed in flow water for 48 hours. Later extracted teeth had been going through histoprocessor Leica TP 1020 with automatically preplanned sequence for 18 hours. At this time the material was in graded alcohols for target time.

Corresponding Author: Marina Anatolyevna Danilova, the State Budget Educational Institution of Higher Professional Education "Perm State Medical Academy Named after Academician Eugenie Anatoljevich Vagner" of Health's Ministry of Russian Federation, Petropavlovskya Street, 26, Perm, 614 990 Russia.

Going forward the teeth were paraffin-embedded with paraffin stock “Histomix”. This process was going on the paraffin block Thermo Scientific Histo Star. After following through and paraffin-embedding slices of 2-3 micron thick were made from the blocks by microtome semi-automatic machine Thermo Scientific Microm HM 325. Then they were attached to slide and stained with ematoxylin, eosin as well as Van Gieson formula. Medication analisis was made with the help of a binocular light microscope «Axioscop 40» (x40, x100, x400). The slices research was conducted with the usage of computer vision analysis system (digital camera Infinity 1).

RESULTS AND DISCUSSION

In the majority of cases there is a variable degree of enamel abrasion on a tooth surface. The structure of comparatively preserved enamel areas is non homogenous and unequally stained with the path of enamel prism not visible enough. There is a granular view in the deep layers (Figure 1).

Previously mentioned structural changes can be recognized as derangement of enamel mineralization with further destruction of inorganic components and as a result there is permeability-increasing of enamel surface. These indicated conditions provided an enamel defect in the form of cracks (Figure 2) and lacunae (Figure 3) which are blunt-edged and expand in the range of upper dentinal layers.

In this case enamel and dentine damage simplifies the penetration of infection into the tooth depth.

The border between enamel and dentine with the help of investigational products often looked blunt-edged, obscure signified and serrated. In some cases globular structures had been analyzed closely to dentino-enamel junction on the level of upper dentinal layers which further could provide a defect-causing process of hard tissues mineralization. Globular forms of dentine, located in this zone, are considered as factors of derangement mineralization processes providing dentine destruction. Moreover the path of dental tubules had not been regarded at a level of globular forms (Figure 4).

Therefore, not only defect-causing processes of mineralization have been found out in our research but also the production of dentine matrix – the base of dental tissues in connection with deep alternative process at the cellular level. The absence of clear border between enamel and dentine and dentine structural disturbance can cause pathologic process especially caries.

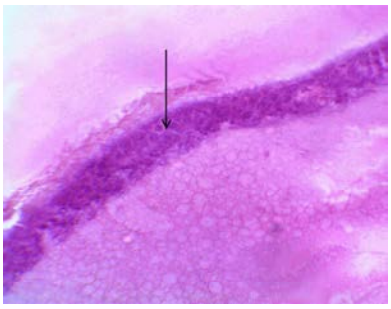

Fig. 1: x400. Hematoxylin and eosin staining. Granular view of enamel

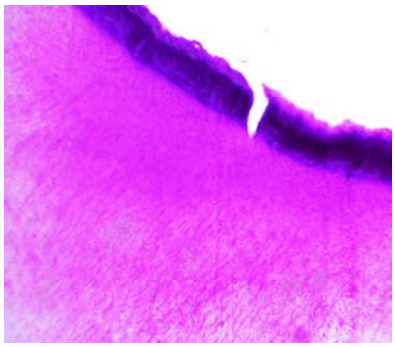

Fig. 2: x400. Hematoxylin and eosin staining. An enamel defect in the form of crack expanding to upper dentinal layers

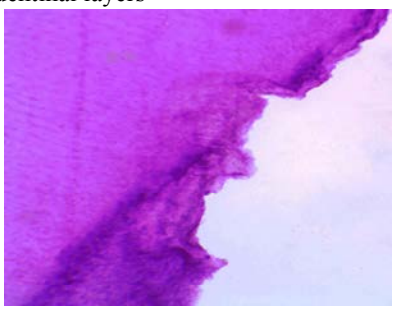

Fig. 3: x 400. Hematoxylin and eosin staining. An enamel defect in the form of lacuna expanding to upper dentinal layers

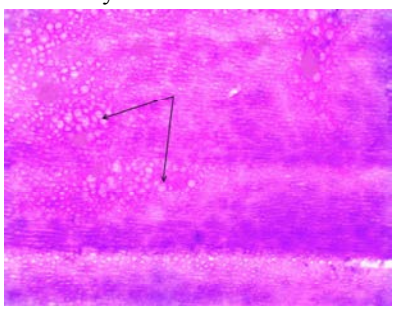

Fig. 4: x400. Hematoxylin and eosin staining. Serious hydropic degeneration of odontoblastic processes and globular forms in upper dentinal layers

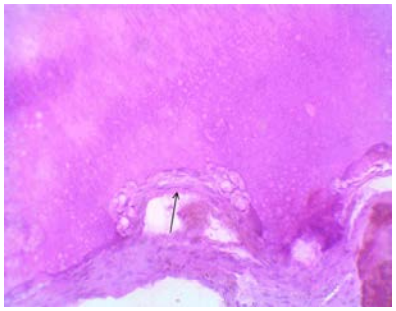

Fig. 5: x400. Hematoxylin and eosin staining. Lacunar defect on a dentine surface consisting of plaque accumulation. Dentine vacuolization is on the defect junction

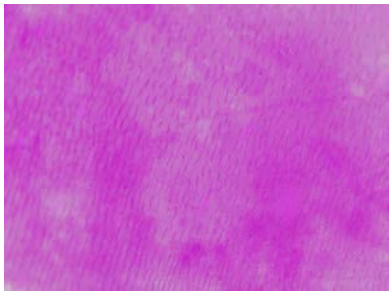

Fig. 6: x400. Hematoxylin and eosin staining. Structural damage of dentinal tubules

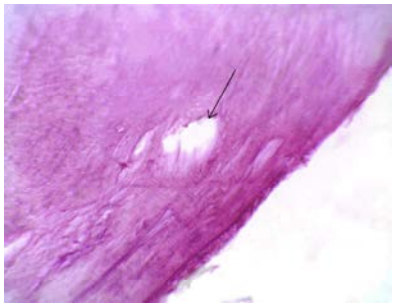

Fig. 7: x400. Hematoxylin and eosin staining. Colliquation area in dentine

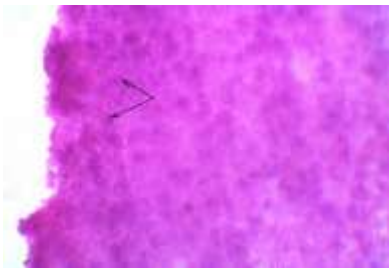

Fig. 8: x400. Hematoxylin and eosin staining. Lack of clear junction dentine-to-predentine transition. There are globules in the very layers

Disorganization and destruction processes affecting enamel have caused extra dental enamel attrition and as a result an underlay dentine exposure, which also becomes more sensitive to negative impacts especially bacterial genesis. On the surface of dentine exposure with skimmed off enamel layer there are also different kinds of defects in a form of lacunae (Figure 5).

In this connection dentine without an enamel layer is more amenable to bacterial adhesion due to dentinal tubules exposure, which have got a wider space in primary teeth (in comparison with permanent teeth).

During the deep dentine layers measurement the deformation of dentinal tubules in apical dentine areas had been noticed. Their path was hardly seen, the structure was homogenized (Figure 6).

These changes demonstrate the damage of cellular dentine component, specific cell processes – odontoblasts. At this time, firstly, there is a defection of peritubular dentine (which surrounds every dentinal tubule and forms its wall) and intertubular dentine producing. As far as dentinal tubules are the components of special cells taking part in a formation of hard tissues described changes have been recognized alterative, causing the death of cell structures and following dentine disorganization. It leads to decreasing activity of the mineralization and fibrillogenesis processes with reducing dentine density. In the process of damage of dentinal tubules they spread, lose shape and dentine permeability increases. It can cause rapidity of decay.

In case of increasing permeability of dentine under conditions of integrity or damaging of tooth enamel, on the one hand and damaging of cell structures, on the other hand, indicate deep and irreversible damage of water mineral turnover at a local level. In upper dentine layers hydropic degeneration in a form of inordinately arranged vacuoles had been noticed. In some places this fact has an expanding nature which demonstrates multifocal alteration processes. In some cases, vacuole fusion causes extended colliquation areas formation with clearing long spans at different levels of dentine areas (Figure 7).

In our opinion the changes in dentine structure can provide easier permeability and unhampered expansion of tooth hard tissues demineralization.

Changes also affected parapulpar tissues especially predentine layer. This layer is visualized indistinctly. It has got irregular thickness, partly dwarfed, minimally visible. The border between dentine and predentine is light significant: partly flat, partly serrated, commonly distorted. Sometimes on the border between dentine and predentine there are round globular

formations which are pathological dentine forms connected with defection of its structural organization and calcification processes (Figure 8).

Lack of clear transition of layers, existence of calcification areas all these factors also can cause rapidity of caries processes.

CONCLUSION

All mentioned processes occur in a period of primary teeth "wearing out" and described structure functional changes are the factors reducing caries resistance.

Dental abrasion and structural imperfections of layers concerning hard tissues make teeth vulnerable to negative impacts. As a result different kinds of defects can appear on the surface which prolong and intensify tooth vulnerability to infectious agent.

Involutional processes in enamel and dentine lead to irreversible processes of deformation and disorganization in component structures with permeability-increasing in the observed layers that causes such pathological process as caries.

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