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ETIOPATHOGENETIC CONSIDERATION AND DEFINISHON OF THE CLINICAL MANIFESTATION OF EROSIVE DENTAL DEFECTS

ABSTRACT: Dental defects of errosive nature are defined as irreversible losses of dental tissue, caused by long lasting and repeated action of acids that disolve top layer of hydroxyapatite and fluorideapatites cristal structure, under assumption that agressive factor is not of bacterial nature. Acids that cause changes on teeth according to their origin are gastric, dietetic, or they are of environmental origin. Current way of life, as well as nutritional habits create potentially dangerous conditions for the hard dental tissue, for prevention of mineralization process causes defects of oral system homeostasis. Defects occur on primary teeth, as well as on permanent teeth. However, this happens once and a half time more frequently on primary teeth due to the weaker primary maturation. In initial phases, changes are localized in enamel and by their development the bottom locates in dentine. Defects appear as smooth, shainy, round concavities on caries immune positions, or as cupping of occlusal surfaces. The depth of an eroded lesion consists of the depth of the crater plus the depth of tissue demineralisation at the base of the lesion. Early verification of the etiological factor, together with good knowledge of the manifested shape change has influence to the prevention of the crown of tooth loss, complete occlusion, mastication and speech.

KEY WORDS: dental erosions, diagnosis, etiology, pathogenesis

INTRODUCTION

Enamel, highly mineralized crystal structure, the densest and the hardest biological tissue of the human organism after teeth eruption remains exposed to the aggressive and biologically active environment of the oral ecosystem. Enamel minerals have crystal structure, but hydroxyapatite is not stoichimetric; it does not have fixed element proportion, for relation of calcium and phosphates is always lower than the assumed one in theoretic formula. Along with calcium, phosphorous, carbonate and fluoride enamel contains over forty other elements. Presence of carbonate and magnesium impurities in hydroxyapatites

petals increases its solubility in acid environment, while fluoride and strontium make enamel more stable.

Enamel content is not a homogenous category. Changes are evident toward referent surface, as well as toward observed depth. Enamel top layers contain significantly higher fluoride concentration than those on enamel and dental enamel junction, while remaining elements such as magnesium, carbonates, water and organic compounds are in an inverse proportion. Cervical enamel has significantly lower mineral density from one that is on occlusal surface of a tooth, or on incisal edge (Sturdevant et al., 1995, Vulović et al., 2002). Mineralization level of primary teeth is connected to the period of teeth eruption into oral cavity. During posteruptive maturation, the duration of which is one to two years, into enamel top layers about 10% more minerals are incorporated.

Enamel layer, tartar and saliva are specific and unique biosystem. In cases of neutral saliva pH values, there is equilibrium in mineral quantity that goes into and out of the enamel. If pH values lower under 5.5, or more, calcification is more intensive from decalcification, and whole activity is determined above all by saliva content, such as calcium, phosphates, fluoride, but also by saliva buffer capacity, protein quantity and quantity of stimulated and unstimulated saliva (Leach, 1986).

By local fluoride application we can influence to enamel "solubility". If its concentration is higher than 100 ppm, changes are manifested to the depth of 0.1—0.2 mm. Saliva free calcium react with fluoride ions, and calcium fluoride that deposits to unmineralised enamel is created on a teeth top layer. Fluoride can be considered anticariogenic, but tooth wear factor as well.

PATHOGENESIS OF THE EROSIVE CHANGES

Inadequate function of saliva puffer system, as well as eventual existence of xerostomia together with long lasting and frequently repeated incorporation or existence of different kinds of acids in oral microenvironment causes lowering of saliva pH values and occurence of deficient balans between demineralization and mineralization. Using interprismatic and intercrystal layers hydrogen ions penetrate enamel and cause solution of hydroxyapatite that cases reduction of the crystal size itself (Vulović et al., 2002). Ultra structural studies suggest that erosive lesions are seen in prismatic enamel as characteristic demineralization patterns where either the prism cores or interprismatic areas dissolve, leading to a honeycomb structure. In aprismatic enamel the pattern of dissolution is more irregular and areas with various degrees of mineral loss are seen side by side. In dentine, the first area to be affected is peritubular dentine. With lesion progression, the dentinal tubules become enlarged, but finally disruption is seen also in the intertubular area. According to current knowledge, there are no differences in lesion shape, size or depth, depending upon kind of acid that caused the defect occurence (Meurman, Ten Cate, 1996).

The depth of an eroded lesion consists of the depth of the crater plus the depth of tissue demineralization at the base of the lesion that is microradiographic detectable (A m a e c h i, H i g h a m, 2005).

Study of erosive changes occurence led to the conclusion that clinical manifestation of the erosive defect is not only result of erosive agents action, but also consequence of the combined action of demineralization of the tooth structure by erosive agents and abrasive action of the surrounding oral soft tissue, as well as by action of abrasive food during mastication and by use of abrasive foreign substances. Abrasive activity of the specified soft tissue structures determines defect location (A m a e c h i et al., 2003, I m f e l d, 1994).

Lesion expansion is a cause of cumulative process caused by:

— Frequency and time period of exposure to acids;
— Maintaining of oral hygiene basic principles;
— Individual sensitivity.

Dental top layer exposed to the action of acids undergoes process of demineralization making the enamel itself, as well as dentin more fragile to the abrasive factors, especially if the exposure to the abrasive force is performed directly after acid intake without previous leveling of pH values by salivary buffer capacity. Need for shortening of the time period from the acid intake to the moment of teeth brushing that lasts thirty minutes is imposed. Attrition of incisal edges and abfractions on cement enamel junction can increase defect caused by erosion, making by it diagnosis of the defect cause more difficult.

Dental erosions are also diagnosed in primary teeth. Attacked structures, enamel and dentin are of significantly less thickness, weaker mineralization (W i l s o n, B e y m a n, 1989), and enamel porous levels increase (F e j e r s k o v et al., 1987) than in permanent teeth. Weaker mineralisation and big pulp cavity cause fast "wearing out" of the dental tissue and formation of larger defects and earlier occurence of dental hypersensitivity, causing also opening of the pulp cavity accompanied by pulp development (S h a w, O'S a l l i v a n, 2000). Fast development of the process causes complete loss of the crown of a tooth and dental organ in whole, which can disturb bite, mastication and speech. Dental erosions in primary teeth can be predictors of the higher risk of erosive dental defects in permanent teeth. *In vitro* conditions they exhibit once and a half time higher susceptibility to erosion than permanent teeth.

ETIOLOGY AND CLASSIFICATION

Dental erosions are defined as irreversible loss of the hard dental tissue caused by long lasting and repeated action of acids that dissolve top layer of the hydroxyapatite and fluoroapatites crystal structure, even when aggressive cause is not cased by bacteria (P i n t b o r g, 1970, E c c l e s, 1974, K i d d et al., 2003, S u t a l o, N j e m i r o v s k i, 1981). Occurence of the saliva low pH value is cased by different kinds of acids, according to their chemical composition, chloral hydrogen acid, ascorbic acid, lemon acid: phosphorous acid, milk acid, but also gastric juices, remedies with the vitamin C, fruit, and car-

bonated beverages. Classification of dental erosions was performed according to the origin of acids that can cause defect occurence to the following ones:

— Endogenous;
— Exogenous;
— Idiopathic.

Endogenous erosions — develop as a consequence of penetration of gastric chlorine hydrogen acid into oral cavity in such a high quantity, often long lasting, that saliva buffer system proved unsuccessful (S c h e n t z e l, 1996). Sometimes gastric acid has pH serial value that starts from one, reaches oral cavity through gastroesophageal reflux or by chronic vomiting (Figure 1).

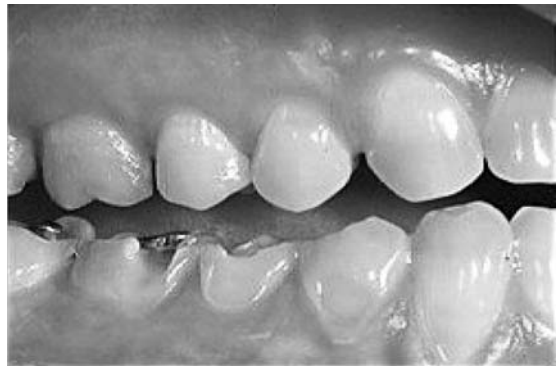

Fig. 1. — Endogenous erosions

The gastroesophageal reflux can occur in the aspect of a disease or it can be provoked. It occurs as a disease in a case of increased abdominal pressure, in a case of inadequate relaxation of lower esophageal sphincter, or in cases of increased production of gastric juices. Exaggerated consuming of a chocolate, coffee, peppermint, spices, as well as fat food causes provoked regurgitation that, when frequently repeated, can be a cause of such dental defects.

Chronicle vomiting in cases of nutritional disorders, such as anorexia, bulimia neurosis, rumination, chemotherapy, alcoholism, and even gravity, peptic ulcers, as well as gastritis place chlorine hydrogen acid into oral cavity and enable its negative action to the surrounding tissue structures, i. e. negative effect of life habits and style is exhibited. Erosions emerged as a consequence of xerostomia make special category. According to the origin of the acids themselves, they could be classified into mixed changes, for the presence of endogenous acid, as well as many exogenous acids introduced with food often coexist. At the same time there exist reduced quantity of saliva and all its elements that make buffer capacity. Some medical diseases and conditions cause occurence of xerostomia:

— Endocrine diseases — diabetes, hyperthyroidism;
— Autoimmune diseases — HIV infections, Systemic lupus, Rheumatic arthritis, Sjögren's syndrome;

— Medicine intake — vitamin C, antipsychotics, antidepressants, appetite suppressants, diuretics, sedatives, hypnotics, antihistamines, and antihypertensives (A m a e c h i et al., 2005, M a r o n, 1996, H a y s et al., 1992).

Exogenous erosions — develop under the action of acids that reach oral cavity from environment by dietetic pathway, or in certain environments as air pollutants (Z e r o, 1996). Dietetic acid source can be food such as: fruit (lemon, apple, plum), tomato, mustard, ketchup, carbonated soft drinks (Coca Cola, carbonated water), squeezed juices (orange, grape, kiwi), alcohol drinks (vine, bear). Several studies have proved that consummation frequency is of the identical importance as the acid level (M i l l w a r d et al., 1994), as well as the method of intake for it is better to take drinks by straw, than to drink directly from glass, and the drink should be kept in mouth as shortly as possible. Time period of teeth brushing is also important, for after food intake teeth brushing should be postponed to thirty minutes from the last intake of acid drink in order to allow buffer capacity of saliva to increase pH value and reduce abrasive action of toothpaste and a brush. This all has significant role in determination of defect size (G a n g a r a et al., 1999).

Consummation of cocaine and ecstasy is related to intake of greater quantity of acid drinks due to the occurence of dehydration and hyposalivation that cause erosive dental changes (D u x b u r y, 1993).

Dental erosions can also be classified into category of professional diseases. Persons who test vine or carbonated beverages on regular basis, as well as professional swimmers can detect this type of the defect on their teeth. (M a n d e l, 2005). Evaporation of industrial acids from battery plants, sanitary cleaning solutions, crystal glass are also dental erosion causers (M i l o š e v i ć, 1998).

Idiopathic erosions — are erosive changes whose existence can not be explained by any of the currently known causer (S u t a l o, N j e m i r o v s k i, 1981).

Current way of life and nutritional habits create conditions that are dangerous for hard dental tissue, for they prevent mechanism of enamel mineralization and in this way disturb homeostasis of the oral system (S m i t h, R o b b, 1996). This is especially expressed in childhood. Therefore, due to inadequately large quantity of juices, carbonated beverages and fruit, this kind of disease is considered typical for the standard of living. In age of adulthood it represents part of the clinical picture of psychosomatic diseases and the cause of certain therapy procedures. In sporadic cases it can also be considered as a professional disease.

According to the clinical picture changes on dental top layers are classified to:

— Superfficial and profound
— Localized and generalized (Z e r o 1996, I m f e l d, 1996);
— Manifested and latent;
— Eccles's and Jenkin's scale for erosive changes in cavity depth 0—3,

And according to the attacked surface they can be localized to:

— Palatal and occlusal surface of the upper jaw teeth;
— Buccal and occlusal surface of the lower jaw teeth.

CLINICAL PICTURE

Due to the lack of any method or procedure that could be used for early detection and quantification of the change, diagnostic of dental erosions is problematic. In early stadium, by erosion changed surface is smooth, shine, without macroscopic defects (A m a e c h i, H i g h a m, 2005), but it can be dim, without expressed colored lines, or clear frontiers toward unchanged part of the dental tissue.

If the defect is localized at inicisal edge, the incisial groove at the dentin is formed (Figure 2). However, if endogenous source is an acid, defect occurs at palatinal surface of the incisors, that becomes smooth, shine and hard. Vestibular diameter is also reduced making inicisal edge thinner and transparent and gingival region existence of an enamel collar (Figure 3).

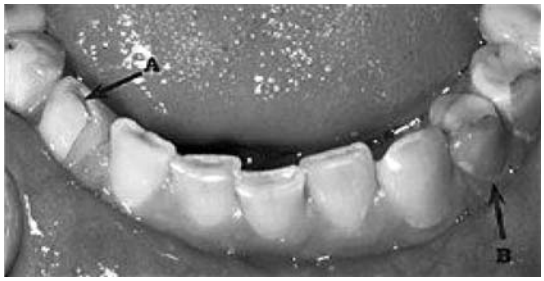

Fig. 2. — Incisal grooving and broad concavities

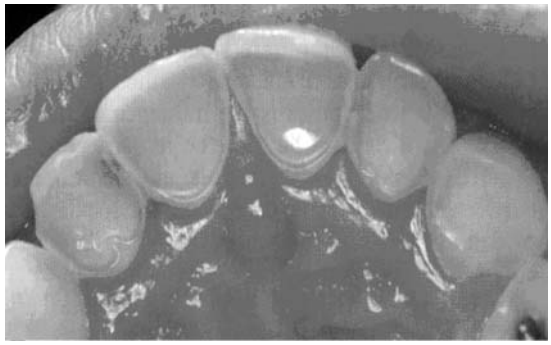

Fig. 3. — Endogenous erosion-enamel collar

At vestibular surface changes are manifested as broad concavities (Figure 4), and at occlusal surface as cup-like recesses (Figure 5). Amalgam fillings on such teeth appear as grown out, i. e. they occur above dental structure (Figure 6).

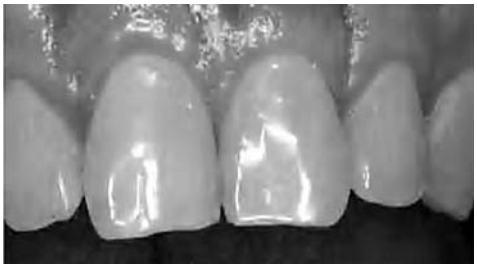

Fig. 4. — Broad concavities

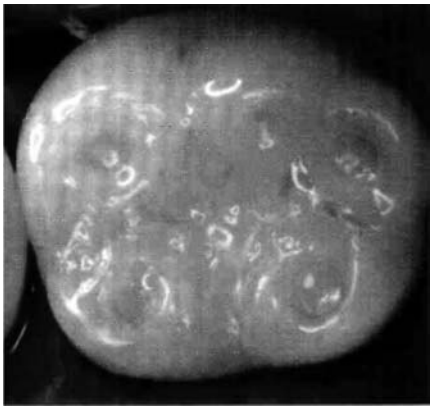

Fig. 5. — Capping of occlusal surfaces

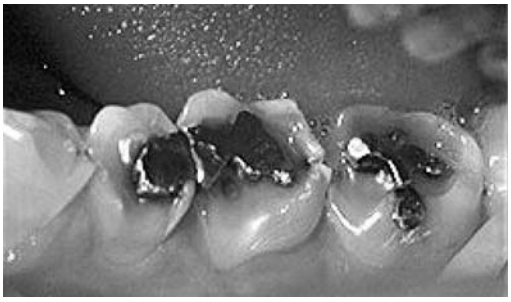

Fig. 6. — Raised amalgam restorations

Difficulties in diagnostics of erosive changes increase significantly at primary teeth (S h a w, O'S u l l i v a n, 2000). Enamel and dentin are thinner, less mineralized and porous, and acids aggressive activity appears even more, i. e. primary teeth are more susceptible to erosive changes. Initial phases are hard to detect. These changes on childrens' teeth are most frequently localized at occlusal surfaces of molars and inicisal surfaces that result in morphology loss (Figure 7), occurence of the dentine hypersensivity, as well as complete loss of the crown of the teeth, the pulpites, and premature extraction of the

primary teeth with all resulted consequences. Dental defects at primary teeth must always be observed from cumulative multi factor aspect. Attrition of incisal edges in primary dentition is frequent during exfoliation and it is than very hard to estimate cause of the change.

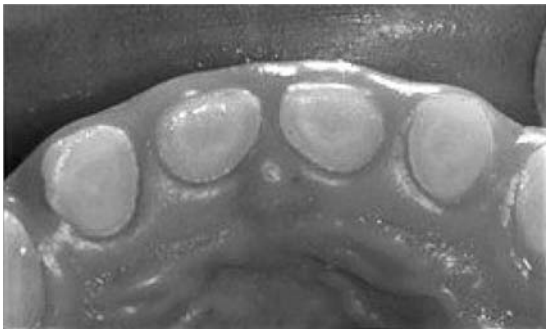

Fig. 7. — Loss of surface characteristics

Diagnostics of dental erosions requires serious, objective examination, analysis and evaluation. Well made questionnaire with precisely defined questions in regard to etiology is necessary. Saliva analysis related to determination of stimulated and unstimulated saliva quantities, extent of calcium and phosphates, buffer capacity, urea quantity is also of the highest importance.

After established diagnosis, progradation dynamic is necessary to be monitored. For that purpose silica index, dental erosion index and study models according to Wickens and taking a photograph are used (G a n d a r a et al., 1999).

PREVALENCE

At the beginning of the 19th century, the first data on existence of this disease were registered (M a h o n e y, K i l p a t r i c, 2003). It occurs in each life time, and it is distributed evenly between sexes. In child age it is considered a disease of living standard (B a r d s l a y et al., 2004), for it is a consequence of intake of great quantities of carbonated beverages, as well as juices and fruits.

Changes are more frequently located at upper jaw tooth, then lower one. The most common changes are on incisives, that are followed by changes on caninus and molar teeth. In lower jaw, defects are predominantly located on caninus and molars.

Regurgitation erosions appear at palatal surface of the upper front teeth, as well as at occlusal and buccal surface of lower lateral teeth. Dental erosions localized at vestibular surface of front teeth with hole-like defects are determined as professional diseases.

DIFFERENTIAL DIAGNOSIS

Erosions as causers of the health tooth tissue loss are a part of a detailed picture of dental defects to which attrition, abrasion and abfraction also belong.

Attrition — causes defects of dental tissue as well as of the established filling, and it is caused by teeth contact during mastification or parafunction. Occlusal surface are smooth, shiny, flat and hard, and at amalgam filling are observed shiny mark. Bottom of the defect can be located in enamel, as well as in dentin.

Abrasion — is caused by direct contact of teeth and foreign substance such as whitening toothpaste, antinicotin paste, sodiumbicarbonate. Changes localized in cervical region are always wider than deeper, and they are most frequently found on premolars and molars.

Abfraction — is characterized by dental tissue loss in cervical region caused by compression and pulling force that occurs during dental flexure. Changes are localized vestibular and they are wedge-shaped (G a n d a r a et al., 1999).

Based upon all stated, it can be concluded that dental erosions represent problem of human population. Many aspects require further research and more precise defining. This is especially important in relation to early diagnostics and quantification of changes for further longitudinal monitoring. Further studies are to be started by epidemiological studies for the purpose of more adequat prevalence, seriousness and spread of changes in different populations. Widening of knowledge from the aspect of pathophysiology is necessary, as well as detection of protective factors, preventive techniques and hemioterapeuticals. It is essential to develop preventive strategy, that should be followed by procedures for limitation of further damages. Protection of the remaining tissue with adequate reconstruction by contemporary dental materials is also of the highest importance. We are expected to fullfil a huge task.

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ЕТИОПАТОГЕНЕТСКО РАЗМАТРАЊЕ И ДЕФИНИСАЊЕ КЛИНИЧКЕ МАНИФЕСТАЦИЈЕ ЗУБНИХ ДЕФЕКАТА ЕРОЗИВНЕ ПРИРОДЕ

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Резиме

Зубни дефекти ерозивне природе дефинишу се као иреверзибилни губици зубних ткива, изазвани дуготрајним и понављаним дејством киселина које растварају површински слој кристалне структуре хидроксиапатита и флуороапатита а да агресивна нокса по свом пореклу није бактеријске природе. Киселине које изазивају промене на зубима по свом пореклу су гастричне, дијететске или потичу из животне средине. Данашњи стил живота као и нутрициона навике креирају стања која су потенцијално опасна за тврдо зубно ткиво јер се спречавањем механизма реминерализације ремети хомеостаза оралног система. Дефекти се јављају како у млечном тако и у сталном зубљу али један и по пут више у млечном због слабије примарне матурације. Промене су у иницијалним стадијумима локализоване у глеђи да би се прогредирањем дно лоцирало у дентин. Дефекти су у виду глатких, сјајних конкавитета овалног облика, на каријес имуним местима, или у виду шољастих удубљења на оклузалним површинама. Дубина дефекта настала денталном ерозијом одређена је дубином кратера којој је додата дубина ткивне деминерализације. Рана верификација етиолошког фактора уз ваљано познавање манифестног облика промене утиче на спречавање губитка комплетне крунице зуба што би довело до ремећења загризаја, мастикације и говора.