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Dental Erosions – Extraesophageal Manifestation of Gastroesophageal Reflux

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1. Introduction

Dentists are often the first healthcare line that can – diagnose certain systemic diseases through their oral manifestations. One of such diseases is the gastroesophageal reflux disease (GERD) which may be recognised through its extraesophageal manifestation in the form of dental erosions (Howeden, 1971; Myllarniemi & Saario, 1985; Jarvinen et al., 1988; Bartlett & Smith.1996; Lazarchik &Filler, 1997; Lussi, 2006). The exact cause of dental erosion is the gastro-esophago-pharyngeal reflux, also called proximal reflux, which takes place in a minority of patients with GERD. Usually, the gastroesophageal reflux is confined to the lower portion of the esophagus, where it may cause esophagitis.

Erosion comes from the Latin verb erodere, erosi, erosum, meaning gnaw, corrode and it is described as a process of gradual degradation of a surface by an electrolytic or chemical process. In clinical terms, dental erosions are defined as a physical result of pathological, chronic, localised, painless loss of dental hard tissue, the outer surface of which is chemically destroyed by acid or chelates. Acids that come into contact with tooth surfaces and cause these changes are not products of intra-oral bacterial flora (Pintborg, 1970; Eccles, 1982; Imfeld, 1996). Dental erosions are of multifactorial aetiology and each factor has a significant role not only in the formation of a defect but also its prevention. The interaction of all factors may cause a synergistic effect. They are usually described as a surface phenomenon, although the process may enter the subsurface structure (Young & Tenuta, 2011). According to the depth of the lesions they may be divided into surface and deep ones. According to the localisation they may be divided into generalized and localised, while according to pathogenic activities, into on manifesting and latent ones. According to the origin of the acid they may be divided into endogenous, exogenous and idiopathic. Idiopathic erosive changes are those which, based on medical history and objective findings, we are not in the position to define the origins of the erosive agent. Exogenous erosive changes have occurred as a consequence of acidulous reaction on dental hard tissues, when the acid enters the oral cavity from an external environment. Exogenous acids, originally, may be dietary, medicational or environmental (Allan, 1967; Gandara&Truelove, 1999). Endogenous ones develop under the influence of gastric hydrochloric acid. This acid comes to the oral ecosystem by recurrent vomiting, regurgitation or reflux (Imfeld, 1996). Psychosomatic disorders (neurotic vomiting, anorexia nervosa, bulimia) are common causes of regurgitation and vomiting, which are self-induced (Klein&Walsh, 2004). On the other

hand, there are somatic causes. These include pregnancy, alcoholism and the antabuse therapy for alcohol abuse (Robb & Smith, 1990). This group also comprises gastrointestinal disorders such as gastric dysfunctions (Holst&Lange, 1939), chronic constipation (Bargen &Austin, 1937), hiatus hernia (Howeden, 1971), duodenal and peptic ulcer (Allan, 1969) and the gastroesophageal reflux disease (Gregory-Head & Curtis, 1997).

GERD is a usual condition that encompasses 65% of the population of highly developed countries in a certain period of their lives (Lussi, 2006). On average, 7% of patients have daily problems, and 36%, once a month (Nebel, Fornes&Castell, 1976).

GERD was for the first time connected to dental erosions in the case study presented by Howden (Howeden, 1971) and the hypothesis that it might become a diagnostic mark of an earlier acidulous reflux in the mouth cavity was formulated by Myllarniemi and Saario (Myllarniemi & Saario, 1985). Numerous scientific papers, both case studies and epidemiological studies, published in the past thirty-five years, point to GERD as a risk factor in the formation of erosive changes on hard tooth tissue, as well as to the possibility of using this tooth defect as a diagnostic marker of this gastrointestinal disorder (Jarvinen et al., 1988; Bartlett & Smith.1996; Lazarchik &Filler, 1997; Lussi, 2006).

Dental erosions associated with GERD also occur in children until their teenage years, they especially often occur in children with cerebral palsy (Goncalves et al., 2008). In adults, the value of the prevalence of dental erosion in patients diagnosed with the disease observed is in the range of 5-28%, while the prevalence of GERD in patients with erosive changes is in the range of 21-83%. In children, the prevalence of dental erosion in patients diagnosed with the reflux disease ranges from 17-87% (Vahil et al., 2006). The diversity of the percentages of dental erosion among the subjects covers a very wide range due to the non-standardised scales, estimates of examined surfaces, specificity of the population tested and the subjective factors – assessment capabilities of examiners.

The connection between this common medical condition and the erosive changes on teeth is not absolute because not everyone with a diagnosed reflux disease has them (Bartlett, Evans&Smith, 1996). On the other hand, there are those that have no subjective problems, but have changes on a specific location that proves the existence of regurgitation and reflux (Dene, 2002). GERD may be a risk factor for the appearance of dental erosions only if it is in combination with regurgitation (Addy, Embery&Edgar, 2000). The refluxate is composed of gastric acid, a small quantity of undigested food, pepsin, and in cases of duodenogastric reflux may contain bile acid and trypsin. The intensity of erosive changes is determined by the content and the pH value of the regurgitated material, number of regurgitation episodes, by the length of time this content stayed in the mouth, which is in direct connection with the quantity of secreted saliva, its pH value, its buffer capacity and its ionic content. To all the above mentioned we must add the influence of habits in terms of oral hygiene maintenance (Gilmour&Beckelt, 1993).

2. Pathophysiology of change appearance

Erosion is a disorder in which the characters such as: structural characteristics of teeth, physiological characteristics of saliva and dental pellicle, characteristics of acids and habits act as very important factors in their development and, therefore, must be carefully

analysed. The seriousness of erosive changes is determined by the sensitivity of dental tissue to dissolution. Enamel is mineralised with less soluble minerals than dentine, therefore its surface is eroded more slowly (Lussi et al., 2011).

Minerals, protein, lipids and water are the basic constituents of the hard dental tissue. They are of similar chemical composition and different morphology. Table 1.

COMPONENTS	VOL % enamel	WEIGHT % enamel	VOL % dentine	WEIGHT % dentine
Carbonate hydroxyapatite	**85**	**96**	**47**	**70**
water	**12**	**3**	**20**	**12**
Proteins and lipids	**3**	**1**	**33**	**18**

Table 1. Chemical composition of enamel and dentine in per cents

2.1 Enamel

Enamel represents the hardest tissue in the human organism. Regardless of the high percentage of mineral phases in its structure in the form of hydroxyapatites, enamel is semi-permeable. It is the consequence of the organic matrix that forms the interprismatic and intercristaline coats which are morphologically defined as enamel pores (Roberson, Heymann&Swift,2002). Semi-permeability is the consequence of both the existence of enamel cavities and not adequately mineralised enamel prisms that form the defects. This fine net of macro and micro pores enables the process of enamel diffusion. Water passes with diluted ions and small molecules through the organic part between enamel crystals and establishes a fluid flow which is directly dependent on the tooth morphology and the age of the patient. Permeability decreases with age because organic channels are sealed by deposition of crystals as well as by the formation of a biofilm on its outer surface (Vulović, 2005)

The mineral phase is presented by hydroxyapatite Ca_{10} $(PO_4)_6$ $(OH)_2$. Pure, natural hydroxyapatite is a very stable and poorly soluble compound. However, apatite of the human enamel is not absolutely pure, it contains about 2-4% of carbonates and 1% of other chemical elements, therefore this apatite is called carbonated. Its solubility is greater than that of pure hydroxyapatite. Certain calcium ions may be substituted by other metal ions, such as sodium, magnesium and potassium. Certain OH ions may be substituted by fluoride ions when fluorapatite is created. The formula of pure fluorapatite is Ca_{10} $(PO_4)_6$ F_2. Fluorides affect hardness, chemical reactivity and the stability of apatite crystals. This type of hydroxyapatites is a less soluble mineral. Phosphate ions are replaced by carbonate ones, but not in a one to one ratio, therefore, the formula of such hydroxyapatite is Ca_{10-x} Na_x $(PO_4)_{6-y}(CO_3)_z$ $(OH)_2$. The sensitivity of carbonated apatite depends on the orientation of crystals. The presence of various "impurities", especially carbonates and magnesium in the crystal petals of hydroxyapatite crystals and it increases its solubility, while the presence of fluoride, strontium and other elements are stabilise enamel (Roberson, Heymann&Swift, 2002; Vulović, 2005) The

density and hardness of the enamel tissue decreases with the increase of the distance from the tooth surface (He & Swain, 2009) and solubility increases (Theuns et al., 1986).

2.2 Dentine

Dentine differs from enamel not only by its morphology but also by its chemical structure. The mineral composition is much smaller while the organic one is much greater than in enamel Table 1. The organic component is represented by type I collagen and the non-collagen protein component is represented by phosphoproteins, glycoprotein, proteoglycanes (Lussi et al., 2011). In terms of weight 1% goes to lipids (Odutuga & Prout, 1974). The mineral phase is here also represented by hydroxyapatite crystals, but these are much smaller. The percentage of carbonate is greater in dentine than in enamel (Lussi, 2006) therefore it is sensitive to acidulous solutions. The mineral composition of dentine grows with age.

Acid as an erosive agent, in order to demineralise the crystals on the tooth surface, must be in direct contact with the tooth substance and this is only possible if it gets through plaque or the dental pellicle and by passing through the protein lipid layers of prisms and crystals through processes of diffusion, it reaches the single crystals.

2.3 Dental pellicle

Dental pellicle represents a cellular organic material from saliva which is deposited on the surfaces of the clinical crown (Eisenburger et al., 2001). The pellicle is formed a couple of seconds after exposure of the tooth surface to the oral environment, by sedimentation of salivary proteins and glycoprotein's and contains lipids and some enzymes. It continuously regenerates during the life cycle of a tooth. It is believed that the function of pellicle is: the protection of enamel as a terminal tissue, reduction of friction between teeth and providing the matrix for the re-mineralization of the enamel surface. The build-up of the pellicle from salivary proteins enables the third function because the some components have a base group through which they absorb phosphate ions, and others, which have acidulous proteins, absorb calcium ions. The composition, thickness and maturation time affect the protective properties of the pellicle. This membrane demonstrates continuously selective potential (semi-permeable membrane) and they suppressively influence the diffusion of acids and thereby reduce the dissolution rate of hydroxyapatite (Hanning, Hanning&Attin, 2005). In situ studies have shown that the thinnest pellicle is formed on the palatal surface of the upper front teeth (0.3-0.38 micrometers) and the thickest on the lingual surfaces (0.96-1.06 micrometers) after one hour of intraoral exposure (Amaechi et al., 1999).Tooth surfaces with the thickest pellicular formations show the lowest percentage of erosive changes 1.7 -2% (Young & Khan,2002)

2.4 Saliva

Saliva is considered the most important biological factor in the prevention of erosive changes by both its indirect, the formation of dental pellicle, and direct effects. It affects directly the dissolution, elimination, neutralization of acidulous compounds, and, at the same time, reduces the level of demineralisation and remineralisation with its ionic composition of calcium, phosphate and fluoride.

Saliva is a complex secretion of three pairs of major salivary glands and numerous minor mucous glands. The total average amount of saliva secreted varies within 0.5 to 1 l per day (according to some authors even up to 1.5 l per day). The total quantity of saliva depends on the individual characteristics of each person, as well as on the type, length and intensity of stimuli. It can be non-stimulated and stimulated.

Un-stimulated saliva represents a mixture of secretions of the parotid, sub-mandibular, sublingual and other minor mucosa glands. It also contains gingival cervical liquid, desquamated epithelial cells, bacteria, viruses, leucocytes, food residues and blood. The average quantity of secrete saliva is 0.3 ml per minute, while the individual column may range between 0.01 -1.9 ml per minute. Un-stimulated saliva of subject with reflux disease has a significantly lower pH and calcium concentration. Phosphate and urea concentrations were lower but not statistically significantly lower (Stojšin, 2009).

Stimulated saliva is a secretion product of the parotid salivary gland as a direct response to a stimulus and enables physiological functions during the periods of intensified activities as well as protection of the oral tissue integrity. The quantity of this kind of saliva secretion varies from 0.5 – 7.0 millilitres per minute (Vulović, 2005).

Saliva contains water (99 %) and the rest (1%) comprises organic molecules (proteins, glycoproteins and lipids), small organic molecules (glucose and urea) and electrolytes (sodium, calcium, chlorine and phosphorus).

The acidity of the oral environment directly depends on the level of acidic products, speed of elimination and the ability to neutralise acids. The speed of eliminating acids is conditioned by the speed of salivation and the quantity of saliva. At lower speed of saliva pH synthesis it may be 5.3, and at higher speed of synthesis in the parotid glands it may be up to 7.8 (Anđić,2000).

Saliva represents an ionic reservoir of chemical elements which compose hydroxyapatite. It is oversaturated by ions of calcium, phosphates and hydroxyl ions, which enable the remineralisation of teeth, but also disable the dissolution of tooth tissue in saliva with pH values below 7 and down to the critical value. The picture clearly shows normal morphological characteristics of enamel, the enamel dentine border and dentine after one hour exposure to artificial saliva with pH 7. Oversaturation with ions that compose hydroxyapatite is present even in the extracellular liquid phase of dental plaque which is in direct contact with the tooth surface. Such state of the saliva decreases only when the pH value in the plaque drops low enough so that the concentration of hydroxyl and phosphate ions reduces below the critical value by binding phosphate ions PO_4 in HPO_4(Anđić, 2000).

Numerous salivary proteins (staterin, acidic proteins rich with prolines and many phosphoproteins) help remineralisation of the sub-surface lesion of the enamel. These proteins are capable of binding calcium and preventing calcium salts from precipitation in an oversaturated solution such as saliva and plaque. With the increase of saliva secretion, staterin secretion increases as well.

The chemical protection of saliva is reflected in maintaining certain acidity in the mouth. The control of the optimal acidity in the mouth is based on physical effects (dilution and rinsing) and on the buffering abilities of the saliva. The buffering system consists of bicarbonates, phosphates, proteins and urea (Edgar, 1992; Edgar & O'Mullane, 1996).

3. Chemical aspects of demineralisation

The basic cause of enamel demineralisation is the existence of the critical pH value, which, in case of enamel, is 5.5. The critical pH value is the value when the solution (saliva or the liquid component of plaque) is saturated with relevant mineral particles that enamel is composed of. If the pH value of the solution is above the critical value, the solution is oversaturated and it causes precipitation. If the pH value of the solution is below 5.5, the solution us not saturated and it causes demineralisation. In people with a low concentration of calcium and phosphate in the saliva and in the plaque liquid, the critical pH value may be even 6.5(Daves, 2003).

Hydroxyapatite dissolves because there are products of solubility marked by Ksp (*product of solubility*) and it is $[Ca]^{10}[PO_4]^6[OH]^2$. The value within the brackets shows the effective concentration, i.e. the activity of component ions. The product of concentration of component ions is labelled with mol/L and for hydroxypatite it is 10^{-117}. Ksp is a constant concentration for each component individually. In any kind of liquid, saliva, liquid component of plaque, a refreshment beverage, gastric juice and hydroxyapatite dissolves into its *ionic products* (Ip)[1].

$$Ca_{1o}\,(PO4)_6(OH)_2 \_\_\_\_ 10\,Ca^{2+} + 6\,PO_4^{3-} + 2\,OH^- \quad (1)$$

If Ip = Ksp, then the solution is saturated with elements constituting hydroxyapatite and there is a balance between the concentration of ions and the concentration of products. If Ip less than Ksp, the solution is not saturated and demineralisation takes place, and if Ip is larger than Ksp, the solution is oversaturated and remineralisation or precipitation take place. There are two basic reasons for dissolution of enamel in acids. Hydrogen ions of acid react with the ionic product (OH) and water is created. By disrupting the concentration of products of hydroxyapatite the stability of the concentration of products is also disrupted and demineralisation takes place. The other reasons are inorganic phosphates that appear in saliva and the liquid component of plaque in four different forms, such as H_3PO_4, $H_2PO_4^-$, HPO^{2-} and PO43-. Their proportion directly depends on the pH values of the environment. Low pH value causes low values of PO_4^{3-} which influences Ip of hydroxyapatite. As soon as Ip < Ksp, demineralisation takes place (Daves, 2003; Lussi, 2006)

The chemical process of development of the erosive process is complex. Hydrochloride acid of the regurgitated stomach content reduces the pH value within the mouth. It undergoes electrolytic dissociation in a water environment of the oral ecosystem. The increased concentration of hydrogen ions in the saliva contributes to ionic exchange between saliva and the pellicular or plaque liquid. Disharmony in the ionic concentration starts a chemical reaction[2]

$$Ca_{10-x}Na_x\,(PO4)_{6-y}\,(CO_3)_z\,(OH)_{2-u}\,F_u + 3H^+ \_\_\_\_\_$$

$$(10-x)\,Ca^{2+} + x\,Na^+ + (6-y)(HPO_4^{2-}) + z\,(HCO_3^-) + H_2O + u\,F^- \quad (2)$$

Hydrogen ions directly react with the mineral component of tough tooth tissue; it dissolves them, reacts with carbonate ions and phosphates as the chemical equation shows. Ions of chlorine have no effect in the process of demineralisation.

The non-ionised form of acid passes through the interprismatic area and dissolves the minerals under the surface layer. This causes the calcium and phosphate ions to mobilise and consequently, the pH value rises within the salivary pellicle, i.e. the saliva on the contact surface (Featherstone & Rodgers, 1981; Ganss, Klimek & Starck, 2004). The process halts if there is no new inflow of acid. The next regulating phase or sipping sour beverages or transfer of liquid from side of the mouth to the other again lowers the pH value and a new demineralisation cycle takes place.

Identical processes take place in the dentine as well; they are just much more complex because of the larger quantity of organic matter in this tissue. Structural characteristics of dentine influence the possible penetration of hydrogen ions, demineralisation and evacuation of elements formed during demineralisation (Kleter et a., 1994)

Erosive changes may appear on primary teeth as well. The mineral content of deciduous teeth enamel is lower than in permanent ones. In situ, the enamel of primary teeth is much more sensitive to acidic influence than the enamel of permanent teeth (Johanson et al., 2001). while the dentine of milk teeth is less sensitive to acidic influence than in case of permanent teeth (Hunter et al., 2000).

Hipersalivation which is a reflex occurring before vomiting represents the response of the vomiting centre in the brain (Feldman, Scharschmidt & Sleisenger, 1998) which significantly reduces the process of erosion. Such reactions may be noticed in eating disorders, rumination and chronic alcoholism. Patients with gastroesophageal reflux disease cannot expect the protective effect of hypersalivation before the episode of reflux because the reflux of the gastric juice is an involuntary event and therefore there is no coordination with the autonomous nervous system (Lussi, 2006). From the aspect of dental erosions, the daily rhythm of salivation is especially significant, according to which saliva secretion practically cease from midnight to six o'clock in the morning and then there is a spontaneous increase until 6 p.m., when non-stimulated salivation reaches its maximum, and then it goes on to decrease until cessation at midnight (Anđić, 2000). Night regurgitations episodes are especially important risk factors for the occurrence of erosive changes because there is no protective effect of saliva.

Erosive demineralisation of the tooth crown is characterised by initial softening of the enamel surface in a nanoscopic scale which, in the course of time, grows into microscopically observable morphological changes, which through prolonged exposition lead to macroscopic defects. The erosive defect is determined by the depth of the cavities and the thickness of the demineralised substrate. The level of demineralisation is determined by the immersion time and acid. The thickness of the initial demineralisation ranges between 0.2 and 3 micrometers (Amaechi & Higham, 2001; Lussi et al., 2011). Partial loss of superficial minerals affects the reduction of the superficial hardness, which makes enamel vulnerable against physical forces. Cheeks, tongue, abrasive food, tooth brushes, as well as ultrasound processing of dental tissue may lead to the elimination of the demineralised organic filling of hard dental tissues (Eisenburger, Shellis &Addy, 2004). Enamel remains sensitive to abrasive forces even one hour after having been exposed to acid (Lussi et al., 2011).

4. Pathohystology of erosive changes

The specific morphology of hard dental tissues affects the formation of the characteristic pathohystological images of erosive changes.

4.1 Enamel

The basic structural unit of enamel is enamel prism with its crystals of apatite which show signs of inclination towards the edge of the prism. Enamel prisms are in strings lining from the enamel-dentine border towards the surface of the tooth. In the outer-most surface layers as well as in the region of the enamel-dentine border the presence of aprismatic enamel can be observed. Enamel prisms from the neighbouring lines are linked because their shape resembles a keyhole. They tend to gather into groups. They are radially placed around the tooth axis, which can clearly be seen in the photographs made by an electronic microscope Figure 1. Each prism consists of a head and a tail. The head of one prism is joined with two tails of the two neighbouring prisms and vice versa (Roberson,Heymann&Swift, 2002.) Each enamel prism has its coating and it represents the entrance of the aggressive noxious, so the process of demineralisation goes from the outer surface of the prism towards the central part and the emerging defects remind of honeycombs (Meurman & Frank, 1991), which can clearly be seen in the central part Figure 2. Ultra-structural examinations (scanning electron

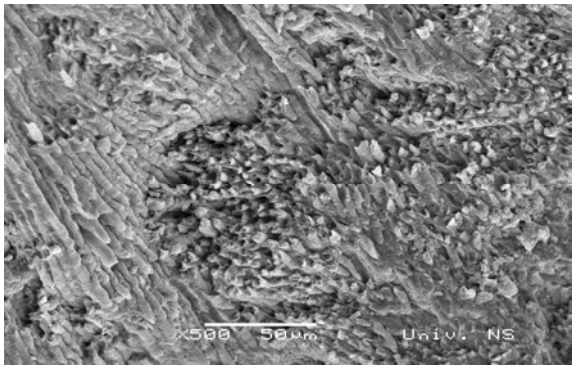

Fig. 1. SEM photographs of tooth enamel enlarged 500 x. Enamel prisms are in strings lining from the enamel-dentine border towards the surface of the tooth. Enamel prisms are grouped in bundles.

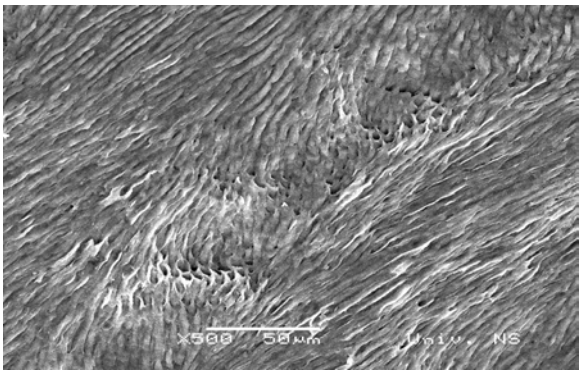

Fig. 2. SEM microphotograph of enamel after 15 minutes of exposure to the solution of HCL pH 1.Enlarged 500 x. The cross section of enamel prisms resembling honeycomb. It is recognizable rounded lines of fracture.

microscopy - SEM, atomic force microscope - AFM) have shown that demineralisation causes changes both in the prismatic and the aprismatic enamel, both on prisms and in the interprismatic region (Amaechi &Higham, 2001; Meurman & ten Cate, 1996). In the aprismatic enamel, demineralisation is irregular and zones of changed mineral content in different places are formed.

4.2 Dentine

Dentine makes the largest part of the dental tissue. The basic structural unit is a dentine tubule. The system of dentine tubules starts at the enamel-dentine border in the form of very thin branches, then they change into broader little channels and in the region of pulp they end as wider tubules. Between the numerous tubules, there is the intertubular dentine. Within the tubules themselves, there is a stem of dentine productive cells – odontoblast, the activity of which produces a new type of dentine called peritubular dentine (Roberson, Heymann & Swift, 2002) Figure 3.

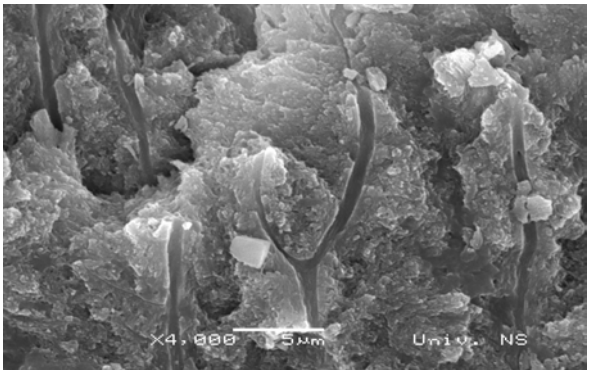

Fig. 3. SEM photographs of tooth dentine enlarged 4000x. Present dentine tubules are lined with peritubular dentin. Between the tubules is intertubular dentin.

When dentine is exposed to acid, the first signs of demineralisation are observable at the border of the peritubular and intertubular dentine, after which loss of peritubular dentine follows and widening of the lumen of dentine tubules and eventually a superficial layer of demineralised collagen matrix is, formed (Schlueter et al., 2011). In the initial period, this layer of collagen matrix protects the down lying tissue from further demineralisation, but it is very sensitive to the effects of mechanical forces and proteolytic enzymes and is fast eliminated and dentine tubes become exposed. Continuous exposure to acids causes' reduction of the demineralisation rate and at a certain thickness, the mineral loss is much less (Lussi et al., 2011), which may be explained by the buffering characteristic of collagen. The organic matrix may be degraded by specific and non-specific proteolytic enzymes (Schluter et al., 2010). Figure four depicts in the scanned electronic microphotograph the image of dentine at a longitudinal crosscut, exposed to the effects of pure hydrochloric acid pH 1 within 15 minutes. After only fifteen minutes the edges of the breakage become rounded and slowly the image of normal morphology is lost. Figure 4.

Scanning electronic microphotography (SEM) of the exposed enamel, the enamel-dentine border and dentine in artificial saliva during a period of one hour shows no changes of

dental structure and clearly defined morphological characteristics, however, if we submerge the sample of dental tissue into a centrifugal filtrate of stomach content within the same time interval, then the microphotograph is completely different Figure 5. Intensive erosions of the exposed surfaces with loss morphological characteristics prove the aggressive effect of stomach content on hard dental tissue (Stojšin, 2009).

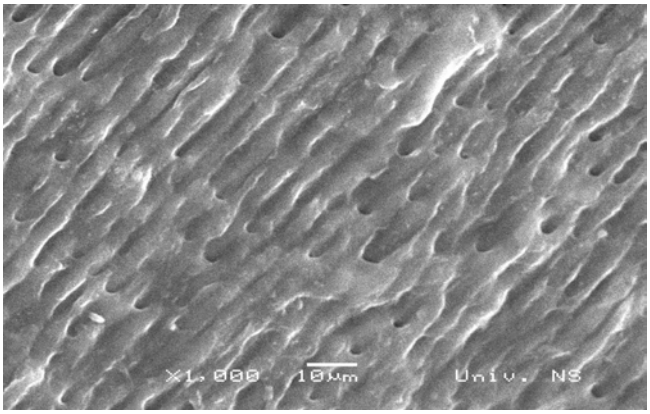

Fig. 4. SEM of dentine exposed to HCL pH1 for 15 min. After only fifteen minutes the edges of the breakage become rounded and the extent of dentinal tubules entrance.

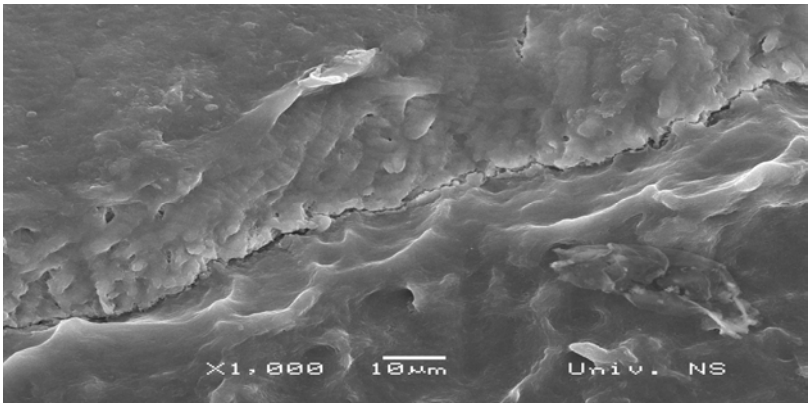

Fig. 5. SEM photograph of enamel, enamel-dentine border and dentine after one-hour exposure to centrifugal filtrate of stomach content. Clearly visible histo-morphological total loss characteristics observed tissue. Rounded bearing ameloblasts in the dentine.

Continuous erosive demineralisation with loss of hard dental tissue and exposure of dentine affects the activation of protective activities of pulp cells – of odontoblast and the synthesis of reactive and reparatory dentine which causes obturation of dental tubules themselves. It is a biological compensatory response. If the process continues the reparatory capacities wear off, pulp cavum exposes, inflammatory processes on the pulp disuse develop as well

as necrosis and periapical pathology. Chronic inflammatory processes in the perapical region may be focal points with consequences to overall health.

5. Clinical assessment

Diagnosing dental erosions is difficult because there is not a single method or a procedure that would indicate early detection and quantification of these changes. In the early stadium, the changed surface of the enamel is smooth, shiny, without macroscopic defects (Amaechi & Higham, 2005). Sometimes it may be dull and without emphasised coloured lines as well as clearly established borders towards the changed part of the dental tissue.

In patients with the reflux disease, the defects are observable on the palatal surface of upper anterior teeth. The palatal surface becomes smooth, shiny and hard and the vestibule-oral diameter shrinks. The incisal edge seems thinned, translucent and slashed. In the gingival region existence of enamel collar may be detected. With loss of enamel, the tooth becomes yellowish, because of the bare dentine. The oral surfaces of the upper premolar and molar lose their morphology. Palatal lumps are becoming rounded or in their places cuplike dents occur and in advanced stadium a steep plane is formed, i.e. a complete loss of morphology Figure 6.

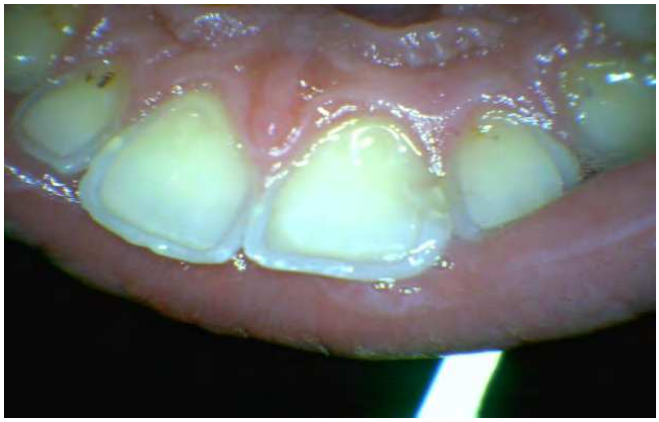

Fig. 6. Palatal surfaces of upper anterior teeth. The surface becomes smooth, shiny and hard and the vestibule-oral diameter shrinks. The incisal edge seems thinned, translucent and slashed

If the defect is localised on the lower anterior teeth, the incisal edge becomes a surface and later a groove is formed with its bottom in the dentine Figure 7.

The changes are much more often observable on the occlusal and vestibular surfaces of lower side teeth than in the anterior ones, because the lower anterior teeth bathe in excretions of the sublingual and submandibular saliva gland. The dorsum of the tongue directs the regurgitated content into the side region of the mandible, therefore, on the occlusal surfaces, the morphology is lost and cuplike defects are formed. On the vestibular surfaces, the changes are manifested through wide concavities. If the changes appear on the vestibular surfaces of the bottom anterior teeth then the direction of flow of the regurgitated content may clearly be seen Figure 8.

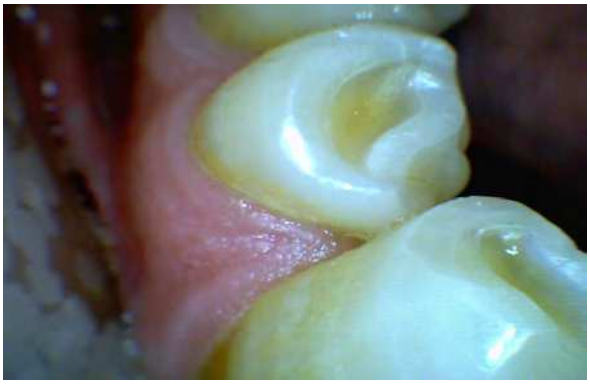

Fig. 7. Incisal surface of the lower anterior teeth with a groove in the dentine

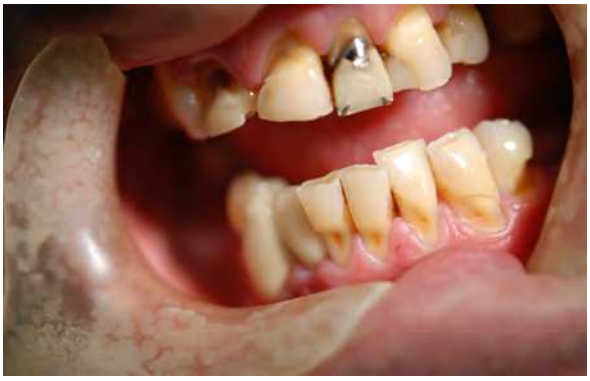

Fig. 8. Vestibular surfaces of the bottom anterior teeth. The gingival part of the vestibular surfaces of teeth has the defects that are wider than deep.

Amalgam or composite fillings on such teeth seem as grown and are located above the tooth structure Figure 9.

Fig. 9. Definitive fillings have "grown" from the dental tissue

Loss of enamel leads to the opening of dentine tubules and a consecutive phenomenon of dentine hypersensitivity as one of the symptoms. The pain is manifested as a shooting sensation to physical, chemical, thermal and evaporative triggers. Exogenous erosions are characterised by spherical defects on the occlusal surfaces. The vestibular surfaces may have defects of different shapes with the basic characteristic that they are rather wide than deep, but in all above mentioned shapes, there is loss of dental tissue (Lussi, 2006). The synergic effect of endogenous and exogenous agents brings about generalised changes that disturb the function of the masticatory apparatus.

It is even more difficult to diagnose erosive changes on primary teeth (Show &Sullivan, 2000). Enamel and dentin are thinner, less mineralised and more porous, so the aggressive effect of acid is even more expressed. The defects are smooth, shiny and rather wide than deep. In children, the changes are most often localised on the occlusal surfaces of molars and the incision edges of anterior teeth, which leads to loss of morphology, dentine hypersensitivity as well as complete loss of the crown of the tooth. All this leads to pulp inflammation and premature extraction of a milk tooth with all its consequences. When it is about defects on primary teeth they always have to be observed from the aspect of cumulative multifactoriality. Attrition of incisal edges in deciduous dentition is frequent during exfoliation and it is very difficult to assess then what the cause of the change is (Lussi, 2006).

In order to diagnose dental erosion, we need a thorough anamnesis, objective examination, analysis and assessment. Therefore a good questionnaire is needed with precisely defined questions which would enable easy diagnostics of the etiological factors as well as saliva analysis (determining the daily quantity of saliva, ph value, quantity of calcium and phosphate, the buffer capacity).

After an established diagnosis, it is necessary to follow the progress dynamics and a silicon index is used for this purpose, as well as the index of dental erosions, study models by Wicken and photographs (Daves, 2003).

Erosive effects of acids are only one of the mechanisms for the occurrence of dental defects. Numerous indices can be found in literature and they are mainly modifications of indices suggested by Eccles, Smith and Knight. The indices often used are also those suggested by the British Children's National Health, National Diet and Nutrition Surveys as well as the index suggested by Lussi (Lussi, 2006). All of them include diagnostic criteria for differentiating erosive changes from other forms of dental defects and criteria for the qualification of hard dental tissue loss.

6. Differential diagnosis

Erosions as causes of dental tissue loss are part of a much broader picture of dental defects, such as attrition, abrasion and abfraction.

Attrition is a defect of both dental tissue and restoration, and is caused by tooth to tooth contact during mastication or para-functions. Occlusal surfaces are smooth, shiny, evened and hard and on amalgam fillings facets are observable. The bottom of the defect may be located both in enamel and in dentine (Gandara&Truelove, 1999).

Abrasions occur with direct contact between the tooth and an alien substance (tooth whitening paste, anti-nicotine, soda...). The changes are usually localised in the cervical region of premolars and molars, always rather wide that deep (Roberson, Heymann & Swift, 2002).

Abfraction is a defect which is characterised by loss of dental tissue in the cervical region. It is caused by compression and stretching forces which take place during dental flexure. At inadequate occlusal relation, the changes are localised mainly vestibularly and they are of a wedged shape (Attin et al., 2004).

7. Prevention of erosive changes

The first step in every prevention is the identification of the etiological factor and its elimination. Many general medical diseases and conditions have repercussions in the mouth, both on soft tissue as well as on teeth. Therefore, an adequate therapy requires cooperation between experts of different specialties – gastroenterologists, oncologists, psychiatrists and dentists. Good prevention should include pharmacists who must, when issuing medication to patients, indicate the side effects of certain medications such as xerostomia (antihistamines, antidepressants, appetite suppressors ...). Some drugs directly express a high degree of erosiveness (chewing vitamin C, acetylsalylic acids ...) and it is necessary to inform patients how to minimize this effect (rinsing with re-mineralising agents) (Amaechi & Higham, 2005; Toumba, 2001).

If, after taken anamnesis, it is confirmed that the acid source is of exogenous source, education and consultation take a dominant role along with long-term examination of the health status of hard dental tissue (Gandara&Truelove, 1999).

All prevention measures may be divided into:

7.1 Measures that regulate the frequency of inflow and quantity of aggressive noxious factors

The first preventive measure is the regulation of regurgitation and reflux and that is the duty of physicians and specialists in gastroenterology. The proton pump inhibitors (PPI) represent the most effective therapy for gastroesophageal reflux. The dietetic measures include the reduction in the amount of intake of food and drink that are known to have erosive potential, change in the manner of their intake, especially beverages, is an important preventive factor for avoiding cumulative effects of acid of endogenous and exogenous origin in patients diagnosed with the reflux disease. It is better to consume the beverages through a straw and swallow them right away rather than shake them in the mouth (Gandara&Truelove, 1999). It is a known fact that cold beverages have much less erosive effect than beverages at room temperature (Amaechi & Higham, 2001). Dentists may suggest increased consumption of milk, cheese, almond in order to neutralize acidity in the mouth (Gedalia et al., 1992) as well as rinsing the mouth with soda solution. However, the aforementioned dietetic measures have no sense without the appropriate pharmacological therapy with proton pump inhibitors (PPI) and that basically, without it; the war against dental erosions is lost in advance.

7.2 Measures of enhancing the defence mechanism (salivary flow and pellicular formation)

This measure implies establishing hyper-salivation in the mouth which would intensify the protective characteristics of saliva. Consuming pastilles without sugar initiates salivation. A

significant effect may be achieved by rinsing the mouth with artificial saliva in order to eliminate potential causes. Chewing gums, which are regularly prescribed to patients with exogenous erosions, are not advisable for patients with the reflux disease because of the effects on gastric secretion (Deshpande & Hugar, 2004). With expressed xerostomia it is necessary to prescribe pilocarpine –Salargan (Gandara&Truelove, 1999).

7.3 Measures of enhancing resistance and remineralisation of hard dental tissue

Increase of resistance and remineralisation of hard dental tissue may be achieved by preparations based on fluorine in the form of 2% solution of sodium fluoride and fluoride pastilles, jellies and lacquer.

Pastilles have the most positive effect because on the one hand they contain fluorides, and on the other they cause hyper-salivation (Jarvinen, Rytoma & Heinonen 1992; Stojšin, 2006). In all patients with observable changes on hard dental tissue, the application of fluorine in dental practice twice a year is necessary.

7.4 Measures of achieving mechanical protection

In patients with an evident gastroesophageal reflux it is advisable that they wear overnight occlusal protectors with applied fluorine preparations (Gandara & Truelove, 1999; West, 2011).

7.5 Measures of decreasing the effects of abrasive forces

The softened hard dental tissue is susceptible to the effects of aggressive tooth brushes and abrasive tooth pastes. Inadequate application of oral hygiene maintenance agents as well as the technique cause increase in the incidence and prevalence of other types of non carious dental tissue defects. That is why it is necessary to engage dentists more intensely in the dental health education. After waking up, the mouth should be first rinsed and then, after 30-60 minutes, the teeth should be cleaned. People with the reflux disease should use a soft toothbrush and non-abrasive pastes and brushing movements should be moderate and not too rough, whilst retaining the Bass methodology of brushing the teeth. Low pH value tooth pastes should be avoided.

8. Therapy

The detection of erosive changes either by patients or by a dentist is not easy. The patient turns for help only when it comes to short sharp pain sensations to thermal, evaporative, tactile, osmotic stimuli or with the occurrence of major defects that disrupt the aesthetics and function. General indications for restorative treatment are:

- Presence of dentine hypersensitivity
- Bad aesthetics
- Loss of vertical dimension of occlusion
- Endangered pulp – dentine complex
- Necessary rehabilitation of toothless areas.

The choice of treatment in dentine hypersensitivity depends on the type of patient's personality, history of the disease and an objective diagnosis (Amaechi & Higham, 2001).

There are two basic principles of medication effects and they are closure of dentinal tubules, or desensitisation of teeth. Desensitisation is achieved by preparations that contain potassium nitrate. The greatest success may be achieved by a 15% solution, 10% gel or toothpastes containing 10% potassium nitrate (Colgate R). Desensitization is achieved by the use of low-energy lasers (Kargul & Bakkal, 2009).

The therapeutic procedure in which the obliteration of dentinal tubules is emphasised may go in two directions. The agents that mimic the natural processes are fluorine preparations (sodium fluoride, sodium monofluorophosphate, tin fluoride) strontium chloride, calcium hydroxide and oxalates. The second group comprises the agents and procedures for closing the dentinal tubules and are represented by materials that mechanically or chemically bind to tooth surfaces, which are primers Copalit, Duraphat), adhesives (Gluma adhesive, Micro prima) and finally, composites and glass ionomer used in the case of dental defects of more extensive dental defects (Jarvinen et al., 1988; Živković, 1998).

The reconstruction of defects localised on non-occlusive surfaces is not problematic. If the bottom of the defect is localised in the enamel, the use of composites with micro-fillers is recommended with previous acid treatment of enamel. In cases where the bottom of the lesions is localised in the dentin or event the cement has been affected, it is necessary to use of dentin bonding systems in combination with composites or glass ionomers. For deeper defects, a layered technique is used with the use of adhesive systems, glass ionomer cement and the last generation of composites, compomers (Kargul & Bakkal, 2009; Jarvinen et al., 1988). Of all the composites, it would be the best to use a Nano DCPA – Whisker composite, which is capable of emitting calcium and phosphate ions [DCPA – Dicalcium Phosphate Anhydrate] (Show & Sullivan, 2000). The defects may be reconstructed also with ceramic facets, but more rarely because they are considered to be sensitive to acidic fluoride gels (Kargul & Bakkal, 2009; Mahoney & Kilpatrick, 2003).

The reconstruction of erosive defects on palatal surfaces of anterior teeth may be achieved with facets where the metal base is made of nickel-chrome alloy or gold. Preparation of the dental tissue is minimal and consists only of evening the enamel collar in the gingival third of the palatal surface of the eroded tooth and roughens the contact surface. In patients with the defected vertical dimension of the occlusion, first the Dahl's apparatus is placed in order to intrude an antagonist or a facet is constructed which is higher than necessary (Lussi et al., 1991). The binding mass for the facet may be resin or glass ionomer cement. Extensive defects of the palatal surface of the tooth may be reconstructed with purely metal facets. Vestibular defects and defects of the incisal edge may be reconstructed with non-metal ceramic facets or composites, but here, we must note that the same effect may be achieved with classical crowns.

The erosive effects in the side region in most cases are localised on the occlusal surfaces in the form of cuplike cavities. The deformed occlusal surfaces may be reconstructed with composites with micro-fillers or gold onlays with minimal tooth preparation and in absolutely dry working area. Conventional glass ionomer cements are not suggested because they have low resistance to wear and sensitivity in acidic environments. In cases when the defects are more extensive and encompass both occlusal and proximal surfaces of the tooth or they extend as far as cement, a good reconstruction is established with ceramic or conventional metallic crowns. In cases of generalised erosion of the side region, the reconstruction of the eroded dental tissue may be achieved with adhesive or ceramic onlays,

metal, metal-ceramic crowns or non-metal ceramics with or without extension of the clinical crown (Lussi et al., 1991).

9. Conclusion

Dental erosion has to be recognized as extra esophageal manifestation of gastroesophageal reflux disease. It can result in tooth sensitivity, poor esthetic, loos of occlusal vertical dimension and functional problems. Clinicians must have thorough understanding of the causes of dental erosion as identification of the cause is the first step in its management. The inspection of the oral cavity in search for dental erosion should become a routine maneuver in patients who have GERD.

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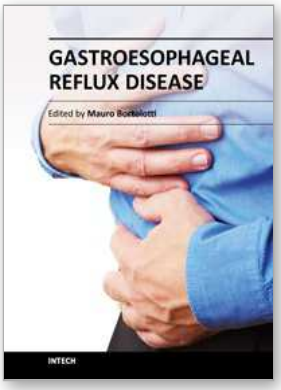

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Gastroesophageal reflux disease affects many patients. This disease not only lowers their quality of life, but it also threatens some of them with an underhand risk of cancer. Additionally, it becomes an economic burden for the patients and society. The aim of this book on gastroesophageal reflux disease is to provide advice and guidance to gastroenterologists to help them understand and manage some aspects of this proteiform disease.

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