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CLINICAL MANIFESTATION AND INCIDENCE OF DENTAL ENAMEL EROSION IN PATIENTS UNDER ANTIHYPERTENSIVE THERAPY

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Abstract: Introduction: Dental erosions are defined as an irreversible loss of hard dental tissue caused by long-lasting and repetitive acid action, which dissolves the surface layer of hydroxyapatite crystal structure and fluorapatite, and the aggressive noxis not being created by the bacteria. Dental erosions can also be classified as occupational diseases. People who taste wine or carbonated drinks on a daily basis, as well as the people who are professionally engaged in swimming can spot this type of defect on their own teeth. Evaporation of industrial acids in battery factories, sanitary materials, or crystalline glass can also lead to dental erosions.

Aim: The purpose of this study was to determine the frequency of dental erosion in patients under antihypertensive therapy and compare them with the frequency of dental erosion in patients who did not take the same therapy.

Material and method: This research involved 62 respondents, aged from 20 to 70. Patients were classified into two groups, the experimental group with 31 patients being on a therapy with antihypertensive medications for more than 5 years, and the other one, control group, with 31 patients not being on the mentioned therapy. The subjective dental anamnesis obtained from the patients was noted along with tooth status, soft tissue intraoral examination and the degree of erosive changes according to BEWE index (Basic Erosive Wear Examination). Results: Comparing the statistical significance in difference between erosive index value of all teeth, there is obvious statistical difference of the average value. In the experimental group ($p<0.05$), higher values of tooth erosion index were noted in comparison with the control group. The average value in the experimental group is 2.25, while in the control group it is 1.37.

Conclusion: Erosive lesions are present in both experimental and control group respondents, with dental erosion being more explicit with patients who use antihypertensive medications.

Keywords: dental enamel, dental erosions, antihypertensive.

1. INTRODUCTION

Dental erosions are defined as the irreversible loss of hard dental tissue caused by long lasting and repetitive effects of acids, which dissolve the surface layer of the hydroxyapatite and fluorapatite crystalline structures, without the aggressive noxa being created by the bacteria [1–3]. The inorganic enamel component is mainly represented by hydroxyapatite and tricalcium hydrophosphate as well as magnesium carbonate, chlorides, fluorides and sulphates. Saliva is a medium which, due to its components, flow rate and antioxidant capacity, greatly contributes to the oral homeostasis. The most common factors causing reduced salivary flow rate (subjective feeling of dry mouth) are adverse oral reactions of drugs, such as antihypertensives [4]. Different sorts of acids lead to saliva's low pH value, such as hydrochloric, ascorbic, citric, phosphoric, lactic but also their different origins gastric juice, ready made preparations with vitamin C, fruit. Dental erosion can also be classified as professional illness. People who test wine or carbonated drinks on a daily basis, as well as people who are professionally engaged in swimming, can notice this type of defect on their teeth [5]. Industrial acid evaporation in battery, sanitary, crystal glass factories also leads to dental erosion [6]. Often, the extrinsic and intrinsic

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factors can be present where there are acids in mouth on the one hand, with another impending factor being deterioration in saliva quality, in the sense of lower pH value (oral motoring reduction, various medication effects, breathing through one's mouth) [7]. The enamel represents the hardest tissue of the human body and forms the outer layer on the anatomical dental crown. The organic component is represented by glycoproteins and proteoglycans [8], i.e. the basic ingredient is a tyrosine-rich amelogenic peptide [9]. The enamel proteins form the network in which the minerals are later deposited [10]. The enamel basic structural component is the enamel prism which consists of hydroxyapatite crystals. Enamel prisms consist of millions of small elongated hydroxyapatite crystals which differ in their shape and size. The aprismatic enamel has a more emphasised mineralisation than the prismatic one due to the lack of boundary borderlines, various crystal orientations and their way of storage [10]. The enamel crystals dissolve more quickly if the acids act parallel to their longitudinal axis rather than if they act at the right angle [7]. The erosive changes in the teeth are directly dependent on the number and duration of acidic effect, not on the acid type or the way it is brought into the oral cavity [11,12]. Dental erosions caused by food are divided into three stages. The first stage- early demineralisation is characterised by enamel softening and structural changes on the nanoscopic scale. The second stage is characterised by microscopic enamel loss and structural changes in solid dental tissues. The final stage is characterised by macroscopic enamel defect which is easily diagnosed by the dentist [8]. In the macroscopic defect, the erosive lesion depth is determined by the defect depth plus the tissue demineralisation depth based on defects [13].

2. PATHOGENETIC MECHANISMS AND PATHOHISTOLOGICAL CHANGES

The chemical procedure for the erosive change development is complex. In order for the solution to touch the enamel, it must first diffuse through the salivary pellicle which is an acellular organic substance from the saliva deposited on the surface of clinical crown [14]. It appears a few seconds after exposing the tooth surface to the oral environment by depositing salivary proteins and glycoproteins. The pellicle shows permanently selective potential (semipermeable membrane) that affects suppressively the acidic agent diffusion and reduces the hydroxyapatite dissolution rate.

Hydrogen-ions in contact with the enamel or the agents possessing the chelating capacity, i.e. the ability to bond with released calcium ions, begin to dissolve hydroxyapatite crystal [15,16]. Citric acid has the ability not only to demineralise, but also to bond released calcium ions. The non-ionised acid form diffuses through the interprismatic space and dissolves minerals beneath the surface layer, which leads to the mobilisation of calcium ions and phosphates and the consequent increase in the pH value of the liquids inside the salivary pellicle on the contact surface [17]. The process stops if there is no new acid inflow or chelating substance. By moving the liquids from one side of the mouth to another or by sipping acid drinks, the demineralising acid activity increases, as their new quantities constantly appear. The identical processes occur also in dentine, and are much more complex due to the higher amount of organic substances that sabotage the hydrogen ion penetration, demineralisation and evacuation of the elements formed by demineralisation [18,19].

3. AIM

The purpose of this study was to determine the frequency of dental erosion in patients on hypertensive therapy and compare it to the frequency of dental erosion in patients who did not take the same therapy.

4. MATERIAL AND METHOD

This research includes 62 respondents, both sexes, ages from 20 to 70. The research includes 31 men and 31 women. The subjective dental anamnesis, obtained from the patient, was recorded and also the objective dental history along with the recorded tooth status, the soft tissue intraoral examination, erosive changes degree according to the BEWE index. They are divided into two groups, where the experimental group includes 31 subjects who have been on antihypertensive drugs for more than five years, and the second group is a control group, and includes 31 subjects, who are not responding to antihypertensive therapy. Respondents of the experimental group reported to the Clinic of Cardiovascular Diseases, where they were kept for the presence of ailments.

The research was approved by the Ethics Committee of the Clinical Center in Banja Luka under the serial number: 01-5-355.2 / 12.

Each of the respondents received a form for information, on the basis of which they volunteered for further cooperation. After the written consent of the respondents for review, a precise anamnesis is taken and an objective examination is carried out. Personal questionnaires, life habits, general medical

anamnesis, which primarily relate to symptoms of high blood pressure and the presence of side effects, an objective finding of the head and neck, are included in the prepared questionnaire. Subsequently, the subjective dental anamnesis was obtained from the subjects and objective dental history with recorded tooth status, intraoral examination of soft tissues, degree of erosive changes according to the BEWE index. In this study, an internationally accepted and standardised index BEWE was used. The vestibular, occlusal-incisal and oral surfaces of all the teeth present in both teeth rows are indexed. The index value by tooth and by jaw was recorded. The total value of BEWE index is based on clinical results which is an early warning of teeth being subject to dental erosion.

The expressions of erosive changes according to the BEWE index are determined as follows:

0 No erosive tooth wear
1 Initial loss surface texture
2* Distinct defect, hard tissue loss<50% of the surface area
3* Hard tissue loss>50% of the surface area

*In grades 2 and 3 dentin is often involved.

5. RESEARCH RESULTS

. The statistical significance of difference in the tooth erosive index of both, the experimental and control groups, were obtained.

The results obtained are shown in Tables 1–3.

Table 1. Statistical significance of the difference in tooth erosive index of the experimental and control groups

t-Test:Two samples assuming unequal variances		
All teeth	*Patients*	*Control group*
Mean value	**1.90625**	**1.3125**
Variance	0.92641129	0.350806452
Number of samples	29	31
Hypothetical mean value	0	
Degrees of freedom	52	
t Stat	2.971981765	
P(T<=t) unilateral	0.002236713	
t Critical unilateral	1.674689154	
P(T<=t) bilateral	**0.004473426**	
t Critical -bilateral	2.006646761	
p < 0,05, so the difference in mean value is statistically significant.		

Table 2: Mean value of the upper jaw erosive index in the experimental and control group

t-Test:Two samples assuming unequal variances		
Upper jaw	*Patients*	*Control group*
Mean value	**2.25**	**1.375**
Variance	1	0.383333333
Number of samples	16	16
Hypothetical mean value difference	0	
Degrees of freedom	25	
t Stat	2.975806057	
P(T<=t) unilateral	0.003199542	
t Critical unilateral	1.708140745	
P(T<=t) bilateral	**0.006399084**	
t Critical- bilateral	2.059538536	
p< 0,05, so the difference in mean value is statistically significant.		

Comparing the statistical significance of the difference in the erosive index value of all teeth, it was shown that there was a statistical significance in mean value. The erosive index mean value in the experimental group is 1.90, and in the control group it is 1.31.

There is difference in the tooth erosive index value in the experimental group ($p<0.05$) in comparison with the control group.

Comparing the statistical significance of the difference in the erosive index value in the upper jaw showed that there is a statistical significance of the

mean value. The erosive index mean value in the experimental group is 2.25, and in the control group it is 1.38.

There is difference in the tooth erosive index value in the experimental group ($p<0.05$) in comparison with the control group.

Table 3. Erosive index mean value in the lower jaw with the experimental and control group

t-Test: Two samples assuming unequal variances		
Lower jaw	*Patients*	*Control group*
Mean value	**1.5625**	**1.25**
Variance	0.6625	0.333333333
Number of samples	16	16
Hypothetical mean value difference	0	
Degrees of freedom	27	
t Stat	1.252612333	
P(T<=t) unilateral	0.110543964	
t Critical unilateral	1.703288423	
P(T<=t) bilateral	**0.221087928**	
t Critical- bilateral	2.051830493	
p> 0,05, so the difference in mean value is not statistically significant.		

Comparing the statistical significance of the difference in the erosive index value in the lower jaw shows that there is no statistical significance of the mean value. The erosive index mean value in the experimental group is 1.56, and in the control group it is 1.25. There is no difference in the tooth erosive index value in the experimental group ($p>0.05$) in comparison with the control group.

6. DISCUSSION

Clinically, it is often very difficult to distinguish whether the loss of a dental substance is caused by erosion, attrition or abrasion, since it can be anamnestically identified the simultaneous existence of the causes of all three phenomena, and even their location can be the same [20]. Therefore, the chemically induced erosion can be regarded as the initial cut into the enamel surface which is later superimposed by mechanically-caused attrition and abrasion, which increases further tooth substance loss. The course of events is experimentally proven and reversed, where the mechanically abraded enamel was much more prone to erosions [21].

For the first time, England has included tooth erosion estimation in its national tooth health survey in 1993, which points to this problem's significance. For more than 20 years, numerous studies have shown dental erosion prevalence. Kreulen et al, Kagawa et al. have shown in their research a greater prevalence of dental erosion with age, as it has been shown in this study, too [22,23]. Non-carious cervical lesions are considered, due to their frequency, another cause of tooth decay- it is estimated that approximately 25% of pathological destruction is non-carious [24,25].

Comparing all teeth, in both upper and lower jaw, statistical significance was found ($p=0.0044$), where the mean value in the group of patients is 0.93, and in the control group it is 0.35. Brkić et al. showed a case study where dental erosion is most emphasised in the upper incisors, canines and premolars [26]. The eroded surface is emphasised on the labial surfaces and incisal edges, which is in accordance with this research where, it is shown that the higher mean index value in the upper jaw is 2.25 in comparison with the lower jaw 1.56 in the experimental group, and also in the control group there is a higher mean erosive index value in the upper jaw 1.38, in comparison with the lower jaw 1.25.

It is important to point out that the great similarity of the surface erosive lesion with the earliest manifestations of caries suggests a similar or the same formation mechanism. Differences occur probably in the later stages of enamel destruction when a surface zone is formed in the caries, and the main part of the demineralisation moves to subarea, while in the case of erosion due to the continuous process of self-cleaning and surface cleaning, and thus the abrasion activation, the demineralisation process extends linearly into depth. The explanation lies in the fact that there is a permanent plaque deposit on the enamel as a source of acid in case of caries. And, in case of erosion, acids occasionally appear on teeth through aerosols, food and beverages. A much more trustworthy explanation is provided by Larsen, and Grobler in their researches [27–29]. Significant gingival damage has also been shown in orthodontic therapy which is explained by corrosion of enamel with most 37% orthophosphoric acid or 10% maleic acid, resulting in demineralisation on a limited surface [30,31]. Surface enamel erosion was found to

occur when the aqueous phase (acid- released ions) is undersaturated in comparison with fluorapatite and hydroxyapatite. When the aqueous phase is undersaturated with hydroxyapatite, and supersaturated with fluorapatite, then a lesion similar to caries occurs, with a sub-demineralisation zone which is covered with a rather well-mineralised surface layer. The demineralisation system in which the aqueous phase is constantly renewed, before the saturation with fluorapatite is achieved, creates lesions similar to erosions, even if the fluorides are present in the aqueous phase [32]. Hyposalivation has also been found as one of the clinical manifestations in hypertensive patients. Reduced salivation is associated with increasing both systolic and diastolic blood pressure, particularly pronounced hyposalivation in patients with diuretic therapy. A marked reduction was demonstrated in unstimulated saliva. The results of this study showed that 16.99% (79) of patients undergoing diuretic drugs showed hyposalivation [33]. Medical problems that cause a reduction in salivary flow can affect the extent of dental erosion. Factors which reduce flow of saliva are salivary gland excision, Sjogrens syndrome, radiation treatment to neck and head region, medications (most commonly are antihypertensives and antiparkinsons drugs) [34].

Many of the medical conditions associated with tooth enamel wear off can have serious consequences if they are not treated or diagnosed in time. Above all, noticing the medical cause of enamel wear off increases the likelihood of preventing the deterioration of the individual's health [35].

7. CONCLUSION

In this paper, it is proved that erosive lesions are present in both experimental and the control group, where dental erosions were more enhanced with the patients who are using antihypertensive drugs. The higher incidence of dental erosion in the upper jaw compared to the lower one is explained by the presence of a higher amount of saliva in the lower teeth compared to the upper one. The enamel protects the soft and sensitive parts inside the tooth, and can resist bites, chewing and other processes in which it participates, if the oral and tooth hygiene is taken into consideration.

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ꕥ

КЛИНИЧКА МАНИФЕСТАЦИЈА ДЕНТАЛНИХ ЕРОЗИЈА КОД ПАЦИЈЕНАТА НА ТЕРАПИЈИ АНТИХИПЕРТЕНЗИВНИМ ЛИЈЕКОВИМА И УЧЕСТАЛОСТ ТРОШЕЊА ГЛЕЂИ

Сажетак: Увод: Денталне ерозије дефинишу се као иреверзибилни губитак тврдог зубног ткива изазван дуготрајним и понављаним дејством киселина, које растварају површински слој кристалне структуре хидроксиапатита и флуорапатита, а да агресивна нокса није створена од стране бактерија. Денталне ерозије могу се сврстати и у категорију професионалних обољења. Особе које свакодневно тестирају вино или газирана пића, као и особе које се професионално баве пливањем, могу на својим зубима уочити овај тип дефеката. Испаравања индустријских киселина у фабрикама акумулатора, санитарних средстава, кристалног стакла такође доводи до појаве денталних ерозија

Циљ: циљ овог рада је да се утврди постоји ли статистичка значајност разлике ерозивног индекса зуба код испитаника контролне групе и испитаника експерименталне групе који су на терапији антихипертензивним лијековима. Материјал и метод: У истраживању је учествовало 62 испитаника старосне доби од 20

до 70 година. Пацијенти су подијељени у двије групе, гдје експериментална група обухвата 31 пацијента који су на терапији антихипертензивним лијековима дуже од пет година, а друга група је контролна група, и обухвата 31 испитаника, у коју спадају испитаници који нису на терапији антихипертензивима. Забиљежена је субјективна дентална анамнеза добијена од пацијента и објективна дентална анамнеза са забиљеженим статусом зуба, интраоралним прегледом меких ткива, степеном ерозивних промјена по BEWE индексу.

Резултати: Поређењем статистичке значајности разлике у вриједности ерозивног индекса свих зуба и зуба у горњој вилици, показало се да постоји статистичка значајност средње вриједности. Код експерименталне групе ($п < 0,05$) уочене су више вриједности ерозивног индекса зуба у односу на контролну групу. Средња вриједност у групи пацијенти износи 2,25, а у контролној групи 1,38.

Закључак: Ерозивне лезије присутне су и код експерименталне групе и код испитаника контролне групе, гдје су денталне ерозије израженије код пацијената који користе антихипертензивне лијекове.

Кључне ријечи: глеђ, денталне ерозије, антихипертензиви.

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