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ANALYSIS OF CROWN/IMPLANT RATIO ON IMPLANTS STABILITY

АНАЛИЗА НА СООДНОСОТ КОРОНКА/ИМПЛАНТ ВРЗ СТАБИЛНОСТА НА ИМПЛАНТИТЕ

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Abstract

In total edentulousness treatment the choice is between complete denture or implant therapy. Implant therapy gives better results in terms of prosthetic stability, patient safety, aesthetics and phonation and prevents alveolar ridge resorption. But implant-prosthetic treatment requires optimally defined indications and conditions. The crown/implant ratio is an important factor in the implant treatment success and its prophylactic effect on the bone tissue. From that aspect, this article will review the available published articles on crown/implant ratio in literature. **Key words:** crown/implant ratio, implant, crown, stress analysis, cortical bone, spongiosseus bone, prosthetic restoration, implant treatment.

Апстракт

При третманот на тоталната беззабност изборот е помеѓу тотална протеза или терапија со импланти. Терапијата со импланти дава подобри резултати во однос на стабилноста на протетската изработка, сигурноста на пациентот, естетиката и фонација и ја спречува ресорпцијата на алвеоларниот гребен. Но имплантопротетскиот третман бара оптимално определени индикации и услови. Соодносот коронка/имплант е значаен фактор за ефектот од имплантолошкиот третман и неговото профилактично дејство на коскеното ткиво. Од тој аспект во трудот ќе биде направен преглед на достапните објавени трудови за соодносот коронка/имплант во литературата. **Клучни зборови:** Сооднос коронка/имплант, имплант, коронка, стрес анализа, кортикална коска, спонгиозна коска, протетичка реставрација, имплантолошки третман.

Introduction

Toothloss leads to morphological, functional, and aesthetic disturbance in the functions of the masticatory system.

The treatment is choice between usual fixed prosthetic or mobile prosthetic restorations or implant therapy.

In total edentulousness treatment the choice is between complete denture or implant therapy.

Each option has its own advantages and disadvantages. However, implant therapy gives better results than other available treatment options.

The advantage of implant prosthetic therapy is that prosthetic suprastructures over implants provide more reliable stability and restore 60-80% of lost function. This gives the patient greater comfort. Also, the transmission of occlusal forces on the bone through the implant prevents resorption of the alveolar ridge[4].

As an edentulousness therapy, implant treatment is preferred in clinical practice due to the quality of modern dental implant materials and the resolved osseointegration problem. Still, implant therapy is not without problems.

The determination of the number of implants, their size, the type of prosthetic construction (suprastructure), and the method of implant placement depends on many factors.

Most important are the bone tissue condition, crown height space (CHS) and the patient's finance capacity.

Besides other indications, resorption of alveolar bone tissue may be crucial in treatment planning, especially for determining the location and dimensions of implants.

Although implant treatments provide better dental rehabilitation than other treatments, overloading the implants is considered one of the risk factors for implants survival.

Gómez-Poloet et al.[2] and Teixeira et al.[3] believe that it occurs when greater resorption of alveolar bone tissue is present, which causes two important problems in implantology: there is insufficient bone tissue for implant placement, but there is more space for crown height.

This conditions shorter implants and construction of longer crown suprastructures. In that case, the prosthodontic concept for natural teeth, minimum crown/root or crown/implant ratio of 1:1, is not respected[4].

According to literature, suprastructure height and implant ratio may be one of the reasons for implants overload, i.e., for stress increasement in the peri-implant bone tissue and treatment failure.

In June 2004, in Las Vegas, Nevada, the International Congress of Oral Implantologists sponsored a consensual conference to determine the space needed for crown height[5].

Despite numerous consensus discussions and meetings, no general guidance consensus was developed, thus leaving room for further research.

Aim

The purpose of this article is to analyse the findings on crown/implant ratio in the published literature.

Material and method

The material consists of reviewed articles that examine crown/implant ratio. The articles were acquired by research in international journals, as well as PubMed and EBSCO database in the period from January 2005 to January 2020. Research was done using keywords according to the Mesh index. 325 articles were reviewed, out of which 50 were analyzed.

Discussion

Implantology treatments in modern dental practice are widely accepted methods for edentulousness treatment. Due to osseointegration of implants, the success of implant treatments is over 95%[6].

Implant treatment failure can be attributed to poor planning, inadequate compliance with surrounding structures, improper design and occlusion of suprastructure.

Initially, dental implantology has accepted the prosthetic standards that were applied to natural teeth. Thus, according to traditional prosthetics, the length of the implant placed in the alveolar bone (equivalent to the root) should be greater than the height of the suprastructure. According to the prosthetic principle, crown/root ratio should be ideally 1:2, 1:1.5 ratio would be optimal, while 1:1 ratio should be minimum. The implant is placed in bone tissue, so the base for implants placement is to have sufficient amount of bone. However, teethloss results in vertical bone tissue loss. Vertical bone tissue height is a prerequisite for determining the implant length.

Misch defines the space for suprastructure as "crown height space" (CHS, Crown Height Space)[7].

The crown height space (CHS) is measured from the crest of the alveolar bone to the occlusal plane in the posterior region for the upper and lower jaw, and in the anterior region the upper CHS is measured to the occlusal plane, and the lower CHS is 1-2 mm above the occlusal plane. The ideal CHS for a fixed suprastructure over the implant should be between 8 and 12 mm. This dimension provides space of 3 mm for soft tissue, 2 mm for occlusal suprastructure thickness and more than 5mm for abutment[8].

The clinical C/I ratio is determined by radiography[9].

In situation of greater crestal bone resorption, the space for the implant suprastructure, i.e. C/I ratio, is increased, which is considered as risk factor for success and implant treatment[10].

Grossmann also believes that correct C/I ratio is one of the key factors in achieving a long-term prognosis in prosthetic rehabilitation[11].

The crown/implant ratio is an important factor for the success of implant-supported prosthetic reconstructions and for the implants stability in general. It is basically taken from the prosthodontic concept for natural teeth but still there are fundamental differences. According to Suham et Effie[12] the differences are because the implants have no periodontal ligament, therefore no rotation, and forces are transferred from the implant directly to the bone tissue. For these reasons, increasing the length of the implant cannot compensate for the increased crown height. Instead, he proposes to increase the area of functional load by increasing the number implants placed, the size of the implant used or the implant design.

Excessive stress on the surrounding tissues, which is caused by the loading stress forces, is one of the possible causes of implant failure. Since the stress is transmitted directly to the bone through the implant, careful planning, the correct number of implants, and the positioning of the implants are crucial to ensure proper stress distribution. However, researches on the optimal number of implants necessary to support the suprastructure are insufficient. Recognizing this problem, Gizem, Sercan et Sedat[13] carried out an in vitro study of finite elements analysis to determine the optimal location, number, and diameter of implants needed to support the suprastructure. The study showed that as the number of implants increased, stress values in peri-implant bone tissue

decreased. However, changes in implant diameter had no significant effect on stress.

This can be explained by Rangert et al.'s theoretical analyses[14], who say that axial loads are more favorable for a uniform distribution of stress around the implant, while non-axial forces are danger. Similarly, Papavasiliou et al.[15] found that non-axial forces increased concentration of stress in implant and bone.

These results are also consistent with the literature describing the impact of increased crown height on transfer of occlusal forces[16-24].

There are various opinions in the literature regarding correlation between crown/implant ratio (C/I) and implant treatment success.

Misch et al.[8, 25] say that several factors can increase the mechanical load of the implant, and increasing the height of suprastructure is one of those.

According to Nissan[26] some longitudinal clinical studies for implants with high crowns suggest that this factor does not compromise the predictability of treatment. According to him, CHS is more significant than the C/I ratio in assessing biomechanical-related detrimental effects.

Rokni[27] also reports that there are clinical studies for high-crown implants that prove that this factor does not compromise implant treatment.

Research study and results received by de Moraes et al.[28] suggest that by increasing crown height the stress concentration in the peri-implant bone tissue is increased.

The stress concentration increases by increasing the crown height. It was concluded that: increased C/I ratio increases the stress concentration in the implant components and cortical bone[29].

According to Hadzik et al.[30], no significant correlation was found between C/I ratio and secondary implant stability, as well as between C/I ratio and marginal bone loss.

The crown/implant ratio of 0.9 to 2.2 did not affect the occurrence of biological or technical complications. One tooth restorations with a crown/implant ratio of between 0.9 and 2.2 can be considered a viable treatment option[31].

According to Ramos et al.[32] crown heights of 12.5 and 15 mm caused a statistically significant stress distribution on screws and cortical bone ($p<0.001$) only at non-axial load. Therefore, he concluded that crown increase was a possible deleterious factor to the screws and to the different regions of bone tissue.

According to Bulaqi et al.[33] the increase in crown height space causes the corresponding distribution to become more nonuniform and increases the maximum compressive and tensile stresses in the peri-implant bone.

Also, de Moraes et al.[28] found that increasing the crown height during non-axial load caused a higher concentration of stress in the crown, at the implant/bone level, and increased bone tissue micro movement.

Meijer et al.[34] have researched the impact of the C/I ratio on implant treatment. They selected 154 articles, eight studies met the inclusion criteria. Average C/I ratio was in the range of 0.86 (with 10 mm implants) to 2.14 (with 6 mm implants). Data reviewed in the articles did not show high incidence of biological or technical complications.

Schulte, Flores et Weed[35] monitored the survival of 889 implant supported suprastructures with average C/I ratio of 1.3 (from 0.5:1 to 3:1), in the 2.3 year period (from 0.1 to 7.4 years). He found out that the average survival rate was 98.2%. Sixteen implant failures had an average 1.4 C/I ratio, similar to successful ones. Due to the similarity of the results of the failed and successful implants, they concluded that the C/I ratio was not the cause for implant loss.

Sanz et Naert[36] convey conclusion of the European Association for Osseointegration: „The use of implant supported restorations with C/I ratio up to 2 does not influence crestal bone loss".

Blanes et al.[37] for more than ten years were evaluating the impact of C/I ratio on 142 implants. The respondents were divided into three groups, according to C/I ratio: The first group C/I ratio of 0 to 0.99, the second with 1 to 1.99, and the third with C/I ratio higher than 2. The third group had a success rate of 94.1% (48 of 51 implants). They concluded that restorations with C/I ratio between 2 and 3 can be successfully used. The authors noted that 81.3% of the implants tested were single crown.

Zhao et al.[38] from January 2007 to January 2012, followed 119 patients with 208 ITI implants in posterior region, during period of 6-66 months. Implant restorations were divided into three groups; first group $C/I \leq 1$; second group $1 < C/I \leq 1.5$ and third group $C/I > 1.5$. The clinical C/I ratio did not significantly affect peri-implant bone loss and biomechanical complications of the suprastructures.

Garaicoa-Pazmiño et al.[39] made literature review on 196 articles and 13 were valid for comparison. They found a negative correlation between the C/I ratio and marginal bone loss ($P = -0.012$).

De Moraes et al.[28] have evaluated the impact of crown height on micro movement and stress distribution at implant/bone level by using the three-dimensional finite element method. The implants were sized (3.75 x 10.0 mm) with external hex connections, and the crowns were 10 mm, 12.5 mm and 15 mm high. Axial forces of 200 N and non-axial forces (45°)100 N were applied.

The height of the crown under axial load did not affect the stress distribution and stress concentration, while on oblique forces load they increased. The results of this study suggest that increasing crown height increases stress on implant/bone tissue and increases the micro movement in bone tissue, especially during oblique forces load.

Kyung-Jin et al.[40] evaluated the impact of the C/I ratio on the change in the marginal bone level around the implant in order to determine the location-related factors that influence the correlation between the C/I ratio and the peri-implant marginal bone loss. The study was performed on 259 implants total, placed on 175 patients with an average follow-up period of five years. The implants were divided in two groups according to C/I ratio: ≤1 and >1. Implants with a higher C/I ratio showed less marginal bone loss than implants with a lower C/I ratio.

Urdaneta et al.[41] have done research to evaluate the effect of increased C/I ratio on single tooth implants. The study was performed on group of 81 patients with 326 implants between 2001 and 2003. Patients with at least one single tooth implant were followed. Higher C/I was associated with a significant increase in prosthetic complications, but had no significant effect on the level of bone tissue.

Cinar et Imirzalioglu[42] using the finite element method investigated the amount and localization of functional stress in implants placed in two different bone types (type 3 and type 4) with three different crown heights. They investigated three C/I ratios: 1/1, 1.5/1 and 2/1. The greatest functional stresses occurred around the implant collar with a C/I ratio of 2/1 (430.57 MPa). When doubling the C/I ratio from 1/1 to 2/1 functional stresses were increased, as well as tensile and compression values in cortical and spongiosseus bone in both bone types (type 3 and type 4). As the C/I ratio increased from 1/1 to 2/1, the largest deformations occurred in type IV spongiosseus bone.

Gehrke[43] made an experimental static load at 30° angle to the longitudinal axis of the implant. He concluded that load resistance significantly decreased by increasing C/I ratio.

In the study of Verri et al.[44] an increase in the C/I ratio of 1:1 to 1:1.25 showed increase of average stress in bone tissue by 30% and the increase of C/I ratio from 1:1 to 1:1.5, increased average stress by 51.5%.

Kwan et al.[45] examined the displacement of crown with different height (10, 12 and 14 mm) and different axis of load. The largest displacement was on highest crown with no-axial load.

Most authors agree that there is no established protocol to determine the maximum allowable C/I ratio for dental implant treatment since the experimental studies are not fully consistent with clinical studies[44, 46].

Conclusion

In most articles there is contradiction regarding the importance of crown/implant ratio for stress in peri-implantable bone tissue.

According to the literature, axial forces cause less stress and non-axial forces cause more stress on peri-implant bone tissue.

Stress in peri-implant bone tissue depends more of the direction of the loading force (higher on non-axial-force load) than of the crown/implant ratio.

Most of the studies are paraclinical, made on three dimensional computer models. Clinical studies are mostly with periodic follow-ups monitoring of changes in peri-implant bone tissue.

The results of both methods, in most cases, complement and thus provide corresponding recommendations for clinical practice.

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THE ROLE OF BIOMARKERS PGE2 AND IL-1β IN ORTHODONTIC TOOTH MOVEMENT - REVIEW PAPER

УЛОГАТА НА БИОМАРКЕРИТЕ PGE2 И IL-1β ВО ОРТОДОНТСКОТО ДВИЖЕЊЕ НА ЗАБИТЕ - РЕВИЈАЛЕН ТРУД

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Abstract

Introduction: Tooth movements caused iatrogenically by the application of orthodontic forces for therapeutic purposes are realized through remodeling of the alveoli, with facilitation of numerous biomarkers including inflammatory mediators. The mediators PGE2 and IL-1β have a particular role in these events. **Aim:** The aim of this review is to determine the role and effect of PGE2 and IL-1β in orthodontic tooth movement. **Material and Method:** A total of 39 articles were analyzed, including original articles and reviews published on PubMed and Google Scholar. Only articles published in English were included in the investigation. Key words used for collecting data were Orthodontic treatment, biomarkers, PGE2 and IL-1β, bone remodeling. **Conclusion.** In the treatment of malocclusions the highest values of PGE2 and IL-1β in gingival fluid have been detected in the early orthodontic phase. Both inflammatory mediators have osteoclastic activity, with IL-1β acting in the first hours of the action of mechanical force, and PGE2 activity is synergistic, with the final effect being tooth movement and alveolar remodeling. **Key words:** Orthodontic treatment, biomarkers, PGE2 and IL-1β, bone remodeling.

Апстракт

Вовед: Движењето на забите кое се јавува како резултат на примена на ортодонтските сили во терапевтски цели, се остварува преку ремоделација на алвеоларната коска потпомогнато од бројни биомаркери, во прв ред инфламаторните медијатори PGE2 и IL-1β, кои имаат посебна улога во целиот овој процес. **Цел:** Целта на овој ревијален труд е да се утврдат улогата и влијанието на инфламаторните медијатори PGE2 и IL-1β во ортодонтското движење на забите. **Методи:** За реализација на поставената цел беа анализирани вкупно 39 стручни статии, вклучувајќи ревијални трудови и трудови со оригинални резултати објавени на базите PubMed и Google Scholar. Во испитувањето беа вклучени само трудови објавени на англиски јазик. Клучни зборови користени за прибирање на податоците беа ортодонтски третман, биомаркери, PGE2 и IL-1β и ремоделирање на алвеоларната коска. **Заклучок:** При ортодонтскиот третман на малоклузиите највисоки вредности на медијаторите PGE2 и IL-1β во гингивалниот флуид беа констатирани во раната ортодонтска фаза. И двата инфламаторни медијатори имаат остеокластична активност, при што IL-1β делува во првите часови од дејството на механичката сила, а активноста на PGE2 е синергистичка, при што крајниот ефект е движење на забите и ремоделација на алвеоларната коска. **Клучни зборови:** ортодонтски третман, биомаркери, PGE2 и IL-1β и ремоделирање на алвеоларната коска.

Introduction

In everyday life, in physiological conditions the teeth are in constant motion which is slow, minor and invisible, and is accomplished by permanent remodeling of the alveoli in which they are placed. In healthy periodontitis, although they are constantly in discrete motion, they remain firmly fixed in the alveoli without luxation. In this case the forces are controlled. But throughout life, teeth are constantly exposed to the effects of various stressors (weaker or stronger) due to mastication, phonetics or may be the result of many other causes that are transmitted from the tooth to the alveoli and surrounding support apparatus. The forces are not always within the permissible force or direction. In many situations they overlap with the axial axis of the tooth, and are vertically oriented. But for a variety of reasons, in addition to the vertical direction, they can be more or less longitudinal, sloping, even horizontal.

In essence, the supportive tooth apparatus is the main harmonizing system that balances strength in physiological stresses, and during pathological stresses tolerance is limited, thus if compensatory mechanisms are unable to continue balancing alterations appear. The consequences depend on the strength, duration of the action of force, and the capacity of the tissues around the over-

loaded teeth. Of course, in these conditions the immune response of individuals that has a significant influence on the tissue response, should not be forgotten.

In physiological conditions there is a balance between osteoclastic resorption and osteoblastic bone formation; hence, in the absence of excessive forces these two processes are in equilibrium. The resorption on one side of the tooth is balanced by the deposition of newly created bone on the opposite side. At the same time, these processes are aided by the continuous deposition of cement which throughout life manages to maintain, more or less, a constant relationship between the root surface and the alveolar cup.

For therapeutic purposes in certain situations, tooth movement during orthodontic treatment (OTM) is desirable, predictable and expected. In orthodontic dosing, tooth movement is a consequence of a series of biological events in the teeth, periodontal ligament (PDL) and alveolar bone that causes microscopic, macroscopic, biochemical and other changes in and around the tooth[1,2].

Concerning these processes that occur in the tooth environment, it is well known and understood that orthodontically conditioned tooth loading causes cell changes in the alveolar bone initiating processes of cell differentiation, reparation, migration, vascular changes and inflammation. Bone remodeling facilitates tooth movement to the desired position. On the pressure side during OTM, the blood perfusion is reduced and is associated with hypoxia. This vascular condition creates conditions for the disrupted process of cell proliferation, which in certain situations can cause apoptosis-cell death[3].

In essence, OTM is not a simple activity, it is rather a complex dynamic movement that causes a series of sequential events that are dominated by vascular tissue changes, synthesis and release of molecules, biomarkers, cytokines or neurotransmitters[2]. Each of the released molecules has activity that in the tissue around and in the tooth has an effect on the alveolar wall or in PDL whose ultimate effect is compression (pressure) on one side and the opposite tension (traction)[4], or more precisely, certain biomarkers stimulate resorption processes, and others the opposite[2].

During orthodontic treatment, biomarkers that are released play a key role in biological changes in and around the tooth and are divided into three major groups: inflammatory mediators, metabolic products and enzymes[5]. Each group contains several substances that act as indicators for the events in and around the tooth. They all have different powers of specificity and sensitivity, but one thing is important - each of them has a field of action and informs about the biological status of PDL and alveolar bone at a particular stage of OTM[6].

Beside the periodontiumthat has an important role in the movement of teeth in OTM, the gingival fluid (GCF) plays the auxiliary role. It is an exudate and an indicator of biochemical events in the tooth[5].

In the GCF besides the largest percentage of water, immunoglobulins, bacteria, enzymes, toxins and many inflammatory mediators are present as well[7]. Some of them play the role of markers responsible for the active destruction of tissues in most periodontal diseases[8]. During orthodontic treatment the cytoskeletal configuration and shape of cells in the PDL are changed[9].

Biological tissue changes under the influence of orthodontic forces

The forces applied to tooth movement have different effects on and around the teeth depending on the duration of action[10]. Initially the force exerted causes thinning of the periodontal ligament followed in parallel by certain metabolic tissue changes that intensify several hours later. But these changes are not identical on both sides; on the contrary, metabolic deviations differ on the side where the tooth is directed, i.e. the compression side, as opposed to the side where the correction occurs, i.e. tension[11].

The forces affect all structures of the periodontal tissues (alveolar bone, periodontium, cementum and gingiva), but the dominant effects are observed on the alveolar bone, where the primary effect is related to the cells. They differentiate, migrate, collapse or heal not only as a result of orthodontic forces but also as a consequence of some other factors including obligatory inflammation and vascular disorders[12]. Thanks to these activities remodeling takes place in the alveolar cup which facilitates movement.

The alveolar bone is not only involved in the movement of teeth, the PDL is also subject to force-induced changes. Thus, in the pressure side of the PDL there is hypoxia-induced blood perfusion, which is believed to either influence cell proliferation or cause apoptosis. Definitely, the effects depend on the degree of oxygen present[12].

In the PDL besides the extracellular matrix and collagen fibers there is also a rich cell population composed of fibroblasts, osteoblasts, osteoclasts, cementoblasts, cementocytes, fibrocytes and other cells that contribute to the specific design of PDL, and each has its own role. Together they have the ability of renewal, so PDL can be easily adapted to the action of orthodontic forces if they are moderate and controlled[13]. Referring to all these developments, periodontal tissues allow the teeth to be moved to new positions, thus changing, what for the patients seems important, aesthetics as well as the function[14].

Given the changes in periodontal tissues, it can be concluded that orthodontic tooth movement is a fairly serious and complex procedure resulting from numerous and varied biomechanical changes that, according to Proffit[15], are primarily due to changes in PDL.

According to another group of researchers, tooth movement during orthodontic treatment may be the result of a series of consecutive reactions in the periodontal tissues including alveolar bone, not just in PDL[10].

Zainal Ariffin[10] confirms that these processes release numerous substances from the tooth and surrounding structures called biomarkers that can be identified in different media.

The remodeling process i.e. compression and stretching, alter the vascularization that causes the synthesis of different signaling molecules that generate a cellular response providing resorption or apoptosis processes[2]. Initiate activation processes on various cellular signaling pathways that promote resorption and apoptosis processes[16]. These signals are responsible for changing of the cytoskeletal structure. Some authors describe this process as a series of orchestrated cellular and molecular events in the alveolar bone and in PDL initiated from the application of orthodontic force[17]. Signals are responsible for the changes that initiate alterations in the cytoskeletal structure[7,18].

In addition to the macroscopic and morphological changes that occur during orthodontic treatment, changes are also evident at biomolecular level, where growth factors, prostaglandins, and cytokines in general are released as one segment of the large biomarker group[19].

The role of biomarkers in orthodontic tooth movement

Biomarkers are identified as important mediators during orthodontic therapy. They are the cause of inflammatory disorders, bone resorption and apoptosis, changes in the periodontal ligament, and vascular and neural tissue changes[20-21]. In essence, biomarkers are substances that are objectively measured and evaluated as indicators of normal biological, pathological processes or as pharmacological response status following applied therapeutic procedures in tissue, and most commonly in GCF.

Numerous studies in the 1990s have reported that biomarkers have been linked to alveolar bone destruction and the progression of periodontal disease. Macrophages and neutrophils, in response to bacteria, produce important inflammatory mediators such as TNF-α, IL-6, IL-1, and other cytokines that are associated with bone and tissue destruction[23, 24].

Thanks to studies[25, 26] concerning the orthodontic tooth movement, it is concluded that the inflammatory interleukins of IL-1β, IL-6, TNF-α and low molecular protein molecules are found in the inflamed periodontal pockets[27].

Therefore, it can be concluded that biomarkers can be indicators for other conditions of the periodontal tissues, and not only related to orthodontic tooth movement. They have a particularly important role in the processes of bone homeostasis and bone destruction. Specific inflammatory mediators such as ICTP, RANKL, osteoprotegerin (OPG), and osteocalcin are pointed out for this role. Bostanci[28] measuring the amounts of RANKL and OPG and their ratio showed that in bone destruction, RANKL increased in the gingival fluid and OPG decreased. This condition is reversed in healthy gingiva or gingivitis where other periodontal tissues including bone are not involved in resorption processes.

In addition to the metabolic substances and enzymes in the group of biomarkers that participate in bone shaping during orthodontic tooth movement, an important site belongs to the group of inflammatory mediators. This group includes the following biological substances: prostaglandin E, neuropeptides (calcitonin related gene peptide and substance p), transforming growth factor-α1, epidermal growth factor, α2 microglobulin and insulin-like growth factor-1, interleukin-1 (receptor antagonist) 1β, 2, 6, 8, tumor necrosis factor, macrophages colony stimulating factors, receptor activator of nuclear factor-kappa / receptor activator of nuclear factor-kappa ligand / osteoprotegerin system, myeloperoxidase, markers of root resorption[5].

Prostaglandins are one of the most important group of inflammatory mediators involved in bone loss and bone formation processes.

Prostaglandin E(PGE2)

Prostaglandins are lipid compounds that are derived enzymatically from fatty acids and have important functions in human and animal organisms. Each prostaglandin contains 20 carbon atoms, five of which form a ring. One of them is Prostaglandin E. Specifically, PGE2 is a major mediator of inflammatory and vascular events. It plays a major role in the destructive and absorptive processes that are based on increased osteoclast activity in the body[29].

Dosage of orthodontic force supplemented with PGE2 injection in animal models (monkeys and rats) resulted in advanced alveolar bone resorption which caused increased tooth mobility[31, 32].

Increased levels of PGE2 in GCF by 24-48 hours of application have been reported in the human population

where orthodontic controlled forces have been applied. However, the study reported a decrease in values after 168 hours of research[32].

Studies on the values of PGE2 and IL-1β in GCF indicate that ovarian activity has an influence on inflammatory mediators during the orthodontic tooth movement stage[33].

A comparison of PGE2 values in GCF during orthodontic force application revealed different findings. Namely, the young population has increased levels of PGE2 in the GCF, which increased to 21 days. From 21 to 28 days the concentration of this biomarker has been decreased. No difference was observed in adults before and after the study. Variations of PGE2 in GCF were not detected. In fact the results do not indicate that PGE2 levels vary with age and period of orthodontic activation. This may explain why orthodontic treatment is more successful in younger versus older populations[34].

PGE2 and IL-1β are considered to be strong stimulants of bone resorption and apoptosis and are a type of response to mechanical stress. In fact, the dynamics of this activity is dependent on their concentration, which in turn is reflected in the clinical finding in orthodontic loading conditions[34]. A study concerning the concentration of PGE2 and IL-1β in GCF confirms the recent findings. Values increase at the early stage of orthodontic treatment, and then normalize, i.e. are returned to their initial values in 7 days[35].

A research by Saito[36] showed that periodontal ligament cells responded to mechanical stress with increased production of PGE while IL-1β enriched the tissue response.

A well-known fact among orthodontists and periodontists is that orthodontic load on a periodontally compromised teeth is not very desirable as it leads to additional lesions of the already damaged tissues. To some extent this connection is also a limit for planning and implementing orthodontic appliances. But the adverse impact of whether and how advanced periodontal disease would affect tooth movement during orthodontic dosing, including the ultimate effect, is an issue that has yet to be elucidated. Okamoto[37] investigated the effect of experimentally induced periodontal inflammation on orthodontic tooth movement in mice. Special

Table 1. Relationship between PGE2 and IL-1β with orthodontic tooth movement

Mediator	Model	Medium	Sample	Important findings	References
PGE2	animal	GCF	rats	Tooth movement	[30]
PGE2	animal	GCF	rats	Tooth movement	[31]
PGE2	human	GCF	humans	Increasing from 24-48 hours	[32]
PGE2	animal	GCF	cats	Influence of ovaries	[33]
PGE2	human	GCF	Young population	Increase till the 21 day	[34]
PGE2	human	GCF	adult	No changes	[34]
PGE	human	GCF	humans	Increasing first 24-48 hours. Normalizing after 7 days	[35]
PGE	animal	tissue	cats	Marked staining of the tension side	[36]
PGE2	In vitro		rats	In vitro treatment with PGE2 decreases the extracellular signal- regulated kinase phosphorylation and RANKL expression	[37]
PGE2	Review	PGE 2		use of light continuous forces	[38]
PGE2	humans	GCF	children	Increase in the tension side	[21]
PGE2	humans	GCF	humans	Increase after 24 hours	[39]

staining has shown that during orthodontic treatment the number of osteoclasts was reduced in the pressure zone under conditions of induced periodontal disease.

The expression level of the receptor activator of nuclear factor kappa-B ligand (RANKL) in the pressure zone was reduced in the group where ligatures were applied. In contrast, experimental periodontitis increased the levels of cyclooxygenase-2 mRNA in periodontal tissues, whereas in vitro treatment with PGE2 reduced extracellular signal-regulated kinase phosphorylation and RANKL expression induced by mechanical stress in osteoblasts. These results suggest that force-induced orthodontic osteoclastogenesis in the alveolar bone was inhibited by concomitant periodontal inflammation, at least in part through PGE2 resulting in reduced tooth movement[37].

The activity of PGE2 and IL-1β is known, but their role in bone remodeling is confirmed in Ren's review[38]. The relationships of these two inflammatory mediators has been confirmed, revealing different patterns of regulation. Namely, PGE 2 and IL-1 beta jointly participate in orthodontic tooth movement. IL-1 beta responds primarily to mechanical stress, and PGE 2 is more responsive to the synergistic regulation of IL-1 beta. The results of the analysis suggest the application of light continuous forces in the orthodontic treatment of patients.

Studies have been conducted comparing PGE2 and IL-1β values on the side of compression and tension in patients undergoing orthodontic forces during the early phase of orthodontic tooth movement. Increases of basic values have in most cases been on the tension side. In control teeth throughout the study values of PGE2 and IL-1β remained at baseline. Results suggest that PGE2 and IL-1β in GCF reflect biological activity in the periodontium[21].

Conclusion

During orthodontic tooth movement, the highest values of PGE2 and IL-1β in gingival fluid have been detected in the early orthodontic phase. IL-1β exhibits osteoclastic activity and PGE2 acts synergistically. Both inflammatory mediators have osteoclastic activity with the final effect being tooth movement and alveolar remodeling.

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QUALITY OF ROOT FILLINGS PERFORMED WITH FOUR ROOT FILLING TECHNIQUES. AN IN VITRO STUDY USING MICRO-CT

ЕВАЛУАЦИЈА НА ЧЕТИРИ ТЕХНИКИ НА ОБТУРАЦИЈА НА КОРЕНСКИТЕ КАНАЛИ НА ЗАБИТЕ СО ПОМОШ НА МИКРО-КОМПЈУТЕР ТОМОГРАФИЈАТА. ИН ВИТРО СТУДИЈА

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Abstract

The main aim of the endodontic therapy consists of the tridimensional obturation of the endodontic space. The efforts for an ideally processed and sterilized root canal are questioned if the same is not obturated completely. The aim of this paper is to evaluate and determine the percentage of the volume of empty spaces and spaces of root canals obturated with different materials for obturation with the help of a microcomputer –tomography. This paper includes 80 frontal human extracted teeth. The teeth were divided in two bigger groups according to the technique of processing of the root canal: The first group consisted of 40 human single root teeth processed according to the standard technique of processing and obturated with gutta-flow and Thermafill. The second group consisted of 40 human single root teeth processed according to the ultrasonic technique of root canals processing and obturated with single cone and lateral compaction technique. Statistically significant differences have been registered between the root canals obturated with Thermafill and the Single cone technique ($p<0.05$). Statistically significant differences have been registered between the root canals obturated with GuttaFlow and the Single cone technique ($p<0.01$) and the Single cone and the technique of lateral condensation ($p<0.01$).
Key words: Root canal obturation, materials, techniques, micro-CT.

Апстракт

Главната цел на ендодонтската терапија се состои во тродимензионална оптурација на ендодонтскиот простор. Напорите за идеално обработен и стерилизиран коренски канал се доведуваат во прашање ако истиот не е комплетно оптуриран. Цел на овој труд е да се оценува и одреди процентот на волуменот на празнини и простори на коренските канали обтурирани со различни материали за обтурација со помош на микрокомпјутер- томографијата. Во овој труд вклучени се 80 фронтални хумани екстрахирани заби. Забите беа поделени во две поголеми групи според техниката на обработка на коренскиот канал. Првата група ја сочинуваа четириесет хумани еднокорени заби обработени според стандардна техника на обработка и оптурирани со Gutta-Flow и Thermafill. Втората група ја сочинуваа четириесет хумани еднокорени заби обработени според ултрасоник техниката за обработка на коренските канали и оптурирани со single cone и техниката на латерална кондензација. Евидентирани се статистички значајни разлики измеѓу коренските канали оптурирани со Thermafill и single cone техниката($p<0,05$). Статистички високо значајани разлики се евидентирани измеѓу коренските канали оптурирани со GuttaFlow и single cone техниката ($p<0,01$) и single cone и техниката на латерална кондензација($p<0,01$). Клучни зборови: канална обтурација,материјали, техники,микро-ЦТ.

Introduction

The success of the endodontic therapy depends on the complete chemical and mechanical processing of the canal system of the root of the tooth, followed by an obturation of the root canal[1,2,3].

The main aim of the endodontic therapy consists of the tridimensional obturation of the endodontic space. The efforts for an ideally processed and sterilized root canal are questioned if the same is not obturated completely[26,85].

An ideal obturation would be considered as such if the canal obturation manages to close the side openings of the dentin canals, foramen apical and the entrance of the root canal, i.e. the root canals obturated with a thick filling which reaches 2 mm to the x-ray tip of the root.

The empty places which would remain between the canal filling and the wall of the canal, as well as the air

bubbles inside the material for obturation, may contain these bacteria or create a path which will enable the bacteria or their products to break through to the paradontal tissue and reactivate the inflammatory processes.

The ideal obturation has to prevent their entering in the periapical tissue i.e. disable the conditions for their further development[4,3].

The materials used for obturating the endodontic space do not satisfy their aim according to their characteristics. This is confirmed by the fact that although there are numerous materials for obturation of the root canals further efforts are made to discover material with better performaces[5,1,6].

Thermafill is a method for obturation of the root canals by using warm gutta-percha. Even before 1883 the author Perry used spiked golden wires covered in soft gutta-percha for root canals obturation.

Single cone - technique was developed in 1960 by standardizing the endodontic instruments. The aim of this method is after preparing the apical part of the root canal to be covered with a thin layer of cement and gutta-percha, silver or titanium cone is placed in the tooth canal.

The method for canal obturation called lateral condensation is considered to be safe, cheap and suitable to work.

The method consists of laying the inner walls of the root canals with cement by using a lentulo spiral, thin needle or an ultrasonic system and master cone gutta-percha placed on the determined root length.

GuttaFlow®2 and GuttaFlow®2FAST is a liquid cold system for canal obturation which combines cement and gutta-percha in one unique product. The system consists of matrix of polydimethylsiloxane filled with small gutta-percha.

The aim of this paper is to evaluate and determine the percentage of the volume of empty spaces and spaces of root canals obturated with different materials for obturation with the help of a microcomputer - tomography.

Material and method

This paper includes 80 frontal human extracted teeth which have curves on the root canal smaller than 10 (degrees) selected according to the technique Schnider.

The experiment does not include teeth with underdeveloped roots, with obturated root canals and caries on the root of the tooth.

After the extraction, the teeth were rinsed with saline to remove the blood and were kept in artificial saliva. The teeth are de-crowned with a diamond borer 1 mm above the enamel-cement border to achieve the length of the root of 12 mm.

Determining the length of the root canals of the teeth has been made using an instrument no.10 placed in the root until you see the tip of the instrument in the apex of the root canal.

As exact length the length 1mm smaller than the length determined by the instrument is taken.

The teeth were divided in two bigger groups according the technique of the processing of the root canal:

1. The first group consisted of 14 human single root teeth processed according to the standard technique of processing.
2. The second group consisted of 14 human single root teeth processed according to the ultrasonic technique of root canals processing.

First group - After determining the exact length of the root canals the teeth from this group were processed according the technique described by Ingle (1961). The canals were irrigated with 2ml NaOCL 5% (Produites Dentares SA, Vevey, Switzerland) and dried with paper pins (PRESIDENT DENTAL, Duisburg,Germany).

This procedure is performed until the root canal gets the determined form and has no smell and leftovers of the pulp tissue.

The root canals from the first group are divided in two subgroups according to the obturation technique.

For the obturation of the root canals from the first subgroup we used the material GuttaFlow®2 (Coltene/Whaledent GMBh+Co.KG, Langenau, Germany).

The root canal in the second subgroup is laid with AH-Plus ®JET™ paste (Densplay De Trey GmbH, Kostanz, Germany) and obturated with Thermafill system (Densplay DeTrey GmbH, Konstanz, Germany).

The second group - the root canals from this group were processed using ultrasonic crown-down technique according to Greka et al. (2007). We started the instrumenting with a hand widener size 40 K-file adjusted to the ultrasonic device (Zhengzhou Smile Dental Equipment Co, Ltd. China) in the period of 1 min. During every change of instrument, we rinsed the root canals with a 5% NaOCL and dried them with paper pins. The root canals were divided in two subgroups. The root canal from the third subgroup was obturated with a paste AH-Plus® JETTM (Densplay De Tray GmbH, Kostanz, Germany) with the help of a lentulo spiral, and the root canal was set with only one gutta-percha (Coltene/Whaledent GmbH+Co.KG).

The root canals of the teeth in the fourth subgroup were obturated with the technique of lateral condensation. The walls of the root canals were laid with AH-Plus® JETTM paste (Densplay De Trey GmbH, Kostanz, Germany) with the help of a lentulo spiral.

Micro-computer tomographic method

The samples were measured in a commercially available cabinet for cone beam micro-CT, (μCT 35, SCANCO Medical AG, Brüttisellen, Switzerland).

The examination samples were wrapped in a piece of sponge 2 x 3 cm and placed in special test tubes with artificial saliva. The test tubes were then placed in a scanning machine and the machine was started. We placed five samples at a time.

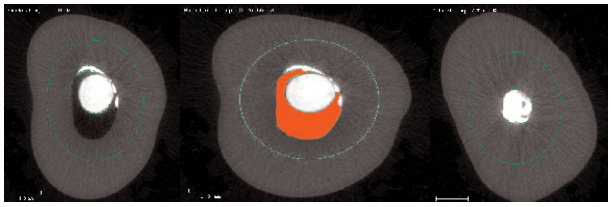
The machine works with a cone beam starting from 7 μм from the point of the x-ray tube. The photons are detected with a CCD-based space detector and the projection data is reconstructed by a computer in a 900 x 900 matrix picture.

The chosen size of the voxel is 20 μм in all three space dimensions.

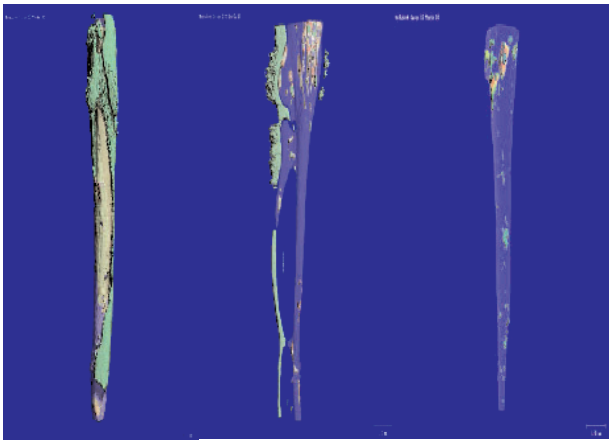
The x-ray voltage is 70kVp, intensity 114.

The empty space in the second evaluation is automatically segmented, based on the grey scale of the values in the CT-layers.

The region of interest (the filling and the empty space) is marked by contours. The script for doing the analyses started by one click on the SCANCO Evaluation program. (picture 20)



Picture 1. *Grey scale of the values of three different teeth. It shows the contours used for the second evaluation to calculate the empty space between the filling and the tooth. The inner contours exclude the volume of the filling.*



Picture 2. *3D-renders on segmented volumes of interest showing the empty space around the filling (seen colored in green)*

In the statistical analyses of the received results from the clinical study we used methods for descriptive and analytical statistical analyses.

For testing the zero hypothesis and reaching valid conclusions we used the following methods of analytics and statistics-

Statistical tests:

- Single factor ANOVA for numeric markings and observation.
- LSD- test-test of the quadrotor of the smallest differences
- Kruskal-Wallis' test
- Mann-Whitney's U test of inversion

Results

Chart 1. The percentual presentation of the empty spaces in the inner side of the canal obturation.

The chart shows the percentage of detected empty spaces in relation to the complete filling.

The statistical analyses showed: ANOVA for numeric markings of observation- F=6,725; DF=3; p<0.1 i.e. there is a statistically significant difference in the percentage of empty space between the researched groups. The smallest percentage of empty space is in 2.1 group, and the biggest in 1.1 group.

The between groups differences with the LSD-test, showed that 1.1 differs statistically significant with 1.2 and 2.1, and does not differ with 2.2.

Group 1.2 differs with 1.1 and with 2.2 and does not differ with 2.1;

Group 2.1 differs with 1.1 and with 2.1 and does not differ with 1.2;

Group 2.2 differs with 2.1 and 1.2 and does not differ with 1.1

The following chart shows the values from the complete percentage of empty spaces and spaces. Chart 2

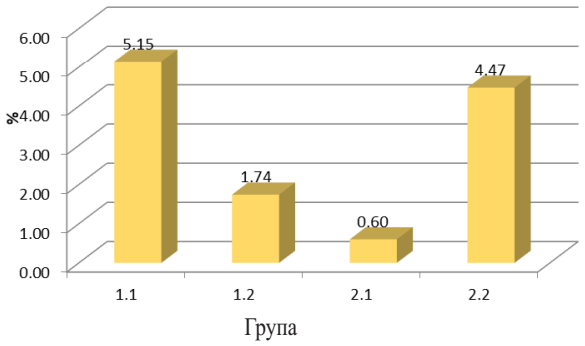


Chart 1. *The percentual presentation of the empty spaces inside the material for obturation compared to the complete filling.*

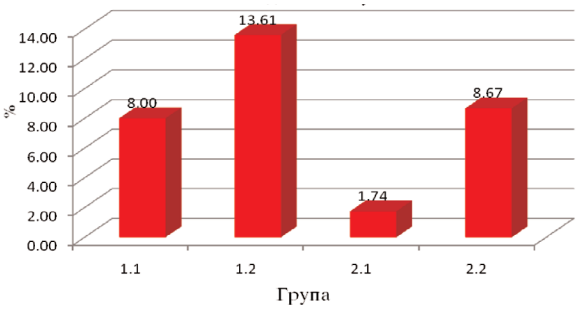


Chart 2. *The percentage presentation of empty spaces inside the material for obturation and the empty spaces in the root canal compared to the complete filling.*

The data analyses showed that a bigger percentage of empty spaces was registered in the root canals obturated with the Thermafill technique (13,61%), while a smaller percentage of empty spaces was registered in the root canals obturated with the Single cone technique.

The single factor ANOVA for numeric markings of observation showed: $F=17.8$; $DF=3$; $p<0.01$

The results show that there is a statistically significant difference in the total percentage of empty spaces in the researched groups:

LSD-tests showed that there was no statistical difference between the root canals obturated with GuttaFlow and Thermafill technique ($p>0.05$), GuttaFlow and the technique of lateral condensation ($p>0.05$) and Thermafill and the technique of lateral condensation.

Statistically significant differences have been registered between the root canals obturated with Thermafill and the Single cone technique ($p<0.05$).

Statistically significant differences have been registered between the root canals obturated with GuttaFlow and the Single cone technique ($p<0.01$) and the Single cone and the technique of lateral condensation ($p<0.01$).

Discussion

A great number of factors might influence the success of the endodontic therapy. After the efforts made to control infections, an adequately processed and obturated root canal should mean success of the endodontic treatment[21].

A well-known fact is that the hermetically sealed root canals of the tooth would mean smaller chances for postoperative complications. The empty spaces in the obturation of the root canals, in theory, might compromise the outcome of the treatment, knowing the possibilities of microorganisms staying in which may find a way to reproduce and penetrate towards the periapical tissue. All techniques, unfortunately, have a lot of registered failures because of the fact that a chemical binding of the material for obturation with the inner walls of the root canals is not achieved. All authors consider the main goal of endodontic obturation to be the prevention of micro-permeability of oral fluids which contain bacteria and their products to move from the coronary towards the periapical part and obstruct the progress of microorganisms towards the periapical space which were resistant to canal disinfection and instrumentation[22,11,5].

The empty spaces may be captured during the canal obturation. The empty spaces which do not communicate with the inner wall of the root canal named as inner empty spaces are considered as non-risk for the prognosis of the endodontic therapy because the bacteria in these empty spaces do not find suitable conditions to survive and reproduce. These empty spaces may communicate with each other and create a path to the inner wall or the periapical tissue.

The production of new materials in endodoncy is connected to technical innovations of finding techniques and materials which will contribute to bigger clinical successes i.e. satisfying all needs of the endodontic materials where the main accent will be given to the hermetic sealing of the root canal.

These new materials should be evaluated carefully[23].

The x-rays made after the treatment are a benchmark for the quality of the canal obturation and the only method to clinically evaluate the homogeneity of the obturation and the presence of empty spaces in the material for obturation[24].

Jung et al. 2005[24] evaluated the potential of the accuracy of micro-CT for the evaluation of canal obturation. The authors confirmed that micro-CT can clearly emphases the wall of the root canal.

According to the findings of all authors, there is no obturated root canal that does not have empty spaces. (Eplay 2006, Gulsahi 2007, James 2007.)[25].

This information rejects the zero hypotheses.

Our research, with the help of micro-CT scanner analyzed the presence of empty spaces in the inner space of the canal obturation. The empty spaces which started from the inside of the material and continued to the outer wall are marked as limited empty spaces.

According to the statistics data a larger percentage of empty spaces are registered in the root canals obturated with GuttaFlow 5.15%, the root canals obturated with lateral condensation 4.47%, Thermafill 1.74% and the single cone technique 0.60%.

When analyzing the total percentage of empty spaces in the material for obturation and the empty spaces between the inner wall of the root canal and the material for obturation, the larger values are from the root canals obturated with Thermafiil system, followed by the root canals obturated with Gutta-Flow and lateral condensation.

While the root canals obturated with the single cone technique gave smaller percentage values, and highly significant statistical values have been achieved.

The authors Peters (2010)[26] and Anbu (2010)[27] confirmed that the obturation of root canals obturated with the technique of lateral condensation is not homogenous and may show more empty spaces between the gutta-percha spikes. The authors state that during the lateral condensation with a widener the air may infiltrate between the spikes which creates empty spaces.

The authors also determined that the obturation with long gutta-percha where the gutta-percha is heated may more easily adapt in all irregularities of the root canal, but these techniques have also their floes. The gutta-percha when heated expands, but when it is getting cold it contracts 1-2% giving bigger empty spaces in the canal.

This finding may explain the highest percentage of empty spaces (13.6) in the root canals obturated with the Thermafill system.

The author Wu M.K. (2009)[28] explains with data the quality of the single cone technique depending on the cement amount placed in the root canal. If during the placement of the cement we use lentulo spiral, the space between the wall of the root canal and the gutta-percha pin is filled. If the obturation is made with a gutta-percha pin only laid with cement, bigger empty spaces may appear and the possibilities of fluid infiltration are bigger.

Angerame (2012)[29] by using the micro-CT system in the root canals obturated with the single cone technique concluded that the root canals obturated with the single cone technique the percentage of inner and outer empty spaces is 0.522±0896%.

The authors favor the single cone technique because of the faster way of processing it and because it can be used in different protocols when processing the canal.

The cements used during this technique and their physical properties act on the sealing possibility. AH-Plus cements have shown to be stable in dimensions and their use is recommended.

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UDC: 616.315-006.2-07/-08:

DIAGNOSIS AND TREATMENT OF NASOPALATINE DUCT CYST – CASE REPORT

ДИЈАГНОЗА И ТРЕТМАН НА НАЗОПАЛАТИНАЛНА ДУКТАЛНА ЦИСТА – ПРИКАЗ НА СЛУЧАЈ

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Abstract

Nasopalatine duct cyst, called the incisive canal cyst, described in literature as a cystis canalis nasopalatine or cystis canalis incisive, is the most common non-odontogenic cyst occurring in the oral cavity. It develops in the midline palate around the incisive foramen from debris of nasopalatine duct's epithelium. The etiology is unknown, although it is believed that some of the irritants may have an impact on its development. Cyst develops slowly destroying the alveolar bone of the maxilla. Typically, the process is asymptomatic and is detected accidentally on radiograph. Clinical diagnosis is made due to X-rays, but only histopathology can confirm the results. The treatment of choice is total enucleation of pathological changes. The following article presents a case of nasopalatine duct cysts, which is diagnosed and treated at the Faculty of Dentistry in Skopje. The case is characteristic regarding the symptomatology, as well as the need of differentiation from other pathological changes within the region, as well as the need for proper treatment. **Key words:** Nasopalatine duct cyst, non-odontogenic cyst, incisive canal cyst.

Апстракт

Назопалатиналната циста, наречена уште и циста на инцизивниот канал, опишана во литературата како cystis canalis nasopalatine или cystis canalis incisive, е најчестата неодонтогена циста во оралната празнина. Се развива во средишната линија на палатумот околу инцизивниот отвор од епителиумот на назопалатиналниот канал. Етиолигијата за нејзиното создавање е непозната, но се смета дека одредени иритативни фактори можат да имаат влијание врз нејзиниот развиток. Цистата се развива многу бавно вршејќи коскена деструкција на околната коска на максилата. Типично, процесот е асимптоматски и најчесто се детектира случајно на радиографска снимка, а клиничката дијагноза се потврдува со хистопатолошка анализа. Терапијата е хируршка со целосна енуклеација на цистичната промена. Авторите презентираат клинички случај на назопалатинална дуктална циста, која е дијагностицирана и хируршки третирана на Стоматолошкиот факултет во Скопје. Случајот е карактеристичен во однос на симптоматологијата, како и потребата од диференцирање од другите патолошки промени кои се јавуваат во таа регија и потребата од соодветен третман. **Клучни зборови:** Назопалатинална дуктална циста, неодонтогена циста, циста на инцизивен канал.

Introduction

Nasopalatine duct cyst is the most common non – odontogenic cyst occuring in oral cavity. It was first described by Meyer in 1914[1, 2]. In the past, known as the fissured cyst, now according to the WHO classification is defined as a non – odontogenic, developmental, epithelial cyst of maxilla[3]. In most of cases, it develops in the midline of the palate near the incisive foramen[4]. In normal development, the nasopalatine duct canal is converted into incisive canal by the disappearance of epithelium. Other cells in the form of epithelial cord can initiate the development of cysts. Mostly it is located in palatal part of canal or superficially[5]. Depending on whether the cyst was caused by palatal or nasal part of the canal, it is lined by stratified squamous or ciliated epithelium[6]. If it appears above, within the nasal part of the canal, cilliary respiratory epithelium can even be observed[3]. It has a capsule built of dense fibrous tissue containing scattered chronic inflammatory cells[5]. Nasopalatine duct cyst is rare and occurs in 1% of the population. Observations show that it is usually detected among people between 4th and 6th decade of life, although there were cases among children up to 8 years old. It is three times more common among men than women[3–5, 7]. It occurs in both human races: white and black[8, 9]. The etiology of the cyst is unknown, although it is suspected that its development may contribute to nasopalatine duct infections, or retention of mucus[3–5, 10]. Of all the factors most likely theory is spontaneous cystic degeneration of the nasopalatine duct remains. This duct, in the fetal period, is a broad connection between mouth and nose. In the course of normal development, shortly after birth, it becomes obliterated and atrophies. Among some peo-

ple, however, it does not disappear completely and its remnants, in the form of epithelial cord, could lead to the development of cysts in the incisive canal. However, the cause of this abnormal development is still unknown[8]. Some authors suggest here the similarity to the lower mammals, in which inside the nasopalatine canal is penetrable by air „nasopalatine duct", which is an auxiliary olfactory organ, called Jacobson's organ[8]. Among the suggested causes of incisive canal cysts formation are also some genetic factors. However, the literature does not describe a lot of evidence for an unambiguous confirmation of any of the hypotheses[3,5]. Cyst develops slowly leading to loss of bone in the maxilla. Most of its development is asymptomatic and is detected incidentally on radiograph. It is observed within the median line of the palate as an oval or heart-shaped radiolucency[3, 5, 13, 14]. If the symptoms appear, swelling in the median line of the palate's front part is the most common[5]. It may also manifest itself on the labial side of the alveolar process of the maxilla, causing rarely facial asymmetry[3, 5, 15]. In some cases, pain may occur as a result of pressure of nasopalatine nerve, reported primarily by people using prosthesis or as a result of palpation examination of incisive papilla's area.

The pain can be caused also by the superinfection. The more advanced change is, the symptoms become more pronounced. Also it is observed that symptoms appear earlier when the cyst is located caudal[5]. Surgical total enucleation is the recommended treatment with pathological findings and shows a very low rate of recurrence[5, 12, 13]. Among all types of cysts diagnosed in dental surgery, a nasopalatine duct cyst is rare. Since 1960, the English-language literature has published only 468 case reports[8]. The aim of our study is to present one case documented by the University Dental Clinic „St. Pantheleimon" - Skopje, in 2019.

Case Report

A 24-year-old male patient was referred by his general dentist to the department of oral surgery, our clinic, complaining to pain in the area of the first maxillary incisors, followed by a panoramic x-ray image showing translucency in the region of the apexes of the central maxillary incisors. (Fig. 1)

The main subjective problem reported by the patient is swelling and pain in the midline, on the inside of the upper jaw. Clinical inspection and examination of the region detects swelling in anterior third of medial line of palatum. (Fig. 2)

Palpation in the area of the incisal papilla produces a painful sensation and a strongly elastic fluctuating prominence that is easily elevated above the level of the gingival mucosa. The panoramic x-ray image shows intact central incisors, with no previous carious lesions and no previous conservative or endodontic treatments (Fig.1). On a vertical percussion, there is a mild painful sensation on them. A vitality test of the central and lateral incisors was performed bilaterally, with a positive result.

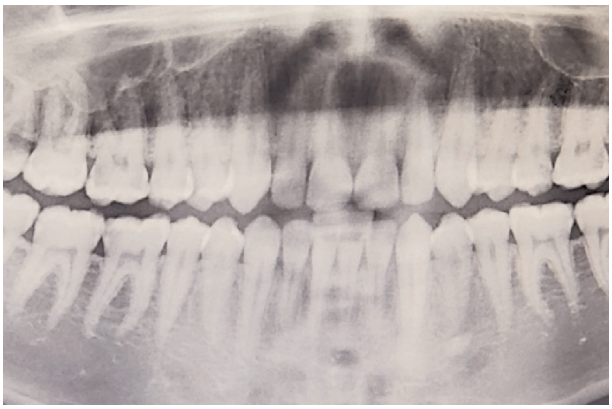

Figure 1. *Panoramic x-ray*

Figure 2. *Intraoral view of the palatum*

By the clinical examinations and differential diagnostic pathways, panoramic X-rays revealed that the diagnosis was a nasopalatinal ductal cyst. The patient is referred for further 3D X-ray imaging, with the aim of precise and accurate localization of the cystic change. We performed a drainage procedure, which produces purulent content and a rubber drain was installed. The 3D x-ray recording clearly reveals a well-restricted radiolucent bone change of non-odontogenic origin, preserving a well-preserved lamellar bone around the roots of all teeth in the frontal region (Fig. 3, 4 and 5). Dimensions of the translucency are 14 mm in craniocaudal direction and 11 mm in transversal direction. After all clinical and paraclinical investigations, the previous differential diagnostic finding confirms that it is a nasopalatinal ductal cyst and that the patient is in need of further surgical treatment, which would result in total enucleation of the cyst.

On several subsequent visits, the pathologically changed tissue was washed and cleaned with potassium permanganate and the rubber drain was changed several

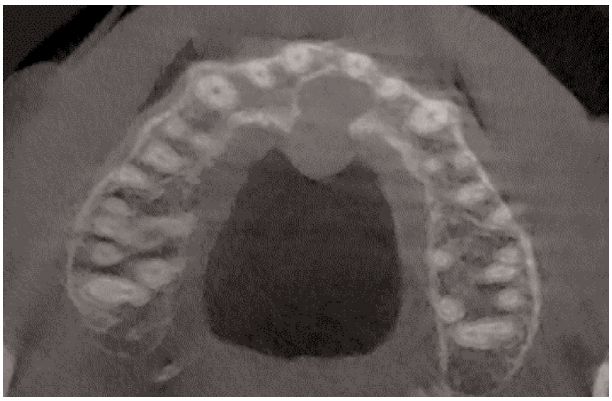

Figure 3. *3D view of the cyst*

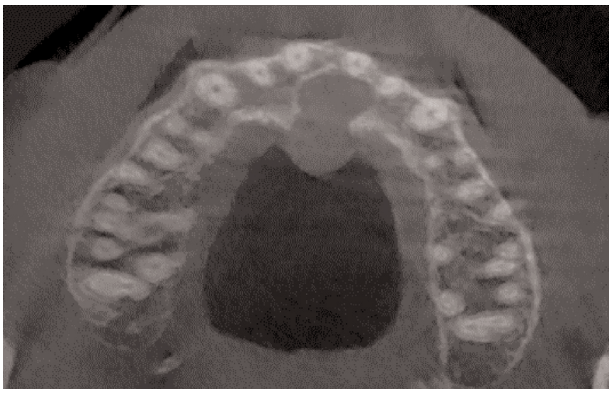

Figure 4. *Transversal view of the cyst*

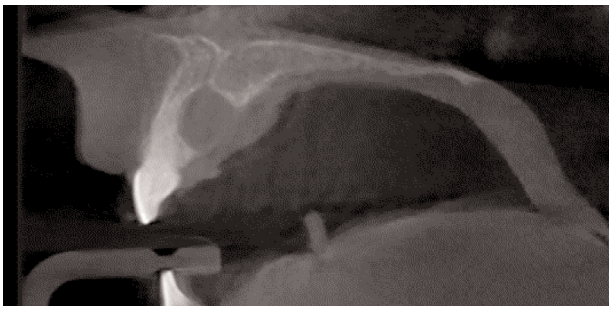

Figure 5. *Sagital view of the cyst*

times every 24 hours. When the subjective problems of the patient disappeared and the purulent content was evacuated, we had complete cystic enuclation. Nasopalatinal ductal nerve block anesthesia was applied, Scandonest 3% in nasopalatine duct and plexus anesthesia in the vestibular support of the upper incisors. We made a marginal, palatal incision in the intercuspid area and raised a mucoperiostal flap with careful prevention of nerve and vascular contents of the nasopalatinal canal (Fig. 6).

After total cyst enucleation, the mucoperiostal flap tissue was returned to its original position and sutured (Fig. 7). A fully enucleated specimen of the cyst is given for

Figure 6. *Intraoral view of the lesion*

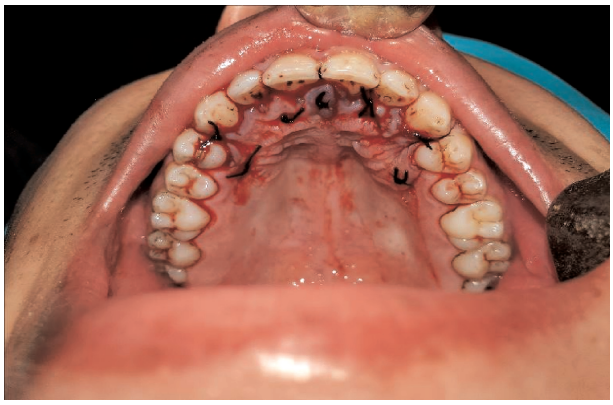

Figure 7. *Postoperative sutured mucoperiostal flap*

pathohistological examination. Postoperatively, oral antibiotic therapy Amoxicillin a 1000 mg twice a day is prescribed, for 5 days after the surgery treatment. In the next few days the patient is followed and advised for rinses with sodium chloride 0.9% for several times in a day.

According to the macroscopic finding of the pathohistological examination, a soft tissue fragment was presented with an aspect of a cyst wall of 0.1 cm thickness. The luminal and outer surface of the cyst wall are smooth, and the overall dimensions of the fragment are 1 cm x 0.7 cm. All material is embedded in paraffin block for analysis.

According to the microscopic finding, the analysis showed connective tissue with areas of edema that contained a cystic formation coated with prominent squamous epithelium without any atypical cells signs. Peripheral to the epithelium is a rich mononuclear inflammatory infiltrate and local areas of fresh bleeding.

Discussion

Diagnosing and differentiation of the nasopalatine duct cyst has to be made very carefully before making the final diagnosis. A presumptive diagnosis suggested

on the base of the anamnesis and precise clinical examination has to be completed with the radiological examination in minimum two projections: periapical and occlusal. However, the only certain confirmation is a result of the histopathological examination. During the clinical examination, the differential diagnosing is very important. Mainly the nasopalatine duct cyst has to be differenced with the radicular cyst to avoid unnecessary endodontic treatment of vital and healthy teeth[5]. For this purpose, the pulp vitality test, the percussion test and the analysis of shape and width of the periodontal space has to be performed[11]. In case of the nasopalatine duct cyst, pulp of the neighboring teeth remains vital and the lamina dura of the periodontal fissure does not lose continuity. All the mentioned tests and analyses have to be performed when periapical granuloma of the upper incisors is suspected. It is very important according to the modern endodontics, which says that most of the periapical granulomas remain 'not infected' because they are caused by the bacteria which are present only in the root canal. That fact implicates the treatment of these lesions, which is based on the antiseptic endodontic treatment without obligatory surgical removal of the periapical granulomas. During the interpretation of the X-ray, those pathological periapical lesions are most frequently suggested as the presumptive diagnosis. In differential diagnosis the rare lesion – median palatal cyst, similar on the X-rays, should be also concerned. However, its etiology is connected with an inappropriate fusion of the maxillary processes and with an injection of the epithelial cells between them[16]. As far as Francoli and Torres claim, diagnostic problems appear also during examination of smaller lesions (average size of the nasopalatine duct cyst is between 6 mm to 17 mm)[3, 8] because they can be similar to anatomical structures like the incisive foramen or widen to 6–8 mm incisive canal. On the other hand, the cyst can reach the size overcrossing 50 mm[8, 11]. The differential diagnosis should concern the supernumerary tooth appearing in this area – the mesiodens in the follicular cyst and also it should concern the primary cyst, the giant-cell granuloma, the osteitis with the palatal fistula and also naso – palatine and palatal – sinus connections[3]. As soon as the final diagnosis of the nasopalatine duct cyst is made, the lesion has to be surgically removed, as the literature recommends, not only because it is destroying the bone, but also a few malignant transformations are known[3]. In English literature, there are a few cases of the squamous cell carcinoma which have developed as a result of the metaplasia of the epithelium which lines the cyst[17]. Gardner observed characteristic symptoms of the cancer developing from the cyst. As an example, it can be a dynamic growth of the lesion, also a resorption of the roots which can be observed on the X-rays and changes in the sensitivity to touch of the upper lip[18]. According to literature, a marsupialization is also a possible alternative treatment for some patients[5, 10]. It is recommended in the cases when the capsule of the cyst shows adhesions with the surrounding area which make it difficult to enucleate. Rounded incision should be made on the biggest circumference and then the cyst's lower wall with surrounding mucosa has to be removed. The procedure should be finished with inserting the sutures between the lining of the cyst and the mucosa of the oral cavity. According to some theories, if the lesion is asymptomatic and does not reach larger sizes, the surgical treatment is not necessary. But they also emphasize that the cyst has to be removed before prosthetic treatment in this area, because the chronic irritation can lead to inflammatory reaction. Considering all those facts, it seems that surgical treatment is the best way of treatment of the nasopalatine duct cyst[3, 16].

A total enucleation should be a surgical treatment of choice because there are some evidenced cases of recurrence reaching from 0% up to 11%. According to Kimberly, in all collected by him 334 cases, only 7 recurrences were observed (2%). Hedin recommends regular control visits, including X-ray examination and pulp vitality tests of neighboring teeth, after the surgery during 3 years[2, 19]. As presented in this study case, the four-week observation period after surgery was performed and then the treatment was considered as completed. However, the total enucleation was performed in our case, the patient was informed that he was obligated to self-control and if any symptoms of recurrence would appear he had to come to clinic as soon as possible. Another rare complication after a surgery which can be observed only in 10% of cases is the paresthesia of the frontal part of the palate. It is caused by removing the part of the cyst's wall which can be connected with the endings of the naso-palatine nerve[3]. Although this does not let us to forget about the proper examination and also if it is necessary to use all the diagnostic sources and make a differential diagnosis to start in the right time, a good treatment to avoid dangerous complications.

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RISK FACTORS IN DIABETIC PROSTHODONTIC PATIENTS FOR PRECANCEROUS CONDITIONS

РИЗИК ФАКТОРИ ЗА ПРЕКАНЦЕРОЗНИ СОСТОЈБИ КАЈ ПРОТЕТИЧКИ ПАЦИЕНТИ СО ДИЈАБЕТ

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Abstract

Diabetes Mellitus has become a global epidemic and presents many complications, usually proportional to the degree and duration of hyperglycemia. Many chronic macrovascular and microvascular complications of diabetes have been reported in the literature with few reports about oral complications. At the same time, the incidence of oral cancer has not decreased over the years despite exhaustive research. Recent research has shown that diabetes is one of the contributing factors in the initiation and progression of certain cancers. In addition, certain drugs used for the treatment of diabetic patients may also have a role in cancer initiation, progression and mortality. Epidemiological studies have shown that diabetic patients are at a higher risk in case of common cancers such as pancreatic, liver, breast, colorectal, urinary tract, etc. This article will explore the relationship between diabetes and oral cancer, with its possible mechanisms of carcinogenesis. **Keywords:** Diabetes, insulin, oral manifestations, oral cancer, precancerous lesions.

Апстракт

ДМ стана глобална епидемија, чии компликации се пропорционални со степенот и времетраењето на хипергликемијата. Многу хронични макроваскуларни и микроваскуларни компликации на дијабетес се сретнуваат во литературата, посочувајќи ја појавата на орални компликации. Во исто време, инциденцата на орален карцином не се намалува во текот на годините и покрај исцрпните истражувања. Неодамнешните истражувања покажаа дека дијабетесот е еден од придонесувачките фактори за започнување и напредување на одредени карциноми. Покрај тоа, одредени лекови што се користат за третман на дијабетични пациенти, исто така, можат да имаат улога во иницирање на карцином, негова прогресија и морталитет. Епидемиолошките студии покажале дека дијабетичните пациенти се изложени на поголем ризик во случај на вообичаени карциноми, како што се карциноми на: панкреас, црн дроб, гради, колон, ректум, уринарен тракт итн. Овој труд ќе ги презентира досегашните истражувања за односот помеѓу дијабетес мелитус и оралниот карцином. **Клучни зборови:** Дијабетес, инсулин, орални манифестации, орален карцином, преканцерозни лезии.

Introduction

Diabetes mellitus is a group of metabolic diseases that leads to high levels of blood glucose and is caused when the body does not make any or enough insulin, or does not use insulin well[1]. Although there are various causes for less common types of diabetes, including drug- or chemical-induced diabetes, exocrine pancreatic disease, or infections (e.g., cytomegalovirus), the two most common subtypes of diabetes are known as Type 1 or Type 2 diabetes[2]. Type 1 diabetes, formerly known as juvenile diabetes, is a chronic autoimmune disease in which the beta cells in the pancreas create little to no insulin[2,3], and accounts for 5% to 10% of all diabetes cases[2]. Type 1 diabetes is generally diagnosed in younger individuals (usually younger than 25 years of age) and has a strong genetic predisposition. Exogenous insulin is needed to regulate blood glucose levels in people with Type 1 diabetes[2].

In contrast, Type 2 diabetes accounts for 85% to 90% or more of diabetes cases and is one of the most common chronic diseases, as well as one of the leading causes of death and disability in the U.S[4]. Type 2 diabetes is characterized by decreased response of target tissues to insulin, requiring increasing levels of insulin for an adequate response, dysregulation of insulin production, and insulin resistance[2]. Type 2 diabetes is associated with excess weight, physical inactivity, family history of diabetes, and certain ethnicities[3]. Although some people with Type 2 diabetes can help improve their glycemic

control with diet, exercise, and weight loss, patients may require insulin sensitizers that help peripheral tissues take up glucose (i.e., biguanides [metformin] or thiazolidinediones) or oral hypoglycemic agents that either stimulate release of insulin (i.e., insulin secretagogues such as sulfonylureas)[2]. Initial symptoms of diabetes include increased thirst and urination. Other symptoms can include unexplained weight loss, fatigue, blurred vision, increased hunger, and sores that do not heal[2].

One of the most common causes of death is cancer[5]. Oral cancer, also known as mouth cancer, is cancer of the lining of the lips, mouth, or upper throat[6]. Oral cavity cancer accounts for approximately 3% of all malignancies and is a significant worldwide health problem[7,8]. Most oral malignancies occur as squamous cell carcinomas (SCCs); despite remarkable advances in treatment modalities, the 5-year survival rate has not significantly improved over the past several decades and still hovers at about 50-60%. In the mouth, it most commonly starts as a painless white patch that thickens, develops red patches, an ulcer, and continues to grow. When on the lips, it commonly looks like a persistent crusting ulcer that does not heal, and slowly grows[10]. Other symptoms may include difficult or painful swallowing, new lumps or bumps in the neck, a swelling in the mouth, or a feeling of numbness in the mouth or lips[11]. Adult males are most commonly affected, with an increased incidence among alcoholics and smokers of sixth and eighth decades of life. The commonly affected sites are buccal mucosa, hard palate, gingiva, floor of the mouth, lips, oropharynx and tongue. The most common risk factor for oral cancer is tobacco. The other risk factors include alcohol, immune defects, genetic factors and viruses such as human papilloma virus, Epstein-Barr virus, hepatitis virus, etc[12]. Many oral SCCs develop from premalignant conditions of the oral cavity[13, 14]. A wide array of conditions have been implicated in the development of oral cancer, including leukoplakia, erythroplakia, palatal lesion of reverse cigar smoking, oral lichen planus, oral submucous fibrosis, discoid lupus erythematosus, and hereditary disorders such as dyskeratosis congenital and epidermolysis bullosa[15].

Recent epidemiological studies have shown a strong link between diabetes and cancer. These studies report that cancer patients with diabetes have a worse prognosis than patients without diabetes after treatment. Though diabetes and cancer has many common risk factors such as obesity, male sex and ageing, both Type I and Type II diabetes are associated with more incidence of cancer[16]. There are differences in carcinogenesis between diabetes I and diabetes II. Studies have shown that the risk of cancers in breast, colorectum, pancreas, etc. in diabetes II is more compared to diabetes I patients[17].

Diabetes mellitus causes many immunologic and metabolic changes in the oral mucosa. Many studies have shown the relationship of diabetes with periodontal disease and inflammatory diseases of the oral mucosa. Emerging evidences suggests that diabetic patients show more precancerous lesions like erythroplakia and leukoplakia that leads to oral cancer. The association between diabetes and oral cancer is unclear till date. Recent discoveries like anti-tumor effect of metformin can help the diabetologists and oncologists in discovering newer drugs for preventing diabetic complications.

Aim

This review discusses the possible association between diabetes and oral cancer with the available data.

Material and method

The material consists of reviewed articles that search the possible association between diabetes and oral cancer. Studies were selected applying the following inclusion criteria: articles published in English, between January 1998 and January 2020, in scientific journals, original research, studies conducted on a human population.

Discussion

Oral complications and manifestations of diabetes mellitus

Several soft tissue abnormalities have been reported to be associated with diabetes mellitus in the oral cavity. These complications include periodontal diseases (periodontitis and gingivitis); salivary dysfunction leading to a reduction in salivary flow and changes in saliva composition, and taste dysfunction. Oral fungal and bacterial infections have also been reported in patients with diabetes. There are also reports of oral mucosa lesions in the form of stomatitis, geographic tongue, benign migratory glossitis, fissured tongue, traumatic ulcer, lichen planus, lichenoid reaction and angular chelitis[18,19,20,2122].In addition, delayed mucosal wound healing, mucosal neurosensory disorders, dental carries and tooth loss has been reported in patients with diabetes[23]. The prevalence and the chance of developing oral mucosal lesions were found to be higher in patients with diabetes compared to healthy controls[24]. The discovery of insulin treatment justified a close correlation between the disorder of the carbohydrate metabolism and oral inflammatory complications[25]. The adequate treatment of diabetes results in marked improvement of the gingival and periodontal lesions. Diabetes patients have further inflammatory complications of the oral mucosa. The decreased rate of

saliva secretion and the low ph value result in chronic cheilitis and glossitis with progressive atrophy of the covering epithelial layer[26].

Histological changes of gingiva

The coronal part of the gingival connective tissue underneath the junctional epithelium shows decreased collagen density. Reduction in collagen synthesis and replication of DNA in dermal fibroblasts are seen more in diabetic patients as compared with non-diabetic patients. It is observed that there is an increase in the collagenase activity and abnormalities in neutrophil degranulation due to gingival crevicular fluid collagenase or other metabolic abnormalities in periodontal ligament fibroblasts. The histological sections in diabetic patients showed thickened basement membrane, swollen and proliferated endothelial cells and obliteration of capillaries with narrow capillary lumen[27].

Diabetes and cancer

The first association between cancer and diabetes was studied in 1885[10]. According to few meta-analysis studies diabetic patients have an increased risk of cancers in endometrium, pancreas, colorectum, etc[28,29,30,31,32,33,34].

Hyperglycemia

Hyperglycemia plays a major role in carcinogenesis. Hyperglycemia generates oxidative stress that damages the DNA and induces carcinogenesis[35].

Anticancer drugs and cancer risk

Insulin analogues used in the treatment of cancer have a role in cancer. Insulin is a mitogen that stimulates mitogen-activated protein kinase (AMPK) pathway, which in turn causes cancer. Many in vitro studies have revealed that, increased insulin levels affect angiogenesis and promote tumor progression by stimulating the mitogen pathway through insulin and insulin-like growth factor receptors. Studies have reported that there is less cancer risk in case of metformin as compared to other antidiabetic drugs. This is because of certain antitumor characteristics of metformin like cell proliferation inhibition. It could also be due to decreased cancer proliferation with the activation of activated protein kinase (AMP), AMP kinase, which is a mediator of tumor suppressor LKB1[36].

Precancerous changes in diabetes

Mihaela et al. reported a case having an eleven-year history of Type II diabetic mellitus with a hyperplastic lesion in the inferior vestibule. The biopsy showed moderate elongation of the rete ridges, epithelial hyperplasia due to a moderate acanthosis, mild orthokeratosis with focal parakeratosis and basal cell hyperplasia of basal cell layer[37].

In the study done by Thomas et al. analyzing the risk factors of leukoplakia, they found that diabetic patients are three times more associated with leukoplakia than non-diabetic patients. This increased incidence might be due to the metabolic and immunologic changes in the oral mucosa[38].

Certain studies have reported a possible association between DM and potentially malignant disorders such as erythroplakia[39,40] leukoplakia[39,41] and lichen planus[41,42 ,43,44]. However, other studies neither demonstrated this association nor found any influence of DM on the duration, distribution, or type of lesion[45,46].

The studies conducted in India[39] and Hungary[40] show an increased prevalence of premalignant lesions among diabetic patients.

A study done by Ujpál et al. revealed 25.6% of Type I and 31.3% of Type II diabetic patients had glossitis and chronic cheilitis that are considered to be precursors of malignant transformations. 10.9% of Type I and 16.9% of Type II had benign tumors. 3.2% of Type I and 11% of Type II had leukoplakia or erythroplakia. There were more incidences of gingival cancer (29%) and lip cancer (24%) as compared to the non-diabetic group[40].

In normal populations, oral cancer mainly involves the tongue, oropharynx and floor of the mouth. The lips, gingiva, dorsum of the tongue and palate are usually not affected[47]. But in people with diabetes, tumors most commonly involve the gums and labial mucosa. Also in contrast to the normal population, in which males are more commonly affected by oral cancer than females, among those with diabetes, tumors are more frequent in females[39,40].

Dikshit Rp et al. analyzed data from randomized oral cancer screening trial in Kerala, India, to study the association between chronic diseases in general, diabetes mellitus in particular, and pre-malignant oral lesions and conditions. They found that the incidence of leukoplakia and lichen planus in diabetic patients was more in comparison with non-diabetic patients[39].

Bastos et al.[48] reported a significantly higher prevalence of lichen planus in dm^2 patients (6.1%) than in control subjects. Van Dis and Parks[25] observed lichen planus in 4% of patients with diabetes.

A study by Mohsin et al. revealed more oral mucosal lesions such as geographic tongue, fissured tongue, coated tongue in diabetic than non-diabetic patients. An association between premalignant lesions and diabetic patients was not significant according to their study[49]. Muralidara et al. also did not find any association between precancerous lesions and diabetes. They have

found only lichen planus in diabetes patients[50]. However, according to the study done by Yadiyal et al. there is no correlation between lichen planus and diabetes[51]. The occurrence of lichen planus in diabetes patients might also be due to the lichenoid reaction of the drugs[52].

There are studies on other site-specific cancers which strongly support an increase in cancer risk and mortality in diabetic patients. The molecular mechanisms associated with diabetes and cancer development are still not clear. However, only few research studies have been done on diabetes and oral cancer. The association between diabetes and oral cancers may be due to shared risk factors between the two diseases, such as diet, aging, obesity and physical inactivity. However, the etiologic factors of oral cancer such as tobacco, alcohol can also contribute to oral cancer in diabetic patients. A few studies on oral cancer and diabetes have shown an association between them, while a few studies have indicated the opposite.

Conclusion

More advanced studies are necessary to show a definitive relationship between diabetes and oral cancer. Proper blood glucose control is mandatory to avoid diabetes related complications. For this reason, clinicians and patients should be aware of possible cancer risks in diabetic patients. This awareness will go a long way in reducing the incidence of oncological complications among diabetic patients.

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ДЕКЛАРАЦИЈА

Кариес на рано детство: Бангкокшка декларација на Меѓународната асоцијација за детска стоматологија

1 | ВОВЕД

Намената на оваа Декларација е да се обезбеди поддршка во светски рамки за една на докази базирана дефиниција и за заедничко разбирање на доказите за етиологијата, факторите на ризик и интервенциите во врска со кариесот на раното детство заради намалување на неговото појавување, како и за да се иницираат заеднички пристапи и политики за сузбивање на таа хронична болест. Имајќи го ова предвид, под покровителство на Меѓународната асоцијација за детска стоматологија (International Association for Paediatric Dentistry, IAPD), 11 експерти од сите краишта на Земјината топка се состанаа и ја составија оваа изјава.

2 | БАНГКОКШКАТА ДЕКЛАРАЦИЈА НА IAPD

Кариесот на раното детство (Early Childhood Caries, ECC) е дефиниран како присуство на една или повеќе кариозни површини (лезии без или со кавитет), површини што недостасуваат или се реставрирани (поради кариес) на кој било млечен заб на дете под шестгодишна возраст. Млечните заби го чуваат просторот за трајните заби и неопходни се за благосостојбата на детето, затоа што забниот кариес на млечните заби може да доведе до хронична болка, инфекции и други заболувања. ECC може да се превенира, но сепак тековно со него се погодени над 600 милиони деца ширум светот и тој останува, главно, нетретиран. Оваа болест има големо влијание врз квалитетот на животот на децата и на нивните семејства и претставува непотребен товар за општеството.

Кариесот на раното детство, како и другите форми на кариес, се смета за една динамична мултифакторна болест, предизвикана од биофилм и поттикната од шеќери, која резултира со нерамнотежа меѓу деминерализацијата и реминерализацијата на тврдите забни ткива. Забниот кариес е предодреден со биолошките, бихевиоралните и психосоцијалните фактори сврзани со животната средина на индивидуата. ECC ги споделува заедничките фактори на ризик со другите непреносливи болести (non-communicable diseases, NCDs), поврзани со претераната консумација на шеќери, како што се кардиоваскуларната болест, дијабетесот и претераната тежина. Претераното внесување на шеќери доведува до пролонгирано производство на киселини од бактериите прилепени на забот и со тоа до промена на составот на типичните микроорганизми во оралната шуплина како и до промена н pH-факторот на биофилмот. Ако внесот на шеќери продолжува континуирано, структурите на забот се деминерализираат. Во некои случаи ECC е поврзан со развојни дефекти на глеѓта.

За намалување на товарот од оваа болест, која што може да се превенира, од големо значење се: соодветно справување со ECC од страна на добро информирани родители, професионалци од областа и општествени здравствени работници, како и здравствена политика базирана на докази. Во овој процес од помош е да се процени кариес ризикот со тоа што ќе се утврди веројатноста за негова појава кај поодделни пациенти или групи деца кај кои се развиваат кариозни лезии. За секое одделно дете, проценката на ризикот е битен, клучен елемент за превенцијата и справувањето со ECC. На локално ниво, проценката на ризикот од појава на кариес може да се искористи за насочување и осмислување на општествени мерки и распределба на времето и ресурсите на оние на коишто тоа им е најпотребно.

Превенцијата и грижата за ECC може да се подели во три фази. Примарната превенција вклучува подобрување на писменоста за оралното здравје на родителите/стрателите и на здравствените работници, ограничување на консумацијата на слободен шеќер во пијалаци и прехранбени продукти од страна на децата како и дневна примена на флуориди. Секундарната превенција се состои од ефективна контрола на почетните лезии пред да настапи кавитација, а може да вклучува почесто премачкување со флуор-препарати и примена на залевачи на фисури и јамички на катниците подложни на кариес. Терцијарната превенција вклучува спречување на ширење на кавитетите и оперативна грижа заради зачувување на забите.

3 | ПРЕПОРАКИ

За да се намали преваленцијата на ECC ширум светот, и неговото оптоварување на општеството, Банкгокшката декларација на IAPD ги препорачува следниве мерки:

Четирите клучни области во кои е потребно да се презема акција од страна на многуте заинтересирани фактори се следниве:

1. Покачување на свесноста за ECC на родителите/старателите, стоматолозите, денталните хигиенолози, лека-

рите, медицинските сестри, здравствените професионалци и другите заинтересирани страни.

2. Ограничување на внесувањето на шеќери преку прехранбени производи и пијалаци и одбегнување на слободни шеќери за деца на возраст под две години.
3. Четкање на забите со флуоридни паста за заби (најмалку 1000 ppm) два пати дневно за сите деца, со користење на количество паста соодветно на возраста.
4. Нудење на насоки за превенција во текот на првата година од животот (онаму каде што тоа е можно, како надградба на постојните програми - на пример, вакцинации) од страна на здравствени професионалеци или општествени здравствени работници и, во идеален случај, упатување на стоматолог заради сеопфатна континуирана нега.

Покрај тоа, се препорачува:

- Заинтересираните фактори да се залагаат за повраток на трошоци и за образовна реформа со акцент на превенцијата заснована на докази и сеопфатното справување со ECC.
- Заради стандардизирање на споредбите меѓу земји и региони, епидемиолошките студии треба да го бележат присуството и на некавитиран и на кавитиран кариес; во идеален случај би требало да се запишуваат почетните, умерените и напреднатите фази на кариес; децата треба да се прегледуваат на тригодишна и на петгодишна возраст за да се согледаат потребите за превенција, и за реставрација.
- На стоматолошките училишта и факултети ширум светот треба да се имплементира наставна програма за ECC и да се придава исто толкава тежина на превентивната нега базирана на докази и на процена на ризикот, колку што му се придава на традиционалниот хируршки третман.
- За да се добие подлабоко разбирање на придобивките од ефективната и навремена нега, треба да се поддржуваат истражувања на ECC-нееднаквостите, истражувања на квалитетот на живот сврзан со оралното здравје, како и истражувања на интервенциите и на здравствената економија.

  Во додатокот што е даден подолу, подготвен од панелот експерти, се нуди едно коминике за кариесот во раното детство, наменето за широк круг професионални и непрофесионални заинтересирани фактори. Ажурираните докази и референци што беа искористени во оваа Декларација се содржани во еден подетален документ, под наслов „Глобален поглед на епидемиологијата, етиологијата, оценувањето на ризикот на кариесот во раното детство и општественото оптоварување што тој го предизвикува, справувањето со него, образованието и политиката во врска со него“ [1].

*Светскиот самит за кариесот на раното детство беше одржан од 2 до 4 ноември 2018 г. во Банкок. Членовите на експертскиот панел што ја составија оваа Декларација, со придонес на Бордот на IAPD, беа: докторите Drs. N.B. Pitts (U.K), R. Baez (USA), C. Diaz-Guallory (USA), K. Donly (USA), C. Feldens (Brazil), C. McGrath (Hong Kong), P. Phantumvanit (Thailand), K. Seow (Australia), N. Sharkov (Bulgaria), N. Tinanoff (USA), and S. Twetman (Denmark).

Членови на Бордот на IAPD по азбучен ред: докторите Drs. M. Bonecker (Brazil), A. O'Connell (Ireland), B. Drummond (New Zealand), T. Fujiwara (Japan), C. Hughes (USA), N. Kramer (Germany), A. Kupietzky (Israel), A.M. Vierrou (Greece), A. Tsai (Taiwan).

РЕФЕРЕНЦА

1 Tinanoff, N, Baez, RJ Diaz-Guillory, C, et al. Early childhood caries epidemiology, aetiology, risk assessment, societal burden, management, education, and policy: Global perspective. Int J Paediatr. Dent. 2019;29:238-248. https://doi.org/10.1111/ipd.12484

ДОДАТОК

Бангкокшка декларација на IAPD: коминике за кариесот на раното детство

Што е кариес на раното детство (ECC)

- **Дентален кариес:** Научна дефиниција - дентален кариес е динамична мултифакторна болест, предизвикана од биофилм и поттикната од шеќери, која резултира со нерамнотежа меѓу деминерализацијата и реминерализацијата на тврдите забни ткива. Забниот кариес е одреден од биолошките, бихевиоралните и психосоцијалните фактори сврзани со животната средина на индивидуата.
- **Кариес во раното детство** е: Лаичка дефиниција - забен кариес кај претшколски деца, којшто е вообичаен, главно нетретиран, а кој може да има длабоко влијание врз животите на децата. Клиничка дефиниција - присуство на една или повеќе кариозни површини (лезии без или со кавитет), површини што недостасуваат или се реставрирани (поради кариес) на кој било млечен заб на дете под шестгодишна возраст.

Контекстот за ECC

- Забниот кариес е најчестото заболување кое што може да се превенира.
- Со нетретиран забен кариес на млечните заби се погодени над 600 милиони деца ширум светот.
- Забниот кариес ги споделува заедничките фактори на ризик со другите непреносливи болести (NCDs), поврзани со претераната консумација на шеќери, како што

се кардиоваскуларната болест, дијабетесот и претераната тежина.

Неприфатливиот товар на ЕСС

- ЕСС и претставува непотребен товар за децата, семејствата и општеството.
- Навремената и соодветна превенција на ЕСС се многу битен чинител во намалувањето на тој товар и играат значајна улога во подобрување на квалитетот на животот на децата во светот.

Како да ги намалиме ЕСС и неговото оптоварување?

- **ЕСС е мултифакторна болест** и не постои лесен начин или само едно единствено решение за сложената „кариесна сложувалка“. Ангажирањето на многуте заинтересирани чинители во третирањето на повеќекратните аспекти на изворите за појавата на кариесот е неопходно за превенирањето на ЕСС.
- **Примарна превенција на ЕСС**
  - Навремени интервенции на ниво на општествената заедница.
  - Превенција на појава на нова болест на индивидуално ниво.
- **Секундарна превенција на ЕСС**
  - Ефективна контрола на почетните лезии пред да дојде до кавитација.
  - Ограничување на понапреднатите лезии, каде што тоа е можно.
- **Терцијарна превенција на ЕСС**
  - Неинвазивни процедури за контрола на кариес.
  - Соодветна реставрациска терапија за зачувување на забите.

Акцијата во врска со ЕСС што треба да ја преземат многуте заинтересирани фактори во четири клучни области

- **Покачување на свесноста за ЕСС** меѓу родителите/старателите, стоматолозите, педијатрите, медицинските сестри, другите здравствени професионалци и другите заинтересирани фактори.
- **Ограничување на внесувањето на шеќери** преку прехранбени производи и пијалаци и одбегнување на слободни шеќери за децата под двегодишна возраст.
- **Четкање на забите два пати дневно со флуоридирана забна паста** (најмалку 1000 ppm) за сите деца, со користење на количество паста соодветно на возраста.
- **Нудење на насоки за превенција во текот на првата година од животот** (онаму каде што тоа е можно, како надградба на постојните програми - на пример, вакцинации) од страна на здравствени професионалеци или општествени здравствени работници и, во идеален случај, упатување на стоматолог заради сеопфатна континуирана нега.

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