PATHOLOGICAL CHANGES OF DENTAL CEMENTUM DURING PERIODONTAL DISEASE

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Abstract: During chronic periodontitis, as one of the most common oral disease, on the hard wall of the periodontal pocket, which is presented by dental cementum significant pathological changes can be noted. These changes participate in maintaining the continuity of the periodontal infection, influencing, at the same time, no less on the complexity and complicate the periodontal treatment. As the pocket deepens, the collagen fibers that insert into the dental cement are mainly destroyed, causing this dental tissue to be exposed to the effects of the oral environment. On the collagen residues of Sharpes' fibers in cement degeneration can be present, creating an environment that favors inhabitation of bacteria, too. These bacteria can penetrate the cement to a depth as far as to the cement-dentin border. In some cases they can penetrate in the dentin tubules, that can cause pulpal inflammation, as a complication. Pathological granules (especually seen by electronic microscopy) that represent regions of collagen degeneration or regions where collagen fibers are not initially fully mineralized can often be detected on the hard wall of the periodontal pocket. The penetration and growth of bacteria leads to fragmentation and carious process of the cement surface, leading to formation of zones of necrotic cement, separated from the tooth by masses of bacteria. In addition, bacterial products, such as endotoxins, have also been detected in the dental cementum of periodontal pockets. Periodontal diseased root fragments also prevent in vitro attachment of human gingival fibroblasts. Most common changes on the hard wall of the periodontal pocket are clinically manifested by softening of the dental root surface, which is usually asymptomatic but painful when a probe or explorer enters the region. The cementum is very thin in the cervical region of the tooth, where therapeutically debridement often removes it completely, leaving exposed dentin beneath it. Regions of increased mineralization of the dental cement are result of exchange or exposure of the dental root surface on the oral cavity sourounding, especially exchange of minerals and organic components on the cement-saliva boundary surface. That is the reason why mineral content of the cement increases. On the periodontal compromised areas of the roots are increased the amount of the following minerals: calcium, magnesium, phosphorus and fluoride. The development of a highly mineralized surface layer can increase the tooth's resistance to dental caries (cervical decay). Hypermineralized zones are associated with enriched crystal structure and organic changes. Loss of or reduction of collagen near the cement surface and subsurface and condensation of organic material of exogenous origin are also reported. Regions of demineralization are usually associated with root caries. Dental cement also can soften and undergo fragmentation and cavitation. Unlike enamel caries, root surface caries tends to progress around the tooth rather than deep into the tooth. Active carious root lesions take the form of yellowish or light brown regions, often covered with plaque and having a relatively soft consistency when probed. Inactive lesions are well-defined darker lesions with a smoother surface and a firmer consistency when probing. When after periodontal therapy (both conservative and surgical) plaque levels and pocket depths increased, there is also a change in the oral bacterial environment, followed by a reduction in periodontal pathogens and an increase of Streptococcus mutans and repercussive caries development in the root.
Keywords: periodontal pocket, pathological changes, hard-wall of periodontal pocket, chronical peridontitis

1. INTRODUCTION

It is very well-known that periodontal disease is generally a bacterial-induced inflammatory reaction. The etiology is dominantly connected with bacteria that are attached to the tooth and root surfaces and progress to form a biofilm environment- known as dental plaque.

All different therapeutic methods tend to result in complete removal of the dental plaque and the pathological changes on the hard-wall and soft-wall of the periodontal socket. One of the most challenging aspects of periodontal therapy is the choice of a predictable approach to root surface modification primarily due to the fact that the biofilm impairs the possibilities for regeneration and needs to be removed in toto. (Eschler & Rapley, 1991)

During the pathological events and changes of the periodontal disease, the root surface of the tooth undergoes a series of changes in its physical and chemical nature, and dental cement also becomes cytotoxic due to the release of

bacterial toxins that are embedded in the cementum of the root. All pathological changes are catogorized in following groups: (1) adhesion loss of collagen fibers on the cement surface, (2) increasing of mineral content on exposed cement (chemical changes) , (3) root surface demineralization and crater formation or presence root caries (structural changes) and (4) presence of lipopolysaccharides on the cement surface (cytotoxic changes). (Sundaran et al, 2014)

Having in mind these numerous changes on the root surface during chronic periodontits, we seted the main aim of this study - to make literary rewiev related to the changes that occur on the surface of the tooth root, primarily using contemporary scientific data.

2. PATHOLOGICAL CHANGES OF DENTAL CEMENTUM DURING PERIODONTAL DISEASE

Periodontal diseases are highly prevalent oral diseases and can affect up to 90% of the world population. Gingivitis, as a mildest form of periodontal disease, is primarly caused by the bacterial biofilm (dental plaque) that accumulates on teeth adjacent to the gingiva. (Minovska & Petrovski, 2014) Periodontitis is defined as "an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both. (Ganhadi &Kothiwale, 2012)

Cementum deposition is necessary for maintenance of periodontal health and defective cementum formation may lead to the development of periodontal pockets. Thus, according to Lindskog & Blomlof (1983) cementum structure may be the major element of susceptibility to microbial invasion and formation of periodontal pockets. Root surfaces of teeth extracted because of aggressive periodontitis have been found to have hypoplastic or aplastic cementum.

During chronic periodontitis, as one of the most common oral disease, on the hard wall of the periodontal pocket, which is presented by dental cementum significant pathological changes can be noted. These changes participate in maintaining the continuity of the periodontal infection, influencing, at the same time, no less on the complexity and complicate the periodontal treatment. As the pocket deepens, the collagen fibers that insert into the dental cement are mainly destroyed, causing this dental tissue to be exposed to the effects of the oral environment. (Bosshardt, 2018) On the collagen residues of Sharpes' fibers in cement degeneration can be present, creating an environment that favors inhabitation of bacteria, too.

These bacteria can penetrate the cement to a depth as far as to the cement-dentin border. In some cases they can penetrate in the dentin tubules, that can cause pulpal inflammation, as a complication. Vibrant bacteria are found on the root surfaces in 87% of periodontically diseased non-carious teeth. (Chandki et al, 2011)

Pathological granules (especually seen by electron microscopy) that represent regions of collagen degeneration or regions where collagen fibers are not initially fully mineralized can often be detected on the hard wall of the periodontal pocket. The penetration and growth of bacteria leads to fragmentation and carious process of the cement surface, leading to formation of zones of necrotic cement, separated from the tooth by masses of bacteria. (Preza et al, 2008)

In addition, bacterial products, such as endotoxins, have also been detected in the dental cementum of periodontal pockets. When dental root fragments of periodontal compromised teeth are placed in tissue culture medium, they cause irreversible morphological changes on the cells in culture, as opposed to a healthy root structure. Periodontal diseased root fragments also prevent in vitro attachment of human gingival fibroblasts. (Könönen, 2019)

Most common changes on the hard wall of the periodontal pocket are clinically manifested by softening of the dental root surface, which is usually asymptomatic but painful when a probe or explorer enters the region. They are also a depot for reinfection of the region, after the treatment.

The cementum is very thin in the cervical region of the tooth, where therapeutically debridement often removes it completely, leaving exposed dentin beneath it. This can result in hypersensitivity to thermal stimuli, especially cold, until the pulp tissue responds with secondary dentin formed. (Nagata et al, 2016)

Regions of increased mineralization of the dental cement are result of exchange or exposure of the dental root surface on the oral cavity sourounding, especially exchange of minerals and organic components on the cement-saliva boundary surface. That is the reason why mineral content of the cement increases. On the periodontal compromised areas of the roots are increased the amount of the following minerals: calcium, magnesium, phosphorus and fluoride. But the quantity of the micro-elements remain unchanged. (Abou Neel et al, 2016)

The development of a highly mineralized surface layer can increase the tooth's resistance to dental caries (cervical decay). Hypermineralized zones are associated with enriched crystal structure and organic changes. These zones have also been verified in microradiographic studies as a layer with a thickness of 10 to 20 μm, regions up to 50 μm thick. No reduction of mineralization was found in the deeper regions, which indicates the fact that the increased mineralization does not come from the surounding regions. (Mamai-Homata et al, 2012)

Loss of or reduction of collagen near the cement surface and subsurface and condensation of organic material of exogenous origin are also reported. Regions of demineralization are usually associated with root caries. Exposure to oral fluid and bacterial plaque results in proteolysis of nested remnants of Sharpe's fibers. (Ho et al, 2007)

Cement also can soften and undergo fragmentation and cavitation. Unlike enamel caries, root surface caries tends to progress around the tooth rather than deep into the tooth. Active carious root lesions take the form of yellowish or light brown regions, often covered with plaque and having a relatively soft consistency when probed. (Deyhle et al., 2011)

Inactive lesions are well-defined darker lesions with a smoother surface and a firmer consistency when probing. The dominant microorganism in the carious lesion on the root surface is Actinomyces viscosus, although its specific role in the development of the lesions has not been established. Other bacteria, such as Actinomyces naeslundii, Streptococcus mutans, Streptococcus salivarius, Streptococcus sanguis and Bacillus cereus, have been found to cause root caries (in some in vitro studies, on animal experimental models). (Klokkevold et al, 2002)

When after periodontal therapy (both conservative and surgical) plaque levels and pocket depths increased, there is also a change in the oral bacterial environment, followed by a reduction in periodontal pathogens and an increase of Streptococcus mutans and repercussive caries development in the root. (Pannu et al,2013)

An important principle in functional tissue engineering is the determination of the biomechanical properties of the native tissue in health and in diseased conditions. Acording to Butler et al (2000), in the case of periodontal tissue engineering, this information can be obtained by analyzing the mechanical properties of both the healthy and the diseased root surface.

In one study, condacted by Sundaram et al in 2014, the SEM characterization revealed the presence of mineralized collagen fibers in the healthy cementum and they were more predominant when compared with the diseased cementum. According to the same authors, SEM micrographs of the diseased cementum showed areas of foreign bodies that could be deposits of calculus.

One of the pathological lessions that can occur during chronical periodontitis is cervical periodontal inflammatory resorption. This cervical (periodontal) inflammatory resorption is also known as cervical root resorption, invasive cervical resorption, extracanalicular invasive resorption or cervical inflammatory resorption. (Frank,1995 and Gulsahi et al, 2007) Although the exact etiology of this pathological condition has not been established, the authors grouped this condition in inflammatory resorptive conditions. (Madison & Walton , 199o and Rodriguez-Pato, 2004) Cervical lesions may develop above the crestal bone and to be supraosseous or below the crestal bone and to be intraosseous.(Benenati, 2002)

Factors that can result with cervical inflammatory resorption include bacteria from periodontal disease, periodontal treatment, trauma, intracoronal bleaching, orthodontics, bruxism, developmental tooth defects or idiopathic causes.(Mock et al, 1997 and Bergmans et al, 2002) The exact pathogenesis of this pathological conditon is not known. However, the process originates in the periodontal ligament in contrast to apical inflammatory resorption, which originates in a necrotic pulp.

Cervical inflammatory resorption is usually asymptomatic and discovered on routine radiographs. The process may appear similar to tooth decay, especially in a supraosseous position in the root. The teeth usually are vital and responsive to pulp tests unless the process begins to involve the pulp. Sometimes additional radiographs should be taken at different angles to differentiate it from internal root resorption. (Andreasen et al, 2006)

Therapy of this condition is complex. The extent of the resorption and accessibility to instrumentation must be identified. Supraosseous lesions may be removed with a large slow-speed round bur with application of trichloracetic acid to the root surface and restored with glass ionomer cement or amalgam without a surgical flap procedure. Intraosseous lesions require a surgical flap procedure to access the lesion or forced eruption with orthodontic appliances prior to restoration. Restoration is performed similarly to that of supraosseous lesions. Conventional root canal therapy may be required if the resorption extends near to the pulpal tissues. (Heithersay,1999)

3. CONCLUSION

Numerous pathological changes occur during periodontal disease on the hard wall of the periodontal pocket. Knowledge of these microscopic changes is of particular importance, especially due to the fact that they provide the basis for the various clinical signs and symptoms that are characteristic for this disease. Recognition of these pathological changes is also important in the aspect of the treatment of periodontal disease, especially in the different advanced regenerative and reparative procedures.

REFERENCES

Abou Neel E. A., Aljabo A., Strange A., Ibrahim S., Coathup M., Young A.M., Bozec L. & Mudera, V. (2016). Demineralization-remineralization dynamics in teeth and bone. International journal of nanomedicine, 11, 4743–4763. https://doi.org/10.2147/IJN.S107624

Andreasen J.O, Bakland L.K & Andreasen F.M. (2006) Traumatic intrusion of permanent teeth. Part 2.A clinical study of the effect of preinjury and injury factors, such as sex, age, stage of root development, tooth location, and extent of injury including number of intruded teeth on 140 intruded permanent teeth. Dent Traumatol. ,22(2), 90-98.

Benenati F.W. (2005) Extracanal invasive root resorption: a perplexing oddity. Gen Dent, 50(1),70-71.

Bergmans L., Van Cleynenbreugel J., Verbeken E., et al. (2002) Cervical external root resorption in vital teeth. J ClinPeriodontol , 29(6), 580-585.

Bosshardt, D.D. (2018), The periodontal pocket: pathogenesis, histopathology and consequences. Periodontol 2000, 76: 43-50.

Butler, D.L., Goldstein, S.A., & Guilak, F. (2000). Functional tissue engineering: The role of biomechanics. J Biomech Eng.122:570–5.

Chandki, R., Banthia, P., & Banthia, R. (2011). Biofilms: A microbial home. Journal of Indian Society of Periodontology, 15(2), 111–114. https://doi.org/10.4103/0972-124X.84377

Deyhle, H., Bunk, O., & Müller, B. (2011). Nanostructure of healthy and caries-affected human teeth. Nanomedicine 7:694-701

Eschler, B.M., & Rapley, J.W. (1991). Mechanical and chemical root preparation in vitro: efficiency of plaque and calculus removal. J Periodontol. 62(12):755-60.

Frank, A.L. (1995) Extracanal invasive resorption: an update. CompendContinEduc Dent. ,16(3), 250-266.

Gandhi, M., & Kothiwale, S. (2012). Association of Periodontal Diseases with Genetic Polymorphisms. International Journal of Genetic Engineering, 2, 19-27.

Gulsahi, A., Gulsahi, K., & Ungor, M. (2007). Invasive cervical resorption: clinical and radiological diagnosis and treatment of 3 cases. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. ,103(3), e65-e72.

Heithersay, G.S. (1999). Treatment of invasive cervical resorption: an analysis of results using topical application of trichloracetic acid, curettage, and restoration. Quintessence Int. , 30(2), 96-110.

Ho, S. P., Marshall, S. J., Ryder, M. I. & Marshall, G. W. (2007). The tooth attachment mechanism defined by structure, chemical composition and mechanical properties of collagen fibers in the periodontium. Biomaterials, 28(35), 5238–5245. https://doi.org/10.1016/j.biomaterials.2007.08.031

Klokkevold, P.R., Newman, M.G., Takei, H., Carranza, F.A., Carranza, F. & Takei, H. (2002). Carranza's Clinical Periodontology, 9e, WB Saunders Co, 859-864

Könönen, E., Gursoy, M. & Gursoy, U. K. (2019). Periodontitis: A Multifaceted Disease of Tooth-Supporting Tissues. Journal of clinical medicine, 8(8), 1135. https://doi.org/10.3390/jcm8081135

Lindskog, S., & Blomlof, L. (1983). Cementum hypoplasia in teeth affected by juvenile periodontitis. Journal of Clinical Periodontology, 10, 443-451.

Madison, S., & Walton, R. (1990). Cervical root resorption following bleaching of endodontically treated teeth. J Endod, 16(12), 570-574.

Mamai-Homata, E., Topitsoglou, V., Oulis, C., Margaritis, V., & Polychronopoulou, A. (2012). Risk indicators of coronal and root caries in Greek middle aged adults and senior citizens. BMC public health, 12, 484. https://doi.org/10.1186/1471-2458-12-484

Minovska, A., & Petrovski, M. (2014). Практикум по претклиничка пародонтологија, Универзитет „Гоце Делчев“-Штип.

Mock, E.S., Wolff, G.K., & Galvan, DA. (1997). Rapidly progressing extracanal invasive resorption. Gen Dent. , 45(1), 66-67.

Nagata, M., Nagata, M., Kanie, T. & Shima, K. (2016). Clinical and Histologic Aspects of Cervical Cemental Tear as a Risk for Periodontal Diseases. Clinical Advances in Periodontics, 6: 167-174.

Pannu, P., Gambhir, R., & Sujlana, A. (2013). Correlation between the salivary Streptococcus mutans levels and dental caries experience in adult population of Chandigarh, India. European journal of dentistry, 7(2), 191–195. https://doi.org/10.4103/1305-7456.110169

Preza, D., Olsen, I., Aas, J.A., Willumsen, T., Grinde, B., & Paster, B. J. (2008). Bacterial profiles of root caries in elderly patients. Journal of clinical microbiology, 46(6), 2015–2021. https://doi.org/10.1128/JCM.02411-07

Rodriguez-Pato, R.B. (2004). Root resorption in chronic periodontitis: a morphometrical study. J Periodontol., 75(7),1027-1032.

Sundaram, S., Ramaseshan, R., Dash, S., & Rao, S.R. (2014). Evaluation of the nanostructure of cervical third cementum in health and hronic periodontitisd: An in vitro study. J Indian Periodontol, 18,560-6