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Gingival Recessions - Pathogenesis and Prognosis: A Literature Review

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Abstract: ***One of the tasks of mucogingival surgery is the prevention and treatment of gingival recessions. Gingival recessions do not lead to tooth loss, but are considered an aesthetic problem and plaque retention factor. Gingival recessions are frequently related to functional problems associated with plaque retention, dentinal hypersensitivity, root exposure, carious and non-carious lesions, alveolar bone loss, and aesthetic shortcoming. The most important prognostic factor for the treatment of gingival recessions is the height of the interproximal supporting periodontal tissues. Successful correction of mucogingival conditions requires a detailed understanding of the etiology and pathogenesis of gingival recessions, because the treating of recessions without addressing the underlying cause can compromise results. The purpose of this study was to provide a literature review of the pathogenesis and prognosis of gingival recessions and to highlight the main etiopathogenetic factors and clinical findings to assist clinicians in their treatment.***

Keywords: gingival recessions, pathogenesis, prognosis, treatment planning, treatment outcome

1. Introduction

One may very well note that is necessary to provide an adequate mucogingival complex in which the mucogingival tissues can retain their biomorphological integrity and maintain a stable attachment with the teeth and the surrounding soft tissues. Generally, there are two ways in which a mucogingival problem occurs. The first one is presented like a close disintegration of the mucogingival complex as a result of which pockets are formed. The second one - like an open disintegration of the mucogingival complex, as a result of which gingival clefts and gingival recession are formed. [1].

The term used to describe the apical movement of the marginal gingiva from its normal position on the tooth crown to the levels on the root surface below the cementoenamel junction (CEJ), is gingival recession. The most widely used classification of gingival recessions is the one by Miller [2], which considers four classes according to the recession extent [3].

One of the tasks of mucogingival surgery is the prevention and treatment of gingival recessions. Gingival recessions do not lead to tooth loss, but are considered an aesthetic problem and plaque retention factor [4]. The prevalence and severity of gingival defects rise with increasing age of individuals [5].

They are more often located on the vestibular surface of the upper frontal teeth and premolars, as well as in the lower incisors [6]. In the aesthetic zone of the maxilla are more common in men [5].

Gingival recessions are frequently related to functional problems associated with plaque retention, dentinal hypersensitivity, root exposure, carious and non-carious lesions, alveolar bone loss, and aesthetic shortcoming. Clinically, a gingival recession with loss of attached tissue can be found, but there are also cases in which no loss of attached tissue is observed [7].

A thorough understanding of the etiology of the gingival recession is necessary for effective correction of mucogingival conditions, as otherwise compromising the results are unavoidable [8].

The purpose of this study was to provide a literature review of the pathogenesis and prognosis of gingival recessions and to highlight the main etiopathogenetic factors and clinical findings to assist clinicians in their treatment.

2. Methods

The article is based on a literature review of various studies (retrospective, randomized, clinical cases) in a database of PubMed and Google Scholar, as well as on an analysis of previous review articles related to the topic.

3. Literature Survey

3.1. Pathogenesis

Gingival recessions can be caused by trauma or bacterial infection and can be the physiological result of tissue ageing and physiological bone resorption, and as a result of anatomical factors due to the incorrect position of teeth in the dental arch [9].
Anatomical gingival recessions are observed in the absence of inflammation and without available loss in the height of the interdental papilla [10].

The pathogenetic mechanism in the formation of gingival recessions of traumatic origin is different from that of bacterial etiology. They can be caused by the individual himself due to improper traumatic brushing, improper use of dental floss, oral piercing, etc., or iatrogenic as a result of the intervention of the attending dentist in orthodontic treatment, improper prosthetic restorations and occlusal relationships [11,12]. In trauma-induced gingival recessions, the etiological factor acts on the healthy oral epithelium on the surface of the gingiva, initially causing abrasion of the

gingival epithelium. As far as is known, the connective tissue that lies between the connective epithelium and the oral epithelium is the area from which the inflammation begins.If the irritation from the external traumatic force continues, it is summed with the irritation from the secondary inflammatory process around the trauma, whereupon gingival connective tissue is directly affected and a gingival ulcer is formed, without any signs of a gingival recession. Upon affecting the entire thickness of the gingival connective tissue, the onset of loss of clinical attachment occurs and, as a result, the root surface is exposed. Upon cessation of trauma, the affected connective tissue re-epithelialize and the gingival recession is clinically detectable [4].

This pathogenetic mechanism is referred to as centripetal, as the pathway to destruction is from the outside-in[4].

The mechanism of pathogenesis is similar in gingival fissures, and in this case, the etiological factor acts from the outside in, causing initially a red lesion confined to the gingival connective tissue and subsequently a white lesion in full-thickness, revealing the underlying root surface [13].

In bacterial-induced gingival recessions, the subgingival bacterial biofilm initially causes an inflammatory response in the connective tissue between the oral epithelium and the sulcular epithelium [14]. If the gingival phenotype is thin, the connective tissue of the gingiva is affected in full by the inflammatory process caused by the subgingival bacterial plaque. As a result, the gingival margin loses support from the underlying connective tissue and proliferates apically from the cemento-enamel junction with clinical attachment loss, forming a periodontal pocket [6]. Therefore, individuals with a thin periodontal phenotype are more prone to the formation of gingival recessions compared to individuals who are characterized by a thick periodontal biotype [15].

In this case, the pathogenetic mechanism is referred to as centrifugal, because the pathway of destruction is inside-out [6].

3.2. Prognosis of gingival recessions

The prognosis of gingival recessions depends on the ability to achieve complete coverage of the exposed root surface. Actually, the most important prognostic factor for the treatment of gingival recessions is the height of the interproximal supporting periodontal tissues (interproximal attachment and the height of the interdental alveolar bone) [16].

The loss of interproximal clinical attachment was analyzed by determining the height of the interdental papillae [17], as well as the height of the interdental alveolar bone by periapical radiography or ConeBeam Computed Tomography (CBCT) [18]. Due to the perpendicular visualization of the teeth, the Bitewing radiograph is also appropriate for the exact measurement of the alveolar crestal bone.CBCT is one of the few methods that allows to analyze the lingual/buccal and palatal surfaces [19,20], as well as provides excellent visualization of the morphology of periodontal defects, which is of great importance in the analysis of dehiscences and fenestration.The new approach allows quality visualization and precise measurement of gingival thickness, dentogingival attachment, and periodontal dimensions, using a tongue/lip retractor. [21].

According to the classification of Nordland and Tarnow (1998) [22], the healthy papilla with normal height - class 0, fill the interdental space to the interdental contact point without loss of interdental clinical attachment (CAL = 0). Periapical radiography and the CBCT radiographic image of a healthy periodontium show no loss of interdental bone and the height of the interdental alveolar crestis at a distance of approximately 1 mm (not more than 2 mm) from the CEJ of the tooth [23].

The normal height of the interproximal supporting periodontal tissues (interproximal attachment and the height of the interdental alveolar bone) enable to cover completely the exposed root surface and restore the correct position of the gingival margin on or coronary to the CEJ level.An accurateidentification of the CEJ location is a key factor in treatment planning [24,25].

Miller`s classification is the most commonly used in practice. In 1985, P.D. Miller has based his classification of gingival recessions mainly on two aspects: the prognosis of gingival recessions, and the degree of interdental loss of soft and hard tissues in the approximal areas adjacent to the gingival defects [2].

The classification includes 4 classes of gingival recessions, taking into account the anatomical criteria and therapeutic possibilities:

a) Class I – marginal tissue recession not extending to the mucogingival junction (MGJ). No loss of interdental bone or soft tissue. The CEJ is visible only on the facial surface of the tooth and the gingival margin of the recession is located coronally to the MGJ without crossing it. The interdental papillae are preserved and fill the interdental spaces. The tooth is not rotated, extruded or malpositioned. Complete root coverage is obtainable [2,26].

b) Class II - no resorption of interdental bone and no clinical attachment loss of the approximal tooth surfaces. The gingival recession reaches or passes apically MGL. The interdental papillae are preserved and fill the interdental spaces. The tooth is not rotated, extruded or in a malposition. It is possible to cover 100% of the recession [2].
   - Subclass IIa - the most apical point of the gingival recession is apically from the MGJ in the adjacent teeth, but apical from the exposed root surface there is a marginal keratinized gingiva.
   - Subclass IIb - the most apical point of the gingival recession passes apically from the MGJ into the alveolar mucosa without the presence of marginal keratinized gingiva apically on the exposed root surface.

   Class I and class II gingival recessions affect teeth with intact periodontal tissues. The difference between class I and class II is based on the presence or absence of facial

keratinized gingival tissues apically to the defect. However, in both classes is possible full coverage (100%) of the exposed root surface.

The division of class II gingival recessions into subclasses is important when planning the therapeutic approach.

c) Class III – marginal tissue recession which extends to or beyond the MGJ and is associated with alveolar bone loss in the interdental area. The tooth may be slightly rotated, malpositioned or extruded. The interdental papillae do not fill the interdental triangles. Partial root coverage is obtainable [2].

The prognosis of this class of gingival recessions is associated with partial coverage. Partial coverage of the exposed root surface relative to the contour of the interproximal soft tissues (50-70%) is possible. This is the prognosis in the treatment of gingival recessions that affect slightly rotated teeth. Other studies suggest that root surface coverage in class III mucogingival defects is unpredictable, but even complete root coverage could be achieved [27,28,29].

d) Class IV - marginal tissue recession which extends to or beyond the MGJ and is associated withseverealveolar bone loss in the interdental area with exposure of more than one proximal root surfaces. The prognosis of this class of gingival recessions is difficult to predict, as in teeth with severe malposition. It is impossible to cover the exposed root surface - predictability below 10% [2].

Therefore, class IV gingival recessions are diagnosed in teeth with interproximal loss of clinical attachment equal to or greater compared to facial loss of clinical attachment.

4. Discussion

The periodontal therapy aims to improve the periodontal health and function of the patient's dentition [7]. However, the aesthetic factor is also an inseparable part of periodontal treatment and today is of leading significance and is of paramount importance to the patient. As a matter of fact, the most common indication in surgical procedures to cover the exposed root surface is the aesthetic element. Other less common indications are dental hypersensitivity, and the role of gingival recessions as a plaque-retentive factor.

Etiological and predisposing factors are leading factorsin planning the treatment of localized gingival recessions [6].

When developing a treatment strategy, clinicians should first focus on susceptibility factors and modifiable conditions while increasing the patient's awareness about gingival recession [30].

Therefore, a correct prognosis of gingival recessions will allow for the proper treatment of these conditions and the improvement of the lifestyle of the affected individuals [7].

The literature review of the pathogenesis and prognosis of gingival recessions in this article provides an opportunity to increase knowledge in different types of gingival recessions and highlights some important diagnostic features.

5. Conclusion

One of the patient's major esthetic complaints is gingival recession. This also exposes the sensitivity and higher risk of root caries on the patients. Therefore, the correct diagnosis, prognosis and selection of the appropriate therapeutic approach by the clinician are mandatory. In these cases, periodontal therapy aims to restore the health, aesthetics and function of the periodontium.

Regarding the proper planning of the treatment of gingival recessions, a good knowledge of the etiopathogenetic factors and their prognosis would facilitate the prevention of recurrence of these conditions and the work of the treating clinician.

Abbreviations

CEJ - cementoenamel junction
MGJ – mucogingival junction
CAL – clinical attachment level
CBCT –cone-beam computed tomography

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