

MARZENA DOMINIAK^{1, A, D}, TOMASZ GEDRANGE^{1, 2, E-F}

New Perspectives in the Diagnostic of Gingival Recession

¹ Department of Oral Surgery, Wrocław Medical University, Poland

² Department of Orthodontics, Technical University of Dresden, Germany

A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation; D – writing the article; E – critical revision of the article; F – final approval of article; G – other

Abstract

Gingival recession (GR) is a common clinical situation observed in patient populations regardless of their age and ethnicity. It has been estimated that over 60% of the human population has gingival recession. It is the final effect of the interaction of multiple etiological factors. Identification and definition of the range of influence is often not possible, with the result that new methods for testing and elimination of potential etiological factors are still being sought. The aim of this study is to present the etiopathogenesis of gingival recessions with regard to the analysis of morphological and functional factors. For the assessment of the bone factors, we will describe the new cephalometric method for measuring sagittal width of the bone in the central incisors area, places when GR are most commonly observed. Also, a review will be presented of modern methods of treatment; in particular classes recessions; usage substitute of autogenous tissue will be emphasized – collagen matrix, and primary culture fibroblasts on collagen net (*Adv Clin Exp Med* 2014, 23, 6, 857–863).

Key words: gingival recession, aetiology, diagnostic, cephalometric analyses, incisors area.

An adequate width of attached gingiva is necessary to maintain a healthy periodontium, especially in orthodontics or restorative treatments in periodontics. The periodontium or mucogingival complex consists of free and attached gingiva, mucogingival junction (MGJ) and the alveolar mucosa [1, 2]. The attached gingiva is defined as the tissue between the MGJ and the projection on the external gingival surface of the most apical portion of the gingival sulcus or periodontal pocket. The width of the attached gingiva is genetically predetermined and varies in different areas of the dentition, and its dimension can change throughout life [3]. The consequences of mucogingival problems could be, on the one hand, pocket formations, due to a close disruption of the complex. Otherwise, open disruption of the mucogingival complex results in gingival clefts and gingival recession [1]. A mucogingival problem is, then, defined as the presence of gingival inflammation and gingival recession in areas with little or no attached gingiva [3].

Prevalence, Etiology and Classification of Gingival Recession

Gingival recession is defined as the displacement of marginal periodontal tissues apically to the cemento-enamel junction [3, 4]. This means that the marginal gingiva shifts from its normal position on the crown of the tooth to the levels on the root surface beyond the cement-enamel junction [2] (Fig. 1). The following consequences of gingival recession are known: tooth hypersensitivity, pain, caries and non-caries lesion, aesthetic distress, gingival bleeding, and plaque retention [4, 5].

Gingival recession is the final result of the impact of many etiological factors. The extent of the impact is often impossible to establish. Finding new and/or rating yet unknown factors and determining their impact is the essence of cognitive dental scientific research, especially in the context of high and ever-increasing incidence of gingival



Fig. 1. Facial view of gingival recession on anterior maxillary right teeth

recession. Morphological factors play an important role in the development of recession. These factors in coexistence with other factors may give rise to changes in the mucogingival complex. Therefore, in today's classification, the following etiological factors are sub-classified in morphologic conditions (I) and driving forces (II–V) giving rise to gingival recession:

I. Primary morphological conditions

- a) bone determinants – type of the alveolar bone (Class D1–D4 by Mish – ratio for cortical and cancellous bone), the anatomic structure of the mandibular symphysis, bone density, size and shape of the alveolar bone,

- b) mucous determinants – thickness of the keratinized gingiva, anatomy and position of the lip frenula, tongue frenulum and the buccinators muscle frenula, the depth of the oral cavity vestibule,
- c) teeth determinants – shape and dimensions of the teeth, abnormal topography of the teeth (Fig. 2).
- d) muscle determinants – strength and length of the muscle attachments, especially the mentalis muscle.

II. Functional factors

a) endogenic (primary):

- 1) oro-muscle dyskinesia, such as infantile swallowing pattern, the habits of the mentalis muscle,
- 2) occlusal and non-occlusal parafunctions,
- 3) posture disorders.

b) exogenic (secondary) – traumatic:

- 1) dental brushing (mechanical trauma),
- 2) centric and eccentric occlusion disorders (mechanical trauma),
- 3) iatrogenic damage during dental treatment (mechanical or chemical) (Figs 3, 4),
- 4) piercing (mechanical),
- 5) smoking (chemical).

III. Inflammation factors (secondary)

- a) poor oral hygiene, b) periodontitis.

IV. Age, sex (secondary factors)

V. General diseases (secondary factors) [6, 7].



Fig. 2. Morphological Miller's classification of gingival recessions in four different classes [6]

Gingival recession is a common clinical occurrence observed in patient populations regardless of their age and ethnicity. It has been estimated that over 60% of the human population has gingival recession [8]. Gingival recession was found in 64% of all tested subjects aged between 20 and 29 in Brazil. 99% of the analyzed individuals older than 40 years showed displacements of marginal periodontal tissue in the same country. Furthermore, the increase in age led to an increase in the mean number of affected teeth [9, 10]. The prevalence of gingival attachment loss in Pomerania, a province in Eastern Germany, was 89.7% in adults (20–81 years of age) with 62.8% of teeth

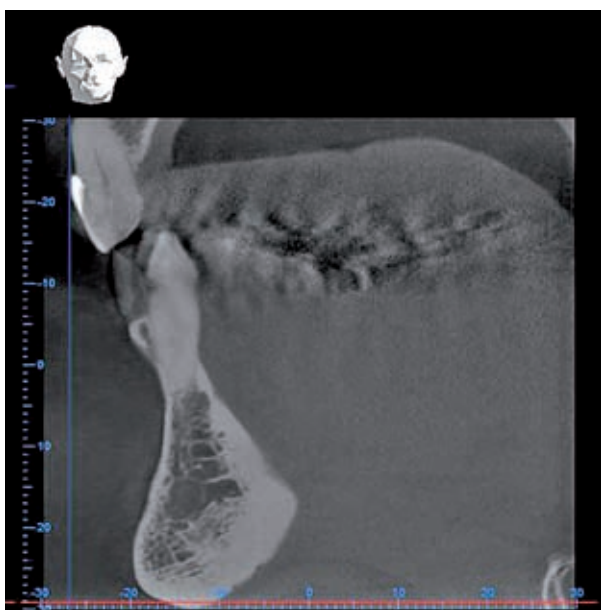


Fig. 3. Lateral X-rays of mandibular incisors reclined front teeth with normal bone level

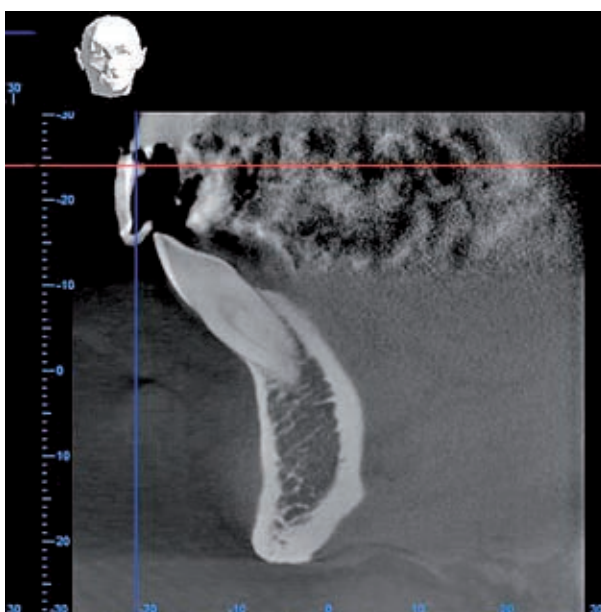


Fig. 4. Proclined front teeth with reduced alveolar bone

being affected [11, 12]. In contrast, in Norway only 51% of the adult subjects over 18 years of age had these defects [9]. This correlates with findings from Germany, where 50% of healthy young adults (19–30 years) presented gingival recession at one or more sites [13]. Interestingly, there was no significant difference in the prevalence of gingival recession between smokers and non-smokers [13]. Dodwad could show that 804 subjects out of 1200 tested subjects showed gingival defects in India. 67% of these patients were males and 33% females [1]. Furthermore, it was demonstrated that about 87% of these patients showed gingival recession in the mandibular anterior region [1]. This is in correlation with the findings from Marini et al. [10]. They showed that the mandibular teeth displayed more surfaces with gingival recession than the maxillary teeth and the mandibular incisors were the most affected teeth.

Similar trends were globally showed in epidemiological studies: i) gingival recession increases with age, ii) buccal surfaces are most commonly affected, iii) lower anterior teeth were more frequently affected and iv) men are more affected than women [4]. The severity of gingival recession can be classified using 2 different classification systems. Sullivan and Atkins categorized the gingival defects into deep-wide, shallow-wide, deep-narrow and shallow-narrow [14]. In contrast, the Miller classification categorized the defects into 4 classes (Fig. 2): class I (marginal tissue recession without extending to the mucogingival junction without bone and soft tissue loss in the interdental areas), Class II (marginal tissue recession extend to the mucogingival junction, but there is no loss of interproximal tissue), class III (marginal tissue recession extend to the mucogingival junction with loss of interproximal soft and bone tissue, secondary malposition teeth), and class IV (class III subsequently followed by severe tooth malposition) [15].

Surgical Therapy of Gingival Recession

Proper mucogingival therapy should lead to gingival augmentation and create a vestibule with adequate depth in the regions with insufficient attached gingival tissues. Several different techniques have been described in order to cover the exposed root surface, and increase the dimensions of the keratinized tissue in gingival recession defects (Table 1). Multiple gingival augmentation techniques with different success are described in the literature [16–19]. Generally, surgical techniques are used to cover recessions with different flaps-ex. rotational and lateral sliding as well as coronally

Table 1. Methods of gingival recession treatment

1. PEDICLE FLAP	
Rotation flap Laterally reposition flap Double position flap	coronally reposition flap trapezoidal flap semilunary flap envelope flap
2. TUNEL TECHNIQUE	
3. FREE GINGIVAL GRAFTS	
Full thickness grafts	connective tissue grafts
4. SOFT TISSUE SUBSTITUTES	
Allo- or kseno- dermal grafts	collagen, gelatin, fibrin or synthetic scaffolds
5. TISSUE ENGINEERING TECHNIQUE	
6. GUIDED TISSUE REGENERATION	
Nonresorbable membrane	resorbable membrane
7. BIOLOGICALLY ACTIVE PROTEINS	
Amelogenins	polipeptyd growth factors recombination growth factors

repositioned flaps or tunnel technique and to augment mucogingival defects, the free gingival auto-graft, the sub-epithelial connective tissue graft, and the acellular dermal matrix grafts [5, 20]. The connective tissue graft in conjunction with a coronally advanced flap is the gold standard treatment for gingival recession defects [21].

Guided tissue regeneration-based root coverage is also one of the approaches for the treatment of gingival defects. This technique was first described by Pini Prato et al. [22]. In this study, results from patients treated with a surgical technique involving membranes were compared to those from patients undergoing mucogingival surgery (free gingival graft and a coronally positioned flap). It was found that the amount of root coverage was similar in both groups, whereas the clinical attachment gain and pocket variation differed significantly [22]. Saczko et al. described recently the isolation of primary fibroblast cells from connective tissue of oral cavity keratinized gingiva and setting on a collagenous carrier [23]. These scaffolds composed of primary fibroblast cells; isolated from connective tissue of the oral cavity keratinized gingiva and a collagenous membrane are effective for gingival augmentation, because recession depth and width was decreased. Furthermore, the height of the keratinized tissue was significantly increased. In addition, the post-procedure aesthetic increased in all cases compared to pre-procedure conditions [24] (Figs 5, 6). In addition, in a 6-month observation a significant decrease in the distance from cemento-enamel junction to

**Fig. 5.** Before surgical treatment**Fig. 6.** Eight years post surgical evaluation of gingival recession after surgical treatment using scaffolds composed of primary fibroblast cells isolated from connective tissue of oral cavity keratinized gingiva and a collagenous membrane

mucogingival junction was revealed. Histologically, mature connective tissue covered by keratinized epithelium was found after 12 weeks. No specimens revealed an inflammatory response. A complete clinical healing was observed after 2 weeks in 8 out of 10 patients [25]. This is in agreement with further studies using human gingival fibroblasts on a biodegradable collagen scaffold [26, 27]. Using these scaffolds a significantly increased amount of attached gingiva at the test site compared to the control site was observed [26].

It is also possible to obtain root coverage without harvesting connective tissue. It was shown recently that Miller Class I and II recession defects can be treated with collagen matrix derived from porcine. A long junctional epithelial attachment as well as connective tissue adhesion was noted [21].

Diagnosis of Gingival Recession

The identification of gingival recession defects is essential, especially in prophylaxis/prevention as well as in dental treatment. Periodontal examination for mucogingival problems in Class I and II defects include measurements of gingival thickness and height, vestibular depth, the distance from cemento-enamel junction to gingival margin (recession depth) and from gingival margin to the mucogingival junction as well as bleeding index, and analyses of recession and inflammation. These studies evaluate a visible recession between the tooth and gingival edge from a clinical view. The second method is the hidden recession. It is covered by the gingiva area between the edge of the gingiva and the farthest point in the direction of the apex, in which the connective tissue trailers. The sum of “visible and invisible” recession matches the loss of connective tissue attachment to the tooth surface [28]. For the assessment of bone width dimension lip-linguistic methods are direct (invasive) and indirect (non-invasive like X-ray). The most important objective is to analyze X-ray images. Extensive use of cephalometric images in orthodontic diagnosis, however, did not result in the creation of many methods for assessing the alveolar width dimension between the tongue and lip in the anterior mandible. There are several limitations in the assessment of the alveolar, width dimension between lip and tongue. This method does not allow an accurate assessment of individual incisors as individual structures in the sagittal plane overlap. Because there is a large picture summation, difficulty in the interpretation of the various anatomical structures in this section appeared. In particular, it is difficult to identify the vestibular alveolar plate [29]. In addition, the resulting image

is enlarged approximately 3.45%, as compared with computed tomography, where the magnification is 1.2%, representing almost 3-fold difference [30]. A semi-optimal method has been presented by Handelman [31]. Artun et al. also measured the width of the jaw in the cephalometric pictures [32]. These studies, however, were very inaccurate.

The construction of a finite element model from computer tomographic and micro-CT images is a suitable method for the analysis of osseous defects [33]. For the identification of Miller Class I and II recession defects, we established a simple method assessing the labial width of bone in the frontal area of the mandible in sagittal direction (angle API-CEJ2-B) and genial muscle width based on lateral X-rays in occlusion. On the model, the author’s angle of bone labial width (CEJ2-B), genial muscle width and bone dehiscence height (CEJ2-B) were determined. Furthermore, measurements enabling the assessment of incisors location in alveolar process were made according to Ricketts, Björk and Steiner’s cephalometric analyses and, additionally, Holdaway’s analysis (Figs 7, 8). Using this diagnostic method a significantly lower value of API-CEJ2-B angle and higher bone dehiscence was found in patients with gingival recession compared to that of healthy subjects. Furthermore, the angle of labial width of bone in the front part of mandible and the genial muscle width was significantly correlated to the height of CEJ2-B and line

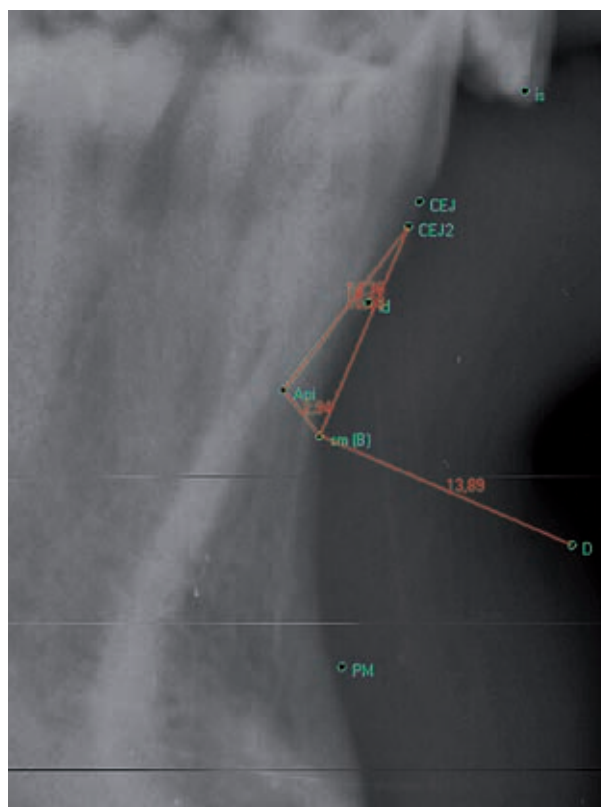


Fig. 7. Measurement on lateral X-rays in occlusion [7]

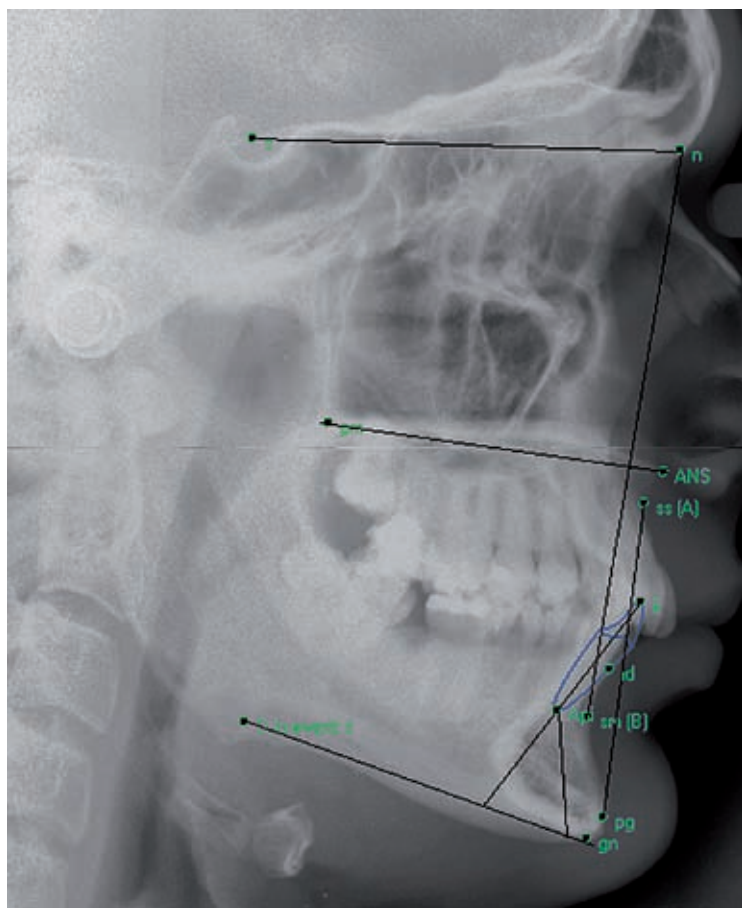


Fig. 8. Cephalometric analyses of angle of bone labial width (CEJ2-B) and bone dehiscence height (CEJ2-B) on lateral X-rays in occlusion [7]

incisor proclination (ii/NBmm) in patients with gingival recession [7].

Conclusion

Taking into account the advantages and disadvantages of all indirect methods of assessing the

width of the mandibular alveolar ridge dimension between tongue and lip and its relation to the roots of the incisors, it is clear that the CT method would be the best way to evaluate these structures. Due to the still limited availability of this method and the inability to assess the relationship of incisors to other craniofacial anatomical structures, the routine photo used for this purpose is still cephalometric X-ray.

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Address for correspondence:

Marzena Dominiak
Department of Oral Surgery
Wroclaw Medical University
Krakowska 26
50-425 Wroclaw
Poland
Tel.: +48 71 78 40 251
E-mail: marzena.dominiak@wp.pl

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