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Gingival Recession in Orthodontics: A Review

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ABSTRACT

By clinical definition, gingival recession refers to the exposure of the root surface by an apical shift in the position of the gingiva. Recession is important because it can lead to poor esthetics, tooth hypersensitivity, loss of periodontal support, difficulties in maintenance of oral hygiene, and increased susceptibility to caries. Although its etiology is not fully understood, periodontal disease and mechanical trauma are considered the primary factors in the pathogenesis of gingival recession. Orthodontic treatment might also promote the development of gingival recessions. It has been well established that orthodontic forces can move roots close to or through the alveolar cortical plates, leading to bone dehiscence. In such instances, the marginal gingiva, without proper alveolar bone support, might be expected to migrate apically and lead to root exposure. This assumption is based on the fact that in areas of recession, a subjacent alveolar bone dehiscence is always present.

Keywords: Orthodontics, Gingival recession.

1. Introduction

By clinical definition, gingival recession refers to the exposure of the root surface by an apical shift in the position of the gingiva. It can be localized or generalized, and associated with one or more tooth surfaces [1]. Several aspects of gingival recession make it clinically significant. First, recession signifies a loss of attachment. In areas of recession, the epithelial attachment has migrated apically from the CEJ. Second, root surfaces exposed as a result of gingival recession are more susceptible to caries. In a sample of 452 adults aged 65 or older, Lawrence et al [2] showed that indicators of poor periodontal status, including gingival recessions, were significantly correlated with an increased risk of root caries development. Additionally, studies that have examined the prevalence of root caries have shown higher levels of lesions in patients with periodontal disease and recession compared to patients without recession [3]–[5]. Third, abrasion or erosion

of the cementum exposed by recession leaves an underlying dentinal surface that can be sensitive. Al-Wahadni and Linden [6] demonstrated that gingival recession of 3 mm or more was the best independent predictor of dentin hypersensitivity. In another study, Costa et al [7] showed that gingival recession was associated with increased dentin hypersensitivity in 1,023 adults aged 35 or older in Brazil. In addition to these complications, authors have proposed other clinical problems associated with recession, including difficult maintenance of oral hygiene [8] and compromised esthetics [8]–[10]. For instance, Rocha et al [10] showed that when the esthetic perception of smiles was evaluated by 160 dental students, there was a statistically significant difference between smiles with gingival recession and smiles without. This presents a problem because esthetics is a major motivational factor for patients to seek orthodontic treatment [11], [12]. For example, Reidman et al [11] showed that for 75% of patients seeking orthodontic treatment, dental esthetics was their primary motive. Therefore, if orthodontic treatment contributes to the formation of gingival recession, there are a number of significant clinical and psychological problems that may result.

2. Anatomy of The Attachment Apparatus

In order to better understand recession, a review of the soft tissue attachment apparatus is necessary. In buccal-lingual cross section, the gingival epithelium forms a crevice around the tooth. On the tooth side, the gingival epithelium is termed the sulcular epithelium, which, along with the tooth, forms the boundaries of the gingival sulcus. The area apical to the unattached sulcular epithelium, termed the junctional epithelium, forms an epithelial attachment to the tooth surface itself. Just apical to the epithelial attachment, densely packed collagen bundles are anchored into the cementum, forming the connective tissue attachment. Apical to the connective tissue attachment is the periodontal ligament (PDL). Therefore, the element of space that healthy gingival tissues occupy between the sulcular base and the underlying alveolar bone is comprised of the junctional epithelial attachment and the connective tissue attachment. The combined attachment width is identified as the biologic width. Garguilo et al [13] found that, in the average human, the connective tissue attachment measures 1.07 mm, and the junctional epithelial attachment measures 0.97 mm.

3. Measurement of Gingival Recession

Gingival recession is typically measured during a clinical examination with a periodontal probe. It is recorded as the distance in millimeters from the CEJ to the gingival crest to better understand recession, however, it helps to distinguish between the actual and the apparent positions of the gingiva. The actual position is the level of the epithelial attachment on the tooth, whereas the apparent position is the level of the crest of the gingival margin. The difference between the actual and apparent position of the attachment is the sulcus depth. As mentioned previously, in a completely erupted tooth with healthy periodontal tissues, the coronal portion of the epithelial attachment is located near the CEJ [1]. In the strictest sense, the severity of recession is determined by the actual position of the gingiva, not its apparent position, and is measured from the CEJ to the actual position. However, measuring the actual position of the gingiva is inherently difficult, and therefore the most widely accepted measurement of gingival recession is from the CEJ to the gingival crest. Methods of measuring recession without having to perform a clinical examination have been developed. The two most widely utilized substitutes are dental casts and intraoral photographs. Dental casts have been successfully used to measure recession as a dichotomous level variable. Renkema et al [14] assessed the validity of using dental casts for scoring recessions on 30 adults. The clinical exam and dental cast comparison produced a mean kappa score greater than 0.80, suggesting good agreement. Intraoral photographs have also been used to measure recession as a dichotomous level variable. To assess the method error in evaluating gingival recession from intraoral photographs, Ruf et al [15] conducted replicate analyses of photographs of 10 randomly selected subjects and found a concordance in 80% of the subjects and 92.5% of the teeth. In both of the studies mentioned above, recession was considered present if the CEJ was exposed. Allais and Melsen [16] measured the reliability of both of these methods and found that the number of unreadable teeth

was larger when performed on casts than when assessed on intraoral photographs, and the variation in the error of the method was smaller for the photographs than for the cast analysis. The authors speculated that the reliability was better for the intraoral photographs compared to the dental casts because the color contrast between the enamel and cementum helped to distinguish the CEJ in the photographs. Also, dental casts may have artifacts around the gingival margin.

A method for measuring recession as an interval level variable using a combination of dental casts and intraoral photographs was established by based on a method originally applied by Coatoam et al [17]. In the study by Coatoam et al [17], the width of keratinized gingiva on the facial surface of the mandibular incisors was measured from slides that were calibrated for size using the corresponding dental casts. They reported an error of the method of 0.1 mm. Trentini et al [18] compared this method of using dental casts and intraoral photographs with the corresponding measurements made during a clinical examination. Using dental casts and intraoral slides, examiner reliability was evaluated on 10 randomly selected patients from their study.

4. Prevalence and Demographics of Gingival Recession

Population-based studies have shown that the development of gingival recessions is correlated with age. The prevalence is lower at younger ages and increases over time [19]–[22]. For example, in a sample of 299 children and teenagers, Ainamo et al [19] showed that at least one gingival recession of 0.5 mm or more, measured from the CEJ to the gingival margin, was present on 5% of 7 year olds, 39% of 12 year olds, and 74% of 17 year olds. Susin et al [22] examined 1,586 individuals aged 14 years and older. Measuring recession clinically from the CEJ to the gingival margin, they demonstrated that recessions of more than 3 mm were present in 6%, 24%, and 54% of patients aged 14 to 19, 20 to 29, and 30 to 39 years, respectively. Using a sample of 9,689 persons from data collected in the third National Health and Nutrition Examination Survey (NHANES III), Albander and Kingman [20] demonstrated that the prevalence, extent and severity of gingival recession increased in individuals aged 30 and over. In this study, 56% of individuals aged 40-49, 71% of individuals 50-59, 80% of individuals aged 60-69, 87% of individuals 70-79, and 90% of individuals aged 80-90 showed 1 mm or more of gingival recession on at least 1 tooth. Some teeth have a significantly higher prevalence of gingival recession than others. For example, from the NHANES III data, Albander and Kingman [20] noted that in the maxillary arch, the two teeth that presented with gingival recession most often were the first premolars and first molars. In patients aged 30-55 years, 20.3% of first maxillary premolars showed recession on the labial surface, compared to 21.1% of maxillary first molars. The same pattern was noted in the maxillary arch for patients aged 56-90 years. For this group, 39.5% of first premolars exhibited recession, and 48.4% of first molars had recession. In the mandibular arch, the central incisors and the first premolars showed the most recession. Approximately 19.7% of central incisors showed labial gingival recession, and 19.5% of first premolars showed recession in the 30-55 year old group. The lateral incisor and second premolar were the next highest, exhibiting 13.2% and 14.5%, respectively. For the 56-90 year old group, 49.7% of central incisors were affected, and 43.5% of first premolars. The lateral incisor was the next highest in this group, with 42% of teeth showing recession. This pattern of recession has been demonstrated in other non-orthodontic samples [19], [22]–[25]. Recession also appears to be much more prevalent on the facial surface when compared to the lingual surface of teeth. In the study by Ainamo et al [19], gingival recession was evaluated on the facial and lingual surfaces of 299 Finish school children aged 7 to 17 years. Of the 5,895 teeth examined, recession was found on the facial aspect of 512 teeth (8.7%) and on the lingual aspect of only 16 teeth (0.3%). The increased prevalence of recession on the facial surface as opposed to the lingual surface is supported by a study by Loe et al [26]. They also demonstrated that lingual recession seems to appear later in life.

Additional demographic features that appear to be related to gingival recession include sex, ethnicity, and socioeconomic class. Using the NHANES III data, Albander and Kingman [20] demonstrated that males

aged 30 or more had significantly more recession than females of the same age. The results of Susin et al [22] corroborate these findings. However, in subjects younger than 30, their sample showed no significant sex differences. Ainamo et al [19] reported similar results. In their sample, no significant sex differences were noted among 17 year olds. Therefore, it appears that there are no sex differences at the younger ages, but as individuals' age, males exhibit more recession than females. The NHANES III data also indicated that of the three race/ethnic groups studied, non-Hispanic blacks had the highest prevalence and extent of gingival recession [20]. Mexican Americans had similar prevalence and extent of gingival recession compared with non-Hispanic whites [20].

5. Attrition and Eruption Effects

The correlation between age and recession has led some investigators to assume that recession may be an age related physiologic process, and that maturation brings about greater exposure of the tooth outside its investing soft tissues. The relative apical movement of the attachment after this result more from vertical growth of the jaws and the accompanying eruption of the teeth than from apical migration of the gingival attachment. By the time vertical growth of the jaws has slowed to the adult rate, which is typically in the late teens, the gingival attachment is usually near the CEJ. At one time, it was thought that *passive eruption*, which is the exposure of the teeth by apical migration of the gingiva, played a large role in this process. However, it now appears that if the gingival tissues are entirely healthy, this sort of downward migration of the soft tissues does not occur [1]. What was once thought to be passive eruption during the teens is really *active eruption*, compensating for the vertical jaw growth occurring at that time. Some reports show migration of the junctional epithelium from its position in healthy individuals (i.e., near the CEJ) to a more apical position on the root surface, with accompanying gingival recession [27]. In other animal studies, however, no apical migration has been noted [28]. With continuing gingival recession, the width of the attached gingiva would be expected to decrease with age, but the opposite appears to be true [29], [30]. This has been termed *physiologic recession*. There is some evidence that supports physiologic recession. Human skull studies have demonstrated the continuous eruption of the mandibular molars throughout life, without a compensatory migration of the surrounding hard tissue [31], [32]. In a study by Whittaker et al [31], the eruption and alveolar bone levels of the mandibular first molars was evaluated on the jaws of an eighteenth-century population whose tooth wear had been minimal. The measurements of eruption and bone levels were made on 122 human skulls using a stable reference point, the inferior alveolar canal. Over a 40-year period, they showed that the mandibular first molar erupted 2.8 mm on average, while the alveolar crest remained relatively stable. The alveolar crest increased by only 0.7 mm over the 40-year period. In a very similar study, Varrela et al [32] showed similar results in a population that had much more attrition. Their sample consisted of 244 individuals from the medieval period. Again, the inferior alveolar canal was used as a stable reference point. Their results showed that the first molar experienced attrition of 2.99 mm over a 30-year period, and erupted 2.25 mm over that same time period. Like the study by Whittaker et al, the alveolar crest remained stable, indicating that despite continuous eruption of the teeth, there was no concomitant growth of the alveolar crest. In this study, only a weak negative correlation was found between eruption and attrition (Spearman's rho = -0.15). However, in other similar studies on human skulls, stronger correlations have been found, such as in the studies by Newman and Levers [33], [34]Levers and Darling [34] and Whittaker et al [35]. Therefore, it is possible that as we age, if there is significant attrition and compensatory eruption, it may be a contributing factor in recession.

6. Etiology

Numerous factors have been implicated in the etiology of gingival recession, including faulty tooth-brushing technique (gingival abrasion) [1], [36], gingival inflammation and periodontal disease [26], [37], friction from soft tissues (gingival ablation) [38], and iatrogenic dentistry. Trauma from occlusion has been suggested in the

past, but its mechanism of action has never been demonstrated. A relationship may also exist between smoking and gingival recession [39], [40]. The multifactorial mechanism may include alterations in the immune response, such as decreases in the phagocytic function of polymorphonuclear leukocytes [41], [42] and reduction in the production of immunoglobulins [43]. Reduction in gingival blood flow as a result of smoking is also a possible contributing factor [44], [45]. Finally, orthodontic treatment has been implicated in the development of gingival recessions [46]–[51]. Orthodontic treatment may cause recession because fixed appliances act as a retention area for plaque. In patients without orthodontic appliances, plaque levels range from 10.3% to 13.3%, depending on the population [52]. In a recent study by Klukowska et al [53], plaque levels ranged from 5.1% of tooth surfaces to as high as 85.3% in patients undergoing treatment with fixed appliances. The mean plaque coverage in their study was 41.9 ±18.8%. If this plaque is not adequately removed, the ensuing gingival inflammation may lead to periodontal breakdown, and therefor recession [1], [46], [47]. Specific anatomical attributes may also place an individual at an increased risk of recession. Susceptibility to recession may be influenced by the position of the teeth in the arch, the root-bone angle, and the mesiodistal curvature of the tooth surface [54], [55]. Moreover, there is also evidence suggesting that thin gingival tissue (thin biotype/phenotype) is more prone to recession [56], [57]. Many authors have hypothesized that on rotated, tilted, or facially displaced teeth, the bony plate is thinned or reduced in height. Subsequent pressure from mastication or aggressive tooth brushing damages the unsupported gingiva and may produce recession. This hypothesis seems to be supported by the literature. To determine the prevalence of gingival recession, as well as the etiologic factors associated it, Parfitt and Mjor [60] examined 668 school children aged 9 to 12 years. Trott and Love [54] investigated a group of 766 high school students aged 14-19 in a similar study. Finally, Gorman [55] examined 164 subjects aged 16-86 years for recession. In teeth in pronounced labioversion, 61% were found to have some degree of gingival recession, as well as 15% of teeth in pronounced linguoversion.

7. Effects of Incisor Proclination on Surrounding Hard and Soft Tissues

In 1981, Steiner et al [46] utilized monkeys to evaluate dehiscence formation during labial tooth movement. They moved the central incisors of five Macaca nemitrina monkeys a mean distance of 3.05 mm over 13 weeks. The movement was followed by a stabilization period of 3 weeks. Afterwards, the teeth and surrounding tissues were evaluated with periodontal flap surgery. Using an amalgam marker on the facial surfaces of the teeth as a stable reference point, they were able to measure the changes in the distance to the marginal bone, the connective tissue attachment, and the gingival margin. Their results showed that all three measurements were significantly different compared to controls. For the marginal bone height, the experimental group showed an average of 3.96 mm of labial bone loss compared to controls. The connective tissue attachment and gingival margin migrated apically, 1.04 mm and 0.81 mm, respectively, compared to controls. In this case, the marginal bone loss was much greater than the amount of soft tissue loss. Eight months later, Engelking and Zachrisson [58] took the same animals and moved the same teeth back lingually into position with fixed appliances. The incisors were retracted a mean distance of 1.8 mm and then retained for 5 months. The animals were sacrificed and clinical and histological examinations were performed. The marginal bone levels recovered, relative to their original levels, an average of 2.5 mm and 3.1 mm for the maxillary and mandibular incisors, respectively. Bone histomorphometric analysis with tetracycline labels demonstrated significant osteogenesis in the periodontium of the retracted teeth. In 1982, Karring et al [59] performed a similar study using beagle dogs. In 1987, Wennestrom et al [47] again showed similar results. In their study, the maxillary central incisors of 5 Macaca Cynomolgus monkeys were protracted an average of 3.3 mm over a period of 3-4 months. The teeth were then stabilized for a period of 1 month. All ten of the experimental teeth experienced facial bone loss. On average, the facial bone migrated apically 2.08 mm compared to control teeth. In contrast, only five of the ten test teeth exhibited apical migration of the gingiva.

8. Effects of Expansion on Surrounding Hard and Soft Tissues

One of the negative aspects of orthodontic expansion is uncontrolled tipping of the clinical crowns. In 1996, Lundgren et al [60] utilized light forces (50cN) in a human model to expand the maxillary premolars. They noted movement in all three planes of space. In fact, the apices of the premolars moved palatally in 49 of 56 cases, and they tipped between 0.2 to 22.9 degrees. Nineteen patients with moderate to severe crowding (5 mm or more) were treated following the published Damon System protocol. Treatment in the transverse dimension was limited to expansion with the normal archwire sequence used in the Damon System. The study showed effective expansion of the dental arches with increased arch perimeter. However, the increase in arch width was in part due to tipping of the crowns. On average, the first and second maxillary premolars expanded more at their cusp tips than at their lingual gingival margins. For the maxillary first premolar, the difference was 1.7 mm, and for the maxillary second premolar the difference was 1.6 mm. In 2011, Cattaneo et al [61] evaluated transverse movements and buccal bone modeling in humans after orthodontic archwire expansion. Sixty-four patients were randomly assigned to treatment with either active (In-Ovation R) or passive (Damon 3MX) brackets. Outcomes were evaluated with digital models and pre and post-treatment cone beam CT radiographs. They found that in all but one patient, transverse expansion was achieved through buccal tipping. Specifically, the Damon group showed 11.7 degrees of tipping at the first premolars and 13.5 degrees of tipping at the second premolars. The In-Ovation group had 11.8 degrees and 13.0 degrees of tipping of the same teeth. Kraus et al [62] used foxhound dogs to evaluate archwire expansion using mechanics similar to those used with the Damon system. Over eight weeks he saw an average of 3.5 mm of tooth movement. The buccal movement was accompanied by a significant amount (15.8 degrees) of tipping. Poor tooth position and excessive tipping are not the only undesirable effects of orthodontic expansion. Buccal or labial crown movement may be producing deleterious amounts of stress on the surrounding hard tissue. The experiment by Kraus et al, mentioned above, showed similar results to those in which the incisors were proclined in monkeys. Namely, a dehiscence can be formed. After expansion of the premolars, he found an average of 2.9 mm and 1.2 mm of marginal bone loss at the mesial and distal roots, respectively [62].

The human clinical trial described previously, a loss of buccal bone height after expansion with the Damon system was also noted. Statistically significant buccal bone height loss occurred at the maxillary first premolars, mandibular first and second premolars, and mandibular first molars. Also, statistically significant facial bone width loss was evident 3 mm apical to the bony crest of the maxillary first and second premolars and first molars, as well as the mandibular right first premolar, second premolars, and first molars. In 2010, Paventy re-evaluated some of the same subjects 6-12 months post-treatment (5 of the 19 subjects could not be contacted). It was observed that all teeth except one showed a small amount of recovery of facial bone height and width, but none of the improvements were statistically significant. In the randomized clinical trial described earlier by Cattaneo et al, loss of buccal bone was also seen [61]. They found that the buccal bone area lateral to the second premolar decreased 23% and 18% (right and left sides) with Damon and 17% and 12% (right and left sides) with In-Ovation. Cattaneo and coworkers also found that the bone loss that occurred with the inter-premolar expansion was positively associated with buccal tipping. In 1985, Quinn and Yoshikawa published a review of the literature on force magnitude in orthodontics [63]. When evaluating different tooth movements, they noted that with tipping, forces were concentrated on the crestal bone, and when these forces surpass physiological levels, they can become deleterious.

9. Recession and Orthodontic Treatment

Without evaluating tooth movements, several studies have shown inconsistencies in the prevalence of recession following orthodontic treatment in humans. In a cross-sectional design, Slutzkey et al [50] found that the prevalence and severity of recession was worse in orthodontically treated patients when compared to

patients who had not received orthodontic treatment. They measured recession clinically as the distance from the CEJ to the free gingival margin on 303 consecutive military cadets, aged 18-22 years. Twenty three percent of patients who had received orthodontic treatment exhibited gingival recession, whereas only 11.4% of patients who had not received orthodontic treatment had recession. Also, 8.4% of patients who had orthodontic treatment had at least one tooth with 3 mm or more of recession, whereas only 0.9% of the patients who did not have orthodontic treatment experienced a recession of 3 mm or more. In contrast, Alstad and Zachrisson [64], as well as Polson et al [65], found no significant difference in gingival recession when comparing groups of orthodontically treated patients with matched control groups. In the study by Alstad and Zachrisson, the periodontal status of the maxillary teeth of teenagers (mean age 11.7 ±1.4 years), excluding the second molars, was evaluated by measuring from the base of the pocket to the CEJ on the facial surface with a periodontal probe. Five months after appliance removal, they reported no significant difference between the 38 individuals in the experimental group and the 39 matched controls. Polson et al [65] evaluated 112 subjects at least 10 years after orthodontic treatment was completed (mean age 29.3 ±4.2 years). Renkema et al [14] investigated both the prevalence and pattern of labial recession during and following orthodontic treatment. They measured recession on the facial surface of all teeth on the dental casts of 302 orthodontically treated patients. Recession was considered present if the CEJ was exposed. Measurements were taken at 4 time points: the beginning of treatment (mean age 13.6 ±3.6 years), the end of treatment (mean age 16.2 ±3.5 years), 2 years after treatment (mean age 18.6 ±3.6 years), and 5 years after treatment (mean age 21.6 ±3.5 years). The authors pointed out that the frequency of recession in their cohort was overall somewhat lower than in non- orthodontic samples [19], [23], [24]. Another important aspect of their results was the pattern of labial gingival recessions. At 5 years post-treatment, the two most commonly affected teeth in the maxillary arch were the first premolar and first molar. Approximately 14% of the maxillary first premolars and 6% of first molars exhibited labial gingival recessions. In the mandibular arch, the central incisors (10%) and the first premolars (8%) showed the highest prevalence of recession. The authors stated that the results from their sample were similar to that observed in epidemiologic studies for both orthodontic [48], [50], [66] and non-orthodontic samples [17], [18], [20].

10. Conclusions

Gingival recession is most common problem encountered in orthodontics after fixed orthodontic treatment and currently many researches are going on to prevent it. Though it is an undesirable side effect after fixed orthodontic treatment, we can reduce the level by modifying the treatment strategies. Thorough diagnosis of the problem and formulation of appropriate treatment plan for gingival recession should be done. Future research should be done to develop newer treatment modalities for preventing gingival recession in orthodontics.

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