

The impact of orthodontic treatment on periodontal support loss

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The increase of life expectancy and purchasing power, along with the changes on esthetic concepts, resulted on the increase of the number of adults seeking orthodontic treatment, combining functional benefits to smile esthetics. Because of this change, now, it is part of the orthodontist clinical routine to treat patients, dealing with individuals with periodontal disease or sequelae of this disease.

It has been well established that dental plaque represents a risk factor that is necessary in the pathogenesis of gingivitis and periodontitis, and the progression of periodontal disease depends on the balance between microbial biofilms and immune and inflammatory host responses.^{2,3}

Regarding gingival inflammation, studies^{4,5} show that the installation of orthodontic appliances increases the amount of plaque, which results in formation of gingival hyperplasia and pseudopockets. This situation changes the subgingival ecosystem and facilitates the increasing of periodontal pathogens levels, which express virulence factors that stimulate host cells to release several types of inflammatory cytokines such as interleukin 1 β (IL-1 β), interleukin 6 (IL-6) and interleukin 8, (IL-8) and growth factors such as tumor growth factor (TGF), which modulate the inflammatory response in periodontal tissues.⁶

In turn, orthodontic movement causes reorientation and remodeling of periodontal support tissues during tooth movement, but the application of appropriate mechanical forces causes expected reactions in the periodontal tissue support. The biological events that occur during orthodontic tooth movement on a periodontium of reduced height are the same as for a normal size periodontium, however, some aspects should be taken into consideration when treating a patient with loss of osseous support:¹ (1) Risk of future bone loss, with eventual loss of teeth, (2) These individuals often have complex malocclusion, and (3) Patients with reduced periodontium requires changes in treatment regarding mechanics and orthodontic appliance.

These considerations are based on recent research,^{1,7,8,9} suggesting that orthodontic load may contribute to the destruction of the bone support of patients with periodontal disease—directly, through the induction of proinflammatory cytokines and also by the decreasing expression of matrix proteins and osteogenic protein and not by gingival inflammation. However, the mechanisms of interference in the regulation of these molecules remain unclear.

There are several ways to assess the impact of orthodontic treatment on periodontal tissues.

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Most studies used clinical periodontal parameters such as plaque index and probing depth associated with microbiological analysis.^{4,10} Another way to analyze changes during orthodontic treatment is through molecular analysis of fluid from the gingival sulcus. This analysis indicates immune and inflammatory reactions resulting from microbial challenge and host defenses, as well as the biomechanical stress caused by orthodontic movement, and can provide instructive answers about the orthodontic management of patients with periodontal disease. It is considered a noninvasive method and is easily performed, because the fluid is collected with tips or strips of filter paper, which are processed in the laboratory and analyzed for the presence of biomarkers.^{5,6,9}

Among the mediators identified in the gingival sulcus fluid, the proinflammatory cytokines have attracted particular attention. Among the most studied are IL-1 β , IL-6, IL-8 and Cyclooxygenase 2 (COX-2). Along with inflammatory mediators, growth factors are also important periodontal homeostasis biomarkers, e.g., growth factor Insulin-like 1 (IGF-1) and tumor growth factor β 1 (TGF- β 1), which promote migration, cell differentiation and proliferation, and extracellular matrix synthesis. As osteogenic protein, important components of the periodontium mineralization.^{7,9,11}

Recently, clinical and microbiological changes were correlated with concentrations of cytokines, particularly IL-6 and IL-8 during orthodontic treatment.⁹ The results showed that increased levels of these interleukins, before treatment, correlated with changes in periodontal parameters, such as increased probing depth, one year after treatment. According to the authors, this knowledge can contribute to the decision to start an orthodontic therapy in susceptible patients.

Similar results were also observed in periodontal⁷ ligament cells, which respond to pro-inflammatory stimuli, modulated by mechanical forces.

On the other hand, it showed that biomechanical forces applied longer and with low magnitude exerted anti-inflammatory effect. According to the authors, this observation suggests that the contribution of orthodontic mechanics in periodontal destruction is not mediated by increased periodontal inflammation, but by interference with the expression of osteogenic and matrix protein. Despite the results, the mechanism to explain why the mechanical force had, initially, a proinflammatory effect and, subsequently, anti-inflammatory effect is still unknown and some speculations are made. The periodontium cells become better adapted to mechanical stress? The anti-inflammatory molecules produced in later stages of movement would cause an imbalance in the inflammatory expression?

In addition to the findings that orthodontic stress influences the levels of proinflammatory cytokines and the expression of osteogenic and matrix protein, it is noteworthy that healthy sites of periodontal patients—compared with healthy sites of individuals without periodontal disease—appear to have higher levels of inflammatory molecules (such as IL-1 β , IL-8 and MMP-8) in the gingival ridge fluid.⁶ This suggests that inflammatory mechanisms may occur in the periodontal tissue before they are detected clinically.

Therefore, biomarkers detected in the gingival ridge fluid could serve to assist in screening patients at risk for bone loss, before the decision to submit them to orthodontic movement. The joint participation of biomechanical stress in the process of bone loss in periodontal patients is well established in the literature, but the mechanisms of how biomechanical forces modulate molecular and cellular events in the periodontium are still not fully understood. Better understanding of the interaction of inflammatory and biomechanical signals will optimize methods of orthodontic and periodontal treatment in patients with reduced bone support.¹

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