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Editorial

Gums and Brain: Does the Evidence Support an Association Between Periodontal Infection and Neurological Diseases?

Yago Leira[1,2]*

[1]*Department of Periodontology, Faculty of Dentistry and Medicine, University of Santiago de Compostela, Spain*
[2]*Health Research Institute of Santiago de Compostela (IDIS), Spain*

***Corresponding Author**: Yago Leira, Department of Periodontology, Faculty of Dentistry and Medicine, University of Santiago de Compostela, and Health Research Institute of Santiago de Compostela (IDIS), Spain.

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In the late 1980s and 1990s, several investigations have arisen focusing on the possible relationship between periodontal infection and cerebrovascular disease [1-5]. Therefore, could it be possible that subjects with periodontitis have a higher risk of developing cerebral ischemia? Very recently, findings from a meta-analysis including data from 8 studies suggested that periodontitis increases the risk of ischemic stroke and that the magnitude of this increase is high (pooled RR = 2.88; 95% CI: 1.53 - 5.41) [6]. In this association, atherosclerosis plays a pivotal role. On the one hand, periodontal bacteria residing in dental plaque could be translocated through ulcerations in the epithelium of the periodontal pocket into the blood stream (i.e., bacteremias) [7]. On the other hand, inflammatory mediators (e.g., IL-1β, IL-6, CRP, TNF-α) produced in periodontal tissues can be systemically disseminated. As a result, inflammatory markers and bacterial products could lead to endothelial dysfunction, which is the first stage of atherogenesis and, therefore, taking part in the onset of cerebral ischemia [8].

Besides ischemic stroke, other neurological conditions drew researchers attention. Alzheimer´s disease is one of the leading causes of primary degenerative dementia affecting elderly population. It is suggested that the association between periodontal disease and Alzheimer´s disease could be bidirectional [9]. Invasion of the brain tissue by periodontopathogens from the dental plaque biofilm and their products via circulation or peripheral nerves may accelerate neuroinflammation [10]. In addition, periodontal-derived inflammatory markers could also reach the brain via the systemic circulation or via neural, therefore, increasing brain cytokines levels [11]. On the contrary, patients with Alzheimer´s disease could have worse oral hygiene compared to non-demented subjects due to diminished manual skill to carry out mechanical plaque control [12]. Thus, a high prevalence of periodontal infection could be seen in Alzheimer´s disease patients.

A similar pattern occurs with the relationship between periodontitis and Parkinson´s disease, where a bidirectional link could be established. Parkinson´s disease is characterized by hand movement impairment, which could predispose to poor daily oral hygiene and may have an influence on the onset or progression of periodontitis [13]. Moreover, swallow and hypersalivation difficulties could further affect the oral environment in these Parkinson´s disease patients. In contrast, periodontal infection can cause translocation of either bacterial products and inflammatory biomarkers (e.g., IL-1β, IL-6, TNF-α), which may be involve in the onset of the conversion of primed microglia into active microglia in midbrain. As a result, Parkinson´s disease may occur by necrosis and apoptosis of dopaminergic neurons [14].

Recently, a possible association between periodontal disease and multiple sclerosis has been suggested [15]. It is proposed that herpes viruses may play a key role in this link. Human herpes virus is thought to be involved in the development of multiple sclerosis and periodontitis [15]. Therefore, chronic infection related to herpes viruses could explain the association between both diseases. However, the exact pathophysiological mechanisms underlining this relationship remain unclear. Indeed, negative results from a study published one

year ago arises the controversy of this link [16]. Discrepancies between studies could be explained by the adjustment of smoking habits in logistic regression analysis and how information of periodontitis was obtained.

Overall, periodontal disease is associated with numerous neurological diseases in which neuroinflammation process plays a pivotal role. However, further research is warranted in order to clarify if this relationship is causal or not. Furthermore, interventional studies are needed to evaluate the potential benefit of periodontal therapy in subjects with periodontitis and neurological diseases.

Finally, further investigations should also focus on other known neuroinflammatory diseases (i.e., migraine) where periodontitis could predispose to their development or chronification.

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Conflict of Interest

Y. Leira did not report any conflict of interest.

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