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Editorial

Some Important Facts about the Periodontal Disease

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The periodontal disease is the most common chronic inflammatory disease of microbial origin, which acts on the supporting tooth tissues, including the periodontal ligament and alveolar bone. The most common form of periodontal disease is the mild form of gingivitis, which is the precursor of periodontitis as the most severe disease in this group and is defined as the apical extension of gingival inflammation, affecting parodontium. The parodontium is a specialized connective tissue that surrounds the root of the tooth and has a role in fixing the tooth for the jaw bone, in the amortization of the mechanical pressure that occurs in chewing and speech, but also in the formation and resorption of bone tissue. Its composition includes gingiva and periodontal ligament, such as soft tissue and alveolar bone and cement (enamel) as solid tissue [1].

The periodontal disease is initiated and aggregated by Gram-negative, anaerobic or micro-aerophilic bacteria that colonize the subgingival sulcus. The bacteria initiate the immune response of the host and destroy the tissues that provide the support of the tooth which leads to apical migration of the gingival tissues, loss of periodontal attachment, and an increase in the depth of the periodontal pocket. The most exposed to the action of plaque microbes is the connection between the gingiva and the tooth, that is, the outer gingival epithelium that communicates with the bone tissue of the jaw through the periodontal ligament [2]. By forming periodontal pockets, conversion of the joint epithelium into the pocket epithelium occurs and culminates with the loss of teeth.

There is a theory that periodontal disease is a pathological manifestation of the host's immune response directed against the bacterial challenge of sub-gingival biofilm. The degree of tissue damage depends on the interaction between host defense mechanisms and biofilm. The host response is mainly determined by genetic factors, the environment, systemic diseases such as diabetes mellitus, rheumatic diseases and acquired factors, such as smoking, and emotional stress [3,4].

Also, periodontal disease, as a chronic reservoir of bacteria and endotoxins, aggravating cytokines and systemic inflammatory mediators, leads to endothelial damage, initiation and exacerbation of atherosclerosis and thrombogenesis, and thus acts on certain systemic diseases [5,6].

In the 2010 US report, 64.7 million people over the age of 30 have periodontitis [7]. In the world, 15 - 20% of people aged between 35 and 44 have a severe periodontal disease pattern [8].

Risk factors

Predisposing factors for periodontal disease include: inappropriate oral hygiene and elderly, hormonal changes - with worsening of the disease activity during puberty, menstruation and pregnancy, diabetes mellitus, various genetic disorders that impair the function of neutrophils and rheumatoid arthritis [9-11]. An effective mechanism of host defense is highly vascularized gingival tissue, an oxidative barrier to penetrate anaerobic bacteria from the dental plaque. Conditions such as smoking and stress modify this barrier causing

vasoconstriction of the peripheral arterioles and reduce the blood flow to the gingival tissue [3]. This provides enough time to survive the anaerobes in the tissues and cause an immune response by activating latent collagenase.

Smoking: The relationship between smoking and periodontal health has been explored since the middle of the last century. More recently, a wealth of epidemiological, clinical and *in vitro* studies have shown irrefutable evidence that smoking negatively affects periodontal health [12-15]. *In vitro* studies have shown a change in gingival cervical fluid with an inflammatory cytokine profile, an increased immune function and an altered proteolytic activity in smokers [16,17].

Diabetes mellitus: Studies have shown an association between poor glycemia control and periodontal disease [18-20]. Taylor., *et al*. suggest a two-way link between periodontal disease and glycemic control [4,21].

Psychological factors: Studies have shown that people with psychological stress are more likely to develop periodontal disease. A possible link in this chain may be the increased production of IL-6 in response to increased psychological stress [22]. Another study suggests that the host's immune response to *P. gingivalis* infection can be compromised in people who are under psychological stress [23].

Genetic factors: Although bacterial infection is an aetiological agent in periodontal disease, studies of identical twins showed a 50% susceptibility to periodontal disease in the second twin [24].

Immune host response: There is an opinion that the occurrence and development of periodontal disease is the result of a hyperimmune response to bacterial infection, rather than a direct destructive effect of the bacterial pathogens themselves [24]. Polymorphisms of the IL-1 gene are associated with periodontal disease [25]. In addition, the evidence suggests the possible interactions between the IL-1 gene polymorphism with smoking and diabetes mellitus, indicating that there is an interaction between genetic and environmental factors, resulting in periodontal disease [26-28].

Deficiency in neutrophil function is associated with periodontal disease [29]. These are the Chedyak-Higashi syndrome [30], cyclic neutropenia [31], lazy leukocyte syndrome, agranulocytosis, and leukocyte adhesion deficiency [32], Down's syndrome [33] and Papillon Lefebvre syndrome [34].

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