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Diabetes Mellitus and Periodontitis: Molecular Interrelationships

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Editorial Article

Diabetes mellitus (DM) and periodontitis are both chronic inflammatory diseases, which contribute significantly to morbidity and are a major health care burden [1]. The interrelationship between DM and periodontitis has been studied for many years. It has been accepted that the supposed inter-relationship between DM and periodontitis is a two-way relationship. In this sense, the presence of one condition tends to increases the risk and severity of the other, and vice versa. However, the mechanisms for this two-way relationship still remain unknown [1,2].

DM is a chronic metabolic disorder, involving impaired glucose homeostasis. DM poorly controlled is characterized by high blood glucose levels that result in an increased formation of protein glycosylation leading to amplified formation of so-called advanced glycation end products (AGEs). AGEs are glucose products that have the ability to attract and stimulate inflammatory cells to produce inflammatory cytokines such as tumor necrosis factor alpha (TNF-α), Prostaglandin E2 (PGE2), and interleukin-1β (IL-1β) and matrix metalloproteinases (MMPs) that together with increased generation of reactive oxygen species (ROS) may raise the risk of periodontal attachment and/or alveolar bone loss and contribute to diabetic complications [3-7]. AGEs combined with pro-inflammatory cytokines can motivate fibroblast apoptosis and impair periodontal wound healing. It is known that, the expression of these pro-inflammatory mediators is regulated by the T helper cells. In addition, the polymorphonuclear neutrophils are important in the first line of defense in periodontal disease process, as periodontitis. However, exacerbate response may transpose this protective action of polymorphonuclear neutrophils into destroyers. Thus, the host immune-inflammatory responses would be protective or destructive [5].

Individuals with DM have greater of developing periodontitis than non-diabetics as well as increased severity [8,9]. The periodontitis is characterized by inflammation of periodontal tissue induced by bacterial biofilm (oral plaque) that leads to tissue destruction and alveolar bone resorption [1,9]. In addition to the biofilm microorganisms, other factors such as environmental, systemic and genetic are also responsible in progression of periodontitis [5]. Consequently, these processes result in destruction of the periodontal tissues and loss of tooth, especially in adults [1,9].

The loss of bone that is increased by hyperglycemia can be reversed by inhibition of cytokines and AGEs. On the other hand, the inflammation interferes with the repair tissue and osteolysis is not reversed by the formation of new alveolar bone [3].

Studies have shown that inflammatory mediators produced during the process of periodontal disease, as well as the microbial product of the periodontal biofilm may fall into the bloodstream and increase the overall inflammatory status of the organism and thus contribute to the onset or aggravation of DM [2]. Low-grade inflammation and a higher serum concentration of pro-inflammatory cytokines such as interleukin-6 (IL-6), TNF-α and C-reactive protein (CRP) may increase insulin resistance and decrease insulin secretion. Furthermore, gram-negative periodontal infection significantly decreases glucose tolerance and can lead, like other types of inflammation, to an increase in the severity of diabetes [10].

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