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Stress and Periodontal Disease (Review Article)

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Abstract: Stress is compatible with good health because it is necessary to cope with the problems of everyday life. Problems begin when the stress response does not match the intensity of the problem. Psychological stress can reduce the cellular immune response. The article describes possible ways of developing periodontal diseases against the background of existing stress. It was reported that patients with ANUG compared with the control group had: Inhibition of chemotaxis and phagocytosis polymer pho nuclear leukocytes and decreased proliferation of lymphocytes upon stimulation with non-specific mitogen.

Keywords: Stress, Periodontal Disease, Behavior Change.

1. Introduction

Stress is a balanced response to constant unfavorable stimuli [14].
Stress is compatible with good health because it is necessary to cope with the problems of everyday life. Problems begin when the stress response does not match the intensity of the problem. Psychological stress can reduce the cellular immune response. Communication between the central nervous system and the immune system occurs through a complex network of bidirectional signals linking the nervous, endocrine and immune systems. Stress disrupts the homeostasis of this network, which in turn alters immune function. A direct relationship between periodontal disease and stress has yet to be proven, due to the difficulty of quantifying the amount and duration of stress, as well as the presence of many factors influencing the incidence and severity of periodontal disease [17]. There are several options for the development of periodontal disease under existing stress.

Gingival microcirculation
The tone of the smooth muscles of blood vessels can be changed by emotions through the autonomic nervous system. With prolonged and ongoing stress, constant constriction of blood vessels can alter the supply of oxygen and nutrients to tissues [24].
Stress and systemic disease
It is well known that cardiovascular diseases, diabetes mellitus, preterm birth, osteoporosis, rheumatoid arthritis, inflammatory bowel diseases, systemic lupus erythematosus, etc. are associated with stress, either as a physiological response to stress or as a behavioral response. Stress may be a significant overall risk factor for the development of diabetes mellitus, cardiovascular disease, preterm birth and osteoporosis, as well as periodontal disease [2].

The role of oxidative stress in periodontitis
Oxidative stress is defined as an imbalance between oxidants and antioxidants in favor of oxidants, resulting in disruption of redox signaling and control and/or molecular damage. Oxidative stress is a key driver of chronic inflammation and, as a consequence, plays a central role in the pathogenesis of a wide range of chronic inflammatory diseases [22] (e.g. type 2 diabetes, cardiovascular disease and metabolic syndrome), indeed it has been proposed as a general relationship between periodontitis and systemic disease.
Normally, there is a balance between oxidants and antioxidants present in all tissues of the body. If this balance is disturbed by excess production of oxidants and/or depletion of local antioxidants, the resulting excess of oxidants causes oxidative stress and is associated with local tissue damage observed in periodontitis [23].
It can cause direct tissue damage by altering molecules, proteins, lipids and DNA, thereby damaging cells directly, or by activating redox transcription factors within the cell, leading to subsequent changes in gene expression and the production of pro-inflammatory molecules. These cytokines or chemical messengers can further enhance and propagate the inflammatory response by increasing local levels of oxidative stress. In susceptible patients, in whom

the body's inflammatory resolution mechanisms do not work effectively, a viscous cycle is established, resulting in a transition from acute to chronic inflammatory lesions, as in periodontitis [7].

Stress and the microbiology of periodontal disease

Microorganisms have the ability to recognize hormones in the host body and use them to adapt to the environment. This supports the idea that psychological stress may contribute to the development of many bacterial infections. Under stress, microbial composition can form under the gum. In vitro studies were conducted to determine whether norepinephrine and epinephrine, which are released during the human stress response, affect the growth of 43 microorganisms found in subgingival microbial complexes. Twenty species in the subgingival biofilm were significantly increased in abundance by norepinephrine inoculation, and 27 species were significantly increased by epinephrine inoculation. There was also variation in growth response within bacterial species and within and between microbial complexes [4, 15].

It is concluded that this change may influence the composition of the subgingival biofilm in vivo in response to stress-induced changes in local catecholamine levels and play a significant role in the etiology and pathogenesis of periodontal diseases. These results are significant because P. gingivalis is the most commonly reported periodontal pathogen implicated in the association between periodontal disease and cardiovascular disease [8].

Endocrine changes

It was assumed that periodontal condition is associated with changes in the concentration of adrenal corticosteroids and changes in the response of oral tissues to bacterial toxins and other hormones involved in the general adaptation syndrome [16].

High cortisol levels can be particularly detrimental to periodontal tissue due to the extremely rapid turnover of certain periodontal components. Elevated glucocorticoid levels may reduce fibroblast formation, collagen production, and sulfated glycosaminoglycan conditions. These changes may be sufficient to cause an imbalance in the synthesis and destruction of periodontal tissue, especially if pre-existing inflammation is present [25].

Changes in salivation and components

It is hypothesized that both increases and decreases in salivary flow caused by emotional distress may adversely affect the periodontium. Emotional distress can also cause changes in salivary pH and saliva composition, such as the secretion of immunoglobulin A [13]. These relationships between salivary physiology and psychological status do not necessarily demonstrate causation of periodontal disease, but they do indicate a pathway through which periodontal health is influenced by changes in saliva [19].

Reduced immunity

Inflammatory periodontal diseases are associated with local and systemic increases in the levels of proinflammatory cytokines, such as tumor necrosis factor a, IL-6 and prostaglandins, and lead to tissue destruction through the participation of matrix metalloproteinases [6, 26]. Stress disrupts the balance between pro-inflammatory and anti-inflammatory responses. The relationship between stress and periodontal disease may be mediated by changes in the levels of IL-1, IL-6 in the gingival crevicular fluid, decreased chemotaxis and phagocytosis of polymorphonuclear leukocytes, and decreased lymphocyte proliferation [9].

Psychosocial stress stimulates the brain, where its stimulation or inhibition depends on adaptive and maladaptive coping, respectively. When the autonomic nervous system is stimulated, prostaglandins and proteases are secreted, which leads to the progression of periodontal diseases. Excessive production of glucocorticoids (cortisol) suppresses the immune system, reducing the secretion of IgA and IgG, thereby increasing the progression of periodontal disease and poor response to treatment [5]. Subsequently, this process can increase the vulnerability of periodontal tissues to pathogenic microorganisms due to the activation of cellular reactions leading to local tissue destruction [11].

Patients with periodontitis under stressful conditions have increased levels of IL-6 and IL-1, and similarly, patients with aggressive forms of periodontitis have increased levels of IL-6 and IL-1 in the blood serum [15].

Stress and behavioral change

Stress influences the consequences of behavioral patterns ranging from neglect of oral hygiene to inadequate nutrition, poor sleep patterns, tobacco use, and alcohol consumption, which contribute to a “vicious circle” of increasingly severe forms of progressive inflammation and periodontal disease [12].

Neglect of oral hygiene
Obviously, proper oral hygiene depends in part on the mental health of the patient. It has been reported that psychological disorders can lead patients to neglect oral hygiene and that plaque accumulation is harmful to periodontal tissues. Academic stress has been described as a risk factor for gingival inflammation with increased IL-b levels in the sulcus and decreased quality of oral hygiene [3, 12, 27].

Changes in diet
It is believed that emotional states change diet, thereby indirectly affecting periodontal health. Psychological factors influence food choices, the physical sequence of the diet, and the amount of food eaten. This may include, for example, consumption of excess amounts of refined carbohydrates and a softer diet that requires less vigorous chewing and therefore predisposes to plaque accumulation at proximal contacts [20]. Stress leads to other behavioral changes such as overeating, especially high-fat diets, which can then lead to immunosuppression due to increased cortisol production [1].

Smoking and other bad habits
Among the many harmful oral habits believed to be caused by emotional disturbances, smoking is perhaps the most important in relation to the worsening of periodontal health [21]. Circulating nicotine results in (1) vasoconstriction caused by the release of epinephrine and norepinephrine, which is thought to result in a lack of nutrients for periodontal tissue; (2) suppression of the secondary antibody response; and (3) inhibition of oral neutrophil function.

Stress and acute necrotizing ulcerative gingivitis
Acute necrotizing ulcerative gingivitis (ANUG) is the most studied periodontal disease in relation to psychosocial predisposing factors. An apsychogenic origin has been proposed for ANUG. Psychogenic factors likely predispose to the disease by promoting bacterial overgrowth and/or weakening host resistance [10]. Host tissue resistance can be altered by mechanisms acting through the autonomic nervous system and endocrine glands, resulting in increased levels of corticosteroids and catecholamines. This can reduce gingival microcirculation and salivary secretion, and at the same time also inhibit the functions of neutrophils and lymphocytes, which promotes bacterial invasion and damage [18].
It was reported that patients with ANUG compared with the control group had:
- Inhibition of chemotaxis and phagocytosispolymorphonuclear leukocytes;
- Decreased proliferation of lymphocytes upon stimulation with nonspecificmitogen.

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