

Influence of Stress on Periodontium: A Review

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ABSTRACT

Psychological condition, particularly stress have been implicated as a risk indicator of many inflammatory diseases including periodontal disease. Stress and psychosomatic disorders causes changes in interactions among nervous, endocrine and immune system. It results in delayed healing of the connective tissues and bone, apical migration of the junctional epithelium and formation of periodontal pocket. This paper describe a overview of association between stress and periodontium and stress interaction with immune system.

Key words: Periodontal diseases; Stress; Psychological distress.

INTRODUCTION

Psychological disturbances can lead patients to neglect oral hygiene with resultant unfavorable effects on the periodontal tissues. It can down regulate the cellular immune response. Communication between the central nervous system and the immune system occurs via 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.¹

Stress is defined as a total transaction from demand to resolution in response to an environmental encounter that requires appraisal, coping and adaptation by the individual. Coping is the response of the individual to stress² Stress is said to influence the host defenses, exerting an immunosuppressive effect, increasing one's vulnerability to disease⁴

Chronic periodontitis is generally considered to be a multifactorial slow progressive

disease. Defined as an infectious disease resulting in inflammation within the supporting tissues of teeth, progressive attachment loss and bone loss. Risk factors of chronic periodontitis are plaque accumulation, diabetes, smoking, emotional stress and genetic factors. Many studies have shown the important role of microbial pathogenesis in periodontitis; however, bacteria alone appear to be insufficient in explaining the progression of the disease⁶

In addition, mechanisms through physiologic pathways may influence periodontal tissues through alteration in saliva, changes in gingival blood circulation, endocrine imbalances and altered host resistance. Psychoneuroimmunologic effects were confirmed by findings of poorer immune functions in persons who experienced stressful life events or chronic stress¹⁹

Effects of stress on immune system

Three types of mode of action of stress mechanism in releasing inflammatory mediators

through neural and endocrine system

1. Through the autonomic nervous system pathways
2. Through the release of neuropeptides
3. Through the release of hypothalamic and pituitary hormones.¹

Sympathetic nervous system

Stress results in the release of adrenalin and noradrenaline from cells of the adrenal medulla. Through interaction with adrenergic receptors, noradrenaline and adrenalin mediate cardiovascular and metabolic effects. This causes an increase in circulating concentration of T-helper lymphocytes, cytotoxic T-cells (CD8+), and natural killer cells, plasma levels of Immunoglobulin IgM, IgG, and complement component C3¹

The release of neuropeptides

Neuropeptides such as substance P (SP), somatostatin, the endogenous opioid peptides (beta-endorphin and enkephalins), vasoactive intestinal peptide (VIP) and nerve growth factor from peptidergic sensory nerves also modulate the activity of the immune system and the release of cytokines. They are also present in gingival and periodontal tissues in close contact with the vascular plexus and penetrate into the epithelium. In response to psychological or certain physiological stressors, an inflammatory reaction occurs through the release of neuropeptides and inflammatory mediators from the sensory nerves and activation of mast cells or other inflammatory cells⁵

Experimental studies suggest long lasting emotional stress may increase SP release, resulting in enhanced and imbalanced inflammatory reactions, which may promote tissue damage. These neuropeptides selectively regulate Th1/Th2 cytokine secretion and may regulate immune responses in, for example, granulomatous infections. Thus, multiple nervous and endocrine factors tend to drive the immune response toward Th2 cell dominance, and therefore emotional stress may be an important predisposing factor in severe and progressive chronic infections.¹

The hypothalamus-pituitary-adrenal (HPA) axis

During a stress response [psychosocial

stressors], the higher center of the central nervous system causes the release of corticotrophin-releasing factor (CRF) and arginine vasopressin from the hypothalamus, which further stimulates adrenal cortex and causes the production and release of glucocorticoid hormones.

These glucocorticoids exert its major suppressive effects by reducing the number and activity (chemotaxis, secretion, and degranulation) of circulating inflammatory cells including lymphocytes, monocytes, macrophages, neutrophils, eosinophils, and mast cells and also inhibits the production of proinflammatory mediators, cytokines (interleukin [IL] IL-1, IL-2, IL-3, IL-6, tumor necrosis factor (TNF), interferon gamma, and granulocyte and monocyte colony stimulating factors) and cascade of the immune response by inhibiting macrophage-antigen presentation, lymphocyte proliferation, and lymphocyte differentiation to effector cell types such as helper lymphocytes, cytotoxic lymphocytes, NK-cells, and antibody-forming B cell .

The two other hormones of the HPA axis, CRF and adrenocorticotrophic hormone ACTH, also separately modulate the immune system activity by regulating production of signal substances from immune cells (cytokines) such as IL-1 by monocytes and blocking the activation of macrophages. They also promote B-cell proliferation, but inhibit antibody production.

Studies approach

Cross sectional studies conducted most of them were at university or private dental practice. Five studies ^{7,8,9,10,11} were epidemiological surveys involving the residents in a particular area or a randomly selected general population. The study by Lopez *et al.* ¹¹ was the only study that included students attending high school, which emphasized the relevance of assessing psychosocial stress and its association with periodontitis early in life. Two studies ^{7,12} involved patients from an academic institution, and one study involved women in the post-partum period ⁹.

A cross-sectional study of 111 patients, to evaluate the psychological stress and salivary cortisol levels and their effect on chronic

periodontitis. Lipp's Stress Symptoms was applied by psychology students, to detect whether a patient presented a clinical stress syndrome. It showed a highly significant association between the mean cortisol levels and periodontal disease, this is in accordance with studies conducted by Genco¹³ in a subsample of individuals with and without periodontitis. This could be attributed to the inhibition of T-cell immune responses mediated by glucocorticoids, leading to a change toward antibody-mediated immunity (Th2-mediated response), enhancing the growth of pathogenic microorganisms that can activate a cellular response⁴.

One study done by Forte *et al* showed a lack of correlation between psychological stress and periodontal disease⁷. This study was performed among periodontally healthy subjects, and they concluded no influence of psychosocial stress on the salivary peptides that could predispose to periodontal disease.⁶

A case-control study on 1196 subjects showed people with aggressive periodontitis were more depressed and socially isolated than people with chronic periodontitis or control group.¹⁸ Patient who had received supportive periodontal care every 3-6 months for a period of 5 years after active periodontal treatment showed stress as one of the variables for progression of periodontal disease at few sites in few patients.¹

In a case control study, 50 adult patients done by Faculty of Dentistry at Tehran University of Medical Sciences in which plaque indices, bleeding on probing, GCF sampling are done. Cytokine production that is the amount of IL-1b and IL-6 in the GCF was analysed by ELISA. Result show higher level of IL-1 α was found in aggressive periodontitis than the other groups and the level of IL-6 was slightly higher in the aggressive group. In addition, in chronic periodontitis strong positive relationship was found between the stress score and the GCF level of IL-1 α ³

In a study by Deinzer *et al.*, a significantly higher amount of GCF IL-1 α level in stress situations was observed and it was concluded that stress might affect periodontal health by increasing local

IL-1 α levels. Besides, some other studies could not show any relationship between the inflammatory process and the stress situation³

Recent studies had confirmed the fact that the concentration of cytokines (IL-6, IL-1 α etc.), cortisol in GCF is higher in person showing depression sign¹

DISCUSSION

Periodontitis is an inflammatory response of the periodontium which involves the destruction of the investing tissues around the teeth, resulting in loss of tooth support, ultimately leading to the tooth loss. The pathogenic processes of the periodontal diseases are largely the results of the host response to microbially induced tissue destruction. The microbes induce destruction of periodontal tissues by initiating inflammatory and immune responses by various inflammatory molecules such as proteases, cytokines, prostaglandins and host enzymes released from leukocytes and fibroblasts⁵

The relationship between periodontal illness and the psychological predisposing factors is well-established in specific conditions, like, Acute Necrotizing Ulcerative Gingivitis (ANUG) is identified to be significantly associated with high levels of trait anxiety, depression, and other emotional disturbances.^{4,8}

It has been shown that stress can effectively increase serum IL-1 α , IL-6, and IL-10 and decrease IFN- α production, suggesting that there is an interaction between endocrine and immune systems in response to a physiological stress⁵. The role of some inflammatory mediators has been more highlighted as elevation of the interleukin-1b (IL-1 α) level in the gingival crevicular fluid (GCF) and in the periodontal pocket tissue

Furthermore, the patients with mood disorders were also found to have an exaggerated inflammatory response to psychological stress compared to healthy individuals⁵

It hypothesized that stress leads to other behavioral changes such as overeating, especially

a high-fat diet, which then can lead to immuno suppression through increased cortisol production. [1 Proper oral hygiene is partially dependent on the mental health status of the patient. It has been reported that psychological disturbances can lead patients to neglect oral hygiene and that the resultant accumulation of plaque is detrimental to the periodontal tissue. Academic stress was reported as a risk factor for gingival inflammation with increasing crevicular interleukin-b levels and a diminution of quality of oral hygiene^{1,15}

The smooth muscle tone of blood vessels may be altered by the autonomic nervous system due to stress. Furthermore, in long or continued emotions, a constant constriction of blood vessels could alter the supply of oxygen and nutrients to the tissues¹ Elevated levels of glucocorticoids can decrease collagen production fibroblasts in vitro and in skin in vivo and sulphate dglycosamino glycans. These alterations may be enough to imbalance the synthesis and breakdown of periodontal tissues, especially if preexisting inflammation is present.²

Emotional distress may also produce changes in saliva pH and chemical composition like IgA secretion. These relationships between salivary physiology and psychological status do not necessarily demonstrate causation of periodontal disease, but they show a pathway in which periodontal health is influenced by salivary changes¹

The release of adrenaline and noradrenaline may not only induce a decrease in blood flow, but possibly also in those blood elements necessary for maintaining resistance to disease-related microbes¹

Cytokines such as IL-1, IL-8, and TNF are extremely important in recruiting phagocytic cells to clear away the damaged tissue and to regulate the rebuilding by fibroblasts and epithelial cells. A decrease in expression in any of these cytokines

could theoretically impair wound healing. Stress could suppress certain aspects of the cellular immune response such as mitogen stimulation, antibody and cytokine production, and NK cell activity. Furthermore, since stress deregulates inflammatory and immune response, stress can alter the course of oral wound healing and affect the management of other oral diseases, e.g., periodontitis¹

CONCLUSION

Direct association between periodontal disease and stress remains to be proven, which is partly due to lack of an adequate animal models and difficulty to quantifying the amount and duration of stress¹.

Since there is changes in inflammatory indices due to stressful satiation, treatment protocols of periodontal disorders should focus not only on medications, but also on the control of the stress situation and psychological support. Studies could not show any relationship between the inflammatory process and the stress situation.³

When periodontal disease severity cannot be explained by established etiological factors and when there is no response to periodontal treatment or when there is a sudden, marked and increase in the rate of periodontal destruction, the practitioner should aware of these factors and taken them into consideration¹⁹. Mostly patient who are above 40 years show association with psychological stress and chronic periodontitis by high salivary cortisol production . hence proper periodontal care should be given for patient who are under stress to avoid initiation and to reduce progression of periodontal disease.⁴

Therefore, further studies for determination of the influence of psychological supportive approaches as therapeutic protocols in periodontal diseases can be recommended³

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