Stress & Periodontitis: Review

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Abstract
Periodontitis is an inflammatory response of the periodontium, which involves the destruction of investing tissues around the teeth and results in loss of tooth support leading to tooth loss. Although, bacterial pathogens are required to initiate the disease process, it has become evident that their presence alone is not sufficient to cause the tissue destruction. The etiological significance of biological and behavioral risk factors for periodontal diseases, such as smoking, advancing age, oral hygiene, and systemic diseases like diabetes mellitus has already been established. Other factors such as stress, depression and anxiety are not yet confirmed as absolute risk conditions but have been identified in some observational studies.

Key words: - Stress, Immune system, Periodontal disease, Glucocorticoids

Introduction

Periodontitis is a chronic bacterial infection that involves the gingiva and the periodontal supporting structures of the tooth. It is a common disease that affects about35% of the US dentate population 30 to 90 years of age, with about 12.6% having a moderate to severe form of the disease. [1]

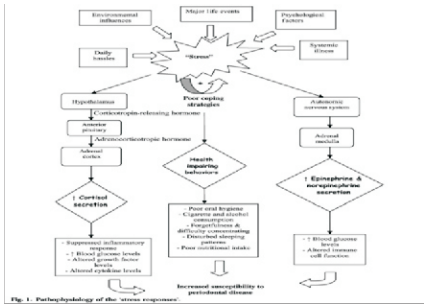
Our understanding of the etiology of periodontitis has changed markedly in the last few years. In the early days of modern periodontal research, periodontal disease" was identified as a single entity, which began with gingivitis and progressed to periodontitis and tooth loss. It is well-demonstrated today that periodontitis occurs in bursts with quiescent periods between bursts of disease activity, and the scars of the disease accumulate throughout the years [2].

Epidemiological evidence indicates that the initiation of periodontal disease is multifactorial with a complex interaction between bacterial infection and host responses often modified by behavioral factors[3]. Moreover, there is a reasonable amount of research indicating that periodontitis may be associated with psychosocial stress, financial stress, distress, and depression[4]. Although additional research is needed to define these relationships more clearly, these findings help to emphasize the impact that these factors may have on individuals' health. Stress is regarded as a cognitive perception of uncontrollability and/or unpredictability that is expressed in a physiological and behavioral response[5]. Thus, stress can be viewed as a process with both psychological and physiological components.

In 1976, Seyle [6] was basically responsible for defining stress as the response state of an organism to physical and mental forces beyond the adaptive capacity that lead to diseases of adaptation and eventually to exhaustion and death. He recognized stressors which act to produce positive changes in the body (e.g. exciting, pleasurable), leading to a response state which he defined as "eustress", or stressors could be negative that induce, threatening homeostasis with pain, discomfort and physical pathology. He defined the negative response state as "distress".

De Marco (1976)[7] coined the term "Periodontal Emotional Stress Syndrome" for individuals with severe periodontitis who had emotional stress associated with active service in Vietnam suggesting a role of occupational stress in the progression of periodontitis.

Pathophysiology of Stress Response



Stress can result in the deregulation of the immune system, mediated primarily through the hypothalamic-pituitary-adrenal and sympathetic-adrenal-medullary axes (Fig. 1).

Activation of the hypothalamic-pituitary-adrenal axis by stress results in the release of an increased concentration of corticotropin-releasing hormone from the hypothalamus. The pituitary gland is connected to the hypothalamus by the infundibulum, a stalk of tissue that contains nerve fibers and small blood vessels.

Corticotropin releasing hormone acts on the anterior pituitary resulting in the release of adreno-corticotropic hormone (corticotropin). Its acts on the adrenal cortex and causes the production and release of glucocorticoid hormones (predominantly cortisol) into the circulation. This glucocorticoids produce a myriad of effects throughout the body such as suppressing the inflammatory response, modifying cytokine profiles, elevating blood glucose levels and altering levels of certain growth factors.[8]

The second major pathway to be activated is the sympathetic nervous system. A well-known example of this is the so-called 'flight or fight' response to potentially harmful stimuli. Stress activates the nerve fibers of the autonomic nervous system, which innervate the tissues of the immune system. The adrenal medulla is actually a modified sympathetic ganglion. Its nerve bodies, instead of possessing axons, secrete their products directly into the bloodstream. The release of catecholamine's results in the hormonal secretion of nor epinephrine and epi-nephrine from the adrenal medulla, which results in a range of effects that may act to modulate immune responses. Catecholamine's released during stress; contribute to the development of hyperglycemia by directly stimulating glucose production and interfering with the tissue disposal of glucose. In addition, the sympathetic nervous system has a role in regulating immune cell activities. So it is apparent that the response of the human body to stressful stimuli is at once helpful and potentially therapeutic, even though a potentially harmful imbalance occurs when the stressful stimuli, or perceived stimuli, are prolonged. Examples include chronic anxiety states and depression.

Stress and Its Role In Periodontal Disease

The mechanisms to explain how stress may affect periodontal disease is not yet clearly understood; however there are 2 pathways which have been proposed how stress plays a role in periodontal disease. These include the biologic model and the behavioral model [9].

The biologic model proposes that periodontal disease may be biologically moderated through the hypothalamic-pituitary-adrenal (HPA) axis to promote the release of corticotropin-releasing hormone from the hypothalamus and glucocorticosteroid from the adrenal cortex.

Figure-2 illustrates the following cascade of events:

1. Under a potential stressful situation, hypothalamus is activated.
2. The hypothalamus induces secretion of corticotropin-releasing hormone (CRH) which flows to the pituitary gland to stimulate the secretion of adrenocor ticotropic hormone (ACTH).
3. ACTH enters the peripheral blood flow, and induces the adrenal cortex to secrete cortisol and other steroids. Increased level of these steroids results inimmuno-

suppression and reduced resistance to infection. This is done by suppressing IgA, which protect by preventing initial colonization of periodontal organisms, and IgG which exert protection by covering the periodontal bacteria with a type of coating that allows the phagocytes to bind and ingest the invading bacteria in addition to suppressing neutrophil functions. All of these immune processes are important in protection against infection by the colonization of periodontal pathogens.

4. This increases susceptibility to periodontal infection resulting in destructive periodontitis. The behavioral model (Figure -3) suggests that psychosocial stress may influence behavioral changes which affect health behaviors (i.e., smoking, poor oral hygiene, poor compliance). Additionally, stress leads to overeating, especially high fat diets which increases cortisol production.

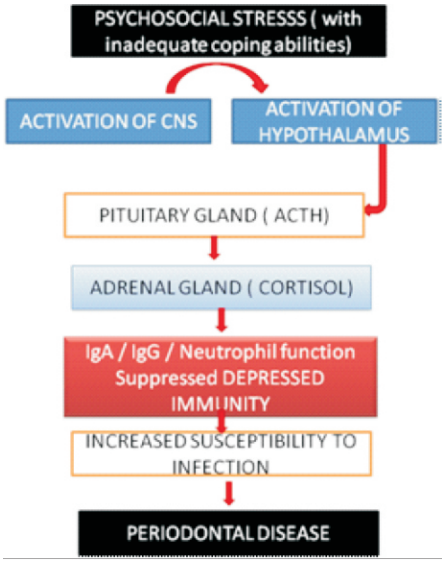
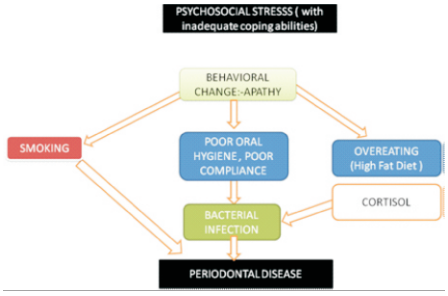


Figure 3 illustrates

Behavioral model to explain the role that stress has in influencing the onset and progression of periodontal disease.



Role of Stress On Periodontal Treatment

A study done by Kamma and Baehni [10] showed that supportive periodontal care was more effective in less stressful patients with aggressive periodontitis

Wimmer et al [11] explained the influence of coping with stress on periodontal therapy and concluded that patients who had maladaptative coping strategies have more advanced disease and thosepatients showed a poor response to non-surgical periodontal treatment

Gamboa et al [12] showed the influence of emotional intelligence which was used as a measure of the coping mechanism in patients with chronic periodontitis on the initial responses to periodontal treatment. The results of this study showed a decrease in plaque formation and reduction in bleeding on probing in patients with active coping strategy.

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 stress. Furthermore, multiple variables affect the severity of periodontal disease and there is uncertainty about the individual's onset of periodontal disease. Moreover, it is not possible to separate the effects of physical stress from emotional stress in these animal studies.

Furthermore, it is likely that systemic diseases associated with periodontal disease such as diabetes, cardiovascular disease etc., may share psychosocial stress as common risk factor. The available scientific evidence thus, does not definitively support a casual relationship between psychosocial factors and inflammatory periodontal diseases. The information reviewed above nevertheless does indicate the possible influence of psychosocial factors in the etiology of inflammatory periodontal diseases though at the moment, the more suggestive evidence relates to ANUG. These studies indicate that psychosocial stress represents a risk indicator for periodontal disease. Consequently, it is noteworthy that the practitioner is aware of these factors and taken them into consideration. The clinical management of inflammatory periodontal diseases might benefit from an exploration of these relationships, principally when 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 in explicable increase in the rate of periodontal destruction.[13,14]

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