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ORIGINAL RESEARCH

Association of Psychosocial Stress and Periodontal Disease

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ABSTRACT

Stress is found to be a predictive factor of periodontal disease. Among proposed mechanisms that mediate possible association between psychosocial factors and periodontitis status, immunological pathways are of great importance. Depressed immune responsiveness, as a result of physical or mental stress, influences stress-released hormones (glucocorticoids), which are present in the gingival crevicular fluid and may provide a source of nutrients that promote subgingival growth of periodontal pathogens, thus, leading to periodontal destruction. The purpose of this review article is to provide the progression of evidence present in the field of stress and periodontal disease for better understanding of the link.

Keywords: Glucocorticoids, Occupational stress, Periodontal disease.

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INTRODUCTION

Stress, a term continually being redefined in scientific study of disease and illness, is nevertheless a confirmed and important factor in the etiology and maintenance of many inflammatory diseases, including periodontal disease. Stress refers to a psychophysiological response of a living organism to a perspective challenge, change or threat.[1] Occupational stress (job stress) is a psychosocial disorder which is an impact of the interaction between the worker and his work environment on the worker themselves. If left unidentified it can cause serious physical and physiological illness to the individual, which affects both the individual and the organization.[2] According to the American Institute of Stress and WHO, the most common form of stress in the world is occupational stress. Thirty-seven percent of subjects who visited physicians and psychiatrist had occupation-related and stress-related problems in a study conducted in United Kingdom.[3]

The WHO reports that 5 to 70% of the population in various countries around the world belonging to the older age groups suffer from severe periodontitis.[4] Periodontal disease is considered more common in developing settings of Asia and Africa than in Europe and America.[4] Periodontitis is a multifactorial disease.[5,6] Periodontopathogens in dental plaque are considered as a main etiological factor for periodontal diseases but, in addition, several modifying factors are also involved in pathogenesis, like systemic diseases, some genetic polymorphisms, socioeconomic or educational status, tobacco smoking and psychological stress.[7] Earlier studies in industrial workers, in Norway and in India, have related periodontitis primarily to smoking as risk factor.[8,9] But, the long known relationship between smoking and stress, especially occupational stress, brings us more closely to associate between periodontitis and occupational stress.[3] Several researches have determined a statistically significant relationship between psychosomatic stress and the onset, course and outcome of periodontal diseases.[10,11]

Evidence for relationship between psychosocial stress and periodontal disease was also observed in a cross-sectional epidemiological study of 1426 aults. Results indicated a significant role for financial strain in relation to greater alveolar bone and periodontal attachment loss.[12] Moss et al collected self-reported information about depression and level of daily strain from such factors as jobs, financial, family and role-related strains, they related this information with adult periodontitis.[13] Their findings demonstrated that periodontal diseases have influences from immune system and psychosocial stress. The deleterious effects of stress are explained by hormonal modifications, behavioral changes, central nervous system and immunogenic causes to affect periodontal disease.[14,15]

The purpose of this article is to provide an overview for dental professionals to better understand the literature that has accumulated over the last 5 decades to support the role stress has in increasing the risk for the onset, rate of progression, and severity of periodontal disease and its resistance to treatment.

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IMMUNE FUNCTION

The biologic model proposes that periodontal disease may be biologically moderated through the hypothalamic-pituitary-adrenal (HPA) axis to promote the release of corticotropic-releasing hormone from the hypothalamus and glucocorticosteroid, from the adrenal cortex.[16] Following are the cascade of events:

- When a potentially stressful situation is appraised as threatening the central nervous system (CNS) and hypothalamus is activated.
- The hypothalamus induces secretion of corticotropin-releasing hormone (CRH) which flows to the pituitary gland where it stimulates the secretion of adrenocorticotropic hormone (ACTH). ACTH enters the peripheral blood flow, and induces the adrenal cortex to secrete cortisol and other steroids. Cortisol is a type of glucocorticosteroid and its increased production results in immunosuppression and reduced resistance to infection.
- Then glucocorticosteroids (hormones made in the body that have major suppressive actions on immune and inflammatory responses), including cortisol, depress immunity. 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. They reduce the number of circulating lymphocytes, monocytes and eosinophils, and inhibit the accumulation of macrophages, eosinophils and neutrophils at inflammatory site. All of these immune processes are important in protection against infection by the colonization of periodontal pathogens.[17]
- This increases susceptibility which leads to the beginning or advancement of periodontal infection eventuating in destructive periodontitis.

STRESS AND BEHAVIOR CHANGES

The behavioral mechanism emphasizes that people suffering from stress and depression may increase poor health behaviors, such as smoking or drinking, more frequently, consuming an unhealthy diet and neglecting their oral hygiene.[17]

Emotional conditions are thought to modify dietary intake, thus indirectly affecting periodontal status. This can involve, e.g. the consumption of excessive quantities of refined carbohydrates and softer diets, requiring less vigorous mastication and therefore predisposing to plaque accumulation at the proximal risk sites.[11]

One classical hypothesis for the role of stress in periodontal diseases was directly caused by bruxism and clenching that directly led to loss of periodontal attachment apparatus in the absence of other factors. The patient may be completely unaware of these repeated and sustained forced contacts of the teeth that seem to have no functional significance in humans.[18]

Use of tobacco and its products during stressful condition also work as risk factor for periodontal diseases.

STRESS AND MICROBIOLOGY

Potentially, pathogenic microorganisms possess the ability to recognize hormones has led to the development of the concept of 'microbial endocrinology'.[19] According to this concept, infectious organisms may utilize hormones present within the host as environmental cues to initiate growth and pathogenic processes.[20] Catecholamine hormone noradrenaline, whose release is up-regulated during stress responses, has been shown to have profound effects on the growth and virulence expression of a large number of microbial pathogens.[20] Thus, the effects of stress might be mediated via two potentially synergistic pathways involving down-regulation of host defences and up-regulation of bacterial pathogenicity. Several bacteria present in oral cavity showed significant catecholamine-induced growth enhancement indicating that stress hormones might directly modulate the growth and composition of the subgingival biofilm. So, this stress-induced cascade plays a significant role in the etiology and pathogenesis of the periodontal diseases.

Other important periodontal diseases of considerable interest are the necrotizing periodontal diseases, e.g. acute necrotizing ulcerative gingivitis. Two important organisms associated with these diseases are *F. nucleatum* ssp. *vincentii*, which showed positive growth effects with adrenaline, and *P. intermedia*, which showed a tendency for increased growth. The positive growth effects observed in these organisms could also provide a potential mechanism linking stress hormones and necrotizing periodontal diseases. The only previous study to investigate bacterial growth effects of catecholamines proposed that they acted as siderophore-like molecules and essentially destabilized iron binding by proteins, such as transferrin and lactoferrin, facilitating iron uptake by microorganisms.[21,22] As transferrin and lactoferrin are both found within the oral environment, this could provide a potential mechanism whereby subgingival organisms can survive in an iron-restricted environment or gain a survival advantage.

STUDIES ON STRESS AND GINGIVITIS/PERIODONTITIS

Emotional stress is actually looked upon as the most important predisposing systemic factor in acute necrotising ulcerative gingivitis (ANUG).[23] This is supported by clinical studies, experimental studies in dogs, and life event studies, indicating that ANUG could be traced back to anxiety and depression.[24] Deinzer R et al analyses the effects of academic stress on crevicular interleukin-1b (Il-1b) both at experimental gingivitis sites and at sites of perfect oral hygiene. Interleukin-1b is thought to play a predominant role in periodontal tissue destruction. These results indicate that stress might affect periodontal health by increasing local Il-1b levels especially when oral hygiene is neglected.[25] Evidences for relationship between psychological stress and periodontal disease was observed in cross-sectional epidemiological study of 1426 adults. Results of this sudy demonstrated that financial strain plays a significant role in alveolar bone loss and periodontal attachment loss.[12]

EFFECT OF TREATMENT

The coping is the effort to try to reduce, control or tolerate the state of stress. Wimmer et al explain the influence of coping with stress on periodontal therapy and conclude that patients with maladaptative coping strategies have more advanced disease and poor response to a non-surgical periodontal treatment.[26]

Kamma and Baehni evaluate the clinical and microbiological status of patients with early-onset or aggressive periodontitis (EOP) who had received supportive periodontal care (SPC) every 3 to 6 months for a period of 5 years, following active periodontal treatment. They found that supportive periodontal care was more effective in patients with aggressive periodontitis harboring less stress.[27]

Gamboa et al investigate the relationship between emotional intelligence (measure of coping mechanisms) and initial response to a simplified non-surgical treatment protocol in 29 patients with chronic periodontitis. An association between emotional intelligence domains and short-term changes in plaque and bleeding, and suggest that initial responses to standardized periodontal treatment may be partly related to emotional intelligence.[28]

PRACTICAL CONCLUSION

A high prevalence of mental stress and periodontitis and the inter-relationship between them is clearly understood through this review. This is recommending to the health ministry to improve the accessibility to free dental care and include psychotherapists services in the public healthcare systems. Health education campaigns promoting the availability of such services and also educating populations on the need for approaching such services is recommended. The employers and administrations should have in office counseling services in addition to the medical services provided within their campus.

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