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Dentistry

BALANCING THE PSYCHOSOMATIC IMBALANCE: ITS IMPACT ON PERIODONTAL DISEASE – AN OVERVIEW

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KEYWORDS :

INTRODUCTION

Health is a state of complete physical, mental, and social well-being and not merely the absence of a disease or infirmity.[1] People of different origin, religion, language, cultural status, socio- economic level are prone to mental illness. George Draper 30 years ago coined a term "Psysomatic" which later changed and was called "Psychosomatic". The word "Psychosomatic" is derived from the Greek terms psyche and soma where earlier "Psyche" meant "soul or mind" now means "behaviour" and "Soma" denotes "physical organism of the body." The American Medical Dictionary, twentieth edition, defines "psychosomatic" as "bodily symptoms of a psychic, emotional or mental origin". The interaction between body and mind has been explained by Sigmund Freud centuries back by the term "Conversion hysteria" which means an unresolved emotion is expressed as a somatic symptom.[2]

Psychosomatic disorders include a group of disorders which results in physiological changes due to emotional factors. They can also affect the oral cavity as the oral environment directly or indirectly is related to human instincts, passions and has a psychological potential. The link between psychosomatic disorders and oral diseases can be explained due to several biochemical disorders which involves neurotransmitters in brain, defect in connections in oral cavity and ill-defined complaints arising from cognitive processes in higher centers of the brain. As there is a link between body and mind, body reacts physically expressing symptoms to psychosomatic diseases.

During any stressful condition an individual's personality and the specific way of coping with environmental stimulus can affect the central nervous system thereby altering individuals host defense mechanism, exerting an immune suppressive effect and increasing their vulnerability to disease. On this stress exposure the individual shows some modifications in order to maintain internal homeostasis. Psychosocial stress generally influences nervous, endocrine and immunological systems resulting in expression of several symptoms. During depression increase in the levels of inflammatory cytokines like Interleukin-6, acute phase proteins like C-Reactive Proteins (CRP) is observed [3] as well as variations in secretion of hormones like insulin, cortisone, adrenaline, thyroxine by endocrine glands are observed.

Oral cavity acts as a window to the overall health status of an individual. Overall health of an individual includes physical, mental and social wellbeing. Oral cavity influences systemic health to a large extent as well as provides clues in identification of several systemic diseases. It is an organ showing certain exclusive characteristics of 'instinctional' cravings and highly charged with psychologic potential. When there is a disturbance in the psyche of a patient, it results in unfavourable changes in the tissues of oral mucosa including periodontium. This could be mainly due to improper maintenance of oral hygiene as well as down regulation of immune response.

Nowadays psychological disorders like anxiety and depression have become so common that a dentist should be in a position to identify oral diseases related to psychological problems. So dentists should also understand the relationship between oral diseases and psychology related disorders and also create awareness among patients regarding this interrelation. Anxiety related to dental treatment or dentist could also be due to an underlying phobia or trait of the patient. Understanding psychology of the patient will aid in providing better quality as well as painless treatment for the patient.

Oral disorders diagnosed as a result of psychological changes include Oral Lichen Planus (OLP), Recurrent Apthous Stomatitis (RAS), Erythema Multiforme (EM) and Mucous Membrane Pemphigoid (MMP), Necrotizing Gingivitis, Burning Mouth Syndrome, Temporo-Mandibular Disorders (TMD) and Atypical facial pain. Out of this most commonly observed conditions are Lichen planus, Recurrent apthous stomatitis and temporo mandibular disorders.

Chronic periodontitis is a multifactorial slow progressive infectious disease resulting in inflammation within the supporting tissues of teeth, progressive attachment loss and bone loss. Risk factors of chronic periodontitis includes plaque accumulation, diabetes, smoking, emotional stress and genetic factors. Psychosocial factors such as stress, distress, state and trait anxiety, depression and inadequate coping mechanisms are also counted as risk factors for degeneration of periodontium. Along with these factors changes in normal physiologic pathways like alteration in saliva, changes in gingival blood circulation, endocrine imbalances and altered host resistance will also result in deterioration of periodontal status of patient. Psycho neuro-immunologic effects were confirmed by findings of poorer immune functions in persons who experienced stressful life events or chronic stress.

An interrelationship between stress and periodontal disease has been suspected for centuries, but evidence to explain the connection has only elucidated in the past few decades. Direct association between periodontal disease and stress remains to be proven, which is partly due to difficulty in quantifying the amount and duration of stress and also there are many factors influencing the incidence and severity of periodontal disease. This overview provides the progression of evidence present in the field of stress, psychosocial factors, depression and ineffective coping, and other mechanisms linking stress and periodontal disease.

HISTORY:

The term "Psychosomatic" was first used in 1818 by the German psychiatrist, Heinroth. Felix Deutsch in 1922 was probably the first author to introduce the term "psychosomatic medicine". The 19thcentury experienced a more robust expansion of psychosomatic ideas. Explaining the mechanism of psychosomatic disorders, H.Freyberger and R.Sifneos stated that emotional reactions occur in two dimensions: psychological (sensual tons of pleasure or displeasure) and vegetative, which performs an important biological function of energy supply for the coherent behavior.

In the course of development of psychology as a science, at first scholars, lexicographers, and later psychologists began to study certain properties of human psyche, in particular, emotions. I. K. Heiroth defined the principles that a century later became the basis of psychosomatics. In 1822 a German psychiatrist M.Jacobi proposed the concept of "somatopsyche" in contrast and at the same time in addition to "psychosomatics", understanding it as the impact of physical defects on the course of mental life. In 1927 an Austrian psychoanalyst

F. Deutsch formulated the general concept of psychosomatics.[4] Back in the 1930s K.Ideler identified the differences in the psychological nature of fear and anxiety, but researchers addressed this problem only in the mid-twentieth century. Briefly his idea can be described as follows. If a human being is unable to overcome his or her fear of anything or anyone, he or she can run away, hide or ask for help for the reasons of fear exist objectively, that is beyond the human being. Therefore, humans often do not know what causes this anxiety, so they need to find the "enemy" not to deal with the causes of fear, but to have a "nervous discharge". If there is no enemy, the human being begins the "fight" on the field, where victory is secured – they start to massacre their own body. Suppressed aggression leads to self-destruction of the body and the occurrence of psychosomatic diseases.

NEUROANATOMY OF STRESS:

The stress response involves an efficient, evolutionarily conserved and complex system, with modulation in several levels of the central nervous system (CNS), governing learning, memory and strategic decision. The first step in the stress response is the perception of a stressor. When a situation is perceived as a threat, the brain recruits several neuronal circuits to maintain physiological integrity even in the most adverse conditions[5] However, detection of different types of stressors requires engagement of different networks. Psychological and physical stressors engage different neuronal networks and cellular activity, leaving distinct footprints within the brain.

PHYSICAL STRESSORS:

Physical stressors are mainly processed by brainstem and hypothalamic region, they usually require immediate systemic reaction, which might be considered reflexive. Thus, first phase of the stress response (Sympathetic Adreno Medullar system—SAM), provides a rapid physiological adaptation, resulting in short-lasting responses, such as alertness, vigilance and appraisal of the situation, enabling a strategic decision to face any challenge in the initial phase of a stressful event. The secondary phase involves the hormonal mechanism (Hypothalamic Pituitary adrenal axis - HPA) which is considered sluggish compared to the synaptic mechanisms that activate SAM, but resulting in an amplified and protracted secretory response (long-lasting responses).

PSYCHOLOGICAL STRESSORS

Psychosocial stressors are generally classified as either major stressful life events or more minor daily stressors or 'hassles.' One of the most important impetuses to investigating the relationship between psychosocial stress and disease states emerged from the work of Holmes[6] and colleagues, who demonstrated significant relationships between important life event changes and the onset and course of disease. First, it was major *negative* life events that more dependably occurred in close proximity to the onset or exacerbation of illness (or sometimes even death when the life event was death of a spouse); second, the relationship between important negative life events and disease was mediated by the immune system.

In addition to external environmental stressors, dysregulation of adaptive functioning by the body as it struggles to mount adaptive responses to the threat of disease or trauma itself constitutes a stressor to the person, having the potential to initiate and maintain a maladaptive spiral of increasingly widespread effects. Thus, the perceptions, attributions, and emotions associated with illness can themselves come to constitute an important set of stressors, exacerbating difficulties for the immune and stress systems as these systems struggle to restore physiological and psychological equilibrium. These changes are accompanied by altered activity at higher brain centres – for example, the amygdala, frontal cortex and hippocampus – which regulate thinking, emotions and behaviour.

STRESS AND INFLAMMATION:

Large bodies of evidence indicate that stress can activate inflammatory response in brain as well as peripherally. There exists a communication between the neuroendocrine and immune systems. Stress activates the HPA axis through hypothalamic secretion of Corticotropin-Releasing Hormone (CRH), which normally suppresses immune responses through release of Glucocorticoids (GCs) from adrenals. GCs are one of the major stress hormones released during stress response that are well known for their immunosuppressive and anti-inflammatory properties. Further, GCs reduce expression of several pro-inflammatory cytokines (e.g., tumor necrosis factor α (TNF- α), interleukin-6 (IL-6)) and enhance expression of anti-inflammatory cytokines (e.g., IL-10, TNF-β). GCs enhance the expression and function of inflammasome NLRP3, promoting secretion of IL-1β in response to ATP. Circulating pro-inflammatory factors such as IL-1, IL-6 and TNFα directly stimulate pituitary-adrenal axis, resulting in increased serum levels of Adreno Cortico Tropic Hormone (ACTH) and GCs, which in turn inhibit production of these pro-inflammatory factors. Interaction of immune system and HPA axis forms the endocrine negative feedback loops. However, when cytokine is over-stimulated in some diseases, these negative feedback loops could be weakened by reduced cytoplasmic GC-receptor (GR) level and decreased expression of GR driven anti-inflammatory genes, thus leading to GC low-responsiveness. Besides GCs, SNS and its main neurotransmitter, Norepinephrine (NE) and Neuropeptide Y (NPY), could regulate several immune and inflammatory function. NE promoted the secretion of inflammatory factors by increasing phosphorylation of Mitogen-Activated Protein Kinases (MAPKs) through a receptor-dependent pathway and NPY could elicit Transforming Growth Factor-β (TGF-β) and TNF-α production in macrophage-like cell line RAW264.7 via Y1 receptor.

Both pro-inflammatory and anti-inflammatory mechanisms depend on the type and intensity of stressors. Acute stressors seem to enhance immune function, whereas chronic stressors are suppressive. Intense stressors over-activate the immune system, leading to the imbalance of inflammation and anti-inflammation.

PERIODONTAL DISEASES

Psychological stress can directly affect periodontal health by various biological mechanisms, and also, it can have indirect effects through the changes in lifestyle such as ignoring oral-hygiene measures, smoking more heavily and consuming more fat and sugar in diet. In an early study, adult subjects under financial strain and exhibiting poor coping behaviours were reported to be at increased risk for severe periodontitis. [7] Periodontitis patients with inadequate stress behaviours strategies (defensive coping) were suggested to be at higher risk for severe periodontal diseases. In a clinical study, academic stress appears to affect periodontal health, shown by more plaque accumulation, gingival inflammation during the examination period of students.[8]

INFLUENCE OF STRESS ON PERIODONTIUM:

Periodontal diseases are inflammatory diseases associated with local and systemic elevations of pro-inflammatory cytokines such as TNF-a, IL-6 and prostaglandins and result in tissue destruction by the contribution of MMPs. Stress impairs the balance between pro-inflammatory and anti-inflammatory responses. The relationship between stress and periodontal diseases might be mediated by alterations in GCF IL-1, IL-6 levels and reduction in polymorphonuclear leucocyte chemotaxis and phagocytosis and reduced proliferation of lymphocytes. Subsequently, this process could increase vulnerability of periodontal tissues to pathogenic microorganisms by activation of cellular responses leading to local tissue destruction' Susceptibility to periodontal diseases seems to be partly explained by the inhibition of T-cell immune responses mediated by glucocorticoids. They can regulate the recruitment of immune cells into inflamed tissues, as well as changing Th1/Th2 balance towards a Th2-dominant response, and by this mechanism, inflammatory processes are able to shut down to prevent host destruction by the prolonged immune activity. Pro-inflammatory cytokines, such as IL-1 and IL-6, are also potent activators of the HPA axis demonstrating an important link between cytokines and glucocorticoids. It is negatively controlled by glucocorticoids and positively controlled by CAs leading to regulation of immune responses, acute phase protein synthesis and hematopoiesis.

Numerous studies have reported correlation between psychosocial stress such as academic stress, job-related stress and clinical periodontal parameters such as plaque accumulation and gingival inflammation. Patients suffering from periodontitis who are under stressful conditions have increased levels of IL-6 and IL-1b in GCF and similarly, patients with aggressive forms of periodontitis have elevated levels of IL-6 and IL-1β in serum. On the contrary, another study failed to find any correlation between IL-6 and IL-1β and cortisol levels in peripheral blood of aggressive forms of patients with periodontitis.[9]

Associations between clinical parameters of periodontal diseases, psychological factors and salivary markers of stress including CgA, cortisol, salivary α -Amylase, and β-endorphin, psycho neuroimmunologic variables and health behaviours are still under investigation mainly concerning mechanisms involved notably immunologic and behavioural changes related to psychological stress. A number of studies have investigated the effects of circadian rhythm on the stress-related markers such as cortisol. Cortisol has increased

concentrations towards the early hours of the morning peaking shortly after awakening and decreasing concentrations over the day and lowest at night. Taking samples soon after post-awakening could overcome this effect.

The possible mechanisms by which psychosocial factors act on periodontal tissues are oral hygiene negligence, changes in dietary intake, smoking, bruxism, changes in gingival circulation, alteration in salivary flow and components, endocrine hormonal changes and lowered host resistance. For example, individuals who smoked, had inadequate dietary habits, consumed alcohol or were physically inactive tended to be more irregular tooth brushers. It is reasonable to assume that such individuals are affected by anxiety or depression and poor coping skills.

Moreover, personality types, mental stability status, psychological disorders, lifestyle, variations in locus of control and coping style may alter the effects of stress. Harmful effects of stress in humans have been associated with undesirable life events such as financial and occupational strain, perceived ill health, loss of a spouse or loved one, academic strain, low socioeconomic status, low level of education, military combat, excessive noise and marital difficulties. One of the most possible mechanisms of influence of psychosocial factors on periodontal health is the alterations of patient's health behavior. Individuals with high stress levels and depression tend to adopt habits which are harmful to periodontal health, such as smoking and alcohol consumption, disruption of sleep patterns, neglecting oral hygiene and poor compliance, bruxism or teeth grinding and nail biting.[10] Besides, the excessive consumption of oily nutrients may cause increased levels of circulating cortisol, thus suppression of the immune system and progression of periodontitis

MODELS TO EVALUATE THE ROLE OF STRESS IN PERIODONTAL DISEASE:

A simple model to evaluate the role of psychosocial stress and coping behaviours in periodontal disease has been illustrated by Genco RJ et al[11] (FIGURE 1). According to this model, psychosocial stress can activate the central nervous system. The hypothalamus releases CRH which, among other things, stimulates release of ACTH from the pituitary, which in turn results in production of cortisol by the adrenal cortex. Glucocorticosteroids, including cortisol, then depress immunity including secretory IgA, IgG, and neutrophil functions, all of which may be important in Mental as well as physical stress can also result in responses being transmitted to the autonomic nervous system and then to the adrenal medulla, resulting in secretion of catecholamines such as epinephrine and norepinephrine. Catecholamines then affect prostaglandin and proteases, which in turn, could enhance periodontal destruction.

MENTAL STRESS RESPONSE LEADING TO BEHAVIORAL CHANGES:

In the second model (FIGURE 2) of the role of psychosocial stress on periodontal disease given by Genco RJ et al (1998)[11], it was hypothesized that the main effects of stress occur through behavioural changes which affect at-risk health behaviours such as smoking, poor oral hygiene, and poor compliance with dental care. There is also a possibility that stress leads to other behavioural changes such as overeating, especially a high-fat diet, which then can lead to immune-suppression through increased cortisol production. There are certainly other possible behaviour that could be affected by stress and inadequate coping and distress, such as depression, which would have significant effects on periodontal disease. Any evaluation of the role of stress in periodontal disease would have to take into consideration these at-risk health care behaviour to determine to what extent they contribute to the interaction between stress and periodontal disease.

STRESS AND AGGRESSIVE PERIODONTITIS:

There is a link existing between aggressive periodontitis and psychosocial factors and loss of appetite. Page RC et al (1983)[12] describes aggressive periodontitis as a particular disease and established the link existing between aggressive periodontitis and psycho-social factors and loss of appetite Kamma JJ and Baehni PC (2003)[13] made a study to evaluate the clinical and microbiological status of patients with early onset periodontitis who had received supportive periodontal care every 3 - 6 months for a period of 5 years following active periodontal treatment. The results showed that supportive periodontal care was effective, but some sites in some patients were still progressive. These variables were related to the progression of the disease: Porphyromonas gingivalis count, Treponema denticola count, total bacterial load, number of acute episodes, number of teeth lost, smoking and stress. The clinical and microbiological status evaluation of patients with early onset periodontitis 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.

SYSTEMIC INFLAMMATORY DISEASE AND PERIODONTAL DISEASE:

A number of chronic recurrent conditions, in addition to periodontal disease, are characterized by a fluctuating course, with ongoing disease punctuated by bouts of greater severity. It is well established that cardiovascular disease, diabetes mellitus, preterm delivery, osteoporosis, rheumatoid arthritis, inflammatory bowel disease, systemic lupus erythematosus etc., are related to stress either as a physiological response to stress or as a behavioural response. It may be that stress is a significant common risk factor for diabetes mellitus, cardiovascular disease, preterm delivery and osteoporosis, as well as periodontal disease (FIGURE 3). Alternately or simultaneously, stress that is modified by perceptions in coping can give rise to at risk health behaviours, which then could affect the same spectrum of chronic diseases. The more severe bouts of all these conditions involve activation of the immune response and an associated increase in inflammation. The body of evidence on the relationship of stress to disease activity appears to be greatest for rheumatoid arthritis; however, because of the types of tissues affected, information on inflammatory bowel disease may be particularly pertinent to periodontal disease.

STRESS AND WOUND HEALING

Stress is an important factor that affects normal wound healing processes and wound healing is less affected in patients with active coping skills than in patients with inadequate coping behaviour.

Altered glucocorticoid level (cortisol) and higher catecholamine level (epinephrine and nor-epinephrine) may lead to any or all of the following:

1. Hyperglycaemia which impairs neutrophil formation and thus impairs initial phase of wound healing.
2. Decreases level of growth factors which may down regulate tissue repair system.
3. Alters cytokine profile that may affect recruitment of macrophages and fibroblast causes impaired wound remodelling.
4. Decreases the MMP level which impairs tissue turnover and causes decrease in wound healing.

EVALUATION OF PATIENTS WITH STRESS RELATED ORAL DISORDERS

The current approaches to measure stress include Self report of stress (for Eg., Perceived stress scale – questionnaire with questions designed to provide information about how unpredictable, uncontrollable and overloaded the respondents felt their lives to be) and Measures of affect (for eg., Profile of moods state; POMS questionnaire – assesses transient, fluctuating affective mood states. Consists of 6 identifiable affective states which are rated by the subjects on a 5 point scale). Measures of stressor exposure (for eg, Major life events stress scale) and lastly the stress biomarkers (for eg., Cortisol, C-Reactive protein and interleukins), or using a composite set of parameters (Allostatic load model) which measure either the stressor or the stress response. The stress biomarkers commonly researched include:

- Metabolic markers – S. cholesterol, high-density lipoprotein (HDL) cholesterol, Total cholesterol-HDL ratio, S. Albumin, Glycosylated hemoglobin
- Immunological markers – Interleukin-6 (IL-6), Tumor necrosis factor (TNF-α), C-Rreactive protein (CRP), Insulin-like growth factor (IGF-1)
- Neuro-endocrine markers – Cortisol, Dehydroepiandrosterone (DHEA), and cortisol- DHEA ratio, adrenaline, noradrenaline, dopamine and aldosterone.

Blood and saliva can be used to monitor the systemic as well as the oral health status. Furthermore, stress is also a factor that can be followed by analysis of saliva, especially by determining the levels of stress-related markers. These markers have biological properties that influence genesis and development of periodontal diseases. There are numerous stress-related molecules involved in different aspects of stress response. The following major salivary and blood stress markers have been studied so far more intensively with regard to psychological stress and periodontal disease interactions.

MANAGEMENT OF STRESS:

Coping against stress is the effort to try to reduce, control or tolerate the state of stress. It needs adjustment, adaptation and confrontation strategies. These coping strategies may be used in generalized stressful situations. Individuals use coping measures to reduce its intensity or to overcome stress altogether. A successful coping is when the subject has the feeling to face the stress and able to control the given situation. An unsuccessful coping is when the subject is submerged by stressor agents and is in the reaction of stress.

Stress Reduction Protocol

The stress reduction protocol includes two series of procedures that when used either individually or collectively, act to minimize stress to the patient during treatment and thereby decrease the degree of risk presented to the patient.

1. Stress reduction protocol for normal, healthy anxious patients. (ASA I)
2. Stress reduction protocol for medical risk patient (ASA II, III, IV)

Stress Reduction Protocol in Dental Office Includes:

1. Recognition of medical risk and anxiety.
2. Medical consultation.
3. Premedication (Antianxiety or sedative-hypnotic drugs given one night before the appointment or one hour before appointment).
4. Appointment scheduling.
5. Minimized waiting time to reduce anxiety.
6. Vital signs monitoring: BP, heart rate, rhythm and respiratory rate
7. Psychosedation.

Stress Reduction by the Patient Includes

There are several ways of coping with stress. Some techniques of time management may help a person to control stress. The Journal of the Canadian Medical Association have recently dubbed "Destressitizers" as any process by which an individual can relieve stress.

1) Regular exercise helps to burn off and use up the stress hormones and neurochemicals.
2) The benefits of meditation and other relaxation techniques for 20 to 30 minute sessions a day can have lasting beneficial effects on reducing the stress levels.
3) Elimination of drug use and not more than moderate alcohol use is key to the successful management of stress.
4) M. Strengthen your relationships by building a strong support network which works as greatest protection against stress. Spend time with the people you love and don't let your responsibilities keep you from having a social life.
5) Learn better ways to manage time. Think about which things are most important, and do those first.
6) Find better ways to cope. Look at how you have been dealing with stress. Be honest about what works and what does not. Think about other things that might work better.
7) Take good care of yourself. Get plenty of rest. Eat well. Don't smoke.
8) Try out new ways of thinking. When you find yourself starting to worry, try to stop the thoughts. Work on letting go of things you cannot change.
9) Speak up. Not being able to talk about your needs and concerns creates stress and can make negative feelings worse. Assertive communication can help you express how you feel in a thoughtful, tactful way.
10) Ask for help. People who have a strong network of family and friends manage stress better.

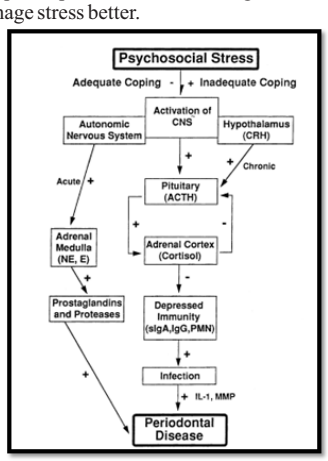

FIGURE 1 - A physiological model for effects of stress on periodontal disease (Genco RJ et al 1998)[11]

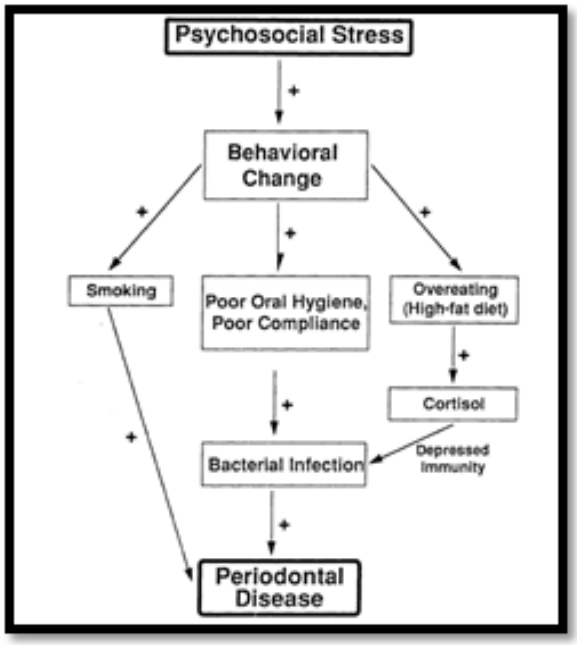

FIGURE 2 - Psychosocial stress and its effect on behavior as manifested by alterations on periodontal disease (Genco RJ et al 1998)[11]

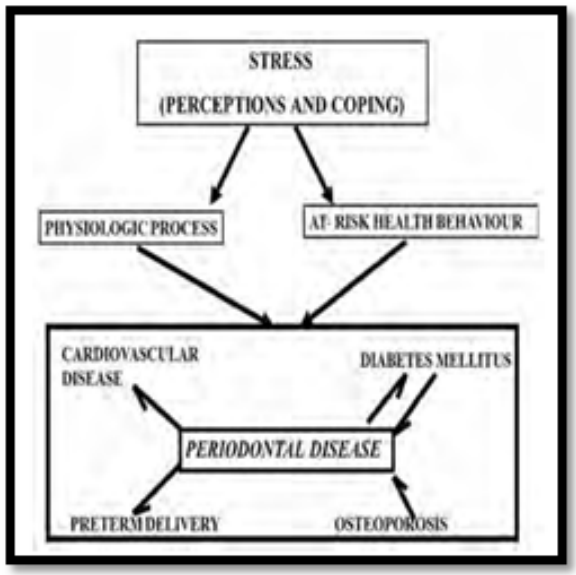

FIGURE 3 - Stress correlation with systemic diseases and periodontal disease (Goyal S et al 2013)[14]

CONCLUSION

Stress is a part of the human being that is present universally with varying degrees and has different effects on individual's health. Chronic stress and depression have been hypothesized to reduce immune responsiveness, resulting in a higher rate of infection with pathogenic organisms and a greater degree of periodontal tissue destruction. Moreover, substantial evidence also indicates that these conditions results in periodontitis through changes in health related behaviours, such as oral hygiene, smoking and diet. The relationship between stress and periodontal disease can be verified in salivary and GCF samples which can be helpful for differential diagnosis in several medical fields. It is essential to develop reliable chair side tests for evaluating the markers to make better diagnosis. Specifically cortisol, catecholamines and alpha-amylase may open up new perspectives in the field of periodontal research to categorise a patient according to the risk factors that may enable prompt medical intervention.

It seems crucial to educate periodontists about the role that psychosocial factors can play in the treatment process. Coping

mechanisms and enhanced perceptions of positive outcomes can better a patient's level of wound healing and future research should focus on developing targeted interventions that periodontists could use to improve the quality of life of their patients during treatment and the treatment outcomes.

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