Stressed Periodontium in the Line of Fire- A Case Report

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Abstract
Periodontitis and its relationship with psycho-neuro-immunological variables, such as stress and cortisol levels, have been explored. "Stress is an association of physiological and psychological reactions of a person confronted to a change of situation he cannot face." The relationship between stress and any disease is explained by hormonal modifications and behavioural changes induced by the stress. This case report supports the concept of stress being a risk determinant for periodontal disease.

Key words: Stress, periodontitis, immune response, cortisol.

Introduction

The term stress in layman terms describes adverse emotions or reactions to unpleasant experiences. "Stress is a state of physiological or psychological strain caused by adverse stimuli, physical, mental or emotional, internal or external, that tend to disturb the functioning of an organism and which the organism naturally desires to avoid."[1]
Genco et al., in 1998 classified stress as a risk determinant for periodontal disease.[1]
Stress is associated with activation of the hypothalamus-pituitary-adrenal axis that links the brain and the immune system. Increased level of cortisol suppresses the immunity and neurotransmitters increase tissue destructive function.
The role of stress in human periodontal disease has a plausible pathophysiological basis. Evidence has suggested that stress is associated with more severe periodontal disease, as well as poorer healing responses to traditional periodontal therapy.[2] This is because stress can cause behaviour modification (e.g. smoking, alcohol abuse, etc.) and immunosuppressive effects (e.g. decreased polymorphonuclear leukocyte function, altered T helper 1 cell/T helper 2 cell ratio, etc.), which may result in greater severity of periodontal disease as well as delayed wound healing.[3]

Pathophysiology of the stress responses

Stress can result in the de-regulation of the immune system, mediated through the hypothalamic-pituitary-adrenal axis or sympatheticadrenal medullary axis (Figure 1).[1] Importantly pro-inflammatory cytokines such as interleukin-1 can also activate the hypothalamic-pituitary-adrenal axis, leading to a feedback loop.[1] Success in the later stages of healing is critically dependent on the early events mediated by pro-inflammatory cytokines. So if the cytokine profiles are modified by stress, it would delay wound healing. The second major pathway to be activated is the sympathetic nervous system. A well-known example is so-called "flight or fight" response to potentially harmful stimuli. Stress activates the nerve fibers of the autonomic nervous system, which innervates the tissues of the immune system.
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, e.g. chronic anxiety state and depression.[3]

Case report

A 40-year-old male, Police personnel, reported to the Department of Periodontics with chief complaint of burning sensation in gums in upper and lower anterior region. Burning sensation occurred while eating spicy food and drinking hot tea since 2-3 months. The sensation used to subside after drinking cold milk. He also noticed bleeding from gums while brushing and also occasional spontaneous bleeding in the lower anterior region since 5-6 months.
Close questioning revealed a link between the episodes of burning sensation, increased gingival redness and stress. Patient gave history of increased psychological stress and night shift since 3-4 months.
Since his childhood, for 20 years, the patient used mishri, ash and powder once daily to clean his teeth. He now uses brush and paste once daily. He also uses lime stick occasionally for cleaning teeth. No positive correlation to the condition was observed with his medical and family history.
Intraoral examination (Figure 2-4) revealed generalized reddish pink, soft-oedematous gingiva with pebbled, granular surface and loss of stippling except in the lower posterior sextants. The patient had generalized bleeding on probing with no exudation and generalized 5mm probing pocket depth. Grade I mobility and recession of 2mm was noticed with 31, 41. Supragingival and subgingival plaque, calculus accounted for oral hygiene index score of 5 which denoted poor oral hygiene.
Following investigations were performed-

1. The radiographic (Intra-oral periapical X-ray) findings, which corroborated with those of the clinical examination, revealed blunting of alveolar crest in the maxillary arch and horizontal bone loss upto middle third of root in the mandibular arch (Figure 5,6).
2. An incisional biopsy was performed from attached gingiva on the buccal aspect from distal of 11 to mesial of 13 region under local anesthesia. The tissue was immediately fixed in 10% buffered formaldehyde solution and sent for histopathologic examination. The microscopic evaluation of the tissue sections (Figure 7) revealed parakeratinized stratified squamous acanthotic epithelia with thin long rete ridges extending into the connective

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tissue. The underlying connective tissue showed dense wavy bundles of collagen fibres containing numerous fibrocytes and fibroblasts. Some sections in the connective tissue exhibited infiltration of chronic inflammatory cells, a few scattered multinucleated giant cells and areas of neovascularization that had red blood corpuscles within the lumen of the blood vessels. So the histopathologic diagnosis was Inflammatory Gingival Hyperplasia without any specific signs of an identifiable cause.

3. Serum cortisol test: This test was done using MEIA/ FPIA AXsYM Immunoassay Analyser. Serum cortisol level was 30µg/dl prior to phase I therapy. (Normal value: 5.0 to 25.0µg/dl).

Based on all these findings, a provisional diagnosis of acute exacerbation of chronic periodontitis with stress as a predisposing factor was confirmed.

Patient initially underwent phase I therapy that comprised scaling and root planing, oral hygiene instructions, adjunctive administration of systemic doxycycline (100mg twice a day on the first day, followed by 100mg once a day for 14 days) and stress management protocol. Stress management included well-balanced diet, consistent sleep schedule, breaking job down into small parts, taking daily exercise etc. Phase II therapy involved periodontal flap surgery in the maxillary arch and mandibular anterior sextant. The patient came for follow up regularly every month for three months during which uneventful healing was observed.

Discussion

Psychological stress, particularly if sustained over an extended period of time can have deleterious effects on the body, representing an important example of the mind-body interaction. This article emphasizes the potential role of psychological stress in periodontal disease progression as well as on wound healing.

Gingival condition at baseline visit clearly reflected the severity and acuteness of signs and ongoing periodontal disease activity. In view of the apparently exaggerated inflammatory response to low levels of plaque exacerbated by episodes of stress, serum cortisol test was performed. Incisional biopsy was performed to rule out desquamative gingivitis. Histopathologic evaluation was suggestive of inflammatory gingival hyperplasia.

Use of mishri and ash for cleaning teeth reflects his unawareness and neglect of oral hygiene, which may be due to his busy work schedule. This finding is supportive of various studies which have established a link between stressful life events and poor oral hygiene.[1]

After complete clinico-radiographic examination and laboratory investigations correlated with the history, case was diagnosed as an acute exacerbation of chronic periodontitis with stress as a predisposing factor.

Management of the case was started with phase I therapy and stress management. Patient was recalled after one month to evaluate the response of initial treatment. At this time, inflammation was less intense, there was no complaint of burning sensation but pockets were persisting (Figure 8-10). There were no deposits. Patient was regularly monitored clinically for improvement in his periodontal condition. When he reported to the hospital after one month for recall visit, generalized chlorhexidine stains with supragingival calculus formation was noticed due to over- use of mouth rinse (more than 14 days).

Patient was prescribed doxycycline for 21 days as an adjunctive therapy. But there was no definite improvement in the clinical condition and pockets were persisting. Patient was scheduled for periodontal flap surgery (open-flap debridement) to eliminate pockets, making plaque control easier and to enable the regeneration or repair of the alveolar bone defect. He responded well to the surgical phase of periodontal therapy as evaluated on subsequent recall visits (Figure 11-13). Serum cortisol level decreased to 13.9µg/dl after psychological and periodontal management of the patient as compared to baseline value of 30µg/dl. At three months recall visit, there were no deposits, pockets, no burning sensation but mild inflammation with edematous papillae was still persisting (Figure 14-16). This could be explained by stress response related to his occupation.

The role of stress in this case requires careful interpretation. Numerous studies have implicated a role for stress in the pathogenesis of periodontal diseases, ever since the early studies linking acute necrotizing ulcerative gingivitis (ANUG) with stress.[4]

In general, human studies lack both a standardized method to define stress and to quantify it. Defining stress ranges from subjective assessments of stressful situations such as during examinations or military service, to physiological measurements such as plasma cortisol levels, cytokine profiles and heart rate.[5,6] A major limitation is that all these parameters show divergent reaction patterns both within an individual at different time points and between different individuals. In addition the types of studies that predominate in this area of investigation within the dental field are case series and cross-sectional studies. These study designs, by their very nature, preclude a cause-and-effect relationship to be concluded. Prospective clinical trials are required at this time.

Another potential confounding factor is that subjects under high levels of stress may be more likely to report symptoms. It is unclear whether it is stress that is causing the reported signs and symptoms or whether the signs and symptoms arising from a disease process cause the subject to feel stressed. Future studies will benefit from a prospective study design incorporating both physiological and psychological measurements of stress.

Conclusion

This case report highlights the co-existence of stress and active periodontal disease. Diagnosis was based on clinical, radiographic, histopathologic and serological assessment.

Stress management may be a valuable component for current periodontal practice. However, at present the majority of the literature consists of case series and retrospective studies. There are even fewer studies dealing with the role of stress and periodontal wound healing. Thus, the exact role of psychological factors in periodontal wound healing remains to be elucidated, and further well controlled, prospective clinical trials are warranted.

Clinical implications

In today's scenario, stressful life events are very well

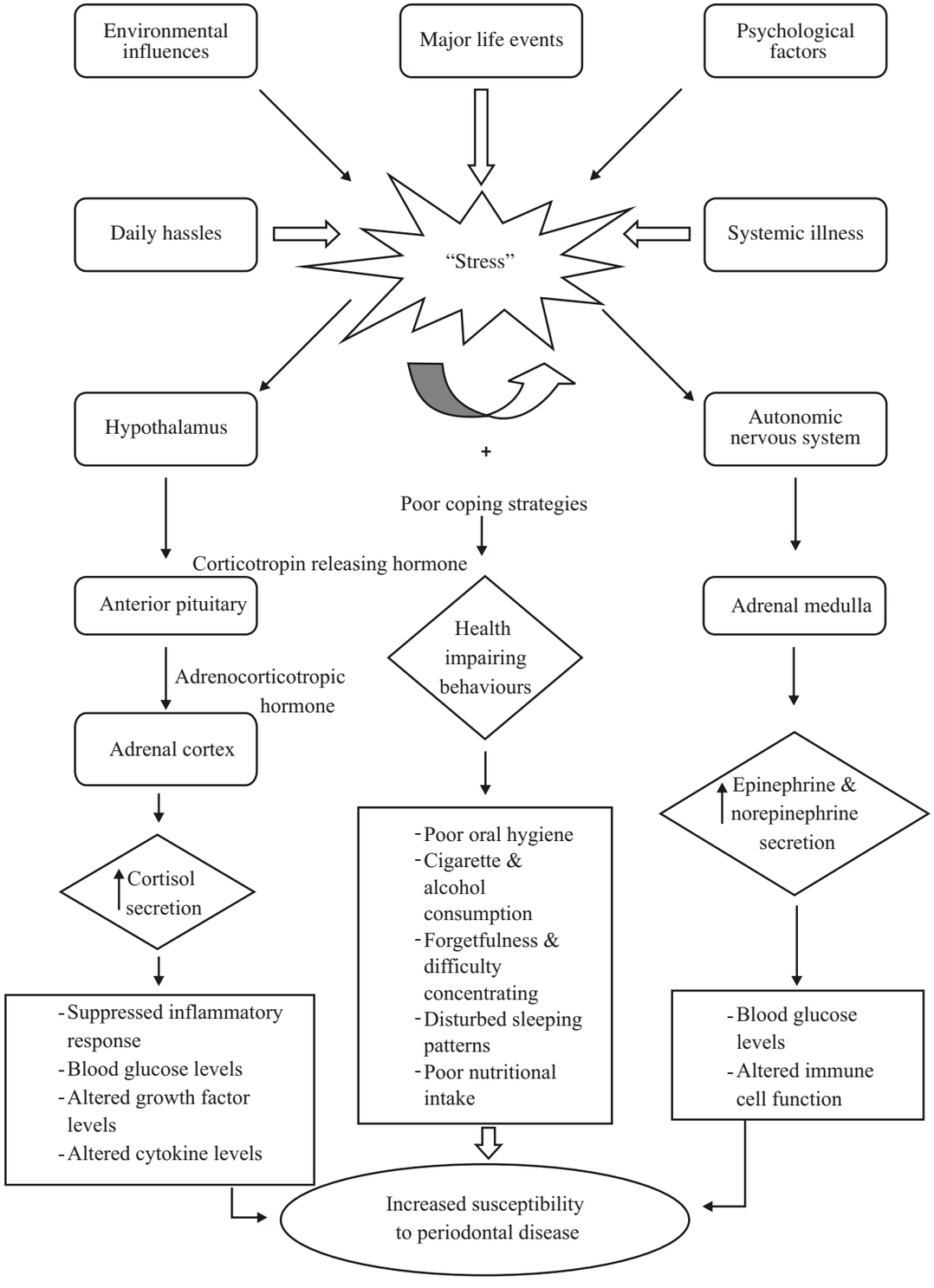

Figure 1: Pathophysiology of the stress responses

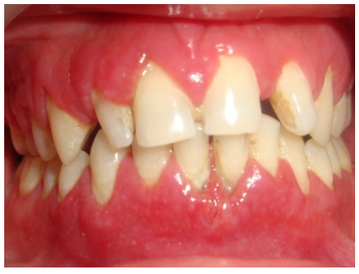

Figure 2: Front view (Reddish-pink, soft-oedematous gingiva at baseline)

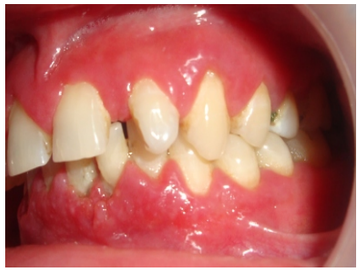

Figure 3: Left lateral view (Pebbled, granular surface of gingiva)

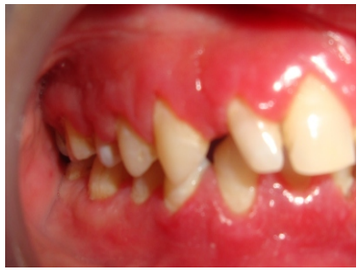

Figure 4: Right lateral view (Stippling lost)

Figure 5: IOPA with 21,22,23 (Blunting of alveolar crest)

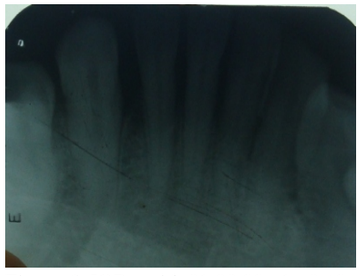

Figure 6: IOPA with 31,32,33,41,42,43 (Horizontal bone loss upto middle 3rd of root)

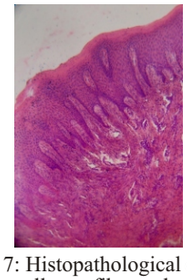

Figure 7: Histopathological examination (Dense collagen fibres, chronic inflammatory cells, a few scattered multinucleated giant cells and neovascularization)

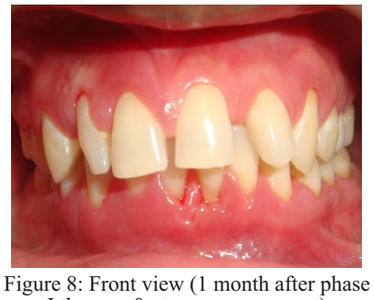

Figure 8: Front view (1 month after phase I therapy & stress management)

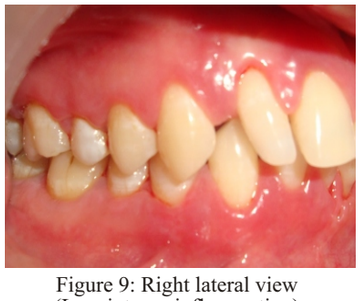

Figure 9: Right lateral view (Less intense inflammation)

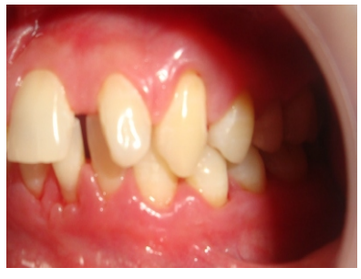

Figure 10: Left lateral view (No deposits)

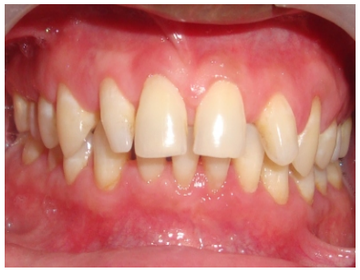

Figure 11: Front view (After periodontal flap surgery)

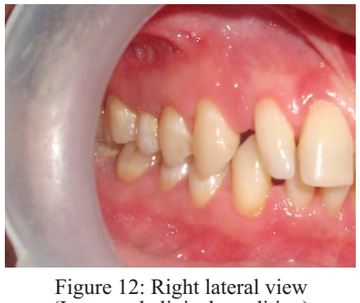

Figure 12: Right lateral view (Improved clinical condition)

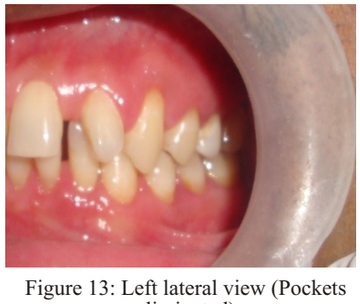

Figure 13: Left lateral view (Pockets eliminated)

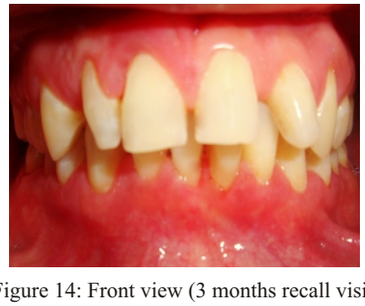

Figure 14: Front view (3 months recall visit)

Figure 15: Right lateral view (No deposits, pockets)

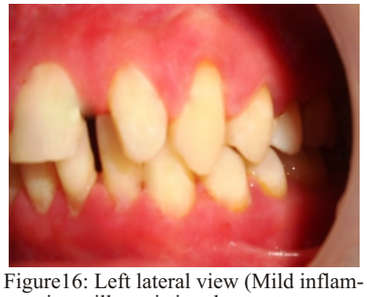

Figure16: Left lateral view (Mild inflammation still persisting due to stress response related to his occupation)

associated with all the individuals. Assessment of a patient's stress levels (perhaps more importantly their ability to cope with stress) and stress-reduction protocols might be of value when instituted as part of routine periodontal treatment.

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