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EFFECT OF TOBACCO USAGE AND PSYCHOSOCIAL STRESS ON CHRONIC PERIODONTAL DISEASE: A COMPREHENSIVE REVIEW OF THIS INIMICAL TRIAD.

Dental Science

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ABSTRACT

Over past few decades, the incidences of stress-induced disease has been on a rise and it has become one of the major reasons for increased prevalence for smoking and tobacco use amongst the young and adults. It has been stated that stress in general is one of the major causal factors for initiation of tobacco use followed by peer pressure. Apart from causing lung cancer, respiratory diseases, cardiovascular diseases, stroke, etc; smoking and smokeless tobacco also leads to detrimental effects on oral health leading to progression of periodontal diseases & oral cancer and significantly affecting the quality of life. Tobacco products appear to have direct local effects on periodontal tissue and can alter the host response. Although smokers benefit from periodontal therapy, clinical improvements are less than those for non-smokers. Also, stress has been considered one of the identifiable risk factor for periodontal disease progression. Analogous to smoking stress hypothesized to reduce immune responsiveness, resulting in more pathogenic infection and concomitant periodontal tissue destruction. This paper addresses the inter-relationship between smoking, periodontal disease and stress.

KEYWORDS

stress, tobacco use, smoking, periodontal disease.

Tobacco abuse has become a major cause of increased morbidity and mortality in our country. In India prevalence of tobacco use is increasing at an alarming rate, despite efforts by World Health Organization (WHO) and Govt. of India in eradicating it. The Global Adult Tobacco Survey, India 2009-10 revealed 46.3% of smokers and 26.7% of users of smokeless tobacco were advised to quit (GATS, 2009; 2010).[1] The nicotine present in tobacco is highly addictive and is accountable for the feeling of pleasure and is considered as a potent stress reliever by smokers or tobacco users. It has been stated that stress in general is one of the major causal factors for initiation of tobacco use followed by peer pressure. Smoking improves concentration, reaction time, and performance of certain tasks and hence alleviates job-related stress/strain, perceives and provides a feeling of job satisfaction.

Apart from alleviating or reducing stress and providing a feeling of improved coping skills, smoking has also been observed to worsen and deteriorate periodontal health. Smokers tend to have more destructive periodontal disease, deeper pocket formation, greater clinical attachment and bone loss and poorer healing capacity as compared to non-smokers. Smoking down regulates the immune mechanism of the body thereby causing rapid and greater periodontal destruction. Also, stress has been considered one of the identifiable risk factor for periodontal disease progression. Analogous to smoking stress hypothesized to reduce immune responsiveness, resulting in more pathogenic infection and concomitant periodontal tissue destruction. This paper addresses the inter-relationship between smoking, periodontal disease and stress.

STRESS AND TOBACCO USE

Over past few decades, the incidences of stress-induced disease has been on a rise and it has become one of the major reasons for increased prevalence for smoking and tobacco use amongst the young and adults. Stress in general and particularly job-related stress might affect smoking behaviour because smoking has been believed to relieve stress and improve concentration disorders. The literature shows a greater concern not only for youth smoking, but stress and smoking in adults have also been extensively examined.[2,3] As observed in a no. of studies, the inability to quit smoking and the relapse has been mainly associated with negative and stressful life events like divorce, death of a relative, financial problem, illness or injury or a move to a new residence.[4-8] Studies have shown that job stress can be harmful for health and one of the causes for it being poor and destructive habits like smoking. In a study by Hellerstedt and Jeffery (1997)[9], it was found that the smoking intensity is directly proportional to the job demands for men as well as for women. It has been postulated that there are two methods by which stress, in particular job-related stress may be linked with smoking: 1. Stress challenges our body physiologically and psychologically and as a result of which an individual responds to it by indulging into self-medication through smoking so as to maintain homeostasis.[10-12] 2. Stress can negatively influence a person's self-control. Muraven and Baumeister (2000)[13] find evidence that self - control is a limited resource so that coping with stress (e.g., inhibiting negative emotions at work) reduces the amount of self - control available for subsequent tasks (e.g. controlling the urge to smoke). About 70% of smokers lacked the will power to quit smoking though they were willing to do so. About 40% try to quit each year but fail. In the analyses of the cross-sectional data, higher odds of job strain was recorded in current smokers than never smokers (age, sex and socioeconomic position-adjusted odds ratio: 1.11, 95% confidence interval: 1.03, 1.18). Current smokers with job strain smoked, on average, three cigarettes per week more than current smokers without job strain.[14] To reduce the disease burden associated with smoking, it is essential to understand the determinants of tobacco smoking: the two most possible determinants being stress in general and work-related stress in particular. However, recent observational studies of the relationship between work stress and tobacco smoking have produced mixed findings, with positive, negative and null-associations reported.[15-19] It can be noted that those smokers who are unable to cope with job-stress can be more susceptible to smoking-associated disease due to a higher smoking frequency. Amongst the various types of occupational or job-related stress; the police or the law enforcers are ranked as among the top ten most stressful jobs in the world.[20] Law enforcement officers have high level of stress not only due of their nature of work, but also because of the environmental influences linked with their work. Distress and other health problems might occur as a result of not coping with their serious operational duties.[21] It is a universal fact that stress is an obvious part of police life. As a result of such a high magnitude of stress, certain symptoms like fatigue, headache, sleeplessness, irritability, lack of concentration, etc. are reported at a higher frequency and Majority of them resort to smoking and drinking as a coping mechanism.

TOBACCO DEPENDENCE: WHY??

Nicotine is the active ingredient responsible for addiction. All leading

authorities, including WHO, the Royal College of Physicians, and the American Psychiatric Association (APA),[22-24] have supported the three major conclusions of a 1988 report by the Surgeon General of the United States[25] regarding nicotine and tobacco: 1. Cigarettes and other forms of tobacco are addictive. 2. Nicotine is the drug in tobacco that causes addiction. 3. The physiological and behavioural processes that determine tobacco addiction are similar to those that determine heroin and cocaine addiction. Nicotine has a dual effect; it acts as both a stimulant and a depressant. It is reported that it increases attention, concentration, memory and improves the information processing and learning ability. Also, it alleviates anxiety, stress, pain and depression. Because of its stimulating properties and ability to relieve stress, smokers consider smoking to be an appropriate act in a negative or stressful situations.[26] The dopaminergic pathways of the mesolimbic system in the brain,; which is responsible for drug abuse and its reinforcement is stimulated by nicotine.[27] It binds to the nicotinic acetylcholine receptors in the brain (nAchRs), which causes the release of dopamine and the subsequent release of neurotransmitters, that results in number of physiological effects, including behavioural arousal and neuralactivation.[28 29] This release of dopamine, norepinephrine, and serotonin is associated with feelings of pleasure and appetite suppression. When there is excessive secretion of AcH associated with nicotine consumption, there is a tremendous improvement in an individual's attention and concentration abilities[30] increase in vigilance in the performance of repetitive tasks, and memory and mood improvements.[29] There is a reported decrease in anger, depression, tension and stress with a positive improvement in mood after smoking. The perceived calming effect from the reduction of withdrawal symptoms may be what nicotine users find reinforcing. Smoking improves reaction time and performance of certain tasks. The prime reason for the enhanced performance and improved mood may be due to the relief from withdrawal symptoms. Smoking cessation leads to withdrawal symptoms like irritability, depressed mood, restlessness, and anxiety. Because of the highly addictive nature of smoking and the rapid rate of action, tobacco dependence is very high and it's an extremely difficult task to quit smoking.

TOBACCO USE AND PERIODONTITIS

Periodontitis is a multifactorial disease with complex interactions between infectious agents and host factors. Disease expression can be modify by Environmental, acquired, and genetic risk factors which ultimately affect the onset or progression of periodontitis.[31] Among the environmental risk factors, Tobacco smoking usually found to be associated with an increased prevalence and severity of periodontal disease.[32] Haber has described a discrete, smoking-specific disease entity—smoking associated periodontitis—characterized by fibrotic gingiva, lesser gingival redness and oedema in relation to disease severity, deeper pockets in maxillary anterior and palatal sites, gingival recession in anteriors, no correlation between periodontal status and level of oral hygiene.[33 34] Bergstrom et al stated that the smoking frequency is directly proportionate to the disease severity.[35] It is also associated with an increased risk of clinical attachment loss, pocket formation and alveolar bone loss. Grossi and colleagues proposed the relationship between smoking and attachment loss and demonstrated a dose dependent relationship wherein greater attachment loss was observed in smokers when compared with non-smokers, approximately 2.05 mm for light smokers to 4.75 mm in heavy smokers.[36] In smokers, emotional stress and poor oral hygiene seem to have an important interactive role.[37] It has been observed that the bleeding on probing is less severe in smokers as compared to non-smokers. This reduced bleeding on probing is accounted to the vasoconstrictive effects of nicotine on the gingival vasculature.[38] The incidence of gingivitis and bleeding on probing is similar in smokeless tobacco users and non-users but smokeless tobacco use leads to localized gingival recession and alveolar bone destruction at the site of placement due to collagen breakdown and increased collagenase activity.[39] This suggests that tobacco smoking exerts a masking effect on gingival inflammation, which might give smoking patients a false sense of assurance of gingival health.[40] The expression of pro-inflammatory cytokines like IL-1 is up-regulated due to smoking which increases periodontal tissue damage and alveolar bone resorption. Interleukin-1 genotype positive smokers are more susceptible to severe adult periodontitis.[41] The host-bacterial interactions in smokers is altered and results in more aggressive periodontal tissue breakdown. This imbalance between bacterial challenge and host response may be due to changes in the periodontal microflora with increases in the numbers and/or virulence of pathogenic organisms; changes in the host response to the bacterial challenge; or a combination of both. A significant increase in the prevalence of orange and red complex organisms including Eikenella nodatum, Fusobacterium nucleatum ss. vincentii, P. intermedia, Peptostreptococcus micros, Prevotella nigrescens, T. forsythensis, P. gingivalis, and Treponema denticola was seen in current smokers than in former smokers and non-smokers when screened for 29 different subgingival species using checkerboard DNA-DNA hybridization technology. A striking finding of particular interest was that the increase in the prevalence of these pathogenic microflora was due to increased colonization of shallow sites (pocket depth <4 mm) with no differences between smokers, former smokers, and non-smokers in pockets deeper than 4 mm.[42] Smoking exerts deleterious effects on periodontal tissues by down-regulating the immune responses to the pathogenic microflora.[43] When exposed to tobacco smoke in-vitro; neutrophils showed altered chemotaxis and phagocytosis. In addition, smokers have an increased number of T cells in the periodontal tissues, another host response cell that uses the integrin/selectin system to migrate into tissues. Based on these findings it has been hypothesized that in smokers, the neutrophils migrate into the periodontal tissues but their activity and further progress into the periodontal pocket may be impaired due to continual exposure to smoke. Also, IgG2levels which are essential for phagocytosis and bacterial killing are reduced in smokers with periodontitis than non-smokers. On the other hand, the GCF of smokers' demonstrated elevated levels of TNF-α, PGE_2, neutrophil elastase, and matrix metalloproteinase.[44] These data suggest that smoking impairs the response of neutrophils to periodontal infection but may also increase the release of tissue-destructive enzymes and hence leads to an aggressive periodontal breakdown.

STRESS AND PERIODONTAL DISEASE

Chronic inflammatory diseases, such as periodontitis, have a complex pathogenesis and a multifactorial etiology, involving complex host–parasite interactions.[45] Cigarette smoking, poor nutrition, alcohol consumption and low socio-economic status also have been shown to be associated with a higher risk of periodontitis.[46] Literature review indicates that psychological stress and ineffective coping can influence the onset and progression of many chronic diseases, including periodontitis.[47] It has been hypothesized that chronic stress and depression reduces immune responsiveness, resulting in additional pathogenic infection and associated periodontal tissue destruction. It has also been shown that chronic stress and depression mediates an increased risk to and progression of periodontitis by changes in health-related behaviours, such as oral hygiene, smoking and diet.[47] Peruzzo et al. conducted a systematic review of the evidence on the influence of stress and psychological factors on periodontal disease. Of the 14 studies (seven case–control, six cross-sectional and one prospective clinical trial) meeting inclusion criteria for the review, the majority (57%) reported a positive relationship between stress/psychological factors and periodontal disease.[48] In the recent study, it has been shown that stress scores and salivary stress markers (chromogranin A, cortisol, alpha-amylase and beta-endorphin) were significantly associated with clinical parameters of periodontal disease in 100 adult patients with periodontitis.[49] After adjusting for stress variables, it has been demonstrated that salivary cortisol and beta-endorphin were significantly correlated with periodontal clinical parameters and tooth loss specifically in those patients who neglected to brush their teeth during stressful periods, thus suggestive of the fact that stress modifies the host defence and progression of periodontal infections in patients susceptible to periodontitis.[47] In a case–control study, Moss et al. found that smoking and elevated antibody titers to Tannerella forsythia at baseline were associated with more severe periodontal breakdown in patients scoring high on a depression inventory.[50] Susceptibility to periodontal breakdown in response to stressful life events appears to depend, in part, on the effectiveness of a person's coping behaviour. It is consistent with a cross-sectional study by Genco et al. that revealed that psychosocial measures of stress (financial strain) and distress, manifest as depression, were significant risk indicators of periodontal disease severity in adults, after adjusting for gender (male), smoking, diabetes mellitus, T. forsythia and Porphyromonas gingivalis.[51] In a cross-sectional study, Rosania et al. found that stress, depression and salivary cortisol scores correlated significantly with severity of periodontitis and the number of missing teeth, when controlling for age, family history and brushing frequency.[52] Patients with periodontitis were found often to have higher systemic levels of C-reactive protein, interleukin-6, interleukin-1 and tumour necrosis factor-alpha.[53] A combination of factors associated with alterations in behaviour and neuroimmunologic function are probably responsible for the inter-relationship between psychological stress and periodontal

disease.

CONCLUSION

There is a wide body of evidence that documents that psychological stress, job-related stress in particular has a negative influence on the immune system and health. These stressors being chronic in nature results in the down regulation of the both cellular and humoral immune systems resulting in an increase in the pathogenic microflora and a greater amount of periodontal tissue destruction. Chronic stress, when complicated with tobacco abuse can lead to more detrimental effects on periodontal tissues magnifying the severity of periodontal disease and diminishing the host immune mechanism. Moreover, these conditions acts as a risk factor for chronic diseases, including periodontitis, through changes in health related behaviours, such as oral hygiene and diet.

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