REVIEW ARTICLE

Deleterious Effects of Smoking on Periodontal Health: A Review

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ABSTRACT

Cigarette smoking is a preeminent prospect for the cause of jillions of diseases. The development and progression of periodontitis has gained extensive awareness, with remarkable progress over the past decade in various domains. This review covers studies to examine the possible relation between periodontitis and cigarette smoking. Smoking cessation is beneficial as the prevalence and severity of periodontitis in former smokers is lesser as compared to current smokers. Smoking affects reaction to periodontal therapy. Smoking constitutes a major dental public health problem.

KEYWORDS: Periodontal therapy, Periodontitis, Smoking

INTRODUCTION

Periodontitis affects the periodontium, i.e., tissues supporting and lining the radicular portion of teeth.[1] It is with gram-negative anaerobic bacteria which leads to irremediable impairment of periodontium.[2] Periodontitis results in a continuous release of bacterial and inflammatory cytokines into saliva and to a certain degree into blood.[3] These periodontal pathogens and inflammatory markers travel via saliva and blood from the affected tissues to distant sites thus affecting systemic health adversely.[4] It has been found in pregnant women that, periodontal therapy reduces the rate of pre-term low birth weight.[5]

The etiology of active periodontal disease considers three factors: A susceptible host, the presence of pathogenic species, and the absence of so called "beneficial bacteria".[6] Smoking is strongly implicated in the development of periodontal disease amongst a variety of other factors like diabetes, socio-economic status, behavior and stress. Greater levels of alveolar bone loss, probing pocket depth, tooth mobility and tooth loss have been demonstrated to be severe in smokers than in non-smokers.[7]

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SMOKING AND GINGIVITIS

The effects of smoking on periodontium are evident, but smokers show reduced signs of inflammation in response to dental plaque than non-smokers, the key diagnostic indices of gingival bleeding on probing and oedema. This reflects an amendment of the calibre of the blood vessels perfusing the gingival tissues which can be ascribed to the cotinine, a nicotine metabolic by-product, as it has a peripheral constrictive action on gingival vessels that reduces clinical signs of gingival bleeding, redness and oedema.[8,9] Chang et al. have demonstrated altered Cox-2 mRNA expression in gingival fibroblasts in response to nicotine.[10]

SMOKING & PERIODONTITIS

Gelskey (1999) used methodology of Sir Bradford Hill's criteria for causation, as a structure to examine association between cigarette smoking and periodontitis. He stated that smoking meets most of the criteria for causation proposed by Hill (1965). The parameters were consistency, strength of association, specificity, temporality, biological gradient, biological plausibility/coherence, analogy and experiment.[11]

Studies have suggested that smokers exhibit increased bleeding upon probing, higher calculus and plaque deposits, increased clinical attachment loss, gingival recession and tooth mobility independent of age, gender and systemic condition.[12] A report from Calsina et al appreciated a more remarkable effect in male patients and reported that the probability of having disease increased to 3.7 in those who had been smoking for 10 years or more.

Schenkein et al. reported on the clinical status of subjects with varying degrees of periodontal destruction. They reported 20% of subjects with localised aggressive periodontitis, 43% with generalised and 16% of healthy subjects were smokers. These patients had significantly more pronounced periodontitis, greater mean loss of attachment and teeth affected sites than non-smokers. Several authors have also reported high prevalence of smoking amongst patients with aggressive periodontitis.[13]

Krall et al concluded that men who smoke cigars or pipes were at higher risk of tooth loss. Cigar smokers were also at increased risk of tolerating alveolar bone loss. These rise in risk were similar to those observed in cigarette smokers.[14] In a Brazilian study it was concluded that group non-smokers have lower alveolar bone loss, especially in the incisal region, and confirmed that cigarette smoking affects maxilla more as compared to the mandible.[15]

SMOKING AND ORAL MICROFLORA

It has been suggested that periodontal microflora is modified by smoking which results in periodontitis. In vitro exposure of bacteria to cigarette smoking results in a marked decrease in the numbers of viable bacteria.[16] Zambon et al. in a study on 798 subjects, reported that smokers were at greater risk of infection by B. forsythia. They concluded that smokers were 2.3 times more likely to harbour periodontal pathogens than non-smokers.[17] Umeda et al. reported that current smokers harbour T. denticola in periodontal pockets, and there is presence of A. actinomycetemcomitans, P. gingivalis, P. intermedia, E. corrodens or F. nucleatum.[18]

Various in vitro studies, reported that bacteria are extensively affected by smoking and in smokers there is a decreased oxygen tension in periodontal pockets that favours anaerobic colonization. In contrast, clinical studies have shown minor contrasts between smokers and

nonsmokers with respect to periodontal microflora.[19]

SMOKING AND SYSTEMIC MANIFESTATIONS

Smoking of tobacco leads to systemic manifestations like diabetes, pulmonary destruction and renal pathologies. Literature suggests that smokers are at an increased risk for type 2 diabetes and exhibit aspects of the insulin resistance syndrome. Nicotine has a direct effect on the beta cells of the pancreas and has also been associated with larger upper body fat distribution, a marker of insulin resistance, raised plasma glucose concentration and overt diabetes.[20-22]

In a spirometric test Neri et al observed chronic obstructive pulmonary disease (COPD) to be affected five folds or greater in smokers than in non-smokers. Hyman and Ried analysed the relationship between smoking and its effect on the periodontal inflammation. The results suggested that the amount of tobacco smoke patient was exposed to was in association with these two diseases and the extent of pathological involvement.[23,24]

Mentioned systemic diseases have been observed to either exaggerate or cause periodontitis. Diabetics have been observed to have raised response by periodontopathic microorganism thus causing destruction of the periodontium. An association has been suggested to be through activated monocyte response in type 2 diabetes.[25] These chronic systemic diseases are of much clinical consideration when it comes to routine dental and medical practice.

End stage renal disease has been observed to cause increased gingival inflammation and plaque and calculus formation. Yoshikara et al carried out a study the impact of renal function and periodontal disease in Japanese elderly. They concluded that there was a significant association between clinical attachment loss and renal impairment which was independent of gender, oral hygiene practices and previous dental profile.[26,27]

SMOKING AND HOST IMMUNITY

A number of virulence factors are released by bacteria causing periodontal disorders resulting in activation of host response. The release of these virulence factors systemically causes tissue destruction.[28] Studies state that smokers have increased number of neutrophils, but they have decreased activity of neutrophils including chemotaxis, phagocytosis, adherence and capacity to produce cytokines. Evidence even outlined that smoking influences lymphocyte count and production of antibody. It increases the level of CD3+ and CD4+ cells in a dose dependent manner. Immunoglobulins particularly IgG2 have shown to be dwindled in smokers when compared to non-smokers.[29] Unstimulated neutrophils exposed to tobacco elevates the oxidative burst causing tissue destruction by direct toxic effect.[30]

Smoking has a deleterious effect on cytokines as well as it reduces the concentration of interleukin (IL)-1, IL-1β and IL-1ra in gingival crevicular fluid. There are lower amounts of IL-4 in GCF of patients suffering from early onset periodontitis and also in patients with healthy periodontium. Levels of IL-6 and IL-8 increase with smoking whereas the amounts of IL-10 in GCF has been observed to be low in smokers than in non-smokers.[31,32] Smoking and Periodontal Therapy Preber and Bergstrom reported that non-smokers respond better to non-surgical therapy than smokers.[33] Ah et al. and Kaldahl et al., reported less probing depth reduction and attachment gain in smokers who

had been treated by periodontal surgery. They confirmed the finding that smokers were poor candidates for successful periodontal care.[34] Kamma and Baehni did a five-year follow up of 25 young adults diagnosed with early-onset periodontitis who had been receiving regular periodontal maintenance care. They reported that smoking was found to have significant predictive value of future attachment loss in these patients.[35] Mc Guire and Nunn found that smokers have twice the risk of tooth loss undergoing maintenance periodontal care over a five-year period.[36]

Effect of smoking on implant survival appeared to be more pronounced in areas of loose trabecular bone. Type II diabetes mellitus may have a pronounced effect on implant survival rates which again as mentioned above is linked to both smoking and periodontal destruction. A history of treated periodontitis does not appear to adversely affect implant survival rates but it may have a negative influence on implant success rates, particularly over longer periods.[37]

Studies have been carried out to find the use of alternative therapies in smokers, for example, in one of the novel therapeutic approaches, there was enhanced connective tissue breakdown which was due to inhibition of metalloproteinase activity as demonstrated for tetracyclines.[38] Well-evaluated markers of collagen turnover, such as the pyridinoline cross-linked carboxyterminal telopeptide of type I collagen (ICTP), have been used to investigate turnover in bone breakdown and bone change. Administration of low-dose doxycycline leads to lowered ICTP in patients with periodontitis, with no effect on non-treated subjects.[39]

SMOKING CESSATION AND PERIODONTITIS

Investigation of Veterans Administration Dental Longitudinal Study (DLS) members showed the result that the rate of tooth loss among men who left smoking was about 50% lower than the rate of current smokers, but still it was still significantly higher than the rate among non-smokers.[40] In a 12-year follow-up study of 1031 Swedish women, rates of tooth loss were similar in non-smokers and former smokers who had abstained from smoking an average of 10 years before entering the study.[41] These findings were constant with the arrested progression of periodontal bone loss and attachment loss observed when individuals abstained smoking.[42]

The results from few studies suggest that the risk of tooth loss decreases after smoking cessation but it remains elevated with respect to non-smokers for at least 9-10 years. The reason is the loss of alveolar bone which is irreversible, so it is to be expected that the damage to the bone tissue by cigarettes is permanent. Cessation of smoking reduces the likelihood of disease and will become widespread and affect more number of teeth. As time elapses, these other risk factors become more important and the differences due to smoking history becomes uncertain. Finally, there are other lifestyle changes which may occur when an individual decides to quit smoking and may become more pronounced as the duration of cessation increases. Smokers who quit smoking appear to be more health conscious than those who continue to smoke, and they make regular physician visits and use health screening programs at higher rates compared to non-smokers.[43,44]

CONCLUSION

From the literature discussed, it can be concluded that smoking is the most important risk factor for periodontal diseases. It increases the risk of periodontitis irrespective of the

genotype. This risk is further aggravated in subjects bearing particular alleles of the polymorphically expressed genes studied. While the precise mechanisms whereby cigarette smoking can exert an effect on periodontal tissues are not completely understood, it is clear that it is still the most significant preventable risk factor for periodontitis. Its effects are related to the duration and number of cigarettes consumed. It is hoped that the evidence of the harmful effects of smoking presented in this review might serve to stimulate dentists to give informed advice to help their patients who smoke to stop.

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