

The Impact of Smoking on Periodontal Tissues: Review Article

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Abstract

Smoking have potential biological mechanisms that might effects the human's immune system, altering microflora of the mouth, periodontal tissues destruction including clinical attachment loss, mobility and loosening of teeth, oral malodor and nonsurgical and surgical periodontal treatments. Furthermore, in this article will review the impact of smoking on periodontal tissues give importance to the impact of smoking on periodontium, oral microflora of smokers, impact of smoking on host response, gingival diseases, periodontal diseases, frequencies smoking, response to treatment of periodontium, discontinuation of smoking and systemic manifestations of smokers.

Keywords: Smoking; Periodontal Tissues; Microflora

Introduction

Periodontal problems consider to be a major reason for the universal burden of oral disease. However, small amount of care has been given by oral health carer and public health educator to periodontal problems in a lot of regions [1]. In the last 10 years, researchers have started to give more care about smoking and their major impact on the severity and incidence of periodontal problems, subsequently started to consider smoking one of the main risk factors [2]. Furthermore, previous researches reported that life anticipation of individuals who smoke is reduced by 14 years relying on the period of time they smoke [3]. Smoking not only leads to halitosis and break down the human's immune system but it raises the risk of periodontal problems by 2 - 7 fold. The impact of smoking on the periodontium relies on consumption and the period of smoking in an individual [4]. Males show more predilection in periodontal diseases than females due to smoking. Cigarettes contains over than 4000 components that retard recovery through periodontal therapy which includes carbon monoxide, formaldehyde, arsenic and ammonia. By-products of cigarettes retard the mechanisms of forbidding the sprouting of bacteria in the mouth which make a convenient flora for bacteria in the oral cavity like *P. gingivalis*, *P. intermedia* and *A. actinomycetemcomitans* [5].

Oral microflora of smokers

Zambon reported in his study of 798 participants with various histories of smoking, that smokers show significantly greater levels of *B. forsythia*, and has higher chance of infection. Also, smokers showed 2.3 times more chance to harbor *B. forsythia* than who smokes in the past or never smoked [6]. Periodontal pockets of present smokers exhibit an increased chance of harboring *T. denticola* and that the presence of *A. actinomycetemcomitans*, *P. gingivalis*, *P. intermedia*, *E. corrodens* or *F. nucleatum*. Also, rise the chance of having a mean periodontal pocket depth of ≥ 3.5 mm in smokers [7]. Haffajee and colleges concluded in their study that the main variance between non-smokers and smokers was in the spread of colonies i.e. periodontal microorganism colonized a larger amount of species, rather than counts or portion. With excess colonization shown among smokers in their shallower periodontal pockets. Moreover, they reported that maxilla show more proportions of sites colonized by *B. forsythus* and *P. nigrescens* than mandible [8].

Impact of smoking on host response

Bacteria consider the primary etiology of periodontal diseases, however, patient's chance to be affected it depends by the host response. Borbour and colleagues provide strong proof that immune host responses effects by smoking, while BERGSTRÖM and Lie reported decreased inflammation [9-11]. Furthermore, Kinane and Radvar stated that smokers show reduced gingival crevicular fluid volumes as compared to nonsmokers, which suggest that smoking reduced gingival blood flow [12]. Mosely and Riebel reported impairs revascularization of bone and soft tissues due to smoking, which might be a major impact on healing of wounds, particularly in regenerative periodontal and implant therapies [13,14]. Smokers have significantly higher numbers of neutrophils in the peripheral circulation, which consider the primary line of defense against infection, but their function is impaired [15]. Smoking also altered another protective host mechanism which is antibody production, with a consistent finding that smoking decreases serum IgG concentration [16,17].

The relationship of tobacco and gingival diseases

Smoking and its clinical manifestations on periodontal tissues is evident, but contradictory, clinical signs of inflammation in response to dental plaque reported to be less in smokers than non-smokers, specifically gingival bleeding on probing and edema are the crucial diagnostic signs. Suggesting that this might reflects changing of the diameter of the blood vessels that perfuse the gingiva, which might be related to the nicotine and cotinine metabolic by-product, as it has a narrowing action on peripheral gingival vessels which reduces gingival clinical signs of redness, bleeding and edema [18,19]. Poor level of oral hygiene has been reported in smokers when compared to nonsmokers [20-22]. Effectiveness of tooth brushing in smokers reported to be lesser, and dental plaque showed to be significantly raised in calcium concentration than in non-smokers, suggesting an impairment of routine oral hygiene and favorable environment for calculus formation [23]. Regarding plaque, it has been reported to be significantly more in males than females where plaque adherence of smokers reported to be doubled the percentage of nonsmokers in both genders [24].

The relationship of tobacco and periodontal diseases

Periodontitis have been defined as an inflammation of supportive tissues of teeth caused by particular pathogens which initiate advanced resorption of alveolar bone and periodontal membrane, with increased of clinical attachment lost. Divided opinions regarding effect of smoking on inflammatory periodontal problems. Earlier reviews regarding periodontal problems epidemiology stated that smoking considered a possible causative factor. Conclusively demonstration of any related pathological alteration in the periodontium credited to smoking have been reported in few studies. Smokers reported to be at increased risk to have *P. gingivalis* in subgingival region, despite this was not reported to be statistically significant. Investigators In the same study reported that smokers were triple the times probably to have *A. actinomycetemcomitans* [25]. Machuca studied a community of youthful Spanish healthy male soldiers to see the correlation of smoking habits and periodontal problems. The report included increased attachment loss and probing depths in smokers, although raised in plaque and bleeding in nonsmokers [26]. Aggressive configuration of periodontitis diagnosed in youthful smokers with more affected teeth and raised mean of clinical attachment lost than non-smokers. Bone loss and significantly greater probing depths reported in smokers than non-smokers, regarding tooth mobility no difference was founded [27]. Bergstrom., *et al.* reported smokers have significantly increased probing depths, alveolar bone loss and increased tooth mobility. Moreover, regarding the most region to be affected, clinical investigators have reported substantial periodontal destruction to be in maxilla in smokers with different types of periodontitis [28]. In a Brazillian investigation it was noticed that alveolar bone resorption raised in smokers comparing to nonsmokers, confirming that cigarettes utilization resorb maxillary jaw more than mandible, specifically anterior proportion [29].

The relationship of frequencies smoking and intensity of periodontal diseases

One of the major risk-determining factors is daily number of cigarettes smoked, increasing it sixfold in the subgrouped who smoked over than thirty cigarettes daily and doubling the risk for smokers in the lowest consumption category [30,31]. Several papers have reported a relationship between intensity of periodontitis and the number of smoked cigarettes. A correlation has been described between

the daily consumption of cigarettes and the spread of moderate to severe periodontal disease [32-36]. Severity of attachment loss was reported to be increased by 0.5% for smoking 1 cigarette per day, while smoking up to 10 and 20 cigarettes a day increases attachment loss by 5% and 10%, respectively [34].

Impact of smoking cessation on periodontium

Little knowledge on the impact of smoking cessation related to the risk of teeth loss reported, but literature suggests linked between teeth loss and smokers. Veterans Administration evaluation for men who cease smoking showed an average of 50% of teeth loss lower than the average of smokers, but remains remarkably higher than nonsmokers [35]. However, changing in increasing length of cessation was not addressed in the analysis. In a 12-year prospective research of 1031 Swedish ladies, who had cessation of smoking within an average of 10 years prior to be involved in the research showed similarity in previous smokers and who never smoked [37]. Progress of periodontium loss is observed to be cease in individuals who quit smoking [10]. The periodontal status of former smokers shows levels between that of never smoked and current smokers, which suggests that irreversible changes happened to the periodontium due to smoking, but the deterioration does not continue after cessation [10,35]. Furthermore, encouraging to note that respond to periodontal therapy in former smokers similar to nonsmoker [38,39].

Periodontal treatment responses in smokers

Smokers had approximately 50 percent less recovery in clinical attachment gain and probing depth than nonsmokers in a six-year longitudinal study [40]. Numbered of papers have reported that 90 percent of refractory periodontitis showed in smokers [41-43]. Success of gingival grafting for root coverage reported less in smokers than nonsmokers in majority of studies [44-46]. De Bruyn and Collaert believes that cigarettes smoking is a relative contraindication to dental implant therapy. Early failures before loading in maxilla were higher in smokers 9% than non-smoker 2% [47]. Implants supporting mandibular fixed prostheses in a fifteen-year longitudinal study showed only 1% loss of implants, but more bone loss demonstrated in smokers than former or who never smoked [48]. Several studies reported that smoking inhibit outcomes of attachment gain and probing depth after nonsurgical or surgical treatments [39,49,50]. Differences numerically among nonsmokers and smokers related to probing depths is ≥ 5 mm, where individual who smokes show 0.4 mm to 0.6 mm less recovery in clinical attachment gain after nonsurgical treatment [39,51]. Regarding surgical debridement, smokers show up to 1 mm less recovery in clinical attachment gains in probing depths that were previously ≥ 7 mm [52].

Systemic manifestations in smokers

Smokers are exhibiting a number of ways regarding insulin resistance and they are at high risk for developing type 2 diabetes. Diabetic nephropathy, retinopathy, neuropathy, macrovascular problems, and peripheral vascular problems reported to be at increased risk to happened to smokers [53-55]. Analyzing the correlation of smoking and its impact on the relationship between chronic obstructive pulmonary disease and periodontal inflammation reported by Hyman. The results showed that smoking is a co-factor in the correlation of these both diseases and the range of pathogenesis action count on the number of tobacco consumption by the patient [56]. Smoking at initial onset of nephritis stated by Orth and colleagues to be a separated risk factor for accelerating established end stage renal problems [57]. Ritz recognize that smoking is the cause of damaging endothelial cells, restrict the fibrinolysis systems and oxygen regeneration, perhaps these factors are the reasons of the relationship of the two [58]. A substantial factor for preventing of having osteoporosis and good prognosis after therapy is to cease or decrease in smoking [59].

Conclusion

Smokers exhibit an increased chance of harboring different pathogens in the periodontal pockets and mean periodontal pocket depth of ≥ 3.5 mm. Maxilla show more proportions of colonization than mandible. Smoking effects immune host responses decreased inflammation and reduced gingival crevicular fluid volumes. Also, clinical attachment lost but no difference in tooth mobility. Smokers showed poor

level of oral hygiene and double the percentage of dental plaque in nonsmokers. Daily number of cigarettes smoked is a major risk-determining factor. Cessation of periodontium loss in individuals who quit smoking. Responding to periodontal therapy in former smokers and nonsmoker is similar. Systemic manifestations including pulmonary destruction, diabetes mellitus, renal problems and osteoporosis.

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