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Effect of cigarette smoking on periodontal health

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Abstract

Cigarette smoking has adverse effect on systemic health. Smoking is associated with cardiopulmonary disease, cancer, diabetes mellitus, insulin resistance and thyroid disorders. In this review, effect of cigarette smoking on oral microbiota, periodontal inflammation, periodontal disease progression and response to periodontal therapy is discussed.

Keywords: Cigarette smoking, Inflammation, Periodontal therapy.

INTRODUCTION

Cigarette smoke condensate (CSC), the particulate matter of cigarette smoke, is comprised of thousands of chemicals (e.g., nicotine, cadmium, phenol, anthracyclic hydrocarbons, nitrosamines, heavy metals and chemical carcinogens) [1]. Nicotine, one of the most important components of tobacco, has a plasma half-life of approximately 30 minutes [2]. Nicotine is quickly converted to its major metabolite, cotinine. Cotinine is used as a biochemical marker of tobacco use [3]. Plasma half-life of cotinine is longer than that of nicotine, ranging from 10 to 30 hours[4] and is detectable for several days (≤1 week) after the use of tobacco. Because of relatively constant cotinine levels for long periods of time, its measurement is used as a method for determining and monitoring smoking behaviour.

Cigarette smoking and periodontal disease

There is a strong positive correlation between cigarette smoking and the increased incidence of periodontal disease [5-8]. Prevalence of periodontitis is correlated to number of cigarettes consumed per day [5]. Smoking increase prevalence of periodontal pockets [9]. Level of periodontal destruction is proportional to nicotine levels [10] with more destruction reported with high yield cigarettes containing higher levels of nicotine, carbon monoxide and tar than low yield cigarettes [11]. CSC increase collagen degradation by altering the levels of membrane bound matrix metalloproteinases and tissue inhibitor of matrix metalloproteinases [12].

Prevalence of periodontal disease is also influenced by salivary cotinine concentrations [13]. Positive correlation is reported between serum cotinine levels and measures of periodontal destruction i.e. clinical attachment loss and crestal bone loss [8]. Environment tobacco smoke (ETS) also known as second hand smoke (SHS) increases prevalence of periodontitis [14].

Smoking not only increase prevalence but also aggravates severity of periodontitis [7]. Smokers also have increased periodontal destruction [15].

Cigarette smoking and periodontal microbiota

Some studies reported difference in plaque scores between smokers and non smokers [16] whereas others demonstrated no appreciable difference [6,17]. Smoking affects subgingival microbiota [18]. Presence of pathogenic bacteria in sulcus is influenced by number and duration of smoking [19]. However, one study reported no difference in subgingival microbiota among smokers and non smokers [9].

As compared to non smokers, smokers have higher levels of calculus [20]. Increased calculus is also reported in teenagers involved in smoking [21].

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Cigarette smoking and vascular response

Smokers have higher percentage of smaller blood vessels and a lower percentage of larger vessels but vascular density is similar to non-smokers [22]. In other words, smoking does not influence vascular surface density and number of vessels [23]. Smokers have less oxygen saturation in gingiva and less tendency to increase oxygen saturation in presence of inflammation [24]. Smokers have lesser gingival blood flow as compared to non smokers [25]. Pocket oxygen tension is also lower in smokers [26].

Cigarette smoking and periodontal inflammatory response

In smokers, sites with increased probing depth have decreased tendency for bleeding on probing (BOP) representing decreased inflammatory response in smokers [27]. Smoking has dose dependent effect on reduction of BOP [28]. However, other study reported no difference in gingival inflammation [15].

Cigarette smoking and immune response

Immune function is altered in smokers with decreased levels of serum immunoglobulin (Ig) G and IgA in smokers compared to non-smokers representing that depressed Ig production [29] especially IgG2 [30] may be responsible for increased severity of periodontal disease in smokers [31]. Smoking also alters humoral immunity in response to *Porphyromonas gingivalis* [31].

Cigarette smoking and periodontal treatment

No difference in disease progression is noted in highly motivated smokers and non smokers on maintenance therapy [32]. Smokers have less improvement in response to periodontal therapy [33]. In smokers, scaling and root planing (SRP) results in less improvement in clinical parameters as that in non smokers [34]. Smokers also have decreased benefit from SRP in combination to systemic antibiotics as compared to non smokers [35].

Former cigarette smoking and periodontal disease

Risk of periodontal disease declines proportionally to number of years since quitting [5]. Former smokers had decreased risk for periodontal disease as compared to current smokers [5,36] depicting importance of smoking cessation in maintaining periodontal health [36].

Other effects of cigarette smoking on oral mucosa

Due to presence of nicotine and benzpyrene, higher pigmentation is reported in current smokers[37] and passive smokers [38]. Tobacco smoking is an important risk factor for precancerous lesions like leukoplakia and oral cancers [39]. Anti apoptotic effect[40] induced by smoking may be responsible for such lesions. Volatile fraction such as acrolien and acetaldehyde disrupt cytoskeleton of human gingival fibroblasts and impair their adhesion [41]. Increased amount of metallothionein, a free radical scavenger, is found in gingival biopsies of smokers in response to increased levels of free radicals [42].

CONCLUSION

Smoking has negative effect on plaque microbiota, alters immune system, is cytotoxic to fibroblasts and also decrease response to periodontal therapy. Due to these negative effects, American Academy of Periodontology's parameter of care promote tobacco cessation counselling by dentists to promote oral as well as general health.

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