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The Impacts of Smoking on Periodontal Health

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ABSTRACT

The present study reviewed the literature about the influences of smoking on health periodontal tissue. Smoking is significantly associated with chronic periodontitis and the impact seems to be dose independent. The prevalence of chronic periodontitis depends on smoking status and ranges from 16% in moderate smokers to 38% in heavy smokers. Smoking increases the risk of periodontitis by four folds.

Keywords: CAL: Clinical Attachment Level; PD: Probing Depth; BoP: Bleeding on Probing

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Introduction

The periodontium involves different tissues such as gingiva, periodontal ligament, cementum, and alveolar bone. Gingivitis is the inflammation of gingiva that is caused by a bacterial dental plaque. Periodontal disease is developed from advanced gingivitis that causes the irreversible loss of the periodontal ligament and supporting bone. Periodontal diseases lead to tooth loss and they are associated with some conditions and systemic diseases such as diabetes mellitus, and cardiovascular diseases (Calsina et al. [1]; Palmer et al. [2]). Periodontitis cases can be determined based on some clinical parameters such as clinical attachment level (CAL), probing depth (PD), and bleeding on probing (BoP) (Bunaes et al. [3]). There are variations in the prevalence of periodontitis in different population groups (Shchipkova et al. [4]). At global level, about of 11% of population have severe periodontitis (Griffiths et al. [5-8]).

There are several risk factors participating to increased burden of periodontitis including smoking, educational and socioeconomic status, diabetes mellitus, health care availability, and oral hygiene habits (Tymkiw et al. [9]; Bunaes et al. [3]). Smoking is likely to participate in elevated prevalence of periodontitis in a dose dependent matter Lutfioglu et al. [10]. However, some risk factors that are known to increase the susceptibility to periodontal disease such as smoking, stress, and obesity are considered modifiable factors Ainamo et al. [11]. Cigarette smoking is a considerable reason for chronic periodontitis O Leary et al. [12]. There are about 5000 different molecules inhaled through the oral and nasal cavity before the vaporized gases absorb in the lungs Borgerding et al. [13]. Smoking is involved in chronic periodontitis in various aspects such as increased tooth loss, aggravated bone and attachment loss, and deeper periodontal pockets compared with non-smokers (Tomar et al. [14-18]).

It has been reported that about 50% of patients with periodontitis are smokers Tomar et al. [19]. Furthermore, smokers are more likely to develop chronic periodontitis four times compared with non- smokers, and this is proportional to dose dependent Heitz Mayfield [19]. Based on the smoking status, smoking participation in de eloping chronic periodontitis has been estimated to range from 16% for moderate smoking to 38% for heavy smoking Susin et al. [15].

The effect of Smoking on Gingival Health Status

In their study, Cicek et al. [20] put focus on gingival pigmentation and defined Pigmentation as a kind of discoloration of the oral mucosa and gingiva that interfere with esthetics of which melanin pigmentation is the most common form. They identified a set of variables to cause melanin pigmentation in the oral mucosa such as physiological or racial pigmentation, smoker's melanosis, pigmented nevus, melanotic macula, Addison disease, Peuutz-Jeghers syndrome, HIV infection and drugs such as minocycline and anti- malarial drugs. The prevalence of oral pigmentation has been shown to

be the highest among Indians (89%) and the lowest among the Europeans (15%) Hedin et al. [21]. It is worth to mention that melanin pigmentation of the oral mucosa has negative effects on esthetics. Due to the fact that melanin pigmentation is involved as a clinical manifestation of systemic diseases and drug usage, it is crucial to involve melanin pigmentation in the differential diagnosis of these conditions Sreeja et al. [22]. In the study of Hanioka et al. [23], the authors stresses on the appearance melanin pigmentation in smokers as a special phenomenon which is called smoker's melanosis.

The authors suggested that melanin pigmentation could be induced by the stimulation of melanocytes by stimuli present in tobacco smoke such as nicotine and benzopyrene. Furthermore, a cause and effect relationship between cigarette smoke and melanin pigmentation has been suggested because of the observation that melanin pigmentation decreases as the number of years the person has quit smoking increases. Various studies across the literature have indicated that the effects of cigarette smoke reached other people who are present in the same environment. Researchers have also demonstrated a significant relationship between smoking and some side effects such as appearance of childhood asthma Fernando et al. [24], dental caries Aligne et al. [25], spontaneous abortion George et al. [26], periodontal disease Arbes et al. [27], and children's behavior problems and childhood cancers Boyaci et al. [28]. It has been noted that there is a dilemma in which children who are exposed to cigarette smoke usually do not complain and when they express their complaints the parents do not pay attention to them or reprimand.

So, children suffer from environmental tobacco smoke and the house is the most important site of this exposure Boyaci et al. [28,29] conducted a study to explore the association between gingival pigmentation and exposure to ETS. The reason beyond conducting such a study is that such an association with active smoking has already been established while it is still remaining to be established with ETS. A case-control study was carried out involving 59 nonsmoking children. Gingiva was examined by two calibrated examiners independently who observed labial gingiva via oral photographs. Study findings have indicated that about 61% of children had at least 1 smoking parent. About 71-78% of children showed gingival pigmentation. It has also been shown that there was a higher percentage of smoking parents among children with gingival pigmentation (70-71%) than in those who lacked pigmentation (35%). Taken together, researchers concluded that excessive pigmentation in the gingiva of children is associated with exposure to ETS. Furthermore, researchers put emphasis on the observation that visible pigmentation effect in gingiva of children could be useful in terms of parental education.

Several studies in literature reported the presence of brownish or black pigmentation in human gingiva in several countries. Furthermore, there is a variation in the prevalence of gingival pigmentation which may be attributed to race and country (Sarswathi et al. [30,31]). Pigmentation in human gingiva has been shown to originate from melanin granules in melanocytes Hedin et al. [32]. Melanocytes have been identified as dendritic cells at the basal layer of gingival epithelium Halaban et al. [33]. Several studies revealed that melanin pigmentation in gingiva to be correlated with active smoking so that smokers presented a greater tendency toward pigmentation than did nonsmokers. Furthermore, a dose-response relationship was detected Hanioka et al. [23]; Hedin [34-36]. It has also been reported that prevalence of pigmentation was decreased in relation to the number of years after smoking cessation. Taken together, previous findings pointed to a causal association between tobacco smoke and melanin pigmentation in gingiva. Gingival pigmentation was shown mainly in the labial area of anterior teeth (Hanioka et al. [23]; Hedin [34]; Sarswathi et al. [36]).

According to Hedin et al. [37], it is not frequently to see excessive pigmentation in palatal mucosa as a result of tobacco smoke except in cases of reverse smoking. It has also been shown that there is an increased prevalence of gingival pigmentation among smokers Hanioka et al. [38]. Hajifattahi et al. [39] conducted a study to evaluate the role of parental smoking on pigmentation of their children's oral mucosa. The study involved 400 healthy children, within the age 10 to 11 years, who did not use any drugs. Participants were categorized into two groups: passive and control groups. The passive smoker group included 200 children and defined as having at least one smoker member in their family. The control group also included 200 children who did not have a smoker in their family. Furthermore, two groups were matched in the point of view of skin color. Oral pigmentation in the two groups was examined and recorded. Study findings showed that the relative risk of oral pigmentation for children who were exposed to passive smoking was 1.23. John et al. [40] conducted a study in the light of the fact that ETS exposure is a distinguished health hazard for children.

Furthermore, poor urban children may be at increased risk of exposure from non-parental sources of tobacco smoke. The study had two main objectives: to identify the sources of ETS exposure for children and to explore caregivers' perceptions of the pediatrician's role and action in addressing ETS exposure. Study findings indicated that 14% of caregivers were smokers; about 49% reported caring for at least 1 smoke-exposed child. It was interesting to find smoke exposure to occur mainly in the home; it was permitted to smoking in the home of 75% of the children who live with a smoker. About 34% of the caregivers who reported no smoking in the home reported having a child who spends time in homes where other people smoke. Results showed that the perception of all caregivers that tobacco smoke is harmful. The findings of the previous study are in line with other studies in which the burden of ETS exposure is of prime concern among pediatricians Gergen et al. [41]. Furthermore, several studies have considered advising parents

to quit smoking to be a recommended part of pediatric practice (American Academy of Pediatrics [42]).

Taken together, reducing children's exposure to ETS is realized as a significant objective for the practicing pediatrician. It has been warned that pediatric subpopulations at increased risk of smoke exposure are ignored. It is expected to find highly smoke-exposed children in locations with high parental smoking prevalence, greater numbers of cigarettes smoked in the same room as the child, and higher degrees of crowding Irvine et al. [43]. Furthermore, the amount of time children spend indoors in a given area has been described to create greater potential for ETS exposure. According to previous context, it has been indicated that ETS may be considered an urban toxicant as well as an individual residential exposure Weaver et al. [44]. It has been recommended to restrict smoking in the home which, in turn, can have a strong influence on the prevalence of pediatric tobacco smoke exposure Eriksen et al. [45]. Some challenges have been reported by various pediatricians in working with children exposed to ETS from non-parental or even nonresidential sources. It has also been reported to encounter such challenges in working with children living in extended families or spending significant time in the homes of family friends and relatives who smoke Eriksen et al. [45].

The Influence of Smoking on Gingival Microvasculature

Vijaya et al. [46] conducted a study in the light of the fact that smoking has been considered as a major risk factor for periodontal disease. Previous studies showed the presence of decreased bleeding on probing and reduced inflammatory response among smokers. These changes have been attributed to the alterations in gingival microvasculature and gingival epithelium. In case of having no smoking, inflammation induces vascular and epithelial changes in the gingiva. The researchers aimed to evaluate the vascular and epithelial changes in gingiva of smokers and non-smokers with chronic periodontitis. Study findings showed that the mean blood vessel density for smokers was 12.388±6.472 and for non- smokers was 14.800±4.91. The mean lumen area of the vessels among smokers and non- smokers was 19.290±8.775 μm2 and 20.044±7.896 μm2, respectively. The mean epithelial thickness among smokers was 150.551±32.994 μ and 134.941±30.63 μ for non- smokers. Taken together researchers concluded that smokers have less vascular density and reduced lumen area and increased epithelial thickness than non- smokers, and these changes were not statistically significant.

It has been show by epidemiological studies that smoking has a marked influence on prevalence, extent, and severity of periodontitis Holm et al. [47]. Other studies have shown that some clinical parameters such as probing pocket depth, clinical attachment loss to be increased in smokers compared to non- smokers. It worth to note that there is a decrease in bleeding on probing and reduced inflammatory response for plaque accumulation among smokers in comparison to non-smokers (Tonetti [48]; Hidalgo [49]). These changes have been utilized through various studies to influence microvasculature of the gingival connective tissue and increased thickness of epithelium among smokers to mask the signs of inflammation (Bergstrom et al. [50]; Scardina et al. [51]; Villa et al. [52]). It has been shown that smokers show reduced vascular density and reduction in the lumen area of the gingival vessels Mirbood et al. [53]. It has been indicated that nicotine can increase rate of proliferation of gingival epithelium, thus increasing epithelial thickness among smokers Gultekin et al. [54]. In his study, Bonkader et al. [55] indicated that inflammation brings about vascular changes like increased density and dilatation as well as epithelial proliferation independent of smoking status [56,57].

Conclusion

The present study showed that smoking has large impact on periodontal and gingival health. Chronic periodontitis is significantly associated with smoking, and this is a dose dependent manner.

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