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THE EFFECTS OF SMOKING ON THE PERIODONTAL CONDITION OF YOUNG ADULT SAUDI POPULATION

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ABSTRACT

Tobacco smoking has a substantial influence on periodontal health and disease. It is associated with an increased disease rate in terms of periodontal attachment loss, periodontal pocket formation and periodontal bone loss. In addition, it exerts masking effect on gingival symptoms of inflammation. The purpose of this study is to examine the clinical influence of tobacco smoking on the periodontal condition of the young adult Saudi patients.

INTRODUCTION

The main etiologic factor, which can cause initiation and progression of periodontal disease, is the infection produced by dental plaque. Although gingivitis and periodontitis are elicited by bacteria, smoking has been shown to represent a strong risk marker and probably true risk factor for the development and progression of periodontal disease (Tonetti, 1998). When it was first discovered that smoking played a role in periodontal disease, it was thought that there was a link with plaque (Arno et al,1958), but Bergström et al,(1991), concluded that smoking is a risk factor even for individuals with good oral hygiene and the combined effect of smoking and plaque infection is likely to be more destructive than either factor alone.

A variety of oral conditions have been associated with smoking. A positive association between cigarette smoking and the prevalence and severity of periodontitis and the occurrence of necrotizing ulcerative periodontitis was first reported more than 4 decades ago (Pindborg, 1947). Smoking may also be an important factor in refractory periodontal disease (Magnusson and Walker, 1996), as most individuals with refractory periodontitis are heavy smokers (MacFarlane et al, 1992). Smoking also increases the risk for osteoporosis (Krall and Dawson-Hughes, 1991), severe periodontitis in HIV-infected individuals (Tomar et al, 1995), progressive periodontal attachment loss and tooth loss in young early-onset periodontitis patients (Schenkein et al, 1995), periodontal disease in diabetics (Karjalaimen et al,1994), root caries (Ravald et al, 1993), oral leukoplakia and oral cancer (Christen, 1970).

Prevalence and severity of periodontitis seem to be greater in smokers than non-smokers (Feldman et al, 1983). In particular, tobacco smoking is associated with an increased disease rate in terms of periodontal bone loss (Bergström et al, 1991), periodontal attachment loss, deep pocket formation (Haber and Kent, 1992), tooth loss (Holm, 1994)

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and is also associated with a greater expression of molar furcation involvement in periodontitis affected subjects (Mullaly and Linden 1996). The postulated mechanisms of increased periodontal disease progression in smokers include alterations in the host response, such as reductions in serum immunoglobulin (lg) G2 levels to *Provetella intermedia* and *Fusobacterium nucleatum*, and impairment of various neutrophil functions, such as phagocytosis and aerobic antimicrobial functions, alterations in gingival blood flow and reductions in the gingival crevicular fluid flow (Al-Ghamdi and Anil 1982).

The severity of smoking-associated periodontal destruction appears to be related to the duration of use; however, recent studies have identified an increased prevalence of periodontitis in young smokers. This was first high-lighted by (Haber et al, 1993), who reported that in the 19- to 30-year olds, they investigated, current smokers were almost four times more likely to have periodontitis and to have more affected sites and a higher proportion of deeper sites than non smokers.

This finding was supported by Linden & Mullally (1994), who reported that smokers had substantially more subgingival calculus, gingival bleeding, periodontal pocketing and attachment loss than non-smokers in regular dental attendees aged between 20 and 33 years in Northern Ireland. A recent study of young (average age 19 years) male army recruits in Spain found that moderate to heavy smoking was associated with accelerated loss of periodontal attachment (Machuca et al, 2000). Schenkein et al, (1995) reported that smokers with generalized aggressive (formerly early onset) periodontitis had more affected teeth and greater mean loss of periodontal attachment than non-smokers. This was confirmed by Mullally et al, (1999), who reported that, in addition, those with generalized aggressive (early onset) periodontitis were significantly more likely to be smokers than nonsmokers. This strong association between smoking and periodontal destruction in the young is of concern because, despite a general reduction in the prevalence of smoking, there are recent reports of increasing levels of smoking in young individuals in developed countries (Wechsler et al, 1998). Although more limited, there is some evidence that smoking is also an increasing problem in the young in developing countries (Warren et al, 2000). The aim of this study is to investigate the effects of smoking on the periodontal condition of a sample of young adults in the Saudi population.

MATERIALS AND METHODS

Study population: One hundred-twenty (120) patients at the outpatient clinic, Faculty of Dentistry, King Abdulaziz University (60 smokers and 60 non-smokers between the age of 18 to 32 years) participated in the present study. Selection criteria for patient inclusion were: (1) systematically healthy conditions, and subjects had no complicating medical condition or pregnancy, nor used any pharmacological agents known to affect the periodontium,;(2) subjects had no history of comprehensive periodontal treatment nor they were under orthodontic treatment; (3) smokers must have smoked at least 10 cigarettes per day at the time of initial examination for at least the previous 2 years; and (4) non-smokers had no previous smoking or tobacco use experience.

Periodontal examination: The periodontal assessment for smokers and non-smokers included measurement of attachment loss, probing depth, gingival bleeding index (Löe and Silness, 1963), plaque index (Silness and Löe, 1964), calculus index (Green and Vermillion, 1964) furcation involvement (Hoag and Pawlak, 1990), tooth mobility (Hoag and Pawlak, 1990) and number of extracted teeth. The measurements were made in millimeters and were rounded to nearest millimeter by using the UNC 15 periodontal probe. Attachment

loss was defined as the distance from the cemento-enamel junction to the bottom of pocket. Probing depth was measured from the gingival margin to the bottom of the pocket. Gingival recession was defined as the distance from the cemento-enamel junction to the gingival margin.

Radiographic examination: Panoramic and standardized bitwings and full set of periapical radiographs were taken for the smoker and non-smoker subjects to evaluate the effect of cigarette smoking on the bone level, and were used to evaluate the percentage of bone loss and number of missing teeth. A patient's bone loss was rated as either mild (0-20%), moderate (20-40%) or severe (greater than 40%). In measuring bone loss, an estimation of the distance from the alveolar crest to the cemento-enamel junction (CEJ) was determined for the entire dentition, with greater emphasis being placed on the molar and premolar teeth. Any patient with greater than 50% bone loss involving four or more premolar or molar teeth was automatically placed in the severe bone loss category.

Statistical Analysis: The results of the present study were analyzed using a 2-sample t-test to compare the parameters of smokers versus non-smokers. The parameters compared were; clinical parameters including plaque index, calculus index, gingival index, clinical attachment loss, pocket depth amount of gingival recession furcation involvement, tooth mobility and the number of extracted teeth**.**

RESULTS

The results of the present study showed that the current cigarette smokers had the worst periodontal status when compared to non-smokers. There was a significantly higher prevalence of mild/moderate periodontitis among current cigarette smokers than in non-smokers group. Also current smokers had higher percentage of subjects with significant attachment loss, gingival recession, and had higher numbers of missing teeth (Fig.2-A&B). The percentage of subjects with gingival bleeding tissue and all the signs of gingival inflammation were considerably lesser in current cigarette smokers than the non-smokers group (Fig.2-A&B). There were also a tendency for higher prevalence and extent of supra and subgingival calculus and plaque formation in the current smokers group (Fig.2A&B). Also, the clinical examination showed that the degree and the incidence of probing depth, furcation involvement and tooth mobility were significantly greater in smokers than in non-smokers. In addition, the radiographic examination showed mild to moderate bone loss in the smokers group, regardless of the amount of local factor or level of inflammation (Fig.1-A&B).

Table 1 shows the clinical values of: plaque index, calculus index, gingival index, clinical attachment level, pocket depth, gingival recession, furcation involvement, tooth mobility, number of tooth loss, and % of bone loss for both smoker and non-smoker groups. Plaque index was significantly greater in smoker group compared to non-smokers (3.3±0.4 versus 2.9±0.5). Calculus index behaved in similar way to plaque index and was significantly greater in smokers than in non-smokers (2.8±0.14 versus 0.86±0.1). In contrast to plaque and calculus indices, gingival index is significantly lesser in smoker group than non-smoker group (1.7±0.5 versus 2.2±0.6). Table 1 shows that the clinical attachment loss is significantly greater in the smokers group than non-smokers (3.2±0.9 versus 0.7±1.04). Table 1 also shows that the probing depth increases significantly in smokers group in comparison with non-smokers group, (2.2±1.2 versus 0.3±0.7). Table 1 shows that the values of gingival recession, furcation involvement, tooth mobility and numbers of missing teeth are significantly greater in smoker group than those values of non-smoker group. The mean value of gingival recession was (1.02±0.8)

of smokers versus (0.32±0.5) of non-smokers, furcation involvement was (1.0±0.3) of smokers versus (0.5±0.2) of non-smokers, tooth mobility was (1.5±0.8) versus (0.6±0.2) of non-smokers, the number of missing teeth was (3.02±1.4) for smokers versus (0.5±0.08) for non-smokers and % of alveolar bone loss is (27.8±6.7) of smokers versus (4.0 ±6.9)

TABLE (1) Mean and standard deviation of clinical and radiographic parameters of smokers and non-smokers.

Parameter	Smokers	Non-Smokers
Plaque index	3.3±0.4*	2.9±0.5
Calculus index	2.8±0.14*	0.86±0.1
Gingival index	1.7±0.5*	2.2±0.6
Clinical attachment level (mm)	3.2±0.9*	0±.04
Probing depth (mm)	2.2±1.2*	0.3±0.7
Gingival recession (mm)	1.02±0.8*	0.32±0.5
Furcation involvement	1.0±0.3*	0.5±0.2
Mobility	1.5±0.8*	0.6±0.2
Number of missing teeth	3.02±1.4*	0.5±0.08
% of alveolar bone loss	27.8±6.7* %	4.0±6.9*%

**Statistically significant difference at p<0.05.* ***Statistically non-significant difference at p>0.05*

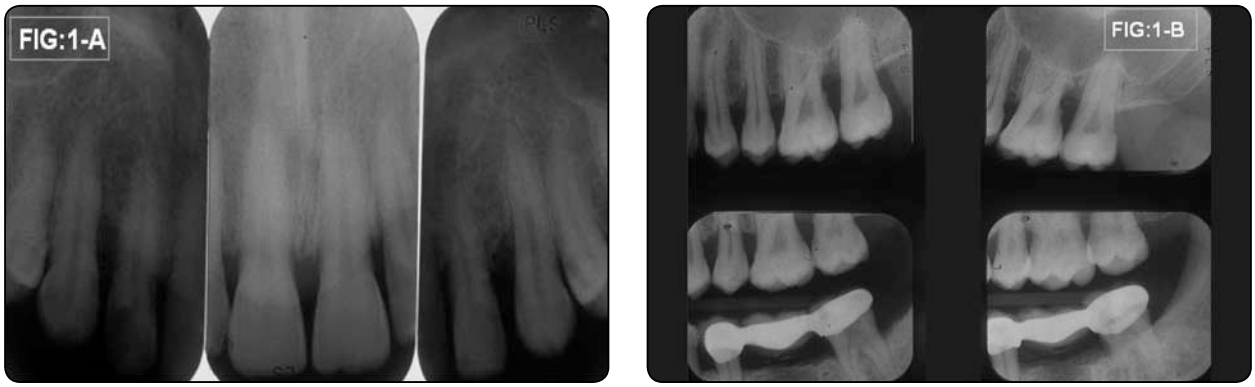

Fig. (1) Periapical and bitwing radiographic x-rays (A&B) for a 19-year-old smoker patient shows moderate to severe alveolar bone loss**.**

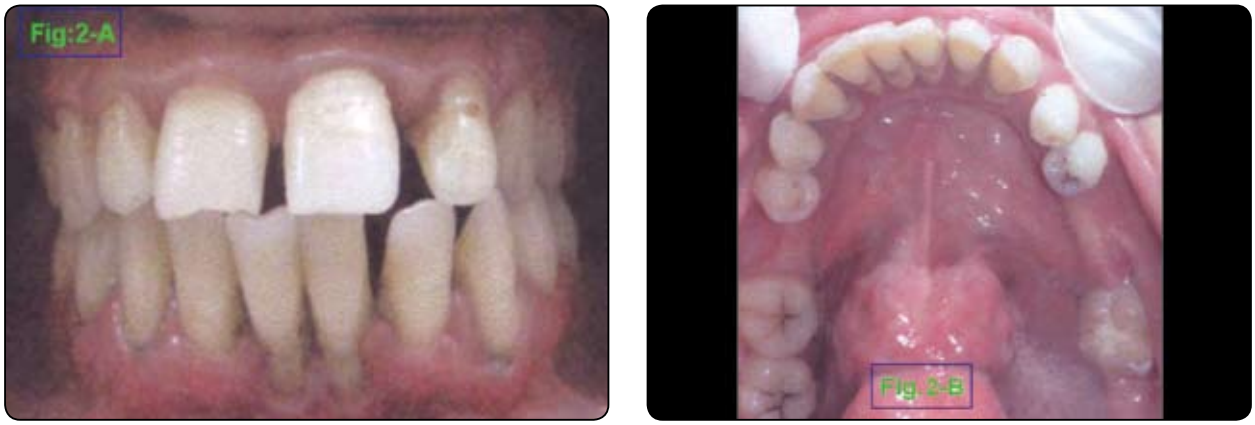

Fig. (2-A&B) Clinical views of a 18-year-old patient with chronic periodontitis associated with smoking. The views show a reduction in all signs of gingival inflammation (redness, edema and bleeding), multiple gingival recession, calculus and plaque formation, root caries and multiple missing teeth.

DISCUSSION

Tobacco smoke contains many cytotoxic substances, such as nicotine, which can penetrate the soft tissue of the oral cavity, adhere to the tooth surface or enter to the blood stream. The oral dose of 60mg of nicotine is lethal to most adult humans. Although the harmful effect of smoking on overall general health is well established, people continue to smoke. Also, the association between cigarette smoking and periodontal diseases represents a significant oral health problem. Most of the previous studies on tobacco smoking and periodontal diseases focused on adult populations of smokers, mostly with severe forms of periodontal diseases in whom smoking of long duration, age, general health problems, and other risk factors may coexist. The impact of cigarette smoking on the overall population has gathered some attention in the last decade, and some studies have evaluated the relationship between smoking and periodontal tissues in young adults. The association between smoking and periodontal bone loss was reported by Mullally et al,(1999) in young subjects diagnosed with aggressive periodontitis. Recently, a relatively high prevalence of aggressive periodontitis associated with smoking, among others risk factors, was found in young urban population in southern Brazil by Susin and Albandar (2005). Tooth loss was found to be associated with smoking in young adults and this may be due to the possible damage to periodontal tissues that have been exposed to tobacco for a short time in young smokers (Susin et al, 2006). Some cross-sectional studies evaluated the relationship between smoking and periodontal clinical parameters in young individuals with minimal or no periodontal destruction. A case-control study reported that smoking was a major factor associated with periodontal destruction in a group of young Jordanian adults (Al-Wahadni & Linden 2003). This is of major concern because the prevalence of smoking in young adults and adolescents, especially in young women, is not decreasing in some developing countries such as Argentina, Chile, and Brazil (Ministry of health of the nation 2007).

Potential molecular and cellular mechanisms in the pathogensis of smoking- associated periodontal diseases has been reported and these include, immunosuppression, exaggerated inflammatory cell responses and impaired stromal cell functions of oral tissues. Tobacco smoking can exert both local and systemic effects. In support of the concept that smoking exerts local affects, study of Feldman et al,(1983) showed that smokers with periodontal

disease had less clinical inflammation and gingival bleeding when compared with non-smokers. This may be explained by the fact that one of numerous tobacco smokes by-products, nicotine, exerts local vasoconstriction, reducing blood flow, edema and acts to inhibit what are normally early signs of periodontal problems by decreasing gingival inflammation, redness and bleeding (Chen et al, 2001).

Alveolar bone is one of the tissues that is most affected by the progression of periodontal disease. Bergstrom et al.(2000), investigated the long-term (10 years) influence of smoking on periodontal bone height and demonstrated a reduction in bone height that was 2.7 times greater in adult smokers. The mechanism of alveolar bone damage produced by smoking is related to the components of tobacco and nicotine metabolites which may act directly as local irritants on the gingival and alveolar bone or systemically because these components are absorbed in the lung, which affects the cellular host defense or bone turnover. Nicotine can suppress the proliferation of cultured osteoblasts while stimulating osteoblast alkaline phosphatase activity (Rosa et al, 2008). Recently, some in vitro studies provided other possible intimate mechanisms by which smoking may affect bone metabolism. (Rosa et al, 2008) reported that nicotine increased the secretion of interleukin-6 and tumor necrosis factor-alpha in osteoblasts and also nicotin increased the production of tissue-type plasminogen activator, prostaglandin E_2 and matrix metalloproteinase, thereby tipping the balance between bone matrix formation and resorption toward the latter process, as reported by Katono et al, (2006).

Receptor activator of nuclear factor-kappa ß ligand (RANKL) and osteoprotegerin (OPG) are members of the tumor necrosis factor super family. RANKL promotes osteoclastic differentiation and activates bone resorption. In contrast, OPG inhibits osteoclasto-genesis and suppresses bone resorption by inhibition of RANKL. Another potential mechanism of bone loss in smokers may be the suppression of OPG production and a change in the RANKL/OPG ratio, as was reported recently in clinical trials conducted by Lappin et al, (2007) who analyzed serum concentrations, and Cesar-Neto et al, (2007) who studied gingival biopsies.

Although bacteria are the primary etiologic factor in periodontal disease, the patient's host response is a determinant of disease susceptibility. Smokers appear to have depressed numbers of helper lymphocytes, which are important to B cell function and antibody production (Ginns et al. 1982). This was manifested by decreased levels of salivary antibodies (IgA) and serum IgG (Bennet and Read, 1982). Serum IgG antibodies to *Provetella intermedia* and *Fusobacterium nucleatum* also have been reported to be reduced in smokers (Haber, 1994). In addition nicotine has been shown to affect monocyte functions (Pabst et al. 1995), and impaired chemotaxis and phagocytosis of both oral and peripheral neutrophils (Kenney et al. 1977). Smoking has been shown to impair the oxidative burst of neutrophils and also nicotine can inhibit neutrophil production of superoxide anion and hydrogen peroxide, which are important to antimicrobial function (Pabst et al. 1995). Bacterial colonization and bacterial shifts may be associated with the inhibitions of neutrophil and monocyte antimicrobial activities (Pabst et al. 1995).

Eggert et al, (2001) proposed several potential mechanisms by which smoking could influence the host control of bacteria. These included the effects of carbon monoxide enhancing growth of bacteria, which in turn provide growth factors for anaerobes, and damaging cells involved in the protection of the periodontal environment such as neutrophils, which could be affected by the formation of advanced glycation endproducts (AGEs) by smoking. AGEs are the final end product of non-enzymatic glycation and oxidation of proteins found in plasma and tissues. AGEs

are biologically active and may initiate a range of cellular responses including stimulation of monocyte chemotaxis, osteoclast-induced bone resorption, proliferation of vascular endothelial and smooth muscle cells, aggregation of platelets, and stimulation of secretion of inflammatory cytokines, collagenase and several growth factors (Dawany &Miller 1998). AGEs can be either external or internal in origin. Internal sources include certain systemic conditions such as diabetes, Alzheimer's disease, and uremia (Yonekura et al.2005). External AGEs are produced by the combustion of nicotine in cigarette smoke. Receptoe of advancd glycation end product (RAGE) are cell surface receptors that are capable of producing a pro-inflammatory response. RAGE has been shown to be expressed by a variety of cell types, including endothelial and smooth muscle cells, lymphocytes, monocytes, and neurons. Furthermore, Hofmann et al,(1999), showed that, once activated by AGE, RAGE can trigger cellular activation with generation of key pro-inflammatory mediators. The binding of AGE to RAGE stimulates expression of RAGE itself and generates oxidative stress, synthesis and secretion of pro-inflammatory cytokines, and chemotaxis. Nornicotine, a nicotine metabolite found in high concentrations in the plasma of smokers, was shown to significantly induce the formation of AGEs. AGEs can induce production of cytokines, such as interleukin-1 (IL-1), IL-6, and tumor necrosis factor-alpha (TNF-ά), and can increase oxidative stress (Dickerson & Janda 2002). These pro-inflammatory responses are mediated by RAGE, which is also upregulated by its ligand.

Epithelial cells line the gingival crevice and constitute an interactive interface with the subgingival bacterial population. The cells are among the first host cells to be affected by periodontal disease (Andrian et al 2006). Alkoid nornicotine, a nicotine metabolite, can increase RAGE expression in gingival epithelial cells (GECs). Also, gingival fibroblasts stimulated with nornicotine can express a higher level of RAGE compared to non-treated cells. Recently it was shown that the predominant AGE of skin (CML collagen) could induce fibroblast apoptosis in vivo (Alikhani et al, 2005). Moreover, in vitro experiments showed that CML collagen induced a time and dose-dependent increase in fibroblast apoptosis compared to control collagen. It is possible that a similar pattern of apoptosis could occur in gingival epithelial tissues, which would contribute to the observed epithelial detachment and cell death involved in periodontitis (Vitkov et al, 2005). AGE deposition and RAGE expression have been associated with other risk factors for periodontal disease (Lalla et al, 1998). Lalla et al, (1998) reported that there is increased formation and deposition of AGEs in the gingiva of diabetic mice when inoculated with *P. gingivalis.* (Katz et al, 2005) examined human diabetic patients with periodontal disease and compared them to age and gender matched controls. Their results showed that the gingiva of diabetic patients with periodontal disease exhibited a higher presence of RAGE compared to their controls. These results suggested that an increased amount of RAGE might contribute to the advanced periodontal destruction commonly found in smokers and diabetic patients.

The combined effect of bacterial colonization and the local and systemic effect of smoking are responsible for the greater severity of periodontal destruction in smokers of the current study. Smokers in this study not only had fewer teeth than their non-smoking counterparts, but also had a significant periodontal attachment loss, deeper periodontal pockets, periodontal bone loss, tooth mobility, furcation involvement and greater amount of gingival recession. These results of the current study are similar to those reported by Linden and Mullally, (1994) , Harber et al, (1993), (Schenkein et al, (1995), (Mullally et al, (1999), Machuca et

al, (2000), and Haffajee and Socransky (2001). All of these studies have shown that compared to non-smokers, young adult smokers have a higher prevalence and severity of periodontitis, despite similar or low plaque levels. They also reported that the prevalence of periodontitis, defined as having a site with attachment loss>2mm and probing depths>4mm, was three to four times higher in young smokers compared to non-smokers. The high periodontal cost of smoking has been calculated as 27 years of disease progression. In-other words, a 32-year old smoker has similar periodontal attachment loss as a 59-year-old non-smoker which does not bode well for the future health of this generation. At the same time, results of the present study showed that the gingival bleeding and gingival inflammatory symptoms appeared to be suppressed in smokers, even though they had a significantly greater plaque and calculus indices. These results are parallel to those reported by Shuller, (1986), Bergström and Preber, (1994), Newbrun, (1996), Bergström and Boström, (2001) and Chen et al, (2001), and Dietrich et al, (2004). They found that the number of gingival bleeding sites, the amount of gingival exudates and the number of gingival sites with distinct redness were significantly less in smokers. There is contradictory evidence from several other studies that have reported increased signs of gingival inflammation and bleeding on probing in young smokers. Both Haber et al, (1993), and Linden & Mullally, (1994) found that young smokers had a significantly higher proportion of sites which bleed on probing than non-smokers. The difference in bleeding response between smokers and non-smokers has generally been attributed to vasoconstrictive properties of nicotine, which results in decreased blood flow to the gingiva and inhibits the early signs of gingival inflammation. The gingival vasoconstrictive episodes during repeated daily exposure to cigarette smoke might in long run result in vascular dysfunction in the gingival tissues (Vapaatalo and Mervaala, 2001). Also, Rezavandi, (2002) suggested that inflamed sites in smokers have reduced vascular density and angiogenesis compared to inflamed sites in nonsmokers, thus impairing inflammatory response and wound healing.

In conclusion, the current study shows that smoking is a major environmental factor associated with accelerated periodontal destruction in this group of young adult Saudi patients. The progression and excessive loss of periodontal support in later life depends to a greater extent upon excessive smoking in youth. The findings highlight the need for preventive strategies aimed at young individuals, many of whom take up smoking as a habit early in life.

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