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Decisive Analysis of Smoking and Periodontal Syndrome at Selected Dental Clinics, Saudi Arabia

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Abstract: ***Background: Cigarette smoking is one of the most preventable sources of morbidity and premature death worldwide. In the United States, smoking is responsible for approximately one in five deaths smoking has been shown to significantly increase the risk of tooth loss from periodontal disease. The effect appears to be dose-related, with heavy smokers exhibiting a significantly greater risk of tooth loss from periodontal disease compared to nonsmokers and lighter smokers. These patients have levels of risk similar to those of non-smokers. The aim of this provide decisive analysis which will consequently, will assist to provide some evidence support for the contribution between smoking and periodontal syndrome. Objective: The aim of this provides a decisive analysis to provide some evidence support for the contribution between smoking and periodontal syndrome. At the selected dental clinics, Saudi Arabia. Method: Structured survey was conducted at ten dental clinics, Saudi Arabia to provide a decisive analysis to provide some evidence support for the contribution between smoking and periodontal syndrome at the selected dental clinics, Saudi Arabia. Results: Several studies have shown that the effect of smoking on periodontal tissues may involve both of these processes. For example, smokers tend to have depressed numbers of T-helper lymphocytes, which are important cells of the immune system to regulate cell-mediated immunity and the activity of B lymphocytes. To boot, the host requires functional neutrophils to deal effectively with bacterial infections. Tobacco smoke has been shown by several studies to have a deleterious effect on various neutrophil functions. Conclusions: The existing research findings discovered that there was strong evidences replicate for the correlation between smoking and periodontal syndrome within the study setting.***

Keywords: Decisive Analysis; Awareness; &Periodontal Syndrome

1. Introduction

Typically, the diseased tissues of smokers tend to have a firmer appearance and less bleeding compared to that of nonsmokers. The term disease masking is used because the vaso-constrictive properties of tobacco smoke hide the inflammatory and destructive changes occurring within the periodontium. (Hennemeyer , 2013).

The periodontal tissues are compromised by the initial vasoconstriction, resulting in decreased blood flow to the gingiva. This masks the normal early signs of periodontal problems by decreasing gingival inflammation, erythematic, and bleeding despite the presence of the disease. The record of diseases origin by smoking has been expanded to include abdominal aortic aneurysm, acute myeloid leukemia, cataract, cervical cancer, kidney cancer, pancreatic cancer, pneumonia, periodontitis, and stomach cancer. These are in addition to diseases previously known to be caused by smoking, including bladder, esophageal, laryngeal, lung, oral, and throat cancers, chronic lung diseases, coronary heart and cardiovascular diseases, as well as reproductive effects and sudden infant death syndrome. (Haber,2003).

Preceding epidemiological results showed that smoking is a significant risk factor for the development of periodontal syndrome.5–7 Smokers have 2.5 to 3.5 times greater risk of severe periodontal attachment loss.8 Risk calculations suggested that 40 per cent of chronic periodontitis cases may be attributed to smoking, with an increased odds ratio of 5.4 for chronic periodontitis in smokers, (Wright,et al., (1993).

According to Bergstrom & Floderus , (2002) , smokers nearby another way when compared with non-smokers. Smokers have deeper probing depths, more deep pockets and more attachment loss, including more gingival recession. Smokers also have more alveolar bone loss and more teeth with function involvement. Smokers also tend to have a higher level of tooth loss than non-smokers after adjusting for oral hygiene, age, gender, and socio-economic level. The effect of smoking on the periodontal tissues is dose-dependent. Both daily consumption quantity and duration of smoking are related. Smokers have a higher prevalence of acute necrotizing ulcerative gingivitis.

2. Participants and Methods

This study was conducted in April, 2016 among periodontal staff at the dental clinics, Saudi Arabia. The study was granted ethical approval by the clinics ethical committee.

The participants were selected from the selected dental clinics. After signing an informed written consent form, the questionnaire was given to each participant. Before administration of the questionnaire, the purpose of the study was explained to each respondent and confidentiality of the information guaranteed.

A cross-sectional survey was conducted at ten dental clinics, Saudi Arabia. Review is to judge the evidence for the involvement between smoking and periodontal syndrome at the selected dental clinics, Saudi Arabia.

3. Results

Whether it is direct heat from the cigarette, the vasoactive response from nicotine, or a change in the host response to periodontal pathogens, the mechanism by which smoking induces periodontal attachment loss is currently unknown. Smoking has not only been shown to increase the severity of periodontal disease, but also to decrease the response of the gingival tissues to periodontal therapy, resulting in a greater incidence of refractory disease. Obviously, there is a plethora of published information correlating periodontal syndrometo both tooth loss and systemic manifestations. These systemic manifestations include increased risk of coronary artery disease; diabetes; osteopenia; and premature, low-birth-weight babies. Further, it has been demonstrated in numerous studies that smoking cessation leads to improved periodontal health and improved response to periodontal therapy, thus improving overall health. Therefore, it would greatly benefit our patients if we, as dental professionals, made a deliberate effort to promote smoking-cessation programs as well as educate our community on the benefits of not smoking.

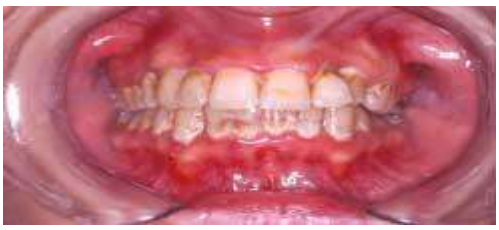
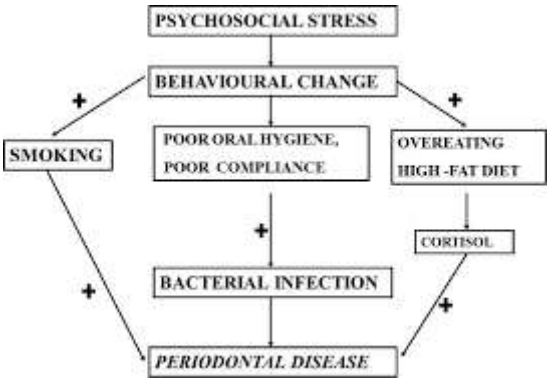


Figure 1: Acute necrotizing ulcerative gingivitis with severe caries.



Risk Factors for Osteoporosis

Risk Factors	Modifiable	Non Modifiable
Gender	No	
Age	No	
Early Menopause	No	
Low Bone Mass	Yes	Treatment of osteoporosis or osteopenia
Thin small framed body	No	
Race	No	
Lack of calcium	Yes	Diet high in calcium or Vitamin D
Lack of exercise	Yes	Weight bearing exercise
Smoking	Yes	Smoking cessation
Alchohol	Yes	Decreased alchohol consumption
Heredity	No	
Diseases (e.g. hyperparathyroidism	To some extent	Treatment
Certain medications (e.g. steroids	To some extent	After treatment if feasible
Propensity to falling	To some extent	physical therapy, neurological treatment if possible

4. Discussion

Whether it is direct heat from the cigarette, the vasoactive response from nicotine, or a change in the host response to periodontal pathogens, the mechanism by which smoking induces periodontal attachment loss is currently unknown. Smoking has not only been shown to increase the severity of periodontal disease, but also to decrease the response of the gingival tissues to periodontal therapy, resulting in a greater incidence of refractory disease.

Obviously, there is a plethora of published information correlating periodontal diseases to both tooth loss and systemic manifestations. These systemic manifestations include increased risk of coronary artery disease; diabetes; osteopenia; and premature, low-birth-weight babies. Further, it has been demonstrated in numerous studies that smoking cessation leads to improved periodontal health and improved response to periodontal therapy, thus improving overall health.

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Tobacco use has been implicated as a risk factor for alveolar bone loss. One hypothesis has been the possible stimulant effect of nicotine on osteoclastic activity, the cells most responsible for bone resorption. A study of porcine bone marrow cells found that nicotine is nontoxic to osteoclasts at clinically relevant levels, and appears to stimulate osteoclast differentiation and the resorption of calcium phosphate, the major inorganic component of bone. It is thought that nicotine-modulated osteoclast stimulation may partially explain the increased rapidity of alveolar bone loss and refractory disease incidence in smokers.

Approximately 90% of patients who were categorized as having failed to respond to conventional therapy were smokers (MacFarlane et al., 1992; Wolff et al., 1994). Recent work by Colombo et l. (1998) has disagreed with this stated proportion, since these investigators found that only 25, of their patients were current smokers, but that 40%, were former smokers.

Bostrom et al. (1 998) suggested that former smokers often begin smoking again, and therefore one must interpret the status of the former smokers cautiously, since self-reporting of smoking status is not reliable (Gonzalez et al., 996). Even though smokers will also benefit from treatment, albeit to a lesser degree, treatment failures tend to predominate among smokers.

Kinane and Radvar (1997) found that the response to non-surgical mechanical therapy is particularly poor in deep pockets in smokers. Although the attachment gain was also greater among the non-smokers than the smokers, this was not significant.

This indicates that, after treatment, a greater degree of recession occurred among the non-smokers compared with the smokers. In the description of the appearance of smokers' periodontal condition, and in studies looking cross-sectional at smokers, a frequently noted feature is the level of recession, which is often noted as worse in smokers than in non-smokers (Martinez-Canutt et a., 1995; Gunsolley et al., 1998).

A recent study in Australia using the National Survey of Adult Oral Health 2004–2006 to investigate the smoking-periodontitis relationship reported that former and current smokers had significantly higher periodontitis prevalence than never-smokers.[19] It was estimated that the population attributable fraction of smoking (classified as both current and former smokers) was 32 per cent for moderate to severe periodontitis (equivalent to 700000 cases).

5. Conclusions

The current study results revealed that there was strong evidences reflect for the contribution between smoking and periodontal syndrome within the study setting. Data available to date seem to suggest that smoking exerts a chronic effect by impairing the vasculature of the periodontal tissues rather than a simple vasoconstrictive effect. The suppressed vasculature has contributed to less gingival redness, less bleeding on probing and may also lead to an impaired healing response by affecting the revascularization.

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