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Critical Reviews of Smoking and Periodontal Disease at Selected Dental Clinics, Saudi Arabia

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Abstract: ***Background: Abundant research of the association between smoking and periodontal disease have been performed over the last 15 years, and there currently exists a substantial body of literature leading which this current review is based. From both cross-sectional and longitudinal studies, there appears to be strong epidemiological substantiation that smoking confers a considerably increased risk of periodontal disease. This evidence is further supported by the statistics emanating from patients who stop smoking. These patients have levels of risk similar to those of non-smokers. The aim of this review is to judge the evidence for the contribution between smoking and periodontal diseases. Objective: The aim of this review is to judge the evidence for the involvement between smoking and periodontal diseases at the selected dental clinics, Saudi Arabia. Method: 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 diseases at the selected dental clinics, Saudi Arabia. Results: With the amount of evidence from clinical and epidemiological studies, linking the adverse effects of smoking to the prevalence of periodontal disease and its severity, and non-surgical, surgical and regenerative treatment responses, dental professionals should consider advising patients about the negative impact of smoking on their periodontal health as well as about the benefits of quitting to the treatment. Conclusions: The current study results revealed that there was strong evidences reflect for the contribution between smoking and periodontal diseases within the study setting.***

Keywords: Safety Measures; Awareness; & Periodontal staff

1. Introduction

Tobacco is packed with harmful and addictive substances. Tobacco smoke contains over 3800 chemicals, including carbon monoxide, hydrogen cyanide, reactive oxidizing radicals and 60 of these chemicals are known or suspected to be carcinogens. In the 2004 United States Surgeon General's report, *The Health Consequences of Smoking*. (Alavi, Palmer, Odell, Coward & Wilson (2001).

According to Alpagot, Wolff, Smith & Tran (1996), the four major conclusions were Smoking harms nearly every organ of the body, causing many diseases and reducing the health of smokers in general. Secondly, quitting smoking has immediate as well as long-term benefits, reducing risks for diseases caused by smoking and improving health in general. Thirdly, Smoking cigarettes with lower machine-measured yields of tar and nicotine provides no clear benefit to health. Finally, the list of diseases caused 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.

Earlier epidemiological studies showed that smoking is a significant risk factor for the development of periodontal diseases.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.(Barbour, Nakashima, Zhang , Tangada , Hahn & Schenkein, et al (1997).

Periodontitis in smokers also presents differently 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. (Bergstrcim, 1989).

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 diseases 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 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. 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.

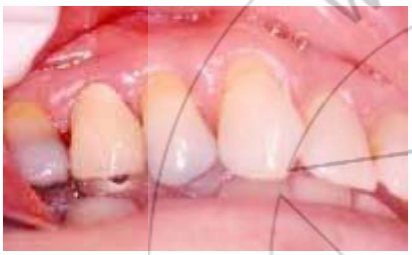


Figure 1: Apparently healthy gingival tissue in a one-pack-per-day smoker.

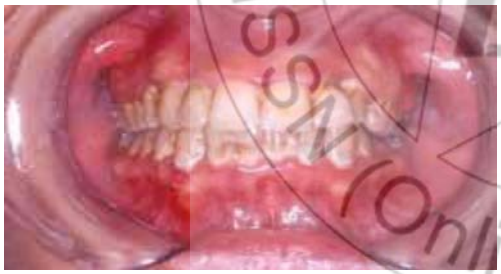
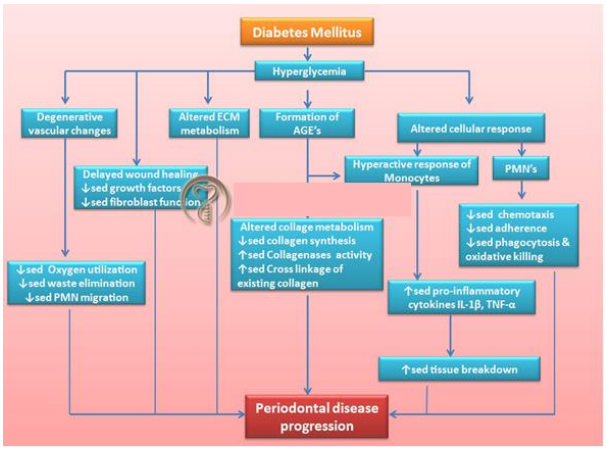


Figure 2: Acute necrotizing ulcerative gingivitis with severe caries



4. Discussion

It is recommended by several studies that smokers may have more disease since their microbial flora may be more 'pathogenic'. Nevertheless, host-related factors must manipulate this sub-gingival niche and the micro flora therein. The host resistance system will comprise the soluble and cellular components of the inflammatory and immune systems as well as the innate immunity afforded by such things as the epithelial barrier and fluid flow (GCF and saliva). The condensed GCF flow reported in smokers will mean that antibodies and other defense molecules derived from the serum will be reduced in quantity. An additional effect of reduced GCF flow would be fewer microbial nutrients and less flushing of the gingival crevice and removal of microbes and waste products as the GCF leaves the gingival crevice by the positive outward flow. These factors may all influence the flora at these sites. Smoking may affect the vasculature, the humoral immune system, and the cellular and soluble inflammatory system, and may have effects throughout the cytokine and adhesion molecule network. The comparative significance of these smoking-related alterations and their precise mode of action in increasing the risk of periodontal disease remains to be elucidated.

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 998a) 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) and 56 per cent for severe periodontitis in the Australian adult population.

5. Conclusions

The current study results revealed that there was strong evidences reflect for the contribution between smoking and periodontal diseases 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.

6. Acknowledgements

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