CLINICAL AND RADIOMETRIC EVALUATION OF PERIODONTAL STATUS IN INDIVIDUALS EXPOSED TO NEGATIVE CIGARETTE SMOKE AS A RISK FACTOR FOR PERIODONTAL DISEASE

Thesis

Submitted in Partial Fulfillment for the Requirements of the Master Degree in Oral Medicine and Periodontolgy

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2008

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Acknowledgment

First of all I thank ***God*** *for paving the way to fulfill this work.*

I would like to express my sincere gratitude and appreciation to ***Prof. Dr. Mouchira Salah El – Din*** *Professor of Oral Medicine, Periodontology and Diagnosis, Faculty of Oral and Dental Medicine, Cairo University, for her generous supervision, her great help and her cooperation in guiding me in every step in this study. I would like to thank her for proposing the topic of the present work.*

I would like to express my deep thanks and appreciation to ***Dr. Shahira Gamal El Ashiry****, Lecturer of Oral Medicine, Periodontology and Diagnosis, Faculty of Oral and Dental Medicine, Cairo University. I was fortune to conduct this work under her valuable supervision. Her sincere help, guidance, continuous encouragement and constructive comments will always be remembered.*

I am deepy grateful to ***Prof. Dr. Adel Salah El Gehini;*** *Professor of Quality Control in Textile Department, Faculty of Engineering, Alexandria University; for spending much of his time and effort in performing the statistical analysis.*

DEDICATION

I would like to dedicate this thesis to my loving and supporting husband and my beloved parents to whom I am greatly indebted.

Contents

مستخلص الرسالة (Abstract)

باللغة الأجنبية:

The present study was conducted for assessment of the periodontal status clinically and radiodensitometrically in patients exposed to passive smoking. The patients were divided into two groups according to exposure to passive smoking. The patients evaluated clinically and evaluation of radiometric and radiodensity measurements was carried out by Digora software at different areas o measure the alveolar bone loss and bone density in the supporting structures of the teeth. The results showed that alveolar bone loss were higher in passive smoker patients than control patients while alveolar bone density were higher in control patients than passive smokers.

Key words:

1· Clinical
2· Radiometric
3· Periodontal
4· Negative
5· Cigarette smoking

Introduction and Review of Literature

Periodontal disease (PD) is a chronic inflammatory disease, affecting the supporting structures of teeth. It is identified by a bacterial challenge that can destroy the host tissues leading to periodontal attachment loss, bone loss and ultimately possible tooth loss **(Nunn, 2003).**

Gingivitis and periodontitis are the two major forms of inflammatory diseases affecting the periodontium. Their primary etiology is bacterial plaque, which can initiate destruction of the gingival tissues and periodontal attachment apparatus. Gingivitis is inflammation of the gingiva that does not result in clinical attachment loss. Periodontitis is inflammation of the gingiva and the adjacent attachment apparatus and is characterized by loss of connective tissue attachment and alveolar bone **(Armitage, 1999).**

Gingivitis is a reversible disease. Therapy is aimed primarily towards reduction of etiologic factors to reduce or eliminate inflammation, thereby allowing gingival tissues to heal. Appropriate supportive periodontal maintenance that includes personal and professional care is important in preventing re-initiation of inflammation. On the other hand, therapeutic approaches for periodontitis fall into 2 major categories: 1) anti-infective treatment, which is designed to halt the progression of periodontal attachment loss by removing etiologic factors and 2) regenerative therapy, which is intended to restore structures destroyed by disease. Essential to both treatment approaches is the inclusion of periodontal maintenance procedures. If periodontitis is not treated, the disease will slowly progress

and painlessly destroy the bone which supports the teeth. Untreated, the disease will eventually cause tooth loss **(Ramfjord, 1993)**.

Inflammation of the periodontium may result from many causes (e.g., bacteria, trauma). However, most forms of gingivitis and periodontitis result from the accumulation of tooth-adherent microorganisms **(Socransky and Haffajee, 1991)**.

Etiology and Pathogenesis of Periodontal Disease:

The mouth, like all external surfaces of the body and the gut, has a substantial microflora living in symbiosis with a healthy host. The microflora of the mouth contains hundreds of species of aerobic and anaerobic bacteria. These organisms grow on tooth surfaces as complex, mixed, interdependent colonies in biofilms and are attached and densely packed against the tooth in the deeper layers, with more motile forms in the superficial layers **(Socransky and Haffajee, 2003)**.

Cultural studies indicated that more than 500 distinct microbial species can be found in dental plaque **(Moore and Moore, 1994).** However, molecular methods of 16S rDNA amplification revealed an even more diverse view of the subgingival bacterial flora and suggested that a large proportion of even this well-studied and familiar microbial environment remains uncharacterized **(Kroes et al., 1999 and Lepp et al., 2004).**

As dental plaque matures to a state that is associated with periodontal disease, the number of gram-negative and anaerobic bacteria increases **(Tanner et al., 1996; Tanner et al., 1998; Ximenez-Fyvie et al., 2000 and**

Ramberg et al., 2003). Bacterial counts above the gums (supragingival) on one tooth surface can exceed 1×10^{9} bacteria. Below the gum, the number of bacteria ranges from 1×10^{3} in a healthy shallow crevice to more than 1×10^{8} in a periodontal pocket **(Socransky and Haffajee, 2003)**.

Tooth cleaning every 48 hours can maintain the biofilm mass at an amount compatible with gingival health. Unfortunately, few individuals achieve this and exhortations to the public to clean teeth more thoroughly are generally ineffective in public-health care **(Watt and Marinho, 2005).**

An enormous research effort has been devoted to the study of periodontal-disease-associated microflora, from classic cultural methods to modern approaches on the molecular, whole genomic and proteomic level **(Socransky et al., 2002 and Sanz et al., 2004)**.

Certain clusters of bacterial species commonly cohabit subgingival sites and are reproducibly associated with disease **(Socransky et al., 1998)**. These putative pathogens include *Porphyromonas gingivalis, Tannerella forsythensis* and the spirochaete *Treponema denticola.* Infection of periodontal tissues with these and other organisms is accompanied by the release of bacterial leucotoxins, collagenases, fibrinolysins and other proteases **(Socransky and Haffajee, 1994).**

Actinobacillus actinomycetemcomitans is another species commonly associated with disease, especially in young adults **(Mandell and Socransky, 1981)**. Recent work implicates herpes viruses in the pathogenesis of periodontitis **(Michalowicz et al., 2000b; Slots, 2004 and**

Kubar et al., 2005) and *Candida albicans* and other fungi in immunocompromised individuals **(Robinson, 2002).**

Although bacteria are necessary for periodontal disease to take place, a susceptible host is also needed. The immune-inflammatory response that develops in the gingival and periodontal tissues in response to the chronic presence of plaque bacteria results in destruction of structural components of the periodontium leading, ultimately, to clinical signs of periodontitis **(Lang et al., 1990)**.

An individual's risk for periodontal disease could be linked to gingival inflammation (bleeding) in response to plaque accumulation **(Joss et al., 1994).** The host response is essentially protective, but both hypo-responsiveness and hyper-responsiveness of certain pathways can result in enhanced tissue destruction **(Preshaw et al., 2004).**

Both the host and bacteria in the periodontal biofilm release proteolytic enzymes that damage tissue. They release chemotactic factors that recruit polymorphonuclear leucocytes into the tissues; if sustained, these cells release various enzymes that break down tissues. Hundreds or even thousands of microbial antigens evoke both humoral antibody-mediated and cell-mediated immune responses. These responses are usually protective, but a sustained microbial challenge results in the breakdown of both soft and hard tissues, mediated by cytokine and prostanoid cascades **(Yamazaki et al., 2003).**

Histologically, non-progressive inflammatory foci tend to be composed predominantly of T lymphocytes and macrophages, suggesting

that the cell-mediated response can control disease. Destructive lesions are dominated by B lymphocytes and plasma cells, suggesting that humoral immunity is not always effective **(Yamazaki et al., 2003)**.

Risk Factors of Periodontal Disease:

Risk is defined as the probability that an event will occur in the future or the probability that an individual develops a given disease or experiences a change in health status during a specified interval of time. Applied to destructive periodontal diseases, it is the probability that periodontitis or measurable periodontal tissue loss, will take place during a specified period of time **(Kleinbaum et al., 1982).**

Risk factors of periodontal disease may be defined as distinctive characteristics or exposures that increase the probability of developing periodontitis or lead to a measurable change (loss) in the status of periodontal supporting tissues. Accordingly, the identification of risk factors should be based on an analysis of the temporal relationship between the presence of exposures (potential factors) and the occurrence of tissue loss over a given time period and this will allow the assessment of the probability of occurrence of this event **(Van Dyke and Sheilesh, 2005).**

A risk factor that can be used to predict the future course of disease, such as an increased probability of disease, is known as a risk marker. Some risk factors can be modified to reduce one's risk of initiation or progression of disease, such as smoking cessation or improved oral hygiene to reduce the

risk of periodontal destruction. Other factors can not be modified such as genetic factors. A risk factor that can not be modified is often referred to as determinant **(Genco, 1996).**

A variety of microorganisms can contribute differently in populations and individuals in the pathogenesis of periodontal disease **(Haffajee et al., 2004)**. However, the manifestation and progression of periodontitis is influenced by a wide variety of determinants and risk factors, including subject characteristics, social and behavioral factors, systemic factors, genetic factors, tooth level factors and other emerging risk factors **(Nunn, 2003).**

I- Subject Determinants:

Aging is commonly associated with periodontal disease, although this relationship is thought to be more related to the cumulative periodontal breakdown over time than to an age-related intrinsic deficiency that contributes to susceptibility to periodontal disease **(Genco, 1996)**.

In a study conducted by **Muzzi et al. (2006)**, they found that 86% of people over 70 years old had at least moderate periodontitis or severe form of periodontitis and over one-fourth of this 86 % had lost their teeth. The study also showed that the disease accounted for a majority of tooth extractions in patients older than 35 years of age**.**

The role of race as a risk factor for periodontal disease is a more complicated issue. For instance, in a study of older community-dwelling

blacks and whites (65 years of age and above), blacks were three times more likely to exhibit advanced periodontal destruction compared to whites in the same age cohort. In addition *Prevotella intermedia* was found to be a risk factor for blacks but not for whites**.** However, in this study, blacks had more indicators related to socioeconomic status than whites **(Beck et al., 1990)**.

When limited to subjects in the same socioeconomic status, differences in periodontal status between blacks and whites often disappeared **(Grossi et al., 1994)**. Furthermore, it was found that racial differences in the distribution of certain genetic risk factors might also contribute to differences in the prevalence and severity of periodontal disease among different races **(Grossi et al., 1995)**.

II- Systemic Factors:

Diabetes Mellitus

The disease most commonly found in medical histories of patients with tooth loss is diabetes mellitus at over 19 % **(Khalaf et al., 2005).** There is much evidence showing a link between type 1 and 2 diabetes and PD. People with these diseases have 15 times the risk of the non-diabetic population **(Golla et al., 2004).** Diabetes causes abnormalities in blood vessels and increases the levels of interleukins. Both of these complications significantly increase the chances of PD. In addition, high levels of triglycerides which are common in type 2 diabetes appear also to impair periodontal health **(Genco et al., 1998).**

In addition to the risk progression of periodontal disease posed by poorly controlled diabetes, it has also been suggested that effective periodontal therapy can have a positive effect on the control of diabetes **(Thorstensson, 1995)**. In a longitudinal study of non-insulin dependent diabetics, severe periodontal disease at baseline was found to be a significant risk factor for poor glycemic control **(Taylor et al., 1996).** Similarly, poor glycemic control in non-insulin dependent diabetics has been shown to be associated with significantly greater alveolar bone loss over time compared to well-controlled non-insulin dependent diabetics **(Taylor et al., 1998)**.

Atherosclerosis

Atherosclerosis is a progressive disease involving large-to-medium-size muscular and large elastic arteries. The advanced lesion or atheroma consists of elevated focal intimal plaques with a necrotic central core containing lysed cells, cholesterol ether crystals, lipid-laden foam cells and surface plasma proteins including fibrin and fibrinogen. The presence of an atheroma provides a surface for enhanced platelet aggregation and thrombus formation. According to data compiled by the American Heart Association, nearly one of every two Americans dies of cardiovascular disease that is attributed to complications of atherosclerosis manifested as coronary thrombosis and myocardial infarction **(Consigny, 1995).**

Periodontitis patients and coronary heart disease patients have a number of common characteristics, including variables such as age (positive correlation), education (negative association), gender (males higher), finances (negative association), tobacco use (positive correlation), alcohol