
بسم الله الرحمن الرحيم
وَقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ
عَمَلَكُمْ وَرَسُولُهُ وَالْمُؤْمِنُونَ
سورة التوبة
الآية ١٠٥
صدق الله العظيم

Effect of Periodontal Therapy On Glycemic level of Diabetic Patients

A Thesis
Submitted in Partial Fulfillment of the requirements for master degree in
Oral Medicine and Periodontology

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2010

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تأثير علاج التهاب الأنسجة حول السنية على ضبط مستوى السكر لمرضى السكرى

ر ســـــالة مقدمة من
الطبيب / محمد عمرو حمدى سالم
بكالوريوس طب وجراحة الفم والأسنان
جامعة قناة السويس

توطئة للحصول على درجة الماجستير في طب الفم وعلاج اللثة

كلية طب الفم والأسنان
جامعة القاهرة
٢٠١٠م

المشرفون

الأستاذ الدكتور / محمد شرين حسن على

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مدرس فى قسم طب الفم وعلاج اللثة والتشخيص
كلية طب الفم والأسنان
جامعة القاهرة

Dedication

To My Parents

&

Lovely Sister

Acknowledgement

First and foremost, thanks are due to **ALLAH**, *to whom I relate any success in achieving any work in my life.*

I'm so grateful to professor Dr. **Mohamed Sherien Hassan Ali,** *Professor of Oral Medicine, Oral Diagnosis and Periodontology, Cairo Unviersity, for his support, continuous teaching and Fatherly guidance during this study.*

Also the words are not enough to express my sincere appreciation and gratitude to Dr. **Eman Yossef El-Firt,** *Lecturer of Oral Medicine, Oral Diagnosis and Periodontology, Cairo University, for her patience and beneficial advice and continuous constructive supervision throughout the whole study.*

I would also like to express my deepest appreciation to Professor Dr. **Mahmoud El-Refaey,** *Chairman and Professor of Oral Medicine and Periodontology, Cairo University, and all staff and members of Oral Medicine, Oral Diagnosis and Peiodontology Department, Faculty of Oral and Dental Medicine, Cairo University, for their considerable cooperation, kind feeling and supports.*

Mohamed Amr Hamdy Salem

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INTRODUCTION

Diabetes Mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia **(American Diabetes Association, 2007)**. Factors contributing to the hyperglycemia include reduced insuline secretion, decreased glucose utilization and increased glucose production **(American Diabetes Association, 2007)**.

Diabetes mellitus (DM) and periodontitis are common chronic diseases in adults. Both diseases are highly prevalent in the world population. Approximately 21 million children and adults in the United States (7% of the population) have diabetes and this incidence is increasing annually **(Connel et al., 2008)**.

Both epidemiological studies and case reports have shown diabetes to be a major risk factor for periodonitis. Periodonitis has been found to be more prevalent and more severe in patients with diabetes than the normal population **(Tan et al., 2006)**. This may be explained on the basis that hyperglycemia dramatically alters the function of multiple cell types and their extra cellular matrix. This results in structural and functional changes in the affected tissue **(Nishimura et al., 2000)**.

On the other hand, chronic gram- negative infections and chronic endotoxemia, like those seen in periodontal disease, resulted in elevated secretion of interleukin-1β (IL-1β), tumour necrosis factor-α (TNF- α), interleukin-6 (IL-6), and prostaglandin E_2 (PGE_2). These cytokines could induce insulin resitance and worsen the metabolic control in diabetic patients **(Grossi et al., 1996)**.

Historically, systemic delivery of teteracyline has been extensively utilized as an adjunctive periodontal therapy. Prior to the mid 1980s,

teteracyline was recognized as inhibitory for a vast number of bacteria commonly found associated with periodontally diseased sites **(Walker et al., 2004)**.

The use of doxycyline combined with mechanical periodontal treatment in subjects with diabetes is justified as follows: first, it is a broad spectrum antibiotic that is effective against most periodontal pathogens, and it reaches higher concentrations in the gingival fluid than in the serum, providing an important adjunct for the reduction of periodontal pathogens. Second, it is a potent modulator of the host response in subjects with diabetes, as well as being a metalloproteinase inhibitor. It also inhibits nonenzymatic glycation of extracellular proteins, and it may have a similar effect on the glycation of hemoglobin **(Connell et al., 2008)**.

In a study by **Grossi et al., 1997**, they verified a 10% glycated hemoglobin (HbAlc) reduction after 3 months of non-surgical periodontal treatment combined with systemic doxycyline, whereas there was no significant change in the control group. Moreover; another investigation by **Iwamoto et al., 2001**, showed that administrated 10 mg local minocycline caused a significant reduction in glycated hemoglobin (HbAlc) levels after 1 month.

Accordingly, and due to the clear clinical and epidemiologic evidence in literature that periodontal disease worsen glycemic control in diabetes. the objective of the present study was to perform a clinical and metabolic evaluation of the response to conventional periodontal treatment combined with systemic doxycyclin administrations on the glycemic control of diabetic patients.

Aim of The Study

The aim of the present study is to evaluate the effect of systemic doxycycline administration as an adjunctive therapy to scaling/root planing on glycemic control of diabetic patients suffering from chronic periodontits .

REVIEW OF LITERATURE

Periodontitis is an inflammatory disorder characterized by destruction of periodontal tissues with a subsequent loss of attachment. Although several mechanisms are involved in the activation of the inflammatory process, the destructive process in fact is a consequence of inadequate interaction between oral microflora and host defense mechanism. **(Mariotti et al., 1999)**

The international workshop for a classification of periodontal diseases and conditions in 2002, classified periodontal diseases into: gingival diseases with or without dental plaque, chronic periodontitis, aggressive periodontitis, periodontitis associated with systemic diseases, necrotizing periodontal diseases, abscesses of the periodontium, periodontitis with endodontic lesions and periodontitis with developmental or acquired deformities **(Armitage, 2002)**.

Gingivitis is a non destructive form of periodontal diseases while periodontitis is a destructive form that are characterized by frequent chronic recurrent bacterial infection of the periodontal tissues, resulting from plaque accumulation in the gingival crevice leading to an apical migration of the junctional epithelium and loss of periodontal soft and hard tissues **(Kinane et al., 2001)**.

Chronic periodontitis is the most common form of destructive periodontal disease in adults, yet it can occur over a wide range of age. It can affect both the primary and the secondary dentition **(Flemming, 1999)**.

Microbial plaque is recognized as the primary etiologic agent for periodontal disease initiation and progression **(Oringer et al., 2002)**. More than 500 different bacterial species have been identified within periodontal

pockets. However, relatively few species have been clearly associated with progressive periodontitis. Most putative pathogens are indigenous to the human oral cavity, but possible superinfecting organisms (enteric gram negative rods, pseudomonas, staphylococci, and yeasts) may also inhabit periodontal pockets. Periodontitis lesions usually harbour a constellation of putative pathogens rather than a single pathogenic species **(Paster et al. 2001)**.

It has long been thought that gram-negative anaerobes were the primary pathogens in periodontal pockets, and efforts to identify specific causative microorganisms have been unsuccessful. More recently it has become clearer that in the broad gram-negative profile found at diseased sites, there are several putative pathogens that are consistently found. The predominant group includes Actinomyces actinomycetemcomitans, now called (Aggrigattibacter actinomycetemcomitans) (Aa), Bacteroides forsythus, now called (Tannerella forsythia), Porphyromonas gingivalis (Pg), Prevotella intermedia (PI), Fusobacterium nucleatum (FN), Campylobacter rectus, and Treponema denticola **(Hamlet et al., 2001)**.

The presence of different clonal types of these bacteria is recognized, and it is not known whether all clonal types are pathogenic, which accounts for some of the inconsistent associations found between the bacterial presence in the periodontal crevice and clinical disease **(American Academy of Periodontology, 2005)**.

In a study by **Albandar et al., 2001**, young adults with generalized periodontitis had significantly elevated serum IgG and IgA antibody levels reactive to Pg and Aa compared to healthy controls.