Bacterial Reduction after using different Irrigation protocols in Healthy and Diabetic patients with Apical Periodontitis

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"بسم الله الرحمن الرحيم"

"رَبِّ أَوْزِعْنِي أَنْ أَشْكُرَ نِعْمَتَكَ الَّتِي أَنْعَمْتَ عَلَيَّ وَعَلَى وَالِدَيَّ وَأَنْ أَعْمَلَ صَالِحًا تَرْضَاهُ وَأَدْخِلْنِي بِرَحْمَتِكَ فِي عِبَادِكَ الصَّالِحِينَ"

صدق الله العظيم

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Dedication

The completion of this study could not have been possible without the participation and assistance of so many people whose names may not all be enumerated, who helped, supported, withstand me to make a difference. Their contributions are sincerely appreciated.

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Introduction

Diabetes mellitus (DM) is a group of complex multisystem metabolic disorders due to a deficiency in insulin secretion caused by pancreatic β-cell dysfunction and/or insulin resistance in liver and muscle. DM affects many functions of the immune system and is associated with delayed healing and compromised immune responses (1).

There is a significant association between an increased prevalence of apical periodontitis and type 2 diabetes mellitus (2). when cases with preoperative periradicular lesions were investigated, diabetics had a much lower rate of successful cases compared with non-diabetics patients preoperatively (3).

Apical periodontitis defined as a result to endodontic infection that includes an active interaction between microbial factors and host defenses, resulting in cytokine production and periapical bone resorption (4). It has been shown that root canal treatment of teeth showing apical periodontitis has a lower success rate in comparison with teeth with no apical disease (5).

Bacterial species, it's density and virulence, in addition to host response play an important role in causing apical disease and consequently affect the prognosis (6). There is an inverse relationship between number of cells sufficient to cause disease and bacterial virulence . Better prognosis is found in teeth showing negative culture before obturation when compared with those presenting bacteria in the canals at the time of obturation (7).

The challenge now is to define the bacterial quantification levels to be established during treatment that lead to healing. Quantitative real time

polymerase chain reaction using universal primers or probes, is one of the most reliable techniques to provide quantitative information for bacterial populations (8). One hypothesis that may explain the reason why diabetic host may be more liable to infections is due to the presence of different root canal microbes and virulent bacterial profile (9).

Success of root canal treatment depend upon the effective removal of bacterial biofilms and their by-products from the root canal by using chemo-mechanical means . Even with modern mechanical techniques not all canal walls can be completely addressed (10). Hard and soft tissue debris maintained after mechanical instrumentation can harbor microorganisms which decrease the efficacy of the seal created by obturating materials, which may lead to treatment failure. The use of irrigant agitation is important during instrumentation as a way of chemical debridement to remove debris and bacteria from root canal system (11).

To improve the flow and distribution of irrigating solution various techniques and devices should be introduced to reach this goals. Evaluation of bacterial reduction before and after different irrigating protocols was thought to be of value.

Review of literature

A-Microgiology of apical periodontitis

I- Pathogenesis of apical periodontitis

The elimination of intruding bacteria is one of the main function of immune system. The polymorphonuclear leukocytes (PMNs) phagocytose and kill bacteria as it's the first to reach blood stream and migrate to the site of bacteria. Then inflammation occurs which lead to vasodilatation of blood vessels accompanied by stasis of blood stream followed by pavementation of PMNs on blood vessels wall. After that PMNs reach to inflammation site by chemotaxis of C5a, which engulf and opsonize bacteria after marking them by IgG or C3b. An intense phase in which PMNs dies, then macrophage appear as scavengers and begin their action as link between innate and specific(12).

A prolonged presence of microbial irritants leads to a shift in the neutrophil-dominated lesion to a macrophage-, lymphocyte-, and plasma-cell-rich one. This lesion is asymptomatic with radiolucent lesions. The macrophage derived pro-inflammatory cytokines (IL-1, -6; TNF-α) are powerful lymphocyte stimulators. Macrophages produce cytokines which activate, proliferate and mature B-lymphocytes to be plasma cells which produce immunoglobulines. Macrophage activated by interferon alpha where it produce IL-1 and IL8 that activate PMS. Bone resorption is mediated by TNF-B activated by T-cells and IL1B activated by macrophages. This is a

destructive side effect of the protective pathway against apical periodontitis(13).

The destructive process is down regulated by delayed or absence bone resorption by collagenous connective tissue formation during the chronic phase of apical periodontitis. The radiolucent area formation due to apical bone resorption in acute apical periodontitis is relatively inactive during chronic phase of apical periodontitis(13).

Pathogenesis of apical lesion in diabetic patients

The immune system, wound healing and leukocytes function are impaired in diabetes mellitus. Abnormal leukocyte function in Diabetic patients can be justified by down regulation of adhesion molecules leading to decreased leukocyte endothelial cell interactions and a reduced leukocytes numbers in inflammatory lesions. A study reported decreased chemotaxis of leukocytes and increased detection of obligate anaerobic bacteria in the pulp of type2 diabetic rats on a 30% sucrose solution diet than in control rats(14).

Advanced glycation end products (AGE) which are the end products of enzymatically catalyzed reactions between glucose with proteins and lipids have been linked to long term complications of diabetes(15). AGEs interact with their receptors (RAGEs) on endothelial cells, smooth cells and infiltrating mononuclear phagocytes. RAGEs are at a low level in normal states, but in hyperglycemia the expression of RAGEs is enhanced on critical target cells which affect respond to infection . The alterations lead to increased vascular permeability, enhanced expression of adhesion molecules on endothelial cells, attraction and activation of macrophages, collagen synthesis impairment, and leukocyte function impairment which lead to accelerated and excessive tissue destruction in periapical infection(16).