Evaluation of the efficacy of Subgingival Debridement using ultrasonic scaler versus Modified Widman Flap in managing advanced chronic periodontitis

Thesis

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Dedication

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Introduction and review of literature

Introduction and review of literature

Normal periodontium is defined as those tissues supporting and investing the tooth. It comprises root cementum, periodontal ligament, alveolar bone and dentogingival junction. The dentogingival junction is an adaptation of the oral mucosa that includes epithelial and connective tissue components. The epithelium is divided into three functional compartments, gingival, sulcular and junctional epithelium, and the connective tissue into superficial and deep compartments. The junctional epithelium plays a crucial role since it essentially seals off periodontal tissues from the oral environment maintaining a healthy periodontium. Periodontal diseases set when the structure of the junctional epithelium starts to fail, which is an excellent example of how structure determines function **(Nanci and Bosshardt 2006).**

Tooth loss may be the ultimate consequence of destructive periodontal disease. Periodontitis is thought to account for 30-35% of all tooth extractions while caries and its sequelae for up to 50% **(Papapanou and Lindhe 2008).**

The most common diseases of the periodontal tissues are inflammatory processes of the gingiva and the attachment apparatus of the tooth including gingivitis and periodontitis. Gingivitis is inflammation of the gingiva that does not result in attachment loss and it is readily reversible by removal of etiologic factors and effective oral hygiene. While periodontitis is inflammation of the gingiva and the adjacent attachment apparatus and is characterized by loss of periodontal

attachment and alveolar bone **(The American Academy of Periodontology 2001).**

Classifications of periodontal diseases are useful to help establish diagnosis, determine prognosis and facilitate treatment planning. Different classifications of periodontal diseases have been used over the years. The most recent classification of periodontal diseases classified periodontitis as chronic periodontitis, aggressive periodontitis and periodontitis as a manifestation of systemic diseases **(Armitage 1999).**

Chronic periodontitis

Chronic periodontitis is defined as an inflammatory disease affecting the supporting structures of the teeth. It is caused by groups of specific microorganisms which result in progressive destruction of the periodontal ligament and alveolar bone. Although chronic periodontitis is most frequently observed in adults, it can occur in children and adolescents in response to chronic plaque and calculus accumulation **(Novak and Novak 2006).**

Chronic periodontitis is the most prevalent form of periodontitis and is generally considered to be a slowly progressing disease. It results from extension of the inflammatory process initiated in the gingiva to the supporting periodontal tissues. The primary clinical features of periodontitis include gingival inflammation, clinical attachment loss, alveolar bone loss, periodontal pocketing. In addition, gingival enlargement or recession, bleeding of the gingiva, increased mobility, drifting and/or tooth exfoliation may occur. With few exceptions, most

forms of periodontitis are chronic inflammations that may progress continuously or by bursts of activity **(Flemmig 1999).**

Chronic periodontitis is considered a site specific disease and the clinical signs are believed to be caused by direct site specific effects of subgingival plaque accumulation. As a result of this local effect, periodontal destruction may occur on one surface of a tooth while other surfaces maintain normal attachment levels. In addition to being site specific, chronic periodontits may be described as being localized or generalized. Localized periodontitis occurs when less than 30% of the sites assessed in the mouth demonstrate attachment loss and bone loss while generalized periodontitis occurs when 30% or more of the sites assessed in the mouth are affected **(Armitage 1999)**.

According to disease severity, chronic periodontitis may be differentiated by mild (1 to 2mm), moderate (3 to 4 mm), or advanced (≥5mm) clinical attachment loss (CAL) **(Armitage 1999)**. A study done by **Morris et al. (2001)** indicated that about 5-10% of the adult population has advanced periodontitis while about 40-45% has moderate periodontitis and 50% of the population has chronic gingivitis.

Etiology and pathogenesis

The current concept on the etiology of periodontitis considers three groups of factors that determine whether active periodontitis will occur in a subject. These factors include a susceptible host, the presence of pathogenic species and the absence or a small proportion of beneficial bacteria **(Socransky et al. 2002).**

The primary initiator of periodontal disease is specific invasive oral pathogens that colonize dental plaque biofilms on the tooth root surface. The presence of one or more pathogenic species in sufficient numbers is necessary in the development of periodontitis. Porphyromonas gingivalis (P.gingivalis), Tannerella forsythia (T.forsythia), and Treponema denticola (T.denticola), in addition to Aggregatibacter actinomycetemcomitans (A.a) are considered key pathogens in the initiation and progression of periodontal disease **(Quirynen et al. 2006).**

Bacteria and their toxic products cause direct periodontal destruction. This occurs through lipopolysaccharides of gram negative bacteria, the liberation of fatty acids (butyric and propionic) that are toxic to the tissues and N-formyl-methionyl leucyl phenylalanine. However the host's own immuno-inflammatory response to this bacterial infection can cause even more destruction than that caused by pathogenic bacteria. This indirect periodontal destruction occurs by the production of host derived inflammatory mediators including cytokines and bioactive lipids by neutrophils, monocytes, lymphocytes and fibroblasts **(Wilson et al. 1996 and Madianos et al. 2005).**

The susceptibility of the host is partially hereditary i.e genetic variations or mutations that modulate the individual's response to the intra-oral bacterial insult. In addition, environmental and behavioral factors such as smoking, stress and diabetes influence the manifestation and progression of periodontitis **(Nunn 2003).**

The presence of beneficial species can influence disease progression in different ways. These beneficial bacteria can affect the vitality or growth of a pathogen, affect the ability of a pathogen to produce

virulence factors or degrade virulence factors. These bacteria also occupy intra-oral spaces that may otherwise be colonized by pathogens and limit pathogens adherence to tissues surfaces. An example of such a beneficial action is the production of hydrogen peroxide by streptococcus sanguis which can kill A.a. **(Hillman et al. 1985 and Quirynen et al. 2006).**

Periodontal therapy

Patients need to be informed of the importance of periodontal therapy as there are broader benefits of obtaining periodontal health other than retaining damaged teeth. Previous information showed that active periodontal disease increases the susceptibility to systemic problems, such as atherosclerosis, strokes, and preterm and low birth weight. **(Dasanayake 1998, Jeffcoat et al.2003, Scannapieco et al.2003).**

The goals of periodontal therapy are to alter or eliminate the microbial etiology and the contributing local risk factors for periodontitis. Arresting the progression of periodontal disease and preserving the dentition in a state of health, comfort and function with appropriate esthetics and to prevent the recurrence of periodontitis and reduce tooth loss are the main therapeutic goals. In addition, regeneration of the periodontal attachment apparatus, where indicated, may be attempted. **(The American Academy of Periodontology 2000).**

Periodontal therapy includes mechanical removal of plaque and local factors, chemotherapeutic agents, oral hygiene measures and periodontal maintenance procedures **(Jolkovsky and Ciancio 2006).**

Plaque control can be achieved by mechanical and/or chemical means. Mechanical plaque control includes regular self performed plaque removal and professional mechanical removal of plaque and calculus. Supragingival plaque control is fundamental to the prevention and management of periodontal diseases as plaque induced gingivitis always precedes the occurrence and recurrence of periodontitis. Also subgingival plaque is derived from supragingival plaque so meticulous supragingival plaque control can modify the composition of pocket microbiota and lower the percentage of periodontopathic bacteria. **(Dahlen et al. 1992, Haffajee et al. 2001 and Weijden et al. 2008).**

Oral hygiene measures, including tooth brushing and interdental cleaning devices, are necessary means for supragingival plaque control. Previous studies demonstrated that high standards of oral hygiene will ensure the stability of periodontal tissue support **(Hujoel et al. 1998 and Axelsson et al. 2004).**

Chemotherapeutic agents

Chemotherapeutic agents are either systemic or local. Local chemical supragingival plaque control can be delivered through tooth pastes, mouth rinses, sprays, irrigators, chewing gums and varnishes. Chemical agents such as Chlorhexidine, Quaternary ammonium compounds, phenols and essential oils, fluorides and oxygenating agents are useful in the control of plaque and/or gingivitis **(Addy and Moran 2008).**

Chlorhexidine mouth rinses after periodontal debridement is effective in the reduction of plaque accumulation, inflammation, and probing pocket depth (PPD) **(Faveri et al. 2006 and Cheng et al. 2008).**

Chlorhexidine has a broad antimicrobial action, including a wide range of gram positive and gram negative bacteria, also effective against some fungi including candida and some viruses including Human Papilloma virus and Human Immunodeficiency virus. It is the most effective antiplaque agent as it adsorbs to oral surfaces with a slow release and persistent bacteriostatic action lasting in excess of 12 hours. The chemical agent has no systemic toxicity, microbial resistance or superinfection. But local side effects including dental staining and taste perturbation limit its long term use in preventive dentistry **(Addy and Moran 2008).**

Local chemotherapeutic agents include also local antibiotics which can be placed into the periodontal pocket. They have the potential to provide greater concentrations directly to the infected area and reduce possible systemic side effects **(Jolkovsky and Ciancio 2006).**

Systemic medications can be divided into two major categories; antibiotics and agents for host modulation. A number of periodontal benefits have been associated with systemic medications, including PPD reduction, CAL gain, long-term reduction of periodontal pathogens, elimination of invasive pathogens in periodontal tissues and a decrease in the extent of periodontal surgery. Host modulation includes periostat (Doxycycline hyclate), non steroidal anti-inflammatory drugs, hormone replacement therapy, anti-arthritic medications and alendronate (Fosamax). These agents produce their beneficial effects by a variety of mechanisms, including inhibition of matrix metalloproteinases, inhibition of prostaglandin production, stimulation of osteoblasts, inhibition of osteoclasts, and other anti-inflammatory mechanisms of action **(Ciancio 2002).**

Sanz and Teughels (2008) stated that systemic antimicrobials should be used in conjunction with mechanical debridement, preferably as part of non surgical periodontal therapy. On the other hand there is no enough evidence to support the use of systemic antimicrobials with periodontal surgery.

Common antibiotic regimens used to treat periodontal diseases include Amoxicillin, Azithromycin, Ciprofloxacin, Clindamycin, Doxycycline or Minocycline and Metronidazole **(Herrera et al. 2002)**. In a systematic review done by **Haffajee et al. (2003)**, the authors demonstrated that greater improvement in CAL was associated with the adjunctive use of systemic antibiotics and scaling and root planing (SRP). They also showed that most antibiotics resulted in similar effects therefore the selection of an antibiotic must be based on other factors such as patient's medical history and microbiological plaque sampling.

Guerrero et al. (2005) studied the adjunctive benefits of systemic Amoxicillin and Metronidazole in non surgical treatment of generalized aggressive periodontitis. The test treatment resulted in an additional 1.4mm PPD reduction and 1mm of CAL gain than patients who did not receive antibiotic therapy at 6 months. Moreover 74% of sites with baseline PPD $\geq$ 5mm were 4mm or shallower in the test group compared to 54% in the control group.

A combination of antibiotics may be necessary to eliminate all putative pathogens from some periodontal pockets. Metronidazole is effective against anaerobes such as P.gingivalis and P.intermedia. While

Amoxicillin/Augmentin has extended spectrum that includes gram positive and gram negative bacteria. The later has an additive effect regarding suppression of A.a **(Jolkovsky and Ciancio 2006)**.

Matarazzo et al. (2008) compared the effect of SRP alone or combined with metronidazole or with metronidazole plus amoxicillin in treatment of chronic periodontitis. The results showed that subjects receiving metronidazole plus amoxicillin showed the greatest improvement in mean PPD and CAL over SRP alone in initially shallow, moderate and deep sites. Also significant reduction in the mean counts of periodontal pathogens was demonstrated in this group.

Cionca et al. (2009) studied the use of amoxicillin and metronidazole immediately after completion of full mouth periodontal debridement in patients with chronic periodontitis. The authors found that in the test group a significantly lower mean number of persisting pockets > 4mm and bleeding on probing (BOP) that required further treatment.

Similarly **Ribeiro et al. (2009)** evaluated full mouth debridement with and without the adjunctive use of Amoxicillin and Metronidazole in managing severe chronic periodontitis. The authors showed that sites treated with the combined antibiotic therapy demonstrated significantly greater improvement in bleeding on probing (BOP) than control group. Furthermore, the percentage of sites exhibiting CAL gain ≥ 2mm were 58.03%, compared to 43.52% in the test and control group respectively. The test group also showed an additional reduction of 0.83mm in PPD.