COMPARISON BETWEEN THE USE OF COMBINATION OF CHLORHEXIDINE MOUTH WASH AND CHLORHEXIDINE CONTAINING TOOTH PASTE VERSUS CHLORHEXIDINE MOUTH WASH AS A PLAQUE CONTROL AGENT IN PLAQUE INDUCED GINGIVITIS

Thesis

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BY

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﴿قَالُوا سُبْحَانَكَ لا عِلْمَ لَنَا إِلاَّ مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ﴾

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REVIEW OF LITERATURE

Gingivitis is an inflammatory process limited to the mucosal epithelial tissue surrounding the cervical portion of the teeth and the alveolar processes. Plaque-induced gingivitis is a reversible inflammatory reaction of the marginal gingiva to plaque accumulation. In gingivitis, bacteria induced inflammatory lesions are confined to the gingiva. The infection involves the gingival epithelium and the gingival connective tissue underlying the superficial periodontal structure ***(Roques et al.1998; Stephen 2009).***

Gingivitis has been classified by clinical appearance (e.g, ulcerative, hemorrhagic, necrotizing, purulent), etiology (eg, drug-induced, hormonal, nutritional, infectious, plaque-induced), and duration (acute, chronic). The most common type of gingivitis is a chronic form which involves the marginal gingiva and is brought on by the accumulation of microbial plaques in persons with inadequate oral hygiene ***(Stephen 2009).***

The role of dental bacterial plaque in the development of these diseases was established almost 40 years ago ***(Tatakis & Kumar 2005).***

Contributing factors to plaque retention that may lead to gingivitis include anatomic and developmental tooth variations, caries, highly attached frenum, iatrogenic factors, mal-position teeth, mouth breathing, overhanges, partial denture, lack attached gingiva and recession ***(Tatakis & Trombelli 2004).***

Existing evidence indicates that gingivitis precedes the onset of periodontitis; however, not all gingivitis cases develop into periodontitis and the reason for this is that accumulation of plaque bacteria is necessary

but not sufficient by itself for the development of periodontitis: a susceptible host is necessary ***(Tatakis & Kumar 2005).***

The intensity of the clinical signs and symptoms of gingivitis will vary between individuals as well as between sites within a dentition. The common clinical findings of plaque-induced gingivitis include erythema, sponginess of the gingival tissue, bleeding upon probing, sensitivity, change in contour, tenderness, and enlargement. Radiographic analysis and/or probing attachment levels of individuals with plaque-induced gingivitis will not indicate loss of supporting structures ***(American academy of Periodotology 2000; Tatakis & Trombelli 2004; Trombelli et al. 2004).***

The two earliest signs of gingival inflammation preceding establish gingivitis are increased gingival crevicular fluid production rate and bleeding from the gingival sulcus on gentle probing. Bleeding on probing appears earlier than a change in color or other visual signs of inflammation which is evident in the well established papillary bleeding index ***(Fiorellini et al.2004).***

Gingivitis can occur with sudden onset and short duration and can be painful. A less severe phase of this condition can also occur ***(Horiuchi et al.2002).***

Change in color is an important clinical sign of gingival disease. The normal gingival color is coral pink and is produced by the tissues`s vascularity and modified by the overlying epithelial layers. For this reason, the gingiva becomes red when vascularization increases or the degree of epithelial keratinization is reduced or disappears. Chronic gingivitis produce changes in the normal firm and resilient consistancy of the gingiva, both distructive (edematous) and reparative (fibrotic) changes

coxist, and the consistency of the gingiva is determined by their relative predominance ***(Fiorellini et al .2004)***.

Gingivitis is slightly more prevalent in males than in females because females tend to have better oral hygiene; furthermore adults are most commonly affected. The most common complaint is bleeding gingiva that is noticed during toothbrushing, flossing or eating especially foods with a hard consistency ***(Stephen 2009).***

The bacterial etiology of periodontal diseases has been explored for over 100 years, evolving along with technologic advances in identification and characterization. Although early studies indicated that periodontal diseases occurred in response to plaque mass (nonspecific plaque hypothesis), current thinking implicates specific microbial species in disease causation (specific plaque hypothesis). This bacterial etiology of periodontal disease is strongly supported by clinical studies that have reported that mechanical and chemical antibacterial treatment can prevent or treat gingivitis and periodontitis ***(Loesche & Grossman 2001).***

Dental plaque

In the context of the oral cavity, the bacterial deposits have been termed dental plaque or bacterial plaque**.** A biofilm of dental plaque is defined as single cells and micro colonies enclosed in a highly hydrated, predominantly an ionic exopolymer matrix. These sessile cells behave in profoundly different ways from their free-floating counterparts ***(Tatakis et al. 2005).***

A biofilm describes the relatively undefinable microbial community associated with a tooth surface or any other hard, non-

shedding material. It is an aggregate of microorganisms in which cells are stuck to each other and multiply on surfaces. These adherent cells are frequently embedded within a self-produced matrix of extracellular polymeric substance (EPS). There is multiplication of the adhering bacteria and sequential adsorption of further bacteria to form a more complex and mature biofilm ***(Marsh1992).*** Biofilm EPS, which is also referred to as "slime," is a polymeric jumble of DNA, proteins and polysaccharides. Biofilms may form on living or non-living surfaces, and represent a prevalent mode of microbial life in natural, industrial and hospital settings ***(Wilderer & Charaklis 1989; Hall-Stoodley et al. 2004).***

The interactions among bacterial species living in biofilms take place at several levels including physical contact, metabolic exchange, small signal molecule mediated communication, and exchange of genetic information *(**Kolenbrander 2005**)*.

The primary colonizers form a biofilm by autoaggregation (attraction between same species) and coaggregation (attraction between different species). Coaggregation results in a functional organization of plaque bacteria and formation of different morphologic structures such as corncobs and rosettes. The microenvironment now changes from aerobic/capnophilic to facultative anaerobic. The attached bacteria multiply and secrete an extracellular matrix, which results in a mature mixed-population biofilm. Transmission occurs from other sites, leading to incorporation of new members into the biofilm and the formation of a climax community ***(Kolenbrander et al. 2002).***

A major advantage is the protection that the biofilm provides to colonizing species from competing microorganisms, from environmental factors such as host defense mechanisms, and from potentially toxic

substances in the environment, such as lethal chemicals or antibiotics. Biofilms also can facilitate processing and uptake of nutrients, cross-feeding (one species providing nutrients for another), removal of potentially harmful metabolic products (often by utilization by other bacteria), as well as the development of an appropriate physicochemical environment (e.g. a properly reduced oxidation reduction potential) ***(Roques et al.1998).***

The bulk of the biofilm consists of the matrix or glycocalyx and is composed predominantly of water and aqueous solutes. The "dry" material is a mixture of exopolysaccharides (EPS), proteins, salts, and cell material. The EPS, which are produced by the bacteria in the biofilm, are the major components of the biofilm making up 50–95% of the dry weight. They play a major role in maintaining the integrity of the biofilm as well as preventing desiccation and attack by harmful agents. In addition, they may also bind essential nutrients such as cations to create a local nutritionally rich environment favoring specific microorganisms. The EPS matrix could also act as a buffer and assist in the retention of extracellular enzymes (and their substrates) enhancing substrate utilization by bacterial cells. The EPS can be degraded and utilized by bacteria within the biofilm. One distinguishing feature of oral biofilms is that many of the microorganisms can both synthesize and degrade the EPS ***(Tatakis & Kumar 2005).***

The biofilm provides a protected environment against antimicrobial agents. The biofilm acts as a barrier to diffusion due to the presence of neutralizing enzymes (b-lactamase, IgA protease) and a diffusion-resistant matrix ***(Stewart 2003).***

Clearly, the amount and nature of the plaque are directly influenced by the status of the periodontal tissues, which can be either healthy or diseased, a difference that determines the degree of severity of the gingivitis and bone loss associated with periodontal disease ***(Roques et al.1998).***

Plaque formation

The formation of plaque on a tooth surface is a dynamic and ordered process, commencing with the attachment of primary plaque-forming bacteria. The attachment of these organisms appears essential for initiating the sequence of attachment of other organisms such that, with time, the mass and complexity of the plaque increases ***(Marsh& Devine 2011).***

Research on plaque development has shown that oral bacteria colonize non shedding hard surfaces and shedding soft tissue surfaces. The physical and morphologic characteristics of these surfaces create different ecosystems or niches with distinct bacterial profiles. These niches are the result of a dynamic equilibrium that exists between the adhesion forces of microorganisms and swallowing and mastication forces, salivary and crevicular flow, and oral hygiene measures ***(Tatakis & Kumar 2005).***

Plaque formation follows several distinct phases; beginning with adsorption onto the tooth surface of a conditioning film derived from bacterial and host molecules that form immediately following tooth eruption or tooth cleaning. This adsorption is followed by passive transport of bacteria mediated by weak, long-range forces of attraction.

Covalent and hydrogen bonds create strong, short-range forces that result in irreversible attachment ***(Tatakis & Kumar 2005).***

In the mouth, teeth provide hard, non-shedding surfaces for the development of extensive bacterial deposits. The accumulation and metabolism of bacteria on hard oral surfaces is considered the primary cause of dental caries, gingivitis, periodontitis, peri-implant infections, and stomatitis**.** In 1 mm^3 of dental plaque weighing approximately 1 mg, more than 10^8 bacteria are present. Although over 300 species have been isolated and characterized in these deposits, it is still not possible to identify all the species present ***(Loesche & Grossman 2001).***

Plaque first develops in irregularities on the tooth surface and at the gingival margin, once established, the speed of plaque formation increases over time. Nevertheless, even in the absence of oral hygiene, no more than 1/4 of the tooth surface is covered after 96 hours of undisturbed plaque growth ***(Marsh& Devine 2011).***

Primary colonization is dominated by facultative anaerobic Gram-positive cocci. They adsorb onto the pellicle-coated surfaces within a short time after mechanical cleaning. Plaque collected after 24 hours consists mainly of streptococci; S. sanguis is the most prominent of these organisms. In the next phase, Gram-positive rods, which are present in very low numbers initially, gradually increase and eventually outnumber the streptococci. Gram-positive filaments, particularly Actinomyces spp., are the predominating species in this stage of plaque development, Surface receptors on the deposited Gram-positive cocci and rods allow subsequent adherence of Gram-negative organisms with poor ability to attach directly to pellicle. Veillonella, fusobacteria, and other anaerobic Gram-negative bacteria can attach in this way. The heterogeneity of

plaque thus gradually increases and, with time, includes large numbers of Gram-negative organisms ***(Stewart 2003).***

Apparently the adherence of microorganisms to solid surfaces takes place in two steps: a reversible state in which the bacteria adhere loosely, and later an irreversible state, during which their adherence becomes consolidated. Results from several studies indicate that plaque formed on natural or artificial surfaces does not differ significantly in structure or microbiology ***(Gibbons & van Houte 1980).***

Plaque control

Removal of dental plaque is recognized as advantageous in the maintenance of gingival health and prevention of periodontal disease ***(Biesbrock et al. 2007).***

Any method of plaque control, which prevents plaque achieving the critical point where gingival health deteriorates, will stop gingivitis. Unfortunately, the lack of knowledge of bacterial specificity for gingivitis does not allow targeting or the control of particular organisms except for perhaps the primary plaque formers. Plaque inhibition has, therefore, targeted plaque formation at particular points, bacterial attachment, bacterial proliferation, and plaque maturation ***(John & Moran 2008).***

The mainstay of primary and secondary prevention of periodontal diseases is the control of supragingival plaque ***(Hancock 1996).*** Primary prevention involves preventing inception of disease and includes the concept of health promotion and protection strategies. These health promotion strategies, aimed at enabling groups or individuals to control and improve their health, include providing oral hygiene education and protection strategies such as fluoridation. In developed nations, dentistry

has been successful in these primary prevention areas. This success is illustrated by improvements in attitudes toward the importance of oral hygiene and the provision of fluoridated water supplies. Secondary disease prevention aims to limit the impact of disease by way of early diagnosis and treatment, thereby stopping disease progression in its earliest stages ***(Dentino et al. 2005).***

Failure to perform oral hygiene adequately at the gingival margin results in the formation of a pathogenic plaque that has the potential to initiate gingivitis and, in some individuals and at some sites, to progress to periodontitis ***(John & Moran 2008).***

Agents that could inhibit the development or maturation of supragingival plaque have been classified according to possible mechanisms of action as anti-adhesive, antimicrobial, plaque removal and anti pathogenic ***(Addy et al. 1997).***

Mechanical Plaque Control

Personal and professional mechanical oral hygiene measures to prevent the formation of plaque plays the key role in dental hygiene and remain the most reliable method of controlling supragingival bacterial plaque ***(Amith 2007; Therwil 2009).***

Successful primary or secondary prevention is based on two major factors, the first being proper, thorough treatment during active therapy and the second being patient compliance with daily plaque removal and regular professional supportive care ***(Dentino et al .2005).***

Mechanical cleaning aims to regularly remove sufficient microorganisms to leave a "healthy plaque" present, which cannot induce gingival inflammation. It could be argued that the heavy reliance on mechanical methods to prevent what are microbially associated diseases is outdated. Very few hygiene practices against microorganisms used by humans on themselves, in the home, at the workplace or in the environment rely on mechanical methods alone and some methods are only chemical ***(Papapanou 1994).***

Improved mechanical plaque removal may be achieved through the use of improved toothbrushes and/or flossing ***(Cugini & Warren 2006).***

Toothbrush

The manual toothbrush as known today, man-made filaments in a plastic head was invented as as the 1930s. Evidence for such devices dates back to China approximately 1000 years ago, re-emerging in the 1800s in Europe, but too expensive for common usage ***(Fischman 1997).***

Mechanical tooth cleaning through tooth brushing with toothpaste is the most common and potentially effective form of oral hygiene practiced in developed countries. Tooth brushes can be manual or electric and vary in size, shape, hardness and arrangement of bristle**s** ***(Jepsen 1998)***.

Tooth brushing causes an increased turnover rate and desquamation of the junctional epithelial surfaces. This process may repair small breaks in the junctional epithelium and prevent direct access the underlying tissue by periodontal pathogen ***(Tomofuji et al. 2002).***