EFFICACY OF CHLOROHEXIDINE VARNISH AND POVIDONE IODINE IN THE PREVENTION OF EARLY CHILDHOOD CARIES

Thesis

Submitted to the Faculty of Dentistry,
Ain Shams University

In partial fulfillment of the requirements for the
Master Degree
(Pediatric Dentistry)

By

Gehan Gaber Ibrahim Allam
Ex. Resident in Pediatric Dentistry Department
Faculty of Dentistry
Ain Shams University

Faculty of Dentistry
Ain Shams University
2008

Supervisors

Dr Nadia Ezz El-Din Metwalli
Professor of Pediatric Dentistry
Faculty of Dentistry
Ain Shams University

Dr Noha Samir Kabil

Lecturer of Pediatric Dentistry
Faculty of Dentistry
Ain Shams University

INTRODUCTION

Early childhood caries (ECC) is a particularly virulent variant of human dental disease that may devastates primary dentition of infants, toddlers and young preschool children. Bacteriologic studies indicate that ECC is characterized by dense infection of Streptococcus mutans on dental surfaces[1].

The American Academy of Pediatric Dentistry defines ECC as "the presence of one or more decayed (cavitated or non cavitated lesions), missing (due to caries) or filled tooth surfaces in any primary tooth in a child 71 months of age or younger". The Academy also specifies that in children younger than 3 years of age, any sign of smooth-surface caries is indicative of severe ECC[2].

Studies showed that a new born has a sterile mouth at birth, which contains only mucosal surfaces exposed to salivary fluid flow and is colonized with bacteria within one or two days of life[3].

To date, preventive efforts largely have focused on counseling parents about cariogenic

infant feeding practices. However this approach has had only limited success[4,5].

Preventive measures usually involve a combination of dietary counsels, oral hygiene measures and fluoride applications[6]. These interventions do not specifically target streptococcus mutants, which are the chief pathogen responsible for caries[7].

Chlorohexidine has a significant property, which is its bactericidal-bacteriostatic action on pathogenic bacteria, Moreover, its substantivity made it feasible to use it in low concentration and fewer numbers of rinses per day, those effects are mostly achieved on bacterial plaque interfering with its formation and reduce bacterial colonization on tooth surfaces[8,9,10].

Another approach to prevent Streptococcus mutans from accumulating to pathologic levels is through the topical application of antimicrobial agents, such as iodine solution; a preparation which has been shown to exert a marked effect on the population of Streptococcus mutans in humans[11].

REVIEW OF LITERATURE

Dental Caries:

Dental caries may be defined as a bacterial disease of the calcified tissues of the teeth characterized by demineralization of the inorganic and destruction of the organic substance of the tooth. It is a complex and dynamic process involving physicochemical process associated with the movement of ions across the interphase between the tooth and external environment, as well as biological process associated with the interaction of bacteria in dental plaque with host defense mechanisms[12].

Etiology of Dental Caries:

The development of a clinical carious lesion involves a complicated interplay among a number of factors in the oral environment and the dental hard tissues[13].

Basically, four essential factors must all be present in order for enamel caries to exist. (1) Bacteria, (2) Sugars, (3) Susceptible dental hard tissues, and (4) Time. If any of the four are inhibited or restricted, the caries process will be prevented or arrested[14].

An oversimplified but essentially accurate concept of the etiology and pathogenesis of dental caries has existed for a century and has come to be known as the chemicoparasitic or acidogenic theory. This holds that bacteria present in the mouth interact with retained food particles to produce substances capable of dissolving enamel[15].

Acquisition of Early Childhood Caries

ECC initially presents as smooth surface carious lesions affecting the primary maxillary incisors which then spread to other primary teeth resulting in the eventual destruction of the primary dentition[3,16].

Early Childhood Caries involves three factors:

1. A susceptible tooth: Some variables make teeth more vulnerable to decay. Genetics may influence the pitting and grooving of teeth, alter the tooth enamel, and affect the pH level (degree of acidity) of the saliva. Factors during the mother's pregnancy such as fever or other illness, malnutrition, iron deficiency, lead exposure, stress, or antibiotic use may cause developmental defects in the baby's tooth enamel, known as hypoplasia[17,18,19].

There is some evidence that babies born by cesarean section[20], born preterm or small for gestational age[21], or who are children of smokers[22], are also at increased risk of dental caries.

2. The presence of cariogenic bacteria: the earlier that a child's mouth is infected with Sm, the greater the risk for future caries development.

3. The presence of acid. The normal, neutral pH in the mouth is 6.4 to 7.0 tending toward acidity. The lower (or more acidic) the pH becomes, the more likely it is that dental caries will develop. Sm is able to break down sugar to use it for energy, forming an acidic environment and leading to a process of demineralization and etching of the teeth[18]. The more frequently sugars or fermentable carbohydrates are present in the mouth, the more often the acid-forming cycle occurs, with potential for harm to the teeth. Babies who suck from bottles containing anything other than water throughout the day or night, and toddlers who carry around sippy cups of juice, milk, or other beverages are continually

creating acidic conditions ripe for causing decay[23,24].

Saliva is the first line of defense. It acts protectively to wash away food debris and bacteria and serves as a buffer to acid formation. Saliva may also have a bacteriostatic effect, inhibiting the growth of bacteria without destruction of the bacteria. It further contains calcium and phosphate, which are needed in the remineralization of tooth enamel. Iron deficiency or prenatal exposure to lead can impair salivary gland function and put the child at greater risk for caries[18]. If the oral pH falls below 5.5, saliva is no longer able to counter acid effects, and demineralization of the teeth begins to occur[25].

Conditions such as mouth breathing or the use of medications like antihistamines or drugs for asthma reduce salivary flow which interferes with the buffering effect. Salivary flow is also lessened during sleep, when the salivary glands do not secrete spontaneously[16]. Frequent and or prolonged night feedings may thus become a risk factor for susceptible children.

Certain foods or beverages may contribute to an acidic environment. Carbonated beverages are

especially high in acid as well as sugar. One can of soda contains the equivalent of as much as 11 or 12 teaspoons of sugar. Even carbonated diet drinks that have no sugar content still contain acid and therefore are harmful to the teeth [26].

Pacifiers that have been dipped in sweet solutions, juices, or other sweetened drinks, and liquid medications in sugar syrups are other precursors to acid production. Most of pediatric medications contain sucrose, some as much as 80 percent of their volume. Even bread and cereal that don't appear to be sweet contains fermentable carbohydrates that can be converted to acid, and the digestive enzymes present in saliva can convert these foods into sugar directly within the mouth[16,27].

Simply ECC is associated with prolonged and frequent consumption of cariogenic substrates at bedtime and or naptime and nursing bottle feeding beyond 12 months of age[28].

Consequences of Early Childhood Caries

Decayed teeth can be very painful and abscesses can develop. Some children who develop ECC learn to live with chronic pain and don't know what it is like to live pain-free. The ability to chew and eat is compromised.

Children with ECC show slowed growth rates and may be diagnosed with failure-to-thrive[29]. Speech can also be affected, if primary molars are lost due to decay, malocclusion may occur. Emotional health may be compromised, children may smile less often and their self-esteem may be affected by others' reactions to their browned, decayed, or missing teeth, or a mouth full of silver caps. Dental repair is an arduous and expensive prospect for young children, often requiring hospitalization, general anesthesia, and sometimes intravenous antibiotics to treat, repair, or extract the affected teeth[30].

Ignoring the situation in hopes that secondary teeth will come unaffected is not a good option since having ECC is a strong predictor of decay in permanent teeth[30].

Evidence of the Role of Microorganisms in Caries Process

Until 1954, there was no conclusive evidence that bacteria were essential for the initiation of caries. Orland et al. demonstrated that germ-free rats of a caries susceptible line failed to develop caries even when fed a cariogenic, high-sucrose diet. By implanting bacteria into the mouth of these germ-free animals, caries was produced[15].

Cariogenic Bacteria:

The cariogenic bacteria demonstrate certain properties that account for their participation in the process[14]. Many of these organisms are acidogenic (acid producers), aciduric (thrive in acid environments), and readily produce the sticky polysaccharide dextran. These properties promote sticking of bacteria to the tooth surfaces, sticking of cariogenic bacteria to each other (quantity selection), growth of other aciduric organisms (quality selection), and acid dissolution of enamel (early caries)[7].

These organisms also tend to produce acids that continue to dissolve the calcium hydroxyapatite of dentin. In addition, these organisms produce to some extent proteolytic enzymes and chelating agents. These by products promote destruction of the protein matrix and

dissolution of calcium from the dentin, therefore facilating the spread of the caries process[31,32].

There is abundant support for the so-called specific plaque hypothesis, introduced by Loesche (1986)[13], which proposes that some specific species of the plaque flora are regarded as major pathogens in the etiology of dental caries. Included in the major pathogens, groups of bacteria associated with caries in humans which are also able to induce carious lesions in experimental animals. The most important are the Sm. The second genus closely associated with caries is lactobacillus.

Streptococcus mutans: (Sm)

Sm is a normal inhabitant of the oral cavity and is isolated from most of caries free individuals. However, accumulation of this organism to pathological levels accompanied by frequent and prolonged exposure to cariogenic substrates, results in dental caries[33]. Sm exhibits a strong positive correlation with caries development. A statistically significant correlation between salivary Sm counts and caries indices was frequently reported[34,35,36].

Sm had been implicated as the principal bacterial component responsible for the initiation and progression of dental caries[37]. In humans Sm is the principal micro-organism related to coronal caries[7], and was found to be among the first microorganisms to colonize infants shortly after their teeth erupt[38].

In 1924, Clarke isolated such organisms from human carious lesions and called them Sm because on Gram stain they were more oval than round and thus appeared to be a mutant form of a streptococcus[7].

They are chain-forming gram positive cocci that are facultative anaerobes, non motile and non spore forming[39]. It is one of the streptococcus viridans which grow well on the blood agar. They are alpha hemolytic and cause green coloration around their colonies on the blood agar[40]. They are well seen on Mitis Salivarius agar because it contains selective supplements for Streptococci but Mitis Sucrose Bacitracin allows the growth of Sm only and inhibits the growth of all microorganisms that grow on Mitis Salivarius agar[13].

Factors that may contribute to the virulence of Sm include: acidogenicity, aciduricity,

synthesis of extra polysaccharides that enhance adhesion to the tooth and increase the bulk of plaque, synthesis of intra-cellular polysaccharides which allows acid production when sucrose is absent in diet, moreover, they posses surface proteins called adhesions that bind to salivary glycoprotein on tooth surfaces which is essential to provide resistance to the flow of saliva[7,31,32]. Collectively, these data indicate that Sm plays a major role in the initiation as well as progression of carious lesions.

Previous studies demonstrated that Sm has a feeble capacity to become attached to epithelial surfaces, therefore it seemed unlikely that these organisms could colonize the mouth of a normal infant before the eruption of teeth[41]. However, more recent clinical studies have demonstrated that Sm can colonize the mouth of predentate infants; the furrows of the tongue appear to be an important ecological niche[42,43,44]. Tanner and others[45], using DNA probe technology, reported that Sm was present in 55% of plaque samples and 70% of tongue scraping samples of 57 children 6-18 months of age living in Saipan, Common wealth of the Northern Mariana islands, western Pacific. These recent studies on acquisition of Sm raise a

doubt that a non shedding oral surface is required for colonization[45].

The fact that caries is a transmissible disease involving the cariogenic Sm bacteria has been well established[46].The major reservoir from which babies acquire streptococcus mutants is through vertical transmission from their mothers. The evidence for this concept comes from several clinical studies in which Sm strains isolated from mothers and their babies exhibited similar or identical bateriocin profiles[47] and identical plasmid or chromosomal DNA patterns[48].

Although most infants infected by Sm have mothers with dense reservoirs of these organisms, a few infected infants have mothers with negligible levels of infection[46].

In addition, mothers with dense levels of Sm who are at high risk for infecting their infants are usually in need of extensive and costly dental treatment. On this basis, antimicrobial strategies targeted at the infant may be more successful and cost effective than those directed at the infant's mother[49].

Recent reports indicated that vertical transmission is not the only vector by which Sm