Early childhood caries: etiology, clinical considerations, consequences and management

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Abstract

Early childhood caries (ECC) is a diet-induced disease characterized by early onset and rapid progression. It results in functional, esthetic and psychological disturbances of the child, accompanied by great concern from the parents and the dentist. The late consequences may continue long after its initial treatment as malnutrition, low self esteem, decay and malocclusion in permanent dentition.

Preventive measures cannot, and will not, work unless parents and caregivers follow and adhere to the preventive methods being prescribed. Dentists, other health professionals, and the public community must also recognize that ECC is not simply an individual problem. Rather, it takes a collaborative effort to make the necessary improvements for these young children to be able to receive the proper dental care that they deserve.

Keywords: Early childhood caries; etiology; risk factors; clinical considerations; consequences; management

Introduction

Dental caries affects humans of all ages throughout the world and remains the major dental health problem among school children globally.[1] It is a disease that can never be eradicated because of the complex interaction of cultural, social, behavioral, nutritional, and biological risk factors that are associated with its initiation and progression.[2]

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Caries is plaque-induced acid demineralization of enamel or dentin. With time, the interaction of cariogenic microorganisms and fermentable carbohydrates may induce demineralization of tooth structure.[3]

Childhood and early adolescence are crucial periods in the development of healthy dentition. Early childhood caries (ECC) is a major public health problem, being the most common chronic infectious childhood disease, which is difficult to control. While not life-threatening, its impact on individuals and communities is considerable, resulting in pain, impairment of function, deleterious influence on the child's growth rate, body weight, and ability to thrive, thus reducing quality of life.[4]

To prevent further tooth destruction and encourage better overall health, treatment should be instituted immediately and specifically. This aggressive approach includes atraumatic restorative treatment (ART), fluoride applications, oral hygiene instruction, dietary counseling, and restorative measures. To achieve these interventions, advanced behavioral management is an important part of the treatment plan, as even with these proactive measures, children with ECC are likely to develop recurrent caries.

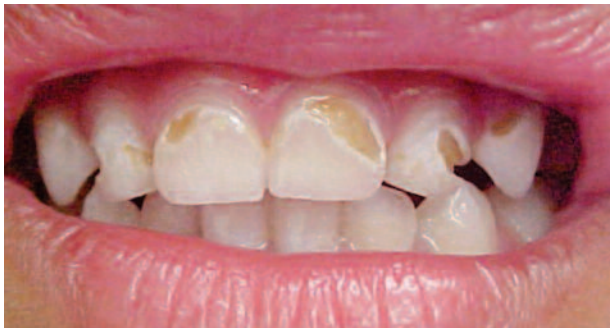

Figure 1: Initial stages of ECC- the lesion can be arrested by the application of fluoride and improved OH habits.

Definition

ECC in pre-school children has been discussed extensively in the scientific literature over the past 50 years. Caries in infants and young children has long been recognized as a clinical syndrome, described by Belterami in 1930s as "Les dents noire de tout-petits" which means "black teeth of the very young."[5] Fass is perhaps the most preeminent in this perspective for defining the term "Nursing bottle mouth."[6] Subsequently, other terms such as "baby bottle tooth decay", "nursing bottle syndrome", "bottle mouth caries", "nursing caries", "rampant caries", "nursing bottle mouth", "milk bottle syndrome", "breast milk tooth decay" and "facio-lingual pattern of decay" have also been used to describe this condition.[7,8]

An enormous diversity of definition and diagnosis of ECC is used worldwide. However, there is currently no universally accepted definition for the term ECC. [9,10] Some researchers defined ECC as the caries on primary maxillary incisors (the number of maxillary incisors ranges from 1 to 4 teeth according to this definition).[7,10,11] Carino et al.,[12] defined ECC as the presence of any decayed, missing, and filled (dmf) teeth, regardless of being anterior or posterior. These definitions all focus on dentinal caries.

A workshop, convened by the National Institutes of Health (NIH) proposed that the term Early Childhood Caries (ECC) should be used to describe the presence of one or more decayed (noncavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces on any primary tooth in children up to 71 months of age.[13, 14] This definition was adopted by the AAPD[15] and, subsequently, by several other researchers. [16,17,18]

Furthermore, the expression severe ECC (S-ECC) was adopted in lieu of rampant caries, in the presence of at least one of the following criteria:

- Any sign of caries on a smooth surface in children younger than three years.
- Any smooth surface of an antero-posterior deciduous tooth that is decayed, missing (due to caries) or filled, in children between three and five years old.
- Decayed, missing, and filled teeth index (dmft) equal to or greater than 4 at the age of 3,5 at the age of 4 and 6 at the age of 5 years.[19]

Etiology

Dental caries is a multifactorial disease that starts with microbiological shifts within the complex biofilm and is affected by salivary flow and composition, exposure to fluoride, consumption of dietary sugars, and by preventive behaviors. The disease is initially reversible and can be halted at any stage, even when some cavitation occurs, provided enough biofilm can be removed.[20]

ECC is an aggressive form of dental caries that begins on tooth surfaces which are usually not affected by decay, such as labial surfaces of maxillary incisors, in contrast to dental caries which usually involves plaque retentive areas.[21] Thus it is thought that there may be distinctive risk factors involved in the progression of ECC.

ECC was historically attributed to inappropriate and prolonged bottle use or breastfeeding. The use of the bottle, especially at bedtime, is believed to be associated with increased risk for caries, but this might not be the only factor in caries development in early childhood. Carious lesions are produced from the interaction of cariogenic microorganisms, fermentable carbohydrates, and susceptible tooth surface. Given the proper time, these factors induce incipient carious lesions.[22] The associated risk factors have also been found to vary from population to population.[23]

Primary risk factors

Substrate

There is sufficient evidence that sugars (such as sucrose, fructose and glucose) and other fermentable carbohydrates play a vital role in the initiation and progression of dental caries. [24,25] Sucrose is the most significant cariogenic food as it converts non-cariogenic/anticariogenic foods to cariogenic. Sucrose promotes an increase in the proportions of *mutans streptococci* and *lactobacilli* and, simultaneously decreases the levels of *S. sanguinis.* Oral clearance of carbohydrates is lowest during sleep, when salivary flow decreases and the contact between plaque and substrates increases, favoring the growth of cariogenic species. A healthy biofilm is thereby converted into a diseased one, consequently enhancing demineralization.

Susceptible tooth/host

Several factors can predispose a particular tooth to dental caries. Host risk factors for the development of caries are reduced saliva, immunological factors, presence of enamel defects, characterized mainly by hypoplasia, immature enamel, tooth morphology and genetic characteristics of the tooth (size, surface, depth of fossae and fissures) and crowded/malaligned teeth.[26, 27]

Saliva is the major defense system of the host against caries. It removes foods and bacteria, and provides a buffering action against the acids produced. It also functions as a mineral reservoir for calcium and phosphate necessary for enamel remineralization. During sleep, the decrease in salivary flow rate reduces its buffering capacity, consequently making tooth susceptible to caries. [26, 27]

Because enamel is immunologically inactive, the main immune defence against *S mutans* is provided largely by salivary secretory immunoglobulin A (IgA) or serum and gingival crevicular fluid. As children become contaminated with oral microorganisms, they develop salivary IgA antibodies.[26] After eruption, the newly exposed enamel surfaces undergo the final stages of post-eruptive maturation and hardening by incorporating orally available ions, including fluoride. The tooth is most susceptible to caries in the period immediately after eruption and prior to final maturation. [28]

Cariogenic microorganisms

Evidence suggests that the first step in the progression of ECC in children is through the acquisition of cariogenic microorganisms. The main cariogenic microorganisms are streptococci, *(mutans streptococci (MS), sobrinus) and lactobacillus*. These pathogens can colonize the tooth surface. When combined with products that contain fermentable carbohydrates, the process of metabolism is initiated by the bacteria, producing acidic end products that ultimately lead to the demineralization of tooth enamel, thus contributing to dental caries.[14,29]

The primary caregiver of the infant, usually the mother, has been shown to harbour the reservoir of MS. Saliva is the vehicle by which the transfer occurs. The exact method of transmission is unknown, but it is suspected to be due to the close contact of mother and child and the sharing of food and eating utensils.[26,30] The AAPD reported that infants and toddlers whose mothers have high levels of MS are at elevated risk for acquiring the organism than children whose mothers have low levels.[31]

Dental Plaque

The presence of visible plaque and its early accumulation have been related to caries occurrence among children.[32,33] Alaluusua and Malmivirta[34] found that 91% of the children studied were correctly classified into caries risk groups, based solely on the presence or absence of visible plaque. Bacteria in the biofilm are always metabolically active, causing fluctuations in the salivary pH. The fermentation of carbohydrates by cariogenic plaque bacteria produce organic acids, which act on a susceptible tooth and result in caries.[26]

Associated risk factors

Bottle feeding

Bottle-feeding, especially nocturnal feeding or, particularly, when children are allowed to sleep with a bottle in their mouth, has been considered cariogenic.[21,35, 36] Du and co-workers[37] found that children who had been bottle-fed had a five times greater risk of having ECC compared to children who were breast-fed. Milk-based formulas for infant feeding, even those without sucrose in their formulation, also proved cariogenic in some studies. However, prolonged milk bottle feeding at night is not the sole cause of ECC.

The use of baby bottles during the night is associated with the reduction in salivary flow, thus decreasing salivary neutralization capacity, which would cause food stagnation in the teeth and prolonged exposure to fermentable carbohydrates.[26] The lower incisors are close to the main salivary glands and are protected from liquid contents by the bottle nipple and the tongue.[21]

Fruit juices and carbonated beverages have also been implicated in children diagnosed with ECC. Fruit juices naturally contain sugar (fructose) and are intrinsically acidic. Carbonated beverages may have a sugar sweetening agent (frequently fructose) and an acidic pH. Both fruit juices and

carbonated beverages lead to a significant decrease in plaque pH thus initiating the process.[37]

Breastfeeding

Breast-feeding has many advantages: it provides optimal infant nutrition, immunological protection and minimizes the economic impact to the family. Despite good practice, there is conflicting evidence regarding breast-feeding in terms of dental health. Prolonged breast-feeding apparently carries a risk of developing dental caries or nursing caries.[38]

Along with the positive health effects of breastfeeding, several epidemiological studies have associated breastfeeding with lower levels of dental caries. The World Health Organisation (WHO) has therefore recommended that children be breastfed until 24 months of age.[1] On the other hand, some reports suggest prolonged exposure of teeth to daytime or nighttime breastfeeding as risk factors for ECC.[21,36] Based on these reports, ADA recommended weaning from the breast soon after the child's first birthday.[39]

The relationship between breast feeding and ECC could not be established in many studies. The period of breast feeding is also controversial. Breastfeeding has been assumed to be associated with ECC when the consumption pattern was ad libitum, and when there is frequent and prolonged breastfeeding, specifically nocturnal.[9,40] Weerheijm and co-workers[41] stated that there is no right time to wean the baby from breast feeding, as long as preventive measures, such as tooth brushing with fluoridated toothpaste and reducing the frequency of feedings, are incorporated.

Oral hygiene

It is generally accepted that the presence of dental plaque is a high risk factor for developing caries in young children.[33,34] Some studies have reported that a child's brushing habit, frequency of brushing, and/or use of fluoride toothpaste are associated with the occurrence and development of dental caries.[42,43] It was found that children who did not have their teeth cleaned at bedtime had a higher risk of developing ECC.[44] As young children lack the ability to clean their own teeth effectively, parents are recommended to clean their children's teeth at least until they reach school age.[40]

Fluoride

The constant maintenance of fluoride in the oral cavity is important for enamel resistance, reducing the amount of minerals lost during demineralization and accelerating remineralization.[21] A number of studies have shown that five year old children living in a fluoridated area have approximately 50% less caries than those in a non-fluoridated area.[45,46] Regular tooth-brushing with fluoridated toothpaste and brushing before going to bed are important measures for the control of caries, since they maintain the concentration of fluoride in the saliva for a longer period.

Education of the parents

The education level of parents has been shown to be correlated with the occurrence and severity of ECC in their children.[47,48] Lower prevalence of dental caries and lower mean dmft scores have been associated with higher levels of parental education.[47] Higher rates of caries have been reported in families, being passed from generation to generation, and children whose parents and/or siblings have carious lesions are at a higher risk for ECC.[31,35] Milgrom[28] showed that the mother is not only the reservoir of cariogenic bacteria, but that her dental knowledge, behavior, as well as the general care of her child are also some of the factors that contribute to caries risk.

Socioeconomic factors

There is evidence of powerful links between the individual's socio-economic experience for disadvantaged children and adverse health events.[37,49] Children born into low-income families are more likely to have low birth weight which impacts on oral health. They also have more difficulty in school and poor oral health can increase school absence. Socio-economic status can further influence health literacy, which, in turn, affects general health.

ECC is more common in children from single parent families and those with parents of low educational level, especially of illiterate mothers.[50] Tsai and co-workers[17] found that, in Taiwan mothers who had full-time jobs were more likely to have children with ECC than those who had part-time jobs or were housewives. Children from low income families tend to make their first visit to the dentist at an older age, less frequently and only when there are established dental problems. In addition, deprived children usually start brushing their teeth later in life and do so less frequently.[51]

Clinical considerations

ECC begins to affect teeth upon eruption and is associated with a characteristic clinical presentation. The initial lesions appear as 'white spots' on the facial tooth surface of maxillary incisors adjacent to the gingival margin, spreading later to the maxillary molars, mandibular molars and, in rare

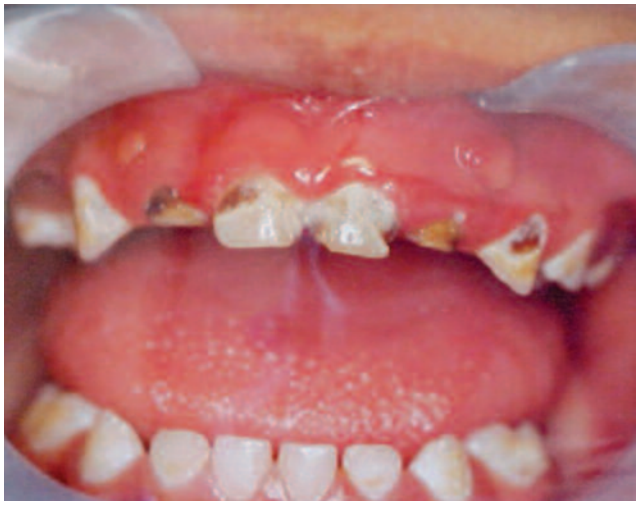

Figure 2: Advanced stage of ECC - requiring restorative treatment or extraction.

cases, the mandibular incisors. The demineralized lesions may become frank lesions or caries within 6-12 months, causing cavities discolored by yellow, brown, and even black stains.[35]

The teeth are affected in the way they erupt.[52] The explanation for this pattern of caries distribution is based on the pooling of milk or sweetened liquid from the nursing bottle around the maxillary incisors and other teeth, while the mandibular incisors are physically protected by the tongue.[53] Clinically, ECC presents as one of the following:[54]

Type I (mild to moderate) ECC

The existence of isolated carious lesion(s) involving molars and /or incisors. The cause is usually a combination of cariogenic semi-solid or solid food and lack of oral hygiene. The number of affected teeth usually increases as the cariogenic challenge persists. This type of ECC is usually found in children who are 2 to 5 years old.

Type II (moderate to severe) ECC

Labiolingual carious lesions affecting maxillary incisors, with or without molar caries depending on the age of the child and stage of the disease, and unaffected mandibular incisors. The cause is associated with inappropriate use of a feeding bottle, at will breast feeding or a combination of both, with or without poor oral hygiene. Poor oral hygiene most probably compounds the cariogenic challenge. This type of ECC could be found soon after the first teeth erupt. Unless controlled, it may proceed to become type III ECC.

Type III (severe) ECC

Carious lesions affecting almost all teeth including lower incisors. This condition is found between the age of 3 to 5 years. The condition is rampant and generally involves tooth surface/s that are unaffected by caries e.g. mandibular incisors.

Consequences of early childhood caries

ECC is not self-limiting. If treatment for ECC is delayed, the child's condition worsens and becomes more difficult to treat, increasing the cost of treatment. The most common immediate consequence of untreated dental caries is dental pain, which affects children's regular activities, such as eating, talking, sleeping, and playing. Children who had caries of primary dentition early in the life are at greater risk of developing additional carious lesions in their primary and permanent dentition. [14]

Severe ECC can lead to the loss of the child's front teeth at an early age. The child may suffer further developmental setbacks involving speech articulation as these years are critical for speech development. Children with ECC can also experience delays in physical development, especially in height and weight. The pain caused by ECC may lead to a decrease in appetite, ultimately resulting in malnutrition. In fact, early extraction or loss of teeth often results in children suffering from psychological trauma from dental procedures required to restore their teeth. Taunting by siblings, peers, and even extended family members may lead to poor self-esteem.[55]

Management of ECC

ECC is an alarming problem because the disease is widespread amongst young children and, if untreated, can lead to serious disability and, in extreme cases, death.[56] A

survey conducted by van Wyk and van Wyk [57] found that 80% of the caries in South African children (4-5 year olds) went untreated. Grobler[58] found that the fluoride levels in all nine provinces of South Africa are very low (<0.1 ppm), with the Western Cape Province having the largest number of municipalities with these low fluoride levels. The management of ECC in South Africa is a serious problem. Science-based standards, guidelines, and protocols for effective clinical, behavioral, and nutritional approaches forassessing individual risk, prevention and management are now needed.

Prevention

ECC is a preventable disease. The physical, psychological, and economic consequences of ECC can be avoided through the education of prospective and new parents on good oral hygiene and dietary practices, using agents such as fluoride and non-cariogenic sweeteners.[59]

Prevention of ECC should begin in the pre- and perinatal period. Attitudes and awareness of pregnant women may be deficient and unfavourable toward preventive dental practices.[55] Prevention should begin in the pre- and perinatal period. It is critical to provide dental care to pregnant women and women of childbearing age, both for their own health and to delay the initial transmission.[22]

ECC is the result of improper parenting, where children are pacified by unsuitable feeding practices such as sweetened beverages in cups, or feeding infants with biscuits or sweets.[60] ECC prevention should therefore be focused on educational programs to improve children's feeding practices, and to reduce levels of *Mutans Streptococci* infection.[61]

There are other preventive measures that can be applied during this period. The effect of fluoride in reducing caries is well established and may be delivered either topically or systemically. The most common method for systematically applied fluoride is fluoridated drinking water, which is shown to be effective in reducing the severity of dental decay in entire populations.[62] In its absence, other modalities such as the administration of salt fluoridation or fluoride supplements (beverages, tablets, drops) should be utilized.[21] Supervised regular use of fluoride mouthrinse and rinsing at certain intervals resulted in the reduction in caries increment in children.[63]

Treatment

Treatment of ECC can be accomplished through different types of intervention, depending on the progression of the disease, the child's age, as well as the social, behavioral and medical history of the child. Examining a child by his or her first birthday is ideal in the prevention and intervention of ECC.[19] During this initial visit, conducting a risk assessment can provide baseline data necessary to counsel the parent on the prevention of dental decay.

Areas of decalcification (early or "white spot lesion") and hypoplasia can rapidly develop cavitation. If lesions are identified early, the use of anticariogenic agents may reduce the risk of development and progression of caries. Parents should be taught how to clean their child's teeth with fluoride toothpaste. The surfaces of the teeth should be carefully scrubbed after each feeding.[23]

Fluoride varnish at one-month intervals, may be a practicable option, especially when targeted at children with carious maxillary incisors. Children should also participate in a school-based fluoride mouthrinsing program.[63] Minimal intervention restorative procedures, such as atraumatic restorative treatment (ART), which do not require the use of local anaesthesia or a dental handpiece are useful to decrease the trauma to both child and parent. The placement of fluoride-releasing glass ionomer cements is efficacious in both preventive and therapeutic approaches.[64]

When cavitation has occurred, more definitive treatment is required. Early stages of cavitation can be treated restoratively, while advanced stages will require more complicated measures such as strip crowns for the anterior teeth and stainless steel crowns for the posterior teeth. Depending on the extent of the lesions, pulpectomies or extractions may be indicated. Such management of caries becomes extremely expensive and difficult to treat because such young children lack the ability to cope with extensive restorative care. General anesthesia or sedation cases are often recommended and can cost thousands of dollars. Recall appointments should be based upon the risk assessment and examination outcomes.[66]

Conclusion

ECC is preventable and manageable with the right information and skills. A closer co-operation between healthcare professionals, dental hygienists and paediatric dentists is required. Distribution of preventive health care literature, early referral and prompt treatment of the children with the signs of decay can help improve oral and dental health of children.

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