Early Childhood Caries - A Review

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Introduction:
Dental caries is the most common chronic disease in children that is five times more common than asthma and seven times more common than hay fever¹. The American Academy of Pediatric Dentistry defines the disease of Early Childhood Caries (ECC) as the presence of 1 or more decayed (noncavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth in a child 71 months of age or younger. In children younger than 3 years of age, any sign of smooth-surface caries is indicative of Severe Early Childhood Caries (S-ECC). From ages 3 through 5, 1 or more cavitated, missing (due to caries), or filled smooth surfaces in primary maxillary anterior teeth or a decayed, missing, or filled score of ≥4 (age 3), ≥5 (age 4), or ≥6 (age 5) surfaces constitutes S-ECC².

The multi-factorial nature of ECC covers but is not limited to commonly used terminologies including “nursing caries”, “baby bottle caries”, and “baby bottle tooth decay”, since inappropriate feeding habits are often associated but are not the sole etiological factors of this condition. The Centers for Disease Control and Prevention suggested the term “Early childhood caries” in 1994 at a workshop in an attempt to focus attention on the multiple factors (i.e. socioeconomic, behavioral, and psycho-social) contributing to caries at such early ages, rather than ascribing sole causation to inappropriate feeding methods³.

Like any other form of dental caries the occurrence of ECC has a multifactorial etiology, inclusive of microbiological factors, diet and other host factors. There are also several factors that escalate the risk of developing ECC but cannot be implicated as specific etiological factors.

Microbiological factor:
It is important to appreciate that ECC is an infectious disease and Streptococcus. The main cariogenic micro-organisms associated with this condition are mutans and Streptococcus sobrinus ⁴. The cultivable plaque flora of S. mutans have shown to exceed 30% in children with ECC as shown by many studies ⁵,⁶. The carious lesions, white spot lesions, and sound
tooth surfaces near the lesions are associated with these bacterial masses. Contrary to this, S. mutans constitutes less than 0.1% of the plaque in children with negligible to no caries activity.

It was always believed that S. mutans has a feeble capacity to attach to epithelial surfaces and hence is less likely to colonize pre dentate infants. It has since been well known that initial acquisition of mutans streptococci (MS) by infants occurs during the window of infectivity. However, recent clinical studies have demonstrated that S. mutans can colonize the mouths of pre dentate infants. The furrows of the tongue appear to be an important ecological niche. Tanner and coworkers, utilizing DNA probe technology, reported that mutans streptococci were found to be present in 55 percent of plaque samples and 70 percent of tongue scraping samples of 57 children aged 6 to 18 months. These recent studies on acquisition of mutans streptococci raise doubts that a nonshedding oral surface is mandatory for their oral colonization.

**Acquisition of organisms:**

Vertical transmission, the transmission of an infection or other disease from caregiver to child is deemed one of the most common sources of organism acquisition in ECC. The major reservoir from which infants acquire Mutans streptococci seem to be their mothers. The early evidence for this concept comes from bacteriocin typing studies where MS isolated from mothers and their infants demonstrated identical bacteriocin typing patterns. Eventually advanced technology utilizing chromosomal DNA patterns provided evidence of vertical transmission. A study carried out by Berkowitz and coworkers reported that infants were approximately nine times more likely to develop caries when maternal salivary levels of the organism exceeded $10^5$ colony-forming units per ml in comparison to the frequency of infant infection observed when maternal salivary reservoirs were less than or equal to $10^3$ cfu per ml. Suppression of maternal microbial reservoirs showed that infection of the baby could be prevented or delayed; only 11 percent of babies whose mothers’ had their mutans streptococci reservoirs suppressed by dental treatment and topical antimicrobial therapy were infected by age 23 months. In contrast 45 percent babies in the control group whose mothers’ levels of mutans streptococci were not suppressed were infected.

More recent trends and studies suggest that horizontal transmission specifically in nurseries also plays a major role in acquisition of organisms in children. Mattos-Graner in a study isolated mutans streptococci from groups of nursery school children (age 12 to 30 months) and genotyped the isolates using primed polymerase chain reaction and restriction fragment-length polymorphism analysis. They found that many children contained identical genotypes of mutans streptococci strains, which indicated that horizontal transmission may be another mode of acquisition of these organisms.

**Dietary factor:**

Breastfeeding provides health benefits to the child along with adequate nutrition. But it has been demonstrated that acidogenic conditions are created followed by softening of enamel due to frequent and sustained contact of enamel with human milk. The duration of contact of fermentable carbohydrate with enamel influences the equilibrium shift from re-mineralization towards de-mineralization. Increased risk of ECC is associated with prolonged and nocturnal breastfeeding, especially when continued after 12 months of age. The reason for this is attributed to the fact that due to less saliva production at night nocturnal feeding leads to increased levels of lactose in saliva and plaque than that expected during the day. Thus there is a shift in balance toward de-mineralization rather than re-mineralization during the night due to insufficient production of saliva.

Studies have also demonstrated that breast - bottle-feeding being practiced together was more
common, and these children had a higher prevalence of caries than the children who were only breast-fed. Also, children who were bottle-fed at night developed more caries lesions\textsuperscript{25}.

There is evidence lacking that human milk alone is cariogenic; factors, such as oral hygiene may also influence caries development than purely at will breastfeeding. There is an ongoing discussion currently which states that breast milk, specifically on demand feeding is only a risk factor in ECC rather than a causative factor. This seems to be supported by the fact that the breastfeeding biomechanics differs from that of bottle feeding, the milk is expressed into the soft palate and is swallowed without remaining on teeth\textsuperscript{26}.

The presence of formula milk or milk with sugar in the feeding bottle while falling asleep, and higher sweet scores in the diet chart have also been demonstrated as factors in the etiology of ECC\textsuperscript{27}.

**Other risk factors:**

Various risk factors have been implicated in the development of ECC. Higher birth order, lower socioeconomic status, non-use of fluoridated toothpaste, and visiting dentist only when a problem was perceived\textsuperscript{27} have been implicated in a study as has sugar and fruit-juice consumption and lack of periodic dental examination been associated in another\textsuperscript{28}.

Another study has found that between-meal snacking and sweetened pacifier use increases risk of ECC development and factors such as increased frequency of toothbrushing, use of toothbrush and fluoridated dentifrice are protective factors against the risk of ECC\textsuperscript{29}.

Increased risk of ECC is seen in children born to single mothers, and those with low educational status\textsuperscript{30,31}.

**Clinical appearance and Diagnosis:**

ECC is initially recognized as a dull, white de-mineralized spot on the enamel surface along the gingival line or on the occlusal surfaces of teeth which coincides with distribution of plaque. It can also appear around the gingival margin, between the interproximal surfaces or on the palatal surfaces and/or in extreme cases it can involve the incisal edges. The first sign of decay is seen on the primary maxillary incisors, simultaneously all four maxillary anterior teeth are involved. Clinically, a yellow or brown cavitated area is evident\textsuperscript{32}.

With progressive, untreated lesions, decay can extend around the circumference of tooth, forming a black collar with extensive hard tissue loss. In advanced cases of ECC crowns of maxillary incisors are completely destroyed leaving root stumps\textsuperscript{33}.

**Detrimental effects/complications and sequelae of ECC:**

Though ECC is preventable by early detection, risk assessment, counseling and educating parents, and by implementing preventive procedures like fluoride application, the progression of ECC can reduce the general health and quality of life of the affected children\textsuperscript{32}.

Untreated teeth can lead to pain, infections, abscesses, chewing difficulties, esthetic deficit, speech problems, poor appetite, and sleep disturbances. Glucocorticoid production is altered due to disturbed sleep and there is depressed erythrocyte production due to suppression of hemoglobin.\textsuperscript{32,33}

Malocclusion can occur due to premature loss of primary teeth. In the long term these can also lead to low self-esteem, malnutrition, and a fear and aversion, to seek treatment\textsuperscript{32,33}.

ECC also predisposes a child to increased caries risk in the permanent dentition\textsuperscript{34}. The risk of hospitalization is increased, increased treatment
cost, absence and inability to concentrate in school resulting in low learning ability and reduced self-esteem. In children less than 2 years old, ECC is also associated with reduction in growth and weight gain due to inadequate food consumption to meet the metabolic and growth needs of the child.

**Preventive measures:**

Recommendation for preventive strategies should be based on:

1. Scientific studies that have tested the efficacy and effectiveness of the interventions that could be used to prevent a disease.
2. A pragmatic, objective, and valid definition of the problem targeted for prevention.
3. A comprehensive rather than a restricted understanding of the condition.

**AAPD Policy**

To decrease the risk of developing ECC, the AAPD encourages professional and at-home preventive measures that include:

1. Reducing the parent’s/sibling’s MS levels to decrease transmission of cariogenic bacteria.
2. Minimizing saliva-sharing activities (e.g., sharing utensils) to decrease the transmission of cariogenic bacteria.
3. Implementing oral hygiene measures no later than the time of eruption of the first primary tooth. Toothbrushing should be performed for children by a parent twice daily, using a soft toothbrush of age-appropriate size. In all children under the age of three, a ‘smear’ or ‘rice-size’ amount of fluoridated toothpaste should be used. In all children ages three to six, a ‘pea-size’ amount of fluoridated toothpaste should be used.
4. Providing professionally-applied fluoride varnish treatments for children at risk for ECC.
5. Establishing a dental home within six months of eruption of the first tooth and no later than 12 months of age to conduct a caries risk assessment and provide parental education including anticipatory guidance for prevention of oral diseases.
6. Avoiding high frequency consumption of liquids and/or solid foods containing sugar. In particular: • Sugar-containing beverages (e.g., juices, soft drinks, sweetened tea, milk with sugar added) in a baby bottle or no-spill training cup should be avoided. • Infants should not be put to sleep with a bottle filled with milk or liquids containing sugars. • Ad libitum breast-feeding should be avoided after the first primary tooth begins to erupt and other dietary carbohydrates are introduced. • Parents should be encouraged to have infants drink from a cup as they approach their first birthday. Infants should be weaned from the bottle between 12 to 18 months of age.
7. Working with medical providers to ensure all infants and toddlers have access to dental screenings, counseling, and preventive procedures.

**Management:**

During this initial visit, it is advisable to conduct risk assessment, plan preventive programs and counsel parents, a baseline data of the disease is necessary for prevention of progression of ECC. Restorative and preventive measures are based on extent of caries and treatment could range from simple restorations to pulp therapies including stainless steel crowns. Extraction may be necessary in few cases followed by planning for space management.

Atraumatic Restorative Treatment (ART) is an approach of treating dental caries in young children. Using hand instruments alone carious tooth tissues are removed and the cavity is restored with an adhesive restorative material.
The current standard of care for treatment of S-ECC may necessitate general anesthesia as the co-operative levels of babies and pre-school children is often less than ideal\(^2\). General anesthesia also serves to complete extensive treatment efficiently in a short time.

Regular recall appointments need to be scheduled for these children to conduct thorough check ups and to catch subsequent lesions early and initiate adequate treatment.

**Conclusion:**

Dental disease and specifically early childhood caries can negatively affect the total well-being of a child. But considering that ECC is largely preventable, it is the onus of pediatric dentists to provide an understanding of its risk factors, consequences, prevention and management strategies to the general public and specifically to new parents. Early detection would also ensure simpler and less invasive management strategies. Establishment of a dental home would also serve the purpose of early detection and where necessary suitable intervention.

**References**


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