

A Review

Early Childhood Caries

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Abstract

Pediatric dentistry exists because children have dental and orofacial problems. The genesis of dentistry for children unquestionably is allied to dental decay, pulpitis, and the inflammation and pain associated with infected pulpal tissue and suppuration in alveolar bone. Dental caries is the most common chronic infectious disease of childhood, caused by the interaction of bacteria, mainly *Streptococcus mutans*, and sugary foods on tooth enamel. Early Childhood Caries (ECC) continues to affect toddlers and preschool children worldwide in both developing and industrialized countries. It is an infectious, transmissible, diet-dependent disease that may begin soon after dental eruption and that may progress rapidly. Preventive measures cannot, and will not, work unless parents and caregivers follow and adhere to the preventive methods being prescribed. Decayed teeth require professional treatment to remove infection and restore tooth function

Key Words

Early childhood caries; etiology; risk factors; clinical considerations; prevention; management

Introduction

Dental caries affects humans of all ages throughout the world and remains the major dental health problem among school children globally.^[1] Early childhood caries (ECC) is the term now recommended by the Centers for Disease Control and Prevention to describe a unique pattern of carious lesions in infants, toddlers, and preschool children.^[2] This begins as soon as the teeth erupts and can rapidly progress to extensive decay of all primary teeth^[4]. ECC is an alarming problem as 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.^[9]

ECC presents as considerable challenge for the pediatric dentists partly due to its rapid progress, but more so due to the age of the affected children, many of whom are below the cooperative age of 29 months old [Holst et al., 1993; Curzon and Preston, 2004]. In addition, the long-term management of these patients is more complex as children with carious lesions in the primary dentition have a greater chance of developing caries in the permanent dentition compared with caries free children [Kaste et al., 1992].

Effective ECC management requires utilising risk-based disease prevention and management approaches that include caries risk assessment, self-management goals and caries remineralization strategies based on which management, "Prevention rather than Treatment" seems to be the best allocation here.^[3]

Definition

Caries is a biofilm (plaque)-induced acid demineralization of enamel or dentin, mediated by saliva. The disease of early childhood caries (ECC) is 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).^[5]

Terminology

Owing to its multifactorial etiology, there are many different names used as synonyms for early childhood caries. Caries in infants and young children has long been recognized as a clinical syndrome, described by Belterami in 1930s as "Les dents noires de tout-petits" which means "black teeth of the very young. Nursing bottle caries (NBC), baby bottle tooth decay (BBTD), nursing bottle syndrome (NBS) and milk bottle syndrome are some other names.^[10] In 1985, the term baby bottle tooth decay was proposed by the Healthy Mothers - Healthy Babies Coalition as an alternative that would be more appropriate for patient acceptance and would focus attention on the potential damage of using a nursing bottles.^[11]

Epidemiology

Despite the decline in the prevalence of dental caries in children in the western countries, caries in pre-school children remains a problem in both developed and developing countries. ECC has been considered to be at epidemic proportions in the developing countries.^[6]

A comprehensive review of the occurrence of the caries on maxillary anterior teeth in children, including numerous studies from Europe, Africa, Asia, the Middle East, and North America, found the highest caries prevalence in Africa and South-East Asia. The prevalence of ECC is estimated to range from 1 to 12% in infants from developed countries.^[6]

Prevalence of ECC is not a common finding relative to some European countries (England, Sweden, and Finland), with the available prevalence data ranging from below 1% to

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32%. However, this figure is rising by as much as 56% in some eastern European countries. In US, pre-school children data from a more recent study indicate that the prevalence of dental caries of children 2–5 years of age had increased from 24% in 1988–1994 to 28% in 1999–2004. Overall, considering all 2–5-year olds, the 1999–2004 survey indicates that 72% of decayed or filled tooth surfaces remain untreated. The prevalence of ECC children in the general population of Canada is less than 5%; but in high-risk population, 50–80% are affected. Studies reveal that the prevalence percentage of ECC in 25- to 36-month olds is 46% and the reported prevalence in Native Canadian 3-year-olds has been as high as 65%.^[6]

Published studies show higher prevalence figures for 3-year-olds, which ranges from 36 to 85% in Far East Asia region, whereas this figure is 44% for 8- to 48-month olds reported in Indian studies. ECC has been considered at epidemic proportions in the developing countries. Studies conducted in the Middle East have shown that the prevalence of dental caries in 3-year-olds is between 22% and 61% and in Africa it is between 38% and 45%.^[6]

Classification

Wyne (1999) has suggested the following classification for ECC according to pattern of caries involving the primary teeth.^[12]

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. This type of ECC could be found soon after the first teeth erupt.



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.



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.^[1]

1. **DENTAL PLAQUE** – In the absence of fermentable carbohydrates, organic acid such as acetate, propionate, and butyrate are produced. In contrast when fermentable carbohydrate are present, lactate is mainly produced, which coincides with a pH drop in plaque bacteria and their alkaline products provide major contributions to the pH rise in plaque and the base generating metabolism of plaque bacteria is considered by many to be a significant determinant for cariogenicity of plaque.^[7]

2. **MUTANS STREPTOCOCCI** : The main bacteria indicated in ECC are termed “mutans streptococci.” Of which the species *S mutans* and *S –robrius* are most commonly isolated in human dental caries.

3. **SALIVARY FACTORS** – Saliva provides the main host defense system against caries has major role in clearance of foods and buffering of acid generated by plaque. Saliva contain anti microbial. Proteins – lactoferrin, lysozymes, saliva also contain several organic compounds – mucin glycoprotein fibronectin, flow rates of saliva are important.

4. **IMMUNOLOGICAL FACTORS** – Host immune mechanism include specific immune factors derived from saliva [secretory IgA] and gingival crevicular fluid [IgG]. And non – specific antimicrobial systems, derived mainly from saliva secretory IgA may inhibit bacterial adherence as well as neutralization of bacterial enzymes.

5. **TOOTH MATURATION AND DEFECTS** – Tooth is most susceptible to caries in the period immediately after eruption and prior to final maturation thus in many infants, a combination of recently imputed immature enamel in an environment of cariogenic flora with frequent ingestion of fermentable carbohydrates would render tooth susceptible to caries.

6. **CARIOGENECITY OF SUCROSE** – Sugars, glucose, fructose found in fruit juices and vitamins and foods are probably the main sugars associated infant caries. This is essential for bacterial adherence and facilitates implantation of cariogenic bacteria in the oral cavity.

7. **FREQUENCY OF CONSUMPTION OF SUGARS** – Children with ECC have a high frequency of sugar consumption, not only of fluids, given in nursing bottle but also of sweetened solid food increased frequency of eating

sucrose acidity of plaque and enhances the establishment and dominance of *S. mutans*.

8. ORAL CLEARANCE OF CARBOHYDRATES – In infants with ECC, the sleep time consumption of sugars is another common characteristic the low salivary flow during sleep decreases the oral clearance of sugars and increases length of contacted between plaque and substrates thus increasing the cariogenicity of substrate.^[14]

9. BOVINE MILK – The cariogenicity of milk is often questioned because plain bovine milk is the common fluid placed in feeding bottle in many cases of ECC and also because prolonged breast feeding has been putatively associated with ECC.

10. HUMAN MILK – Compared to bovine milk human breast milk has a lower mineral content higher concentration of lactose and less protein the relationship breast feeding and dental caries are likely to be complex and confounded by many biological variables such as *mutans streptococcus* infection enamel hypoplasia in take of sugars as well as social variables.

11. ACID FRUIT DRINK Acid in fruit juices and soft drinks many decrease the oral PH. In presence of sugars in the drinks, this fall in pH is likely to enhance the fermentation of carbohydrate thus causing enamel demineralization.^[15]

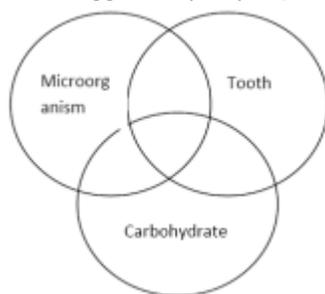
12. FLUORIDES – There is minimal information cariostatic effect of topical fluorides is prevention of ECC. The topical effect of fluorides are complex and include changes in mineralization phase as well as modulation of metabolic effects of *mutans streptococci* and other bacteria in dental plaque.

Current Theories In Etiology Of Caries^[16]

Most of the current theories suggest that, various etiological factors involved in dental caries may be,

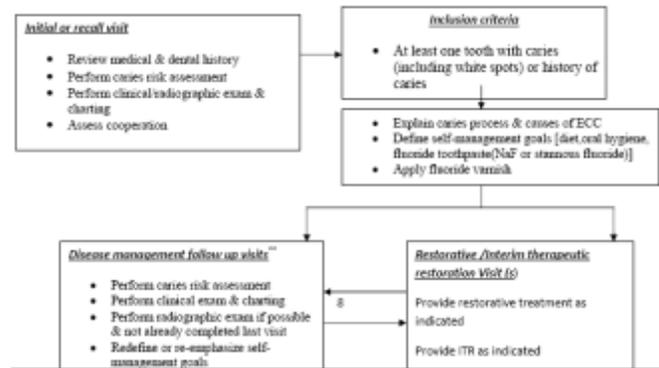
- i) Pathogenic microorganism in mouth.
- ii) Fermentable carbohydrate that metabolize to organic acids and
- iii) Tooth surface that are susceptible to acid dissolution

The fig shows the well known diagram which interlinks these three etiological components and demonstrates the need for simultaneous presence of all three component before caries can occur, which was suggested by Keyes (1962).



However, in order for the lesion to progress and to be clinically diagnosed, these 3 variables must interact over a suitable period of time. Hence a fourth variable 'time factor' was added by Newbrun in 1969.

DISEASE MANAGEMENT PROTOCOL (used in ECC collaborative)^[13]



Prevention

** For children at high risk

- Schedule Next Disease Management Visit in 1-2 month

** For children at medium risk

- Schedule Next Disease Management Visit in 3-4 months

** For children at low risk

Working together with other stakeholders in the community gives dentists access to skills and tools that aid in the prevention of ECC. A dental health promotion program presented in books, brochures, stickers or videos and made available in dentists' offices and community centres can potentially lower the incidence of ECC in communities at high risk for caries. This type of dental health promotion program must be geared to expectant parents or the parents of very young children.

- The expectant mother should be monitored for dental problems during pregnancy and given the appropriate prevention recommendations before the birth of her baby.

- Parents should be taught how to brush their baby's teeth, either by resting the baby against them, or laying the baby on their lap with his head between their legs.

- When the baby reaches the age of one, his teeth should be brushed twice a day with a small toothbrush and water and fluoride toothpaste (about the size of a pea). Between the ages of 18 and 24 months, the child can learn to brush his teeth under adult supervision.^[12]

- In addition, parents should not try to soothe a crying or agitated baby with candy, a pacifier dipped in sugar, or a bottle containing a sweet drink.

- The success of fluoride therapy depends on the parent's motivation and participation, regular check-ups and adjusting the dose depending on the dosage schedule.

- The use of topical fluoride in the form of a varnish or gel is beneficial but not recommended before the child turns one.

- Chlorhexidine varnishes can be used in children between the ages of 3 and 4 with a high risk for caries, in order to reduce the quantity of streptococci within the dental plaque and as a tool for the bacterial control phase.^[9]

- Sealing agents are evidently entirely indicated to prevent occlusal caries of the primary molars, and should be used beginning at age 3 after consideration of the caries risk and clinical recommendations.

- Substituting sugar with xylitol or other artificial sweeteners (sorbitol and mannitol) in candy, and the recent appearance of products made with casein phosphopeptide or amorphous calcium phosphate (in chewing gum and toothpaste) will have interesting applications in preventing EEC in the future.

Despite a dental health provider's efforts to implement a prevention program, sometimes the outcome does not meet expectations. The prevention program must be accompanied by individual counselling of the parents.

Conclusion

The etiological factors of EEC are known, and there is an arsenal of preventive and curative therapies available to help practitioners prevent and properly control EEC. EEC prevention is an essential component in any dental health promotion program, providing a solid foundation for the optimal development of children.^[8]

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