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# Early Childhood Caries Update

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## Abstract

Early childhood caries (ECC) is the most common chronic disease among young children who are less than 71 months of age and is currently represented as a public health problem in various countries worldwide. Dental caries continues to be a major health problem in developing nations because of lack of education, awareness, and poor socio-economic status. It begins with white-spot lesions on upper primary incisors along the margin of the gingiva and leads to complete destruction of the crown. The potential impact of ECC on the general health and development has been widely reported in the literature. The main risk factors in the development of ECC can be categorized as microbiological, dietary, genetic, behavioral, and environmental. Evidences for effective ECC prevention suggest prenatal and immediate postnatal interventions. Population-based early childhood health systems hold great potential to reduce the burden of ECC. This chapter focuses on diagnosis, prevalence, etiology, preventive strategies, and treatment options of ECC.

**Keywords:** early childhood caries, etiology, preventive strategies, treatment options, update

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## 1. Introduction

Early childhood caries (ECC) is one of the most prevalent, infectious, biofilm-mediated, and transmissible childhood diseases with long-term progression and caused developmental implications that affect children worldwide especially in developing countries [1]. Those dental decays in infants and toddlers are also known as baby bottle caries, baby bottle tooth decay, nursing bottle caries, nursing caries or rampant caries. Today, the more commonly used terms are ECC and S-ECC in severe cases [2]. ECC describes dental caries affecting children aged 0–71 months. “According to the American Academy of Pediatric Dentistry (AAPD),

ECC is defined as the presence of 1 or more decayed (non-cavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth in a child 71 months old or younger.” AAPD confirms that any sign of smooth-surface caries in children younger than 3 years of age is indicative of severe ECC. This definition is detailed as severe ECC constitutes 1 or more cavitated, missing (due to caries), or filled smooth surfaces in primary maxillary anterior teeth from ages 3 to 5, or a decayed, missing, or filled score of  $\geq 4$  (age 3),  $\geq 5$  (age 4), or  $\geq 6$  (age 5) surfaces [3]. These lesions involve tooth surfaces that are less prone to caries development.

There are a multitude of risk factors associated with ECC. Feeding habits and a variety of biological, environmental, and socioeconomic factors are involved in the development of ECC [4]. ECC is associated with other general and dental health problems such as ranging from local pain, infections, leading to difficulty in chewing, malnutrition, gastrointestinal disorders, poor child growth or development, and social outcomes [5].

ECC has important etiological bases during the first year of life. Current research suggests that gaps in the knowledge about that disease’s progression prohibit effective and early identification of “at-risk” children [6]. Recent studies have focused on the markers of the disease with multifactorial origin. They investigated the risk factors for ECC, such as a variety of microorganisms, the genome along with the oral microbiome (metagenome) and their interactions (transcriptome, proteome, and metabolome) at the tooth surface level genetics, salivary proteins, proinflammatory cytokines and iron deficiency, and so on. They remain largely unknown in caries risk assessment and personalized clinical decision-making.

Understanding the natural history of ECC has an important effect on implementing effective preventive strategies [6]. Prenatal and immediate postnatal interventions are suggested to prevent ECC in evidence-based dentistry. Training on ECC prevention and preventive programs should be started in the prenatal period and should persist in early childhood. Population-based early childhood health systems hold great potential to reduce the burden of ECC.

Treatment options of ECC range from prevention, minimally invasive approaches to extraction depending on the extension of the caries. As ECC is monitored during early childhood, children in this age group may exhibit behavior problems during dental treatment. Pharmacological behavior control methods such as sedation and general anesthesia may be required.

The objective of this chapter is to make an update of ECC which is a persistent and possibly widespread public health problem. The diagnosis, epidemiology, etiology, preventive strategies, and treatment options of ECC are reviewed.

## **2. Early childhood caries (ECC)**

### **2.1. Diagnosis and classification of ECC**

The lesions progress rapidly; they can be extensive and typically affect free smooth surfaces such as the labial surfaces of maxillary incisors and lingual and buccal surfaces of maxillary

and mandibular molars. ECC has a special caries pattern. The maxillary primary incisors are most vulnerable to the disease and are affected first. In the moderate stage, the caries begins to spread, and the buccal and occlusal surfaces of the primary molars and the buccal surfaces of the primary canine are affected, respectively, in a way that reflects the pattern of eruption. Usually the mandibular incisors are protected by the tongue and saliva [7, 8].

ECC initially presents as opaque white or brown spots on maxillary incisors along the gingival margin and the lesions often cover many surfaces in each of affected tooth. In severe cases, anterior teeth break down during eruption and the process continues with the mandibular molars [2, 7] (**Figure 1**).

Several research groups have attempted to develop classification systems for early childhood caries. Wyne A classified ECC based on the severity and etiology, as Type I (from mild to moderate), Type II (moderate to severe), and Type III (severe). The existence of “isolated carious lesion(s)” involving incisors and/or molars is classified as Type I; labiolingual lesions affecting maxillary incisors, with or without molar caries, depending on the age of the child and stage of the disease, are classified as Type II; carious lesions affecting almost all teeth including the mandibular incisors are classified as Type III. Johnston T, Messer LB, classified ECC based on the pattern of ECC presentation. Lesions that are associated with developmental defects (pit and fissure defects and hypoplasia) are classified as Type I; smooth surface lesions (labial-lingual lesions, approximal molar lesions) are classified as Type II; rampant caries—having caries in 14 out of 20 primary teeth, including at least one mandibular incisor—are classified as Type III [2, 9–11].

## 2.2. Epidemiology of ECC

ECC still remains a serious challenge for health care providers, despite the improvement in dentistry practice and decline in the prevalence of dental caries [11]. The prevalence of ECC differs according to the examined groups’ age, affected teeth, socioeconomic status, lifestyle, dietary pattern, oral hygiene practices, behavioral factors, race, culture, and ethnicity which differ from country to country [2]. The prevalence of ECC is reported as between 1% and 12% in most developed countries [12] and 85% in disadvantaged groups in developing countries [13]. In a systematic review, it is presented that the prevalence of ECC varies from 2.1% in Sweden to 85.5% in rural Chinese children [14]. Some of the highest prevalences of ECC have been reported in some Middle Eastern countries, such as Palestine (76%) and the United Arab Emirates (83%) [15, 16]. Increases in caries prevalences among 2–5-year-olds are reported, respectively, from children in Brazil, North America, China, Australia, and Korea, with the



**Figure 1.** Intraoral images of the severe ECC.

prevalence rates of 27, 60, 67, 80, and 83.3% [17, 18]. Turkey is a developing country; therefore, there is a lack of data about the prevalence of ECC in Turkey. Only two articles are presented about this topic. Gökalp et al. [19] presented a national epidemiologic data which showed that the caries value and prevalence rates were 3.7 and 69.8% for 5-year-old children and 1.9 and 61.1% for 12-year-old children. Doğan et al. [20] presented the regional ECC prevalence in the city center of Kırıkkale, Turkey, as 17.7% in a group with the mean age of  $25.8 \pm 10.11$  months. To realize the severity and extent of the disease, a comprehensive study supported by health ministry is needed in Turkey.

### 2.3. Etiology of ECC

The etiology of ECC is obviously multifactorial and can be viewed from multiple standpoints: molecular/biochemical, microbiological, behavioral, social, health system, and even political [21].

Dental caries arise from the interaction of various etiological factors. Cariogenic microorganisms, fermentable carbohydrates, and susceptible tooth surface/host are the main factors. Factors such as high sugar intake, lack of oral hygiene, lack of fluoride exposure, and enamel defects are some of the major factors and responsible for the development of ECC [22, 23]. Low socioeconomic status, sociocultural differences in oral health, beliefs and practices, minority status, low birth weight, and transfer of microorganisms from mother to child are some of the multitude risk factors documented in epidemiological studies [24, 25]. To understand the complex nature of ECC better, further studies are required.

#### 2.3.1. Diet

There are many risk factors associated with ECC. It is a dynamic pathological process that depends on biofilms, diet, and host salivary constituents. Dietary practices; poor dietary habits and food preferences (including starches and other sucrose-containing foods), frequent exposure to sweet beverages, and night-time meals or drinks promote the proliferation of cariogenic bacteria [26, 27].

Inappropriate feeding practices, such as bottle feeding with sweetened milk or fruit juice, night-time bottle feeding, and sleeping with honey-soaked dummies, have been associated with the initiation and development of caries in children [6, 8]. Inappropriate feeding practices can prolong the exposure of teeth to fermentable carbohydrates; *Streptococcus mutans* converts fermentable carbohydrates into acids, and demineralization starts.

Despite benefits of breastfeeding for systemic health, a well-known effect on ECC is controversial. In one of the systematic reviews, breastfeeding over 1 year beyond tooth eruption has been found to be associated with ECC [6]. The World Health Organization (WHO) recommends exclusive breastfeeding until the age of 6 months, and breastfeeding complemented with food intake is suggested until the child becomes 2 years old [28]. Current scientific evidence suggests that breastfeeding has a greater protective effect against dental caries than bottle feeding, although the proper duration of breastfeeding analyzed in the studies could not be determined [29]. Cultural and social factors directly affect the practices such as prolonged



breastfeeding and sharing the mother's bed. In the systematic review, no association was reported between breastfeeding duration, sharing bed, and ECC, whereas a strong association was determined with nocturnal breastfeeding. They concluded that children who were breastfed more than twice at nights after age of 1 year had a much higher risk of having ECC [6]. Frequency and duration of exposure are critical at this point, but the caries experience also correlates often with social and other behavioral factors within the family [8].

Breastfeeding should be encouraged in accordance with WHO recommendations but to prevent ECC, regular mechanical removal of the dental plaque should be satisfied and parents should stop breastfeeding at nights [29]. Behaviors like limiting added sugar, reducing bottle use, and serving defined meals or snacks have positive implications for oral health. If the baby awakens at night, and has difficulties on leaving his/her behavior about nightfeeding, water should be preferred over milk or juice. Snacking frequency should be limited (no more than two or three snacks per day) to prevent decaying.

According to the European Academy of Pediatric Dentistry (EAPD)'s guidelines on the prevention of ECC, frequent intake of sweet drinks and feeding with sweetened baby bottles on demand should be discouraged, especially at nighttime. It is recommended with the evidence level grade C [30].

### 2.3.2. Cariogenic microorganisms

Cariogenic bacteria were shown to be a significant risk factor for ECC. The pathophysiological etiology of ECC is associated with early colonization and high levels of the cariogenic microorganisms (e.g., *Streptococcus mutans* (SM) and *Streptococcus sobrinus*) and abundance of dental plaque as a consequence of overexposure to sugars and complex interspecies with accumulation of salivary proteins and adhesive glucans. The acidic environment in the dental biofilm causes demineralization of the enamel and dentin [27, 31].

Since cesarean deliveries are more aseptic than vaginal deliveries, infants delivered by cesarean section acquire SM earlier and the atypical microbial environment increases the chances of SM colonization [32]. Bacteria that present in the predentate stage play a significant role in early caries experience [6].

SM is the main bacteria that have strong association with ECC whereas other oral bacteria in the dental biofilm could be involved in the initiation and progression of caries [26]. Another bacterium associated with ECC development is *Lactobacillus* (LB) species, which play an important role in lesion progression [31]. The *Actinomyces* species, especially *Actinomyces gerencseriae*, were also associated with caries initiation. On the other hand, *Bifidobacterium* species were linked with deep caries lesions. There are also a few non-mutans streptococci which have acidogenic and aciduric attributes, related to dental caries. Epidemiological data suggest that in the pathogenesis of dental caries *Candida albicans* also plays an active role [2]. The evidence presented in the systematic review indicates that the prevalence of *C. albicans* in children with ECC is significantly higher than in caries-free children. In addition, it is stated that children with oral *C. albicans* have higher odds of experiencing ECC compared to children without *C. albicans* [33].

However, MS levels in plaque vary depending on the stage of caries development and dietary habits, infant feeding practices such as frequent exposure to sugar, frequent snacking, taking sweetened drinks to bed, sharing foods with adults, as well as maternal caries status and oral hygiene [34].

ECC is an infectious, transmissible disease. SM is the main bacteria in ECC pathogenesis and these bacteria can be transmitted from mother to child and/or also from other primary caregivers. That is commonly termed as “window of infectivity” [35]. It’s known that children who have higher SM levels were five times more prone to have dental caries [23]. The major source of acquiring the SM in children is mainly from their mothers during first 12–24 months via vertical transmission through saliva. Maternal factors such as poor maternal oral hygiene maintenance, frequent snacking, and sugar exposure were shown to influence bacterial acquisition. However, antibacterial treatment of only mothers or children may not be sufficient to reduce infection [36]. Children can be infected with mutans streptococci horizontally between siblings and caregivers [37].

It is well known that saliva and dental biofilm harbor different genotypes of *S. mutans*. However, it is still unknown if genotypic diversities and phenotypic traits of *S. mutans* are related to different caries status or caries severity in children [38]. Regardless of caries status, the genotypic diversity of *S. mutans* was determined to be similar. In addition, *S. mutans* genotypes from caries-active children were found more acid tolerant and presented higher ability to form biofilms than those isolated from caries-free children. The most important *S. mutans* trait related to the pathogenesis of sECC is thought to be tolerance to acid [39].

### 2.3.3. Environmental factors

ECC is a global dilemma, and with the exception of a multitude of risk factors like oral hygiene maintenance, its incidence is associated with additional factors, such as socioeconomic and education status [40]. ECC prevalence in non-industrialized and industrialized countries has reported between 28 and 82% [6]. It is emphasized that children, who are two times more likely prone to dental caries, are from low socioeconomic status [41].

The use of caries-promoting rearing practices was associated with sociodemographic factors such as family income, poverty, ethnicity parental education, number of children that families have, and rural or urban residence [6]. Lack of good oral hygiene practices and lifestyle factors promote the development of ECC. Children should begin receiving oral hygiene care since the eruption of the first primary tooth.

Prenatal conditions such as premature birth and low birth weight are described as some risk factors that could promote the presence of enamel hypoplastic defects [42]. Enamel hypoplasia was reported in 67% of children who have low birth weight in comparison to 10% among children with normal birth weight, in a case–control study [43]. The causal relationship of enamel hypoplasia with dental caries has not been established [2].

According to SIGN 18 recommendations (grade D), when developing community preventive programs, children from low socioeconomic status groups should be considered to have increased risk for ECC [44].

#### 2.3.4. *Teeth*

Dental caries is etiologically heterogeneous but mostly behaviorally driven; half of its observed variance may be attributed to predisposing genomic factors [45]. Since the late 1950s, dental caries has been shown to have a substantial genetic component [46]. Genomics likely influences ECC, susceptibility via control of dental anatomy, enamel quality, salivary properties, immunity, oral microbiome composition, taste preference, and other intermediate characteristics [45]. Recent studies have been designed to validate genetic susceptibility to dental caries in children. It is thought that the developments in this area may be an important step for determining the dental caries risk and prevention.

### 3. Risk assessment update in ECC

As ECC is a highly complex disease, identification of children at risk for ECC before the onset of cavitation remains challenging. There are gaps in the knowledge as to the answers of the following questions: how the risk factors interrelate and why some children suffer a greater burden of disease than others [26]. This limitation is due to the multifactorial (environmental, social, and behavioral) nature of ECC and the limited accuracy or poor validity of existing caries risk screening tests [44]. Up to date, no effective methods in identifying the risk factors of ECC development are presented. In the near future, identification of biomarkers and development of biology-based analytical tools or devices for caries risk assessment would be available. For providing greater predictability of ECC development and improving the accuracy of the existing ECC-risk screening methods, behavioral risk assessments should be combined with microbiome and salivary biomolecule analyses [26].

Recent studies are already focusing on this area. In this point of view, ECCs' relationship with microbial biofilm variability (focused on OMICS-based approaches, such as high-throughput (meta) genomics, transcriptomics and proteomics, as well as metabolomics), specific genetic influences, iron deficiency, prenatal maternal cigarette smoking, and so on is still being investigated. Therefore, further well-designed studies should be planned to get evidence-based results in this area.

Emerging biological insights about the pathogenesis of ECC can help to better understanding, provide personalized approach, provide enhanced preventive care for the susceptible children, and treat this early-onset, aggressive childhood disease. This approach ultimately helps to eliminate this costly and painful disease [26, 47].

For making a clear prediction, there is not any superior method and no evidence exists to support the use of one model, program, or technology over any other [48]. Although ECC prediction tools have limited practical clinical utility, they serve as a valuable resource in dental education and as guides for the development of public health programs [21].

Moderate-to-weak evidence supports the following recommendations that could be made: (1) Caries risk assessment of children should be carried out as soon as their first tooth erupts by dental professionals and this should be reassessed periodically over time; (2) while assessing



caries risk in young children, multiple factors like clinical, environmental, and behavioral should be considered by interviewing the parents. Examples include caries experience, dietary habits, such as frequency of sugary food and drink consumption, socioeconomic status, oral hygiene habits, including use of fluorides, and medical history, with emphasis on conditions that could affect salivary flow rate. Considering the factors associated with the parents such as parental oral health status and parental deprivation, it is very important for assessing children's caries risk. (3) The use of structured forms, although with limited validity, may aid in systematic assessment of multiple caries risk factors and in objective record keeping. (4) Children from low socioeconomic status groups should be considered in increased risk of ECC when developing community preventive programs [44, 48].

#### 4. Management of ECC

ECC affects children's physical (body weight, growth, etc.) and mental health and quality of life (eating, speaking, school attendance, and school performance, etc.) negatively [6]. A single and most powerful predictor for future caries experiences is accepted as past caries experiences. So, early and effective interventions are highly required for preventing ECC [8, 27]. The most effective approach for controlling ECC is based on prevention, not on restorative treatment through the epidemiological data [8].

It is recommended that routine community health awareness programs should be conducted to educate the public about the deleterious effect of ECC and also to emphasize the importance of dental routine check-ups. This may help prevent the disease and improve the quality of life of not only individuals but also their children [49].

There are some barriers in integrating oral health care into overall health care to prevent ECC. ECC prevention should be integrated into the primary care settings. Some strategies such as effective prevention, utilizing the support of professionals, integrated disease management, and innovative insurance structuring might develop to overcome these barriers [50].

A severe ECC experience is an important predictor for adult caries. Creating strategies to prevent and control ECC is important to improve general and oral health.

Primary prevention must start in the prenatal stage during pregnancy. The prevention process should progress through the perinatal period and continue with the mother and infant within the context of the family and then proceed during preschool programs.

Although maternal factors were shown to influence bacterial acquisition and modifying bacterial colonization in children, due to the complex nature of the disease, a relationship between vertical transmission of the bacteria and subsequent caries development in infants was not clarified yet. Further qualified studies are required to understand the complex nature of ECC better. In addition, in the systematic review written by Leong PM et al. [6], it is concluded that bacteria present in the prenatate stage play a significant role in early caries experiences. So the pregnancy and the neonatal period are the important stages to identify 'at-risk' children and early maternal intervention can reduce the possibility of ECC.

Prenatal visits, understanding the importance of regular examinations, especially before the age of 1 year, and brushing with fluoride toothpaste can be considered as important strategies for preventing ECC [27]. There are increasing evidences which suggest that the preventive interventions within the first year of life are critical. For an effective prevention of ECC, conducting a caries risk assessment and providing parental education within 6 months (but no longer than 12 months) of the child's first tooth eruption is recommended [3].

For reducing mother-child (vertical) mutans streptococci (MS) transmission, clinical and educational interventions should start during pregnancy [51]. For this reason, oral health screening, dental treatment, education on oral health hygiene, and supporting a non-cariogenic diet during pregnancy and perinatal period are the most important strategies that can assist in the prevention of ECC [2]. To promote maternal and infant oral health, anticipatory guidance should be a part of standard prenatal health care [51].

Management of ECC can be examined in two sections as preventive strategies and treatment of cavitated lesions.

#### 4.1. Preventive agents

It is important to understand the natural history of ECC in order to implement effective preventive strategies. ECC disease has rampant, acute, and progressive characteristics and it is thought to be largely modulated by behavioral and environmental risk factors, such as diet and fluoride exposure [52, 53]. Early non-operative interventions done by dental professionals, including plaque removal, topical fluoride, and sealant applications, are accepted as the important steps to prevent ECC [50].

Several antimicrobial agents (e.g., fluoride, chlorhexidine, iodine, xylitol, silver compounds) combined with a range of application methods (e.g., mouth rinse, gel, varnish, cleaning wipe, restorative materials) have been used, with remarkable reductions in *S. mutans* and *S. sobrinus* levels [31].

The most commonly used preventive agents in management of ECC include application of fluoride gels, fluoride varnishes, sealants, chlorhexidine varnish, 10% povidone iodine, and xylitol oral syrup [27].

In order to arrest early childhood caries, topical applications of antibacterial agents such as chlorhexidine and povidone iodine are supported and recommended [27].

##### 4.1.1. Fluoride

Fluoride is the most important strategy in the non-invasive management of dental caries. Using fluoride through gels, mouthwashes, and varnishes is a common application in prevention programs [54].

Fluoridated toothpaste is the most commonly used form of self-applied fluoride therapies for fluoride delivery, and its anti-caries effects are proven [55]. The formulation of dentifrice, brushing behaviors, frequency, time, and post-brushing rinsing practices are significant factors influencing its efficacy [56].

While starting to implement oral hygiene, agents should not exceed the time of eruption of the first primary tooth as AAPD recommends. Under the age of 3, toothbrushing should be performed with a smear or rice-sized amount of fluoridated toothpaste. For children aged between three and six a pea-sized amount of fluoridated toothpaste should be used [3]. As EAPD recommends, brushing teeth twice a day with a fluoride toothpaste could prevent ECC [48].

While EAPD [57] recommends brushing teeth with 500 ppm of fluoridated toothpaste, according to SIGN (2005) [58], teeth should be brushed with 1000 ppm of fluoridated toothpaste as soon as they erupt, regardless of age and risk for caries, with no rinsing. Only spitting is recommended. On the other hand, the Centers for Disease Control and Prevention (CDC) (2001) and Australian Research Centre for Population Oral Health (ARCPOH) (2006) recommends that children under the age of 2 years and 18 months, respectively, do not have to use fluoridated toothpastes. Caries risk assessments of these patients should be performed by a professional and those assessments should be taken into account to offer the right amount of fluoridated dentifrice. Brushing is essential and that should start as soon as teeth eruption and must be done by parents with a small quantity of fluoride-containing toothpaste [59].

Fluoride varnishes have been used at concentrations of 1% and 5% for the prevention of ECC. Cochrane systematic review stated that fluoride varnish is an effective way in prevention of ECC and reduces caries in the primary dentition by 33% [27]. According to evidence, fluoride varnish is considered as a safe and effective agent for caries prevention in young children [56]. EAPD policy document informs that professional applications of fluoride varnishes are recommended for groups or individuals who are carrying the risk of caries, at least twice a year (grade B) [31]. Additionally, Silver Diamine Fluoride (SDF) can be an option for keeping tooth decays under control. A total of 30 and 38% concentrations of SDF have potential to be a caries preventive treatment agent for primary dentition and permanent first molars. Standardized SDF protocols must be developed [60].

To reduce dental caries in preschool children, fluoride varnish applications that are made twice a year are recommended in conjunction with toothbrushing twice a day with a fluoride-containing toothpaste and dietary advice [56].

Moderate and limited quality of evidence in support of fluoride toothpaste and fluoride varnish for early childhood caries prevention is reported [54].

#### 4.1.2. Other non-fluoride agents

##### 4.1.2.1. Chlorhexidine

Chlorhexidine (CHX) is an antibacterial agent that is commonly used as an antiseptic. CHX is used at concentrations ranging from 0.1 to 40% in solutions, gels, chewing tablets, and varnishes. In one of the meta-analysis, the caries inhibiting effect of CHX was reported to be around 46% [61].

Chlorhexidine has a long history of use in caries prevention with conflicting results [27]. Some studies have found 0.12% of chlorhexidine gluconate efficacious with multiple applications, already with synergic effects in combination with fluoride therapy. A clinical study performed with children supported this hypothesis and it was reported that separate application of the

chlorhexidine varnish and fluoride varnish was not effective as a combination of the agents in enhancing the remineralization of white spot lesions after 3 months [56].

#### 4.1.2.2. Xylitol

Xylitol is a five-carbon sugar alcohol and therefore it is a good sugar substitute and has sweet taste. It is non-acidogenic, meaning not metabolized by the cariogenic bacteria. It has anti-bacterial properties. Salivary flow rate and buffering capacity can be increased with xylitol. Xylitol arrests the dental caries and helps remineralization [27]. AAPD supports the usage of xylitol as a non-cariogenic sugar substitute. Xylitol is available in many forms (e.g., gums, mints, chewable tablets, lozenges, toothpastes, mouthwashes, oral wipes) [62].

Xylitol showed better results in the reduction of the incidence of caries by decreasing the MS levels in children and maternal transfer from mother to their children [27, 62].

AAPD recommends xylitol syrup (3–8 grams/day in divided doses) to children under the age 4 years and chewing gum, mints, and lozenges (3–8 grams/day in divided doses) to children 4 years of age or older at moderate or high caries risk [62].

- Daily xylitol-wipe application significantly reduced the caries incidence in young children as compared with wipes without xylitol.
- Chewing xylitol-containing gums during the period of primary teeth eruption (6–20 months) may reduce caries in the primary dentition [27].

#### 4.1.2.3. Casein phosphopeptide-amorphous calcium phosphate (CPP-ACP)

One of the calcium-phosphate-based remineralization systems is CPP-ACP which is a bioactive material with a base of milk products. It can provide calcium and phosphate ions in the dental plaque, buffering plaque acidity (pH) and enhancing the remineralization process. In addition, it can participate in the pellicle structure and prevent the adhesion of cariogenic Streptococci to tooth surfaces [63]. CPP-ACP is used as an adjunct preventive agent in patients at a high risk for caries. CPP-ACP is available in the form of chewing gums, mouthwashes, and dental creams. A recent systematic review showed that even though the CPP-ACP seems insignificantly different beside fluorides, it has a remineralizing effect on early caries compared to control or placebo groups. Although some of the studies have shown a synergistic effect of CPP-ACP and fluoride on remineralization, the advantage of it is still controversial when it is used as a first choice supplement instead of fluoride. To confirm the effectiveness of non-fluoride agents, high-quality clinical trials are required while controlling dental caries of preschool children [56].

#### 4.1.2.4. Povidone iodine

Povidone iodine is a solution that releases iodine slowly which has long-term antimicrobial action. It is used as a topical agent in prevention of ECC. Several studies searched the effect of regular applications of 10% povidone iodine and concluded that periodic topical applications reduce risk for ECC development by suppressing the level of *S. mutans* [27].

#### 4.1.2.5. Probiotic supplements

Probiotics are described as viable microorganisms and provide health benefits on the host when used in enough amounts. Most commonly investigated strains belong to the *Lactobacillus*, *Streptococcus*, and *Bifidobacterium* genera as regards probiotics. Within the oral cavity, mechanisms of probiotic action can be explained in two ways as the first as direct interactions with dental plaque by replacement therapy and the second as modulation of both innate and adaptive immune function [64].

In one of the reviews, searching about the prevention effect of probiotic bacteria on ECC suggested that probiotic supplements were better than placebo but concluded that the quality of the evidence was low or very low [65].

The existing evidence supporting the use of silver diamine fluoride, xylitol, chlorhexidine varnish/gel, povidone iodine, remineralizing agents (e.g., casein phosphopeptide amorphous calcium phosphate), and probiotic bacteria for ECC prevention is considered insufficient [54].

Although there is also insufficient evidence for using sealants to reduce incidence of ECC, EAPD recommends fissure sealant application to arrest non-cavitated occlusal caries [48, 54].

## 4.2. Management of cavitated ECC

Although there is insufficient evidence for a traditional restorative approach to the management of caries in the primary dentition; it is well-known that untreated ECC can cause increased treatment costs, delayed physical growth and development, diminished ability to learn, and diminished oral health-related quality of life [54]. Restorative care in the primary dentition is essential for making a significant difference on psychological and social aspects of the child's life.

Although there is insufficient evidence to make recommendations about which material and technique is the most appropriate for restorative treatment in young children, minimally invasive approaches are accepted as advantageous [66].

Atraumatic restorative treatment (ART) was initially developed to provide effective restorative treatment of cavitated ECC, in developing countries where electricity may not be available [66]. ART is a pain-free restorative procedure, low cost, and can be applied outside the clinical setting or when conventional treatment is not available. It involves no local anesthesia or drilling. Caries removal is done by using hand instruments and followed by a restoration with highly viscous glass ionomer cement (GIC). A disadvantage of this treatment is its high rate of failure. Minimally invasive approaches with ART with highly viscous GIC provide some evidence that is beneficial in managing decayed primary teeth. To provide more information on restorative and clinical outcomes of the ART-based approach, further studies with a longer follow-up should be undertaken [67, 68].

New dental materials and techniques for restoring decayed teeth have been developed. However, despite much improvement in dental materials, the failure rates of amalgam, composite fillings and glass ionomers were reported almost at the rate of 58%, 62%, 77%, respectively, over five years. For making decisions to determine the appropriate choice for dental practitioners, limited evidence exists. Primary teeth after pulp therapy or multiple carious surface lesions are generally recommended for restoring with stainless steel crowns. The



Hall technique that involves sealing the carious lesions of primary molars with stainless steel crowns without caries removal and crown preparation has been proposed as a modified technique for stainless steel crowns. The adoption of this technique in a community-based program is considered to be so difficult because of material prices and the operation time required [56].

## 5. Conclusion

ECC is the most common chronic disease among young children and affects children less than 71 months of age, and they currently represent a public health problem in various countries worldwide. There are a multitude of risk factors associated with ECC. Feeding habits and a variety of biological, environmental, and socioeconomic factors are involved in the development of ECC. It can affect a child's well-being, learning ability, and quality of life. It is important to understand the natural history of ECC in order to implement effective preventive strategies. Evidence of effective ECC prevention suggests prenatal and immediate postnatal interventions. Health care professionals should carry out children's caries risk assessments in their first year as part of the children's overall health assessments and children should be reassessed periodically over time. Population-based early childhood health systems hold great potential to reduce the burden of ECC, improve health care efficiency and cost-effectiveness. Early non-operative interventions by the dental professionals, including plaque removal, application of topical fluoride, and usage of sealants, are accepted as important steps for preventing of ECC. Cavitated ECC should be treated restoratively, although there is insufficient evidence to make recommendations about which material and technique is the most appropriate. Restoring is essential for making a significant difference on psychological and social aspects of the child's life.

## Conflict of interest

None declared.

## Notes/thanks/other declarations

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## References

- [1] Hallett KB, O'Rourke PK. Caries experience in preschool children referred for specialist dental care in hospital. *Australian Dental Journal*. 2006;**51**(2):124-129
- [2] Sukuraman A, Pradeep SA. Early childhood caries: Prevalence, risk factors, and prevention. *Frontiers in Pediatrics*. 2017;**5**:157
- [3] American Academy on Pediatric Dentistry; American Academy of Pediatrics. Policy on early childhood caries (ECC): Classifications, consequences, and preventive strategies. Reference Manual. 2016;**39**:617
- [4] Kawashita Y, Kitamura M, Saito T. Early childhood caries. *International Journal of Dentistry*. 2011;**72**(5):320
- [5] Finlayson TL, Siefert K, Ismail AI, Sohn W. Psychosocial factors and early childhood caries among low-income African-American children in Detroit. *Community Dentistry and Oral Epidemiology*. 2007;**35**(6):439-448
- [6] Leong PM, Gussy MG, Barrow SL, de Silva-Sanigorski A, Waters E. A systematic review of risk factors during first year of life for early childhood caries. *International Journal of Paediatric Dentistry*. 2013;**23**(4):235-250
- [7] Ferreira MC, Ramos-Jorge ML, Marques LS, Ferreira FO. Dental caries and quality of life of preschool children: Discriminant validity of the ECOHIS. *Brazilian Oral Research*. 2017;**31**:e24
- [8] Fejerskov O, Kidd E, Nyvad B, Baelum V, editors. *Dental Caries. The Disease and its Clinical Management*. 2nd ed. Oxford: Blackwell Munksgaard Ltd; 2008. 607 p
- [9] Drury TF, Horowitz AM, Ismail AI, Maertens MP, Rozier RG, Selwitz RH. Diagnosing and reporting early childhood caries for research purposes. A report of a workshop sponsored by the National Institute of Dental and Craniofacial Research, the Health Resources and Services Administration, and the Health Care Financing Administration. *Journal of Public Health Dentistry*. 1999;**59**(3):192-197
- [10] Wyne A. Prevalence and risk factors of nursing caries in Adelaide, South Australia. *Pediatric Dentistry*. 1999;**9**:31-36
- [11] Petrola KA, Bezerra ÍB, de Menezes ÉAV, Calvasina P, Saintrain MV, Pimentel GF, Vieira-Meyer A. Provision of oral health care to children under seven covered by bolsa família program. Is this a reality? *PLoS One*. 2016;**11**(8):e0161244
- [12] Congiu G, Campus G, Luglie PF. Early childhood caries (ECC) prevalence and background factors: A review. *Oral Health & Preventive Dentistry*. 2014;**12**(1):71-76
- [13] Thitasomakul S, Thearmontree A, Piwat S, et al. A longitudinal study of early childhood caries in 9- to 18-month-old Thai infants. *Community Dentistry and Oral Epidemiology*. 2006;**34**:429-46

- [14] Ismail AI, Sohn W. A systematic review of clinical diagnostic criteria of early childhood caries. *Journal of Public Health Dentistry*. 1999;**59**(3):171-191
- [15] Azizi Z. The prevalence of dental caries in primary dentition in 4- to 5-year-old preschool children in northern Palestine. *International Journal of Dentistry*. 2014;**2014**:839419
- [16] El-Nadeef MA, Hassab H, Al-Hosani E. National survey of the oral health of 5-year-old children in the United Arab Emirates. *The Eastern Mediterranean Health Journal*. 2010;**16**(1):51-55
- [17] Arora A, Scott JA, Bhole S, Do L, Schwarz E, Blinkhorn AS. Early childhood feeding practices and dental caries in preschool children: A multi-centre birth cohort study. *BMC Public Health*. 2011;**11**:28
- [18] Carvalho DM, Salazar M, Oliveira BH, Coutinho ES. Fluoride varnishes and decrease in caries incidence in preschool children: A systematic review. *A Revista Brasileira de Epidemiologia*. 2010;**13**(1):139-149
- [19] Gökalp S, Güçüz DB, Tekçiçek M, Berberoğlu A, Ünlüer Ş. Oral health profile of children aged five, twelve and fifteen years in Turkey-2004. *Hacettepe Dişhekimliği Fakültesi Dergisi*. 2007;**31**:3-10. (In Turkish.)
- [20] Doğan D, Dülgergil ÇT, Mutluay AT, Yıldırım I, Hamidi MM, Çolak H. Prevalence of caries among preschool-aged children in a central Anatolian population. *Journal of Natural Science, Biology and Medicine*. 2013;**4**(2):325-329
- [21] Divaris K. Predicting dental caries outcomes in children: A “risky” concept. *Journal of Dental Research*. 2016;**95**(3):248-254
- [22] Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children: A systematic review of the literature. *Community Dental Health*. 2004;**21**(1 Suppl):71-85
- [23] Milgrom P, Riedy CA, Weinstein P, Tanner AC, Manibusan L, Bruss J. Dental caries and its relationship to bacterial infection, hypoplasia, diet, and oral hygiene in 6- to 36-month-old children. *Community Dentistry and Oral Epidemiology*. 2000;**28**(4):295-306
- [24] Senesombath S, Nakornchai S, Banditsing P, Lexomboon D. Early childhood caries and related factors in Vientiane, Lao PDR. *The Southeast Asian Journal of Tropical Medicine and Public Health*. 2010;**41**(3):717-725
- [25] Gordon N. Oral health care for children attending a malnutrition clinic in South Africa. *International Journal of Dental Hygiene*. 2007;**5**(3):180-186
- [26] Hajishengallis E, Parsaei Y, Klein MI, Koo H. Advances in the microbial etiology and pathogenesis of early childhood caries. *Molecular Oral Microbiology*. 2017;**32**(1):24-34
- [27] Jayabal J, Mahesh R. Current State of Topical Antimicrobial Therapy in Management of Early Childhood Caries. *ISRN Dentistry*. 2014;**2014**:762458. DOI: 10.1155/2014/762458
- [28] World Health Organization, UNICEF. Global strategy for infant and young child feeding. Geneva: 2003. Available at: <http://www.who.int/nutrition/publications/infant-feeding/9241562218/en/>

- [29] Avila WM, Pordeus IA, Paiva SM, Martins CC. Breast and bottle feeding as risk factors for dental caries: A systematic review and meta-analysis. *PLoS One*. 2015;**10**(11):e0142922
- [30] Guidelines on Prevention of Early Childhood Caries: An EAPD Policy Document. Approved by the EAPD Board November 2008. [Internet]. 2018. Available from: [https://www.eapd.eu/uploads/1722F50D\\_file.pdf](https://www.eapd.eu/uploads/1722F50D_file.pdf) [Accessed: Jan 10, 2018]
- [31] Li Y, Tanner A. Effect of antimicrobial intervention on oral microbiota associated with early childhood caries. *Pediatric Dentistry*. 2015;**37**(3):226-244
- [32] Li Y, Caufield PW, Dasanayake AP, Wiener HW, Vermund SH. Mode of delivery and other maternal factors influence the acquisition of *Streptococcus mutans* in infants. *Journal of Dental Research*. 2005;**84**(9):806-811
- [33] Xiao J, Huang X, Alkhers N, Alzamil H, Alzoubi S, Wu TT, et al. *Candida albicans* and early childhood caries: A systematic review and meta-analysis. *Caries Research*. 2017;**52**(1-2):102-112
- [34] Paglia L, Scaglioni S, Torchia V, De Cosmi V, Moretti M, Marzo G, et al. Familial and dietary risk factors in early childhood caries. *European Journal of Paediatric Dentistry*. 2016;**17**(2):93-99
- [35] Mattos-Graner RO, Li Y, Caufield PW, Duncan M, Smith DJ. Genotypic diversity of mutans streptococci in Brazilian nursery children suggests horizontal transmission. *Journal of Clinical Microbiology*. 2001;**39**(6):2313-2316
- [36] DenBesten P, Berkowitz R. Early childhood caries: An overview with reference to our experience in California. *Journal of the California Dental Association*. 2003;**31**(2):139-143
- [37] Kozai K, Nakayama R, Tedjosasongko U, Kuwahara S, Suzuki J, Okada M, et al. Intrafamilial distribution of mutans streptococci in Japanese families and possibility of father-to-child transmission. *Microbiology and Immunology*. 1999;**43**(2):99-106
- [38] Tabchoury CP, Sousa MC, Arthur RA, Mattos-Graner RO, Del, Bel Cury AA, Cury JA. Evaluation of genotypic diversity of *Streptococcus mutans* using distinct arbitrary primers. *Journal of Applied Oral Science*. 2008;**16**(6):403-407
- [39] Valdez RMA, Duque C, Caiaffa KS, Santos VR, Loesch MLA, Colombo NH, et al. Genotypic diversity and phenotypic traits of *Streptococcus mutans* isolates and their relation to severity of early childhood caries. *BMC Oral Health*. 2017;**17**:115
- [40] Javed F, Feng C, Kopycka-Kedzierawsk DT. Incidence of early childhood caries: A systematic review and meta-analysis. *Journal of Investigative and Clinical Dentistry*. 2017;**8**(4)
- [41] Gaur S, Nayak R. Underweight in low socioeconomic status preschool children with severe early childhood caries. *Journal of the Indian Society of Pedodontics and Preventive Dentistry*. 2011;**29**(4):305-309
- [42] Prokocimer T, Amir E, Blumer S, Peretz B. Birth-weight, pregnancy term, pre-natal and natal complications related to child's dental anomalies. *The Journal of Clinical Pediatric Dentistry*. 2015;**39**(4):371-376

- [43] Lai PY, Seow WK, Tudehope DI, Rogers Y. Enamel hypoplasia and dental caries in very-low birthweight children: A case-controlled, longitudinal study. *Pediatric Dentistry*. 1997;**19**(1):42-49
- [44] Fontana M. The clinical, environmental, and behavioral factors that foster early childhood caries: Evidence for caries risk assessment. *Pediatric Dentistry*. 2015;**37**(3):217-225
- [45] Divaris K. Precision dentistry in early childhood: The role of genomics. *Dental Clinics of North America*. 2017;**61**(3):619-625
- [46] Boraas JC, Messer LB, Till MJ. A genetic contribution to dental caries, occlusion, and morphology as demonstrated by twins reared apart. *Journal of Dental Research*. 1988;**67**(9):1150-1155
- [47] Hajishengallis E, Forrest CB, Koo H. Early childhood caries: Future perspectives in risk assessment. *JDR Clinical and Translational Research*. 2016;**1**(2):110-111
- [48] Kühnisch J, Ekstrand KR, Pretty I, Twetman S, van Loveren C, Gizani S, et al. Best clinical practice guidance for management of early caries lesions in children and young adults: An EAPD policy document. *European Archives of Paediatric Dentistry* Feb 2016; **17**(1):3-12
- [49] Kellesarian SV, Malignaggi VR, de Freitas PC, Ahmed HB, Javed F. Association between prenatal maternal cigarette smoking and early childhood caries. A systematic review. *Journal of Clinical and Experimental Dentistry*. 2017;**9**(9):e1141-e1146
- [50] Douglass JM, Clark MB. Integrating oral health into overall health care to prevent early childhood caries: Need, evidence, and solutions. *Pediatric Dentistry*. 2015;**37**(3):266-274
- [51] Finlayson TL, Gupta A, Ramos-Gomez FJ. Prenatal maternal factors, intergenerational transmission of disease, and child oral health outcomes. *Dental Clinics of North America*. 2017;**61**(3):483-518
- [52] Ng MW, Chase I. Early childhood caries: Risk-based disease prevention and management. *Dental Clinics of North America*. 2013;**57**(1):1-16
- [53] Ballantine JL, Carlson JC, Zandona AGF, Agler C, Zeldin LP, Rozier RG, et al. Exploring the genomic basis of early childhood caries: A pilot study. *International Journal of Paediatric Dentistry*. 2017;**28**(2):217-225
- [54] Twetman S, Dhar V. Evidence of effectiveness of current therapies to prevent and treat early childhood caries. *Pediatric Dentistry*. 2015;**37**:246-253
- [55] Twetman S. Caries prevention with fluoride toothpaste in children: An update. *European Archives of Paediatric Dentistry*. 2009;**10**(3):162-167
- [56] Duangporn D, Kitty JC, Sherry SG, Edward CML, Chun HC. Managing early childhood caries with atraumatic restorative treatment and topical silver and fluoride agents. *International Journal of Environmental Research and Public Health*. 2017;**14**:2-13
- [57] European Archives of Paediatric Dentistry [Internet]. 2009. Available from: [https://www.eapd.eu/uploads/82C0BD03\\_file.pdf](https://www.eapd.eu/uploads/82C0BD03_file.pdf). [Accessed: Jan 20, 2018]



- [58] Scottish Intercollegiate Guideline Network (SIGN) 2005. Prevention and management of dental decay in the preschool child. Available from: <http://sign.ac.uk/pdf/sign83.pdf> [Accessed: Jan 30, 2008]
- [59] Mani AS. Evidence-based clinical recommendations for fluoride use: A review. *Archives of Orofacial Sciences*. 2009;**4**(1):1-6
- [60] Contreras V, Toro MJ, Elías-Boneta AR, Encarnación-Burgos A. Effectiveness of silver diamine fluoride in caries prevention and arrest: A systematic literature review. *General Dentistry*. 2017;**65**(3):22-29
- [61] van Rijkom HM, Truin GJ, van't Hof MA. A metaanalysis of clinical studies on the caries-inhibiting effect of chlorhexidine treatment. *Journal of Dental Research*. 1996;**75**(2):790-795
- [62] American Academy of Pediatric Dentistry. Policy on the use of xylitol. *Reference Manual*. 2015;**39**:617-618
- [63] Zero DT. Dentifrices, mouthwashes, and remineralization/caries arrestment strategies. *BMC Oral Health*. 2006;**6**(Suppl 1):S9. DOI: 10.1186/1472-6831-6-S1-S9
- [64] Allaker RP, Stephen AS. Use of probiotics and oral health. *Current Oral Health Report*. 2017;**4**:309-318
- [65] Jørgensen MR, Castiblanco G, Twetman S, Keller MK. Prevention of caries with probiotic bacteria during early childhood. Promising but inconsistent findings. *American Journal of Dentistry*. 2016;**29**(3):127-131
- [66] Duangthip D, Jiang M, Chu CH, Lo EC. Restorative approaches to treat dentin caries in preschool children: Systematic review. *European Journal of Paediatric Dentistry*. 2016;**17**(2):113-121
- [67] Frencken JE. The state-of-the-ART of ART sealants. *Dental Update*. 2014;**41**:119-124
- [68] Peter A. Restorative outcomes of a minimally invasive restorative approach based on atraumatic restorative treatment to manage early childhood caries: A randomised controlled trial. *Caries Research*. 2016;**50**:1-8