Early Childhood Caries: Causes, Prevention and Management

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Abstract

Early Childhood Caries (ECC) is one of the most prevalent and significant infectious diseases affecting children aged five years and younger worldwide. Defined by the American Academy of Pediatric Dentistry (AAPD) as the presence of one or more decayed, missing, or filled tooth surfaces in any primary tooth in a child under six years of age, ECC poses a substantial burden on both pediatric health and healthcare systems globally. This review article comprehensively explores the etiology of ECC, including microbial, dietary, host, and socioeconomic factors. It further discusses evidence-based preventive strategies such as fluoride application, dietary counselling, oral hygiene practices, and parental education. Clinical management approaches—ranging from non-invasive techniques to pharmacological interventions and restorative procedures—are also addressed. The article emphasises the importance of early dental visits, interdisciplinary collaboration, and community-based public health measures in reducing the burden of ECC.

Keywords: Early Childhood Caries, ECC, dental caries, primary dentition, prevention, management, pediatric dentistry.

Introduction

Early Childhood Caries (ECC) remains one of the most common chronic childhood diseases globally, with a prevalence that continues to challenge public health authorities and dental professionals alike[(1)]. It is an aggressive, rapidly progressing form of dental caries that primarily affects the primary dentition and can have far-reaching consequences on the child's overall health, growth, and quality of life.

According to the American Academy of Pediatric Dentistry (AAPD), ECC is defined as the presence of one 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 of age or younger[(2)]. A severe form, termed Severe Early Childhood Caries (S-ECC), is characterised by atypical, progressive, or acute patterns of caries that manifest in infants and young children.

Epidemiological data indicate that ECC affects approximately 600 million children under the age of five worldwide[(3)]. In developing countries, including India, the prevalence is particularly high, with studies reporting rates between 40% and 85% in preschool-aged children. Despite being largely preventable, ECC continues to be prevalent due to a complex interplay of biological, social, economic, and behavioural factors.

This review aims to provide a comprehensive and updated overview of the aetiology, risk factors, preventive measures, and management strategies for ECC, with the intent to assist dental professionals and public health practitioners in effectively addressing this significant paediatric health concern.

Definition And Classification

The term 'Early Childhood Caries' was formally adopted by the AAPD and the Centers for Disease Control (CDC) in 1994 to replace older and inconsistent terminology such as 'nursing bottle caries,' 'baby bottle tooth decay,' and 'rampant caries.'[(2)] The updated terminology reflects the multifactorial nature of the condition and avoids assigning blame solely to bottle-feeding practices.

Classification

Kaste and Gift (1995) proposed one of the earliest formal classifications:

Type I (Mild to Moderate ECC): Involves caries of the primary molars and/or canines, usually associated with dietary practices.

Type II (Moderate to Severe ECC): Labial/lingual caries of the maxillary anterior teeth with or without involvement of the primary molars, typically associated with inappropriate bottle-feeding or breastfeeding habits.

Type III (Severe ECC): Affects all primary dentition including the mandibular incisors, suggesting extreme dietary habits and poor oral hygiene.

The AAPD further delineated S-ECC as any smooth surface caries in a child younger than three years; or one or more cavitated, missing, or filled smooth surfaces in the maxillary primary anterior teeth in a child aged three to five years; or a decayed, missing, or filled score of ≥4 (age 3), ≥5 (age 4), or ≥6 (age 5) surfaces[(4)].

Aetiology And Risk Factors

ECC is a multifactorial disease influenced by microbial, dietary, host-related, and socioeconomic variables. The interaction of these factors over time determines the onset and progression of the disease.

Microbial Factors

Streptococcus mutans (S. mutans) and Lactobacillus species are the primary pathogens implicated in ECC[5]. S. mutans is highly acidogenic and aciduric, capable of producing organic acids that demineralise enamel. Vertical transmission from mother to child is a well-established mode of S. mutans acquisition, occurring primarily during early infancy through shared utensils, kissing, or other oral contacts.

Candida albicans has also been identified as a synergistic organism in ECC pathogenesis, forming biofilms with S. mutans that enhance virulence and caries progression[5].

Dietary Factors

Frequent intake of fermentable carbohydrates—particularly sugars such as sucrose, glucose, and fructose—provides substrate for microbial acid production[6]. Prolonged and ad libitum bottle-feeding at night with milk, formula, fruit juices, or sweetened liquids exposes the teeth to cariogenic substrates for extended periods, especially when salivary flow is reduced during sleep.

Nocturnal breastfeeding on demand beyond 12 months has been associated with ECC in some studies, though the evidence remains debated. Solid or semi-solid sticky foods and snacking frequency also contribute significantly to caries risk.

Host Factors

The primary teeth, particularly the maxillary incisors, are at high risk due to their location in the oral cavity, thinner enamel compared to permanent teeth, and proximity to salivary gland ducts that pool milk or formula[7]. Defects in enamel formation (hypoplasia), reduced salivary flow (xerostomia), and altered salivary composition may further predispose a child to ECC.

Genetic factors influencing enamel quality, salivary function, and immune response may also play a role in susceptibility. Premature birth and low birth weight have been associated with enamel defects and increased caries risk.

Socioeconomic and Behavioural Factors

Children from low-income families are disproportionately affected by ECC due to limited access to dental care, inadequate nutritional knowledge, and higher consumption of sugary foods and beverages[8]. Parental oral health literacy and attitudes towards child dental health are important determinants. Immigrant communities and certain ethnic groups have shown elevated ECC prevalence, often linked to cultural feeding practices and limited healthcare access.

Clinical Features And Diagnosis

ECC typically initiates on the smooth labial surfaces of the maxillary primary incisors—an area that is easily visible with adequate examination. The earliest sign is the appearance of white spot lesions (WSL) along the gingival margin, representing areas of subsurface demineralisation[3].

If unchecked, lesions progress rapidly to cavitation, brown or black discolouration, and ultimately pulpal involvement. The mandibular incisors are usually spared initially due to protection by the tongue and proximity to submandibular salivary glands.

Clinical diagnosis involves a thorough history of feeding, dietary habits, and oral hygiene practices combined with a visual-tactile examination using a dental explorer and adequate light source. Adjunctive diagnostic tools include:

Bitewing and periapical radiographs to detect interproximal and deep caries.

DIAGNOdent laser fluorescence for early caries detection.

Quantitative Light-induced Fluorescence (QLF) for white spot lesion assessment.

Prevention Of Ecc

Prevention is the cornerstone of ECC management. Strategies must be multifaceted, addressing microbial, dietary, host, and social risk factors simultaneously.

Parental and Caregiver Education

Anticipatory guidance, provided ideally during the perinatal period, informs parents about ECC risk factors, appropriate feeding practices, the importance of oral hygiene from birth, and the need for early dental visits[2]. Educating mothers about their own oral health and S. mutans transmission is a critical preventive measure.

Dietary Counselling

Parents should be counselled to avoid prolonged bottle use, eliminate nocturnal bottle-feeding with milk or sweetened beverages, and transition to a cup by the first birthday[6]. Reducing sugar intake frequency, substituting water for sweetened drinks, and avoiding sticky snacks between meals are key dietary recommendations.

Oral Hygiene Practices

Oral hygiene should begin even before the eruption of primary teeth by wiping the gums with a clean damp cloth. After tooth eruption, a soft-bristled toothbrush should be used[7]. The AAPD recommends using a smear of fluoride toothpaste (grain of rice-sized) for children under three and a pea-sized amount for children aged three to six, twice daily.

Fluoride

Fluoride remains the most effective agent for caries prevention. Its mechanisms include remineralisation of early

enamel lesions, inhibition of demineralisation, and interference with bacterial enzyme function[9]. Fluoride modalities include:

Fluoridated water (optimal level: 0.7 ppm in India/USA).

Fluoride toothpaste.

Professionally applied fluoride varnish (2.26% NaF), recommended every 3–6 months for high-risk children.

Silver Diamine Fluoride (SDF), a newer minimally invasive agent, can arrest active carious lesions in young children.

Xylitol

Xylitol, a naturally occurring five-carbon sugar alcohol, is non-fermentable by S. mutans and has demonstrated significant caries-preventive properties in several clinical studies[10]. Maternal xylitol consumption during the perinatal period has been shown to reduce S. mutans transmission to infants.

Pit and Fissure Sealants

Though less relevant in primary teeth with ECC confined to smooth surfaces, sealants applied to occlusal surfaces of primary molars may prevent caries in high-risk children.

First Dental Visit

Both the AAPD and the Indian Society of Pedodontics and Preventive Dentistry (ISPPD) recommend that a child's first dental visit should occur by the eruption of the first primary tooth or by the child's first birthday, whichever comes first[2].

Management Of Ecc

Management of ECC depends on the extent and severity of carious involvement, the child's age and cooperation level, and available healthcare resources. A stepwise, age-appropriate approach is recommended.

Non-invasive and Minimally Invasive Management

For early or incipient lesions (white spot lesions), non-invasive approaches aim to remineralise the enamel. These include:

Fluoride varnish application.

Silver Diamine Fluoride (SDF): Highly effective in arresting cavitated lesions, particularly useful in uncooperative children or low-resource settings. SDF causes permanent black staining of arrested lesions, which may be a cosmetic concern.

Casein Phosphopeptide-Amorphous Calcium Phosphate (CPP-ACP): Applied as a cream or paste to promote remineralisation.

Ozone therapy: Emerging evidence supports the use of ozone gas or ozonised water for non-invasive caries arrest.

Pharmacological Management

Chlorhexidine gluconate (0.12%) mouthrinse or varnish has antimicrobial activity against S. mutans and may be used as an adjunct in high-risk children, though its role in ECC prevention remains supplementary due to limited long-term evidence.

Restorative Management

When cavitation is evident, restorative intervention is necessary. Options include:

Composite resin or Glass Ionomer Cement (GIC) restorations: GIC is particularly useful in young children due to fluoride release and moisture tolerance.

Stainless Steel Crowns (SSC): Considered the gold standard for multi-surface carious lesions in primary molars, especially following pulp therapy. They offer durability and full coronal coverage.

Strip crowns: Prefabricated aesthetic crowns used for maxillary primary incisors with ECC.

Zirconia crowns: Increasingly popular for their excellent aesthetics and biocompatibility in anterior restorations.

Pulp Therapy

Depending on the degree of pulpal involvement, pulp therapy options include:

Indirect Pulp Capping (IPC): For deep caries with no signs of irreversible pulpitis.

Direct Pulp Capping (DPC): When minimal pulp exposure occurs in a vital tooth.

Pulpotomy: Removal of the coronal pulp tissue, followed by placement of Formocresol, Ferric Sulfate, MTA (Mineral Trioxide Aggregate), or Biodentine as dressing agents.

Pulpectomy: Complete removal of radicular pulp tissue, indicated in teeth with irreversible pulpitis or necrosis, followed by obturation with zinc oxide eugenol or iodoform paste.

Extraction

Severely decayed teeth with poor prognosis may require extraction, particularly when preserving the tooth risks spread of infection, chronic pain, or systemic complications. Space maintenance should be considered following premature primary tooth loss.

Behaviour Management

Managing young children in the dental clinic requires the use of appropriate behaviour guidance techniques, including tell-show-do, positive reinforcement, voice control, and hand-over-mouth exercise (HOM) when appropriate. In uncooperative or very young children, conscious sedation or general anaesthesia may be indicated for comprehensive dental rehabilitation.

Public Health Implications

ECC has significant socioeconomic implications. Untreated dental caries in young children leads to pain, infection, sleep disturbances, difficulty eating, poor weight gain, school absenteeism, and reduced quality of life[8]. The financial costs of treating ECC under general anaesthesia are substantial and place a burden on healthcare systems, particularly in resource-limited settings.

Public health strategies should focus on community water fluoridation, school-based dental health programmes, subsidised dental care, and nationwide oral health education campaigns targeting mothers and caregivers during the prenatal and postnatal periods.

Discussion

ECC is a preventable yet widespread condition that reflects systemic inequities in healthcare access and health literacy. Despite advances in preventive dentistry, high ECC rates persist globally, particularly in low-income communities. The shift towards non-invasive and minimally invasive strategies, exemplified by the growing use of Silver Diamine Fluoride and Atraumatic Restorative Treatment (ART), represents a promising direction for managing ECC in resource-limited and paediatric settings.

The multifactorial nature of ECC demands a multidisciplinary approach involving paediatricians, nurses, nutritionists, and community health workers alongside dental professionals. Maternal health education and the use of xylitol to reduce vertical transmission offer upstream preventive benefits that are increasingly supported by evidence.

Teledentistry and mobile dental clinics have emerged as potential avenues for extending preventive dental care to underserved rural populations. Policy-level interventions—including sugar taxation, regulation of baby food marketing, and integration of oral health into maternal and child health programmes—are critical for sustained reduction in ECC prevalence.

Conclusion

Early Childhood Caries is a complex, multifactorial, and highly prevalent disease with significant consequences for children's health and wellbeing. A thorough understanding of its aetiology, risk factors, preventive measures, and management strategies is essential for dental practitioners and public health professionals. Early intervention, parental education, dietary counselling, fluoride application, and appropriate restorative care form the pillars of an effective ECC management programme. Greater emphasis on prevention, interdisciplinary collaboration, and equitable healthcare access is necessary to reduce the global burden of ECC and ensure optimal oral health outcomes for all children.

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References

1. Drury TF, Horowitz AM, Ismail AI, Maertens MP, Rozier RG, Selwitz RH. Diagnosing and reporting early childhood caries for research purposes. *J Public Health Dent.* 1999;59(3):192–7.
2. American Academy of Pediatric Dentistry. Policy on early childhood caries (ECC): classifications, consequences, and preventive strategies. *Pediatr Dent.* 2020;42(6):60–2.
3. Tinanoff N, Baez RJ, Diaz Guillory C, Donly KJ, Feldens CA, McGrath C, et al. Early childhood caries epidemiology, aetiology, risk assessment, societal burden, management, education, and policy: Global perspective. *Int J Paediatr Dent.* 2019;29(3):238–48.
4. Ismail AI, Sohn W. A systematic review of clinical diagnostic criteria of early childhood caries. *J Public Health Dent.* 1999;59(3):171–91.
5. Berkowitz RJ. Mutans streptococci: acquisition and transmission. *Pediatr Dent.* 2006;28(2):106–9.
6. Marshall TA, Levy SM, Broffitt B, Warren JJ, Eichenberger-Gilmore JM, Burns TL, et al. Dental caries and beverage consumption in young children. *Pediatrics.* 2003;112(3 Pt 1):e184–91.
7. Dye BA, Vargas CM, Lee JJ, Magder L, Tinanoff N. Assessing the relationship between children's oral health status and that of their mothers. *J Am Dent Assoc.* 2011;142(2):173–83.
8. Casamassimo PS, Thikkurissy S, Edelstein BL, Maiorini E. Beyond the dmft: the human and economic cost of early childhood caries. *J Am Dent Assoc.* 2009;140(6):650–7.
9. Marinho VCC, Worthington HV, Walsh T, Clarkson JE. Fluoride varnishes for preventing dental caries in children and adolescents. *Cochrane Database Syst Rev.* 2013;(7):CD002279.
10. Milgrom P, Söderling EM, Nelson S, Chi DL, Nakai Y. Clinical evidence for polyol efficacy. *Adv Dent Res.* 2012;24(2):112–6.