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Review Article

Early childhood caries- Etiology, prevention and management: A Review

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ABSTRACT

Early Childhood Caries (ECC) is a severe debilitating disease affecting the primary dentition of young children in both developing and industrialized countries. Historically, the management of ECC has a global scenario showing operative management of the disease. The contemporary method of preventing the carious process mainly focuses on dietary factors, host factors and removal of plaque biofilm. Enamel demineralization is caused by acidogenic and aciduric bacteria. Colonization of microorganisms in the mouth of children can occur via horizontal or vertical transmission or both. The bacteria present in dental plaque metabolize sugars and starches to produce acids, which lowers the pH in the mouth and promotes loss of minerals from the tooth surface. Sucrose is the most common sugar and when metabolized, produces dextrans which promote superior bacterial adhesion to teeth. Visible plaque on the labial surfaces of the maxillary incisors were strongly associated with the caries development. Saliva buffers plaque acids, aids in oral clearance and acts as a reservoir for minerals to assist in the mineralization of enamel. Most studies have put forward a correlation between bed time use of bottles with sweetened contents especially lactose. One of the factors included by Newbrun in factors causing dental caries is time. The CPP-ACP complex, an effective remineralizing complex preventing the acidic environment reduce incidence of caries. Conventional probiotics comprised of lactobacilli and bifidobacterium which were mainly of intestinal origin; the contemporary probiotic research has brought forward that oral diseases like dental caries, periodontal problems and candida infections have direct link with oral microbial disequilibria. A promising new approach towards primary prevention of the disease is to target the infectious component of Early Childhood Caries by preventing or delaying acquisition of Streptococcus mutans at an early age.

1. Introduction

Early Childhood Caries (ECC) is a severe debilitating disease affecting the primary dentition of young children in both developing and industrialized countries, the term is recommended by the Centers for Disease Control and Prevention (CDCP) to describe a unique pattern of carious lesions in infants, toddlers, and preschool children.[1,2] Severe Early Childhood Caries (s ECC) is the severe form of the disease, seen to develop soon after eruption of primary teeth on smooth surfaces and may leave a lasting detrimental effect on the dentition unlike any other disease.[3] During the era of SARS-CoV-2 pandemic when "social distancing" is the new normal preventive cure of ECC becomes utmost important.[4] ECC is not self limiting and it requires professional intervention to prevent serious complications (dental pain, infections, abscesses) which further may lead to malnutrition and gastrointestinal disorders and may even cause difficulty during sleep, concentration deficits, distraction from learning and restricted activity, thus affecting the overall quality of life and health of the individual and their family.[5]

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Clarke et al suggested that Early Childhood Caries may be a risk factor for iron deficiency anemia which affects the development of the child. Pain and stress caused due to sECC increases the secretion of glucocorticoids which in turn inhibits the growth hormone secretion. Historically, the management of ECC has a global scenario showing operative management of the disease. This has two extremes: The highly interventive, invasive and expensive treatment procedures often under general anesthesia or the cheaper, easier alternative; to wait for the carious primary teeth to exfoliate hoping it does not cause complications in the interim.[6] The contemporary method of preventing the carious process mainly focuses on dietary factors, host factors and removal of plaque biofilm.

2. Etiology

Enamel demineralization is caused by acidogenic and aciduric bacteria as intake of sucrose cause a rapid fall of pH in tooth adherent biofilms to 5.0 or below, leading to a so-called dysbiotic microbiome. Colonization of microorganisms in the mouth of children can occur via horizontal or vertical transmission or both, they utilize fermentable sugar to form weak acids which is the cause of initiation of caries. Thus, exposure to sugar is a determining factor in the causation of caries. (Figure 1).

2.1. Cariogenic microorganisms

Various microorganisms like, *Streptococcus mutans (MS) and Streptococcus sobrinus* have a strong association with initiation of dental caries; *Lactobacilli* helps in progression of the lesion. Other microbes like, *Actinomyces gerencseriae* were associated with caries initiation while *Bifidobacterium* species was associated with deep carious lesions. These bacteria are seen to be vertically transmitted through saliva from mother to child especially during first 19-31 months (*Window of infectivity*). Poor oral hygiene of the mother and frequent exposure to sugar increases the chances of transmission of infection to the child. Horizontal transmission of *MS* may also occur between siblings and care givers. Children having higher *MS* levels in saliva were 5 times more prone to have dental caries.[3]

The bacteria (*MS*) present in dental plaque metabolize sugars and starches to produce acids, which lowers the pH of the saliva and causes loss of minerals from the surface of the teeth. Minerals in the oral cavity including fluoride are redeposited on the tooth surface once the neutral pH is restored (normally after approximately 20 minutes). As this process is dynamic, the tooth surface remains intact and sound as long as minerals are replaced. However, a prolonged pH drop and frequent net loss of minerals lead to weakening and eventual cavitation of the tooth surface. It is also seen that infants delivered by cesarean section acquire MS earlier due to aseptic conditions during delivery and the atypical microbial environment increases its colonisation rates.[1] (Table 1)

2.2. Substrate

There is overwhelming evidence that sugars (such as sucrose, fructose and glucose) and other fermentable carbohydrates (such as highly refined flour) play a role in the initiation and development of dental caries. Among these; Sucrose is the most common sugar and when metabolized, produces dextrans which promote superior bacterial adhesion to teeth. It is therefore considered the most important substrate in the establishment of cariogenic bacterial flora. It now appears that the frequency of intake of sucrose is more important than the total amount of sugar consumed. The smaller sized fermentable sugar molecules allows salivary amylase to split the molecules into smaller parts that can be easily metabolized by plaque bacteria in turn producing acidic end-products causing demineralization of teeth. It has been reported by some authors that there is a positive correlation between incidence of dental caries and intake of sugar where fluoridation is negligible and oral hygiene is bad; the best evidence being countries where level of free sugar consumption is 40-55 gram per person per day, the incidence of dental caries is high.[2]Thus, if sugars are retained in the mouth for prolonged periods like 20-40 minutes, oral bacteria begin to produce acids thus lowering the pH and contributing to demineralization. Authors have reported the oral clearance of sucrose is the quickest while sorbitol and food residues stay longer in the mouth.[3]Although breast-feeding is essential in providing the best possible nutrition to the infants, the American Association of Pediatric Dentistry (AAPD) cautions that frequent breast-feeding at night and on demand after eruption of teeth maybe implicated in contributing to the development of ECC.[2] So, the frequency of sugar, retentiveness of food, consumption of milk based formulas or Non Milk Extrinsic Sugars(NMES) for infant feeding are all considered etiologic factors causing caries.

Many liquid medications including cough drops prescribed for children contain a variety of ingredients that can make the teeth more susceptible to decay. The sugar in the medication combined with the acids lowers the pH levels and cause high acidity in the oral environment which can be a detrimental for a child's teeth.

Ingredients such as high fructose corn syrup and sucrose contribute to decay by dissolving enamel, causing erosion when the cariogenic bacterial strains in the mouth break down the sugars and form acids that attack the enamel of the teeth. Also, certain ingredients such as citric acid and antihistamine syrups can wear down the enamel of the teeth.

Saliva helps to naturally rinse the sugars and acids away from the teeth but in certain medications and cough syrups, the addition of alcohol also has a drying effect on the mouth. — so with less saliva present, the sugars and acids remain

in the mouth for longer time span leading to adverse effect on teeth. These risks can be magnified if the medication is taken before bedtime as less saliva is produced during sleep thus allowing sugar and acids to remain in contact with the teeth surfaces for a longer period further increasing the risk for decay.

Although most parents are aware of the conditions caused by these sweetened syrups and recognize that pediatric medicines can cause teeth problems, a high percentage of the parents are still unable to establish a clear cause and effect relationship between them. Pediatric formulations without added sugar should be made available so that professionals could provide better guidance and more intensive care on the proper oral hygiene maintenance regime after use of these medicines. Therefore, even the pharmaceutical industry needs to be more aware of this problem and should prepare pediatric medicines without the presence of sucrose.

2.3. Plaque

Dental caries is an infectious disease that is modified by diet and a number of other factors. There is very strong evidence that MS is involved in the development of caries in both children and adults. Visible plaque on the labial surfaces of the maxillary incisors were strongly associated with the caries development. The plethora of microorganisms and their toxic by-products in the dental plaque matrix adherent to the tooth surfaces and the inter-dental crevices may cause dental caries and periodontal infections.[7]

2.4. Susceptible tooth/host

Several factors can predispose an individual or indeed a particular tooth to dental caries. These may include immunological factors, reduced saliva, immature enamel and defects of the tooth tissues. Because enamel is immunologically inactive, the main immune defence against MS is provided largely by salivary secretory Immunoglobulin A (IgA), gingival or crevicular fluid. As children become infected with oral microorganisms, they develop salivary IgA antibodies. In addition to providing specific immunological factors, the saliva acts as an important protective factor. Saliva buffers plaque acids, aids in oral clearance and acts as a reservoir for minerals to assist in the mineralization of enamel. Teeth erupt into the mouth with immature enamel following which the process of enamel maturation continues after tooth eruption, so that teeth become less susceptible to decay over time. The enamel maturation takes place by incorporating fluoride and other ions available in the saliva. Therefore, a tooth is most susceptible to caries immediately after eruption until final maturation has occurred.[2]

2.5. Infant feeding pattern

Though dietary practices are significant, its role is secondary in causing dental caries. Inappropriate frequency of use of the baby bottle plays a critical role in the incidence and severity of ECC. Most studies have put forward a correlation between bed time use of bottles with sweetened contents especially lactose. Breast feeding provides perfect nutrition for newborns and it has caused reduced risk of gastrointestinal and respiratory infections. The frequency and duration of contact of human milk with the enamel of teeth has been shown to result in acidogenic oral environment and shifting the demineralization-remineralization equilibrium towards demineralization especially after the age of 12 months.[3]

2.6. Saliva

Prolonged night time use of baby bottle along with less saliva production at night results in higher levels of lactose in the resting saliva. This causes a shift in the equilibrium towards demineralization thus causing dental caries due to reduced salivary flow rate. Saliva gives adequate protection to teeth during sleep and has other factors like oral clearance, buffering capacity and antimicrobial properties which are some of the important determinants marking the reduction in caries development. It has anti microbial proteins like lysozyme, lactoferrin, agglutinins which are significant in caries prevention and also several organic molecules like mucins, fibronectins, glycoproteins, lysozymes, secretory immunoglobulins, etc which agglutinates oral bacteria and helps in its removal.[1]

2.7. Time

One of the factors included by Newbrun in factors causing dental caries is time. The more prolonged the exposure of the teeth to sugar and plaque biofilm, the higher the risk of carious exposure. Hence brushing or cleaning of mouth is essential to avoid the lowering of salivary pH causing an acidic environment which causes demineralization of tooth structure.[8]

2.8. Socioeconomic factors

The correlation between ECC and socioeconomic status is well documented suggesting that children who live in impoverished state belonging to low ethnic or racial minorities, born to single mothers or parents with low educational level are more commonly found with higher incidence and prevalence of ECC. In a study conducted by Tang JM et al they concluded that children whose parents are in the lowest income group have four times higher Decayed Missing Filled Teeth (DMFT) score than children whose parents are in the highest income group.[2]

3. Management

Previously, the aim of managing childhood caries was to remove all tooth tissues that showed any signs of decay and restore the cavity no matter whether the lesion was active or arrested.[4] Nowadays, the aims of management of the lesions have evolved considerably, by regeneration and remineralization through preventive techniques, arresting the disease process and maintenance of as much tooth as possible.[6]

The management of ECC includes prevention, methods to arrest the progession of the carious lesions or restoration of the cavitated tooth.(Tables 2 and 3)

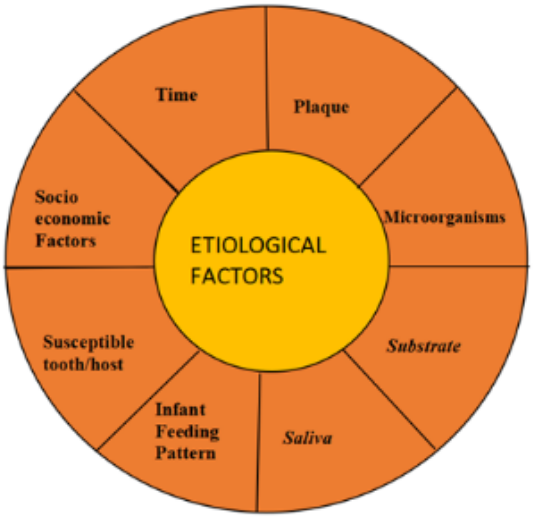

Figure 1: Etiological factors causing ECC

3.1. Preventive measures

3.1.1. Prevention of vertical transmission

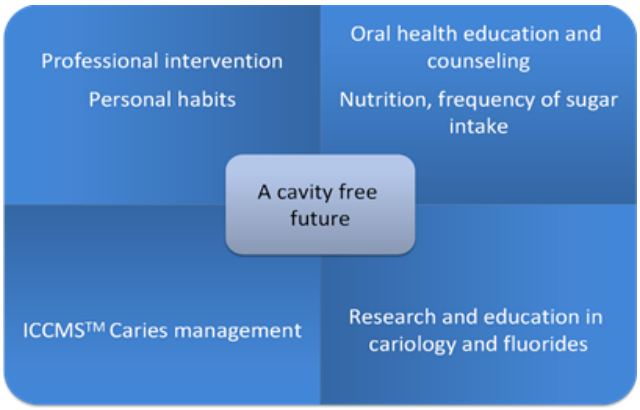

Figure 2: The Caries free future: The multi factorial caries requires multifaceted approach for a caries free future[9]

Table 1: Caries causing microorganisms and their activities

Microorganisms	Activity
Streptococcus mutans	Initiation of Dental caries
S. salivarius	Abundant in oral cavity
Lactobacilli fermentum	Progression of Dental caries
Streptococcus sobrinus	Strongly associated with deep carious lesions
Actinomyces israelli	Most predominent species in plaque of ECC
Bifidobacterium species	Associated with deep carious lesions
Veilonella	Seen in lactate users, abundant in carious dentine.
Enterococci	Found in oral cavity of medically and immunologically compromised patients

Table 2: Methods to arrest the progession of the carious lesions or restoration of the cavitated tooth.

By applying one of the following procedures these above two aims can be achieved
Topically removing the plaque by appropriate brushing of teeth
Sealing in with restorative materials
Use of topical and systemic fluoride
Use of tooth-paste, varnish ,silver diamine fluoride
By reducing the frequency of sugar intake.

Table 3: The preventive measures and the interventive approaches in a glance.

Preventive Measures	Interventive Approaches
Prevention of vertical transmission	Repair of non-cavitated lesion
Oral health education	Sealing in the more extensive lesions
Fluoride application	Repair of active cavitated lesions
CPP-ACP	Selective caries removal
Probiotics	Hall technique

In many of the cross-sectional studies conducted previously, it was confirmed through serotyping, genotyping and bacteriocin profiling that the principal source of MS infection in the infants are from their mothers and these infants may exhibit infection as the same level as their mothers.[13] Early establishment of salivary MS colonies in young children marks the future with extensive caries in the primary dentition[14] It was observed that infants with MS colonisation by the age of 2 years are more likely to develop dental caries later on in primary and permanent dentitions in comparison with children in whom colonisation of MS was delayed or did not occur at all.[13] It has been interesting to find that the mother and close family members often

Table 4: The treatment interventions and management of early childhood caries

Intervention	Management	Examples
Repair of non-cavitated lesion	Managing non-cavitated lesions to stop the progress of caries or reverse the lesion or prevent it from occurring in the first place may involve different types of intervention.[1] These interventions may depend on the child's age, social, behavioural and medical history of the infant and the extent of lesion progression.	Application of topical fluoride, oral hygiene maintenance
Sealing in the more extensive lesions	Fissure sealants (low-filled resin based material) prevents decay of occlusal surfaces and prevent further progression of lesion by preventing dietary sugars from gaining access to the fissures on the tooth surface.[6]	Pit and Fissure Sealants, Preventive Resin Restorations
Repair of active cavitated lesions	Cavitated lesion have exposed collagen fibrils in soft dentin making it difficult to remove the biofilm from the exposed tooth surface so sealing in strategies may be helpful.[8]	Restoration of cavity done with Glass ionomer cement (GIC), amalgam or Composite etc
Selective caries removal	Selective causing hand instruments and the edge of the cavity cleared of any diseased tissue to provide a sound surface for the restorative material to adhere to.[4]	Atraumatic Restorative Treatment (ART)
Hall technique	Recent studies claim it is a superior option where no LA is required, no tooth preparation is needed but provides a good seal.[10]	Pre-formed stainless steel crown of appropriate size is cemented over the tooth.[4]
Fixed dentulous space maintaining appliance for upper anteriors	The factors associated with anterior tooth loss include: tipping of adjacent teeth, over-eruption of antagonist teeth, midline deviation, masticatory impairment, speech problems and lingual dysfunction.[11] The reasons for replacing the edentulous upper anterior span with an aesthetic appliance constitute parental desire, space maintenance, restoration of aesthetics and maintenance of function.[12]	Groper's appliance
Managing a child with dental pain	Child with abscess or swelling associated with decayed tooth may often be anxious or fretful. Use of local anesthetic and radicular procedures may be the mode of treatment.	Direct and Indirect pulp capping for pin point exposure or pulpotomy, pulpectomy for deeper infection.[4]
General anesthesia and sedation	The invasive and anxiety provoking nature of treatment like extraction or restoration may be stressful for the child. Thus completing the treatment under General Anesthesia or Conscious sedation.	Pulp Therapy (pulpectomy, pulpotomy), extraction followed by space maintainersMultisurface or multiple caries lesion, Full mouth rehabilitation with stainless steel crowns and zirconia crowns

have the same strain of cariogenic bacteria.[14] In his study, Ercan E et al. conducted a preventive regimen among mothers of new-born children for the first 2-3 years like oral hygiene instructions, scaling, restorative treatments and daily use of chlorhexidine mouthwashes; which showed a major reduction in the salivary MS levels in the children and also in their subsequent siblings thus causing a decline in their future caries experience.[13] Another study conducted by *Turksel Dulgergil C et al.* he concluded that the mothers who received extensive preventive programme over a period of 2 years showed a decline in the levels of MS in their saliva and their children demonstrated a delay in the colonisation of MS bacteria.[14] Also, *Kishi M et al.* concluded that there is a relationship between the high quantitative levels of S. mutans and S. sobrinus in the saliva of the mothers of the 54 mother-and-child included in his study with the caries incidence in the children due to MS colonisation in the children's saliva.[15]

3.1.2. Oral health education

One of the primary steps of management of ECC starts before the disease has set in. Motivational interviewing of the parents to establish a rapport and trust between parent and clinician. Parents should be advised that regular dental visits are beneficial for the oral health of the child. No added sugar in juice, weaning off from using baby bottle while in bed, chewing xylitol gum, brushing regularly with fluoridated dentrifices are some of the management goals in the prevention of new carious lesions.[16]

3.1.3. Fluoride application

Application of topical fluoride increases the number of fluorapatite crystals on surface enamel, thus reducing enamel solubility to acid attack.[17]Topical fluoride

application treatment are advised to be supplemented with tooth-brushing, using fluoridated tooth paste and mouthwashes.[16,18] Applying fluoridated varnishes by a dental professional 2-4 times per year.[19] Using high fluoride tooth paste containing 2800 parts per million fluoride for children over 10 years old.[11,20]

3.1.4. CPP-ACP

Casein phosphopeptide–amorphous calcium phosphate (CPP–ACP) is obtained from milk and milk products. It has proved helpful in the treatment of white spot lesions, early childhood caries, root caries, dental erosion and remineralization of carious lesion. *Mellanby* found that certain milk derivatives has caries preventive effects. At University of Melbourne, *Reynolds et al.* found that digesting casein, a milk protein by enzyme trypsin, then combining it with calcium phosphate followed by ultra-filtration formed casein phosphopeptide (CPP) at a pH ranging from 5 - 9 and *Aaron S. Posner* was the first to describe amorphous calcium phosphate (ACP) in the mid 1960s as a good bio-active and osteoinductive substance with biodegradability. ACP may produce fluorapatite in the presence of fluoride ions on the surface of the teeth. Thus, the remineralization of the subsurface enamel by the fluoride ions in the presence of unstable ACP becomes a challenge. Then the CPP-ACP complex, an effective remineralizing complex was patented by the University of Melbourne. CPP-ACP can be incorporated into the pellicle to inhibit *Streptococcus mutans and Streptococcus sobrinus* adherence to the tooth surface in exchange of the albumin protein thereby preventing the acidic environment from forming and the calcium and the phosphate ion levels in the plaque maintain supersaturation due to localization of ACP at the enamel surface. Each molecule of CPP-ACFP (CPP-ACP with fluoride ions) has 25 calcium ions, 15 phosphate ions and 5 fluoride ions so that calcium, phosphate and fluoride are available in one product and is considered better than when CPP-ACP is used alone. CPP-ACP has found profound use being available in the form of chewing gums dentrifices, mouth-rinses, lozenges, topical creams, sprays, energy drinks as well as dental restorative cements. Though it is not indicated in patients with lactose intolerance, it has provided a new arena to preventive dentistry as a non fluoridated remineralizing agent.[21,22]

3.1.5. Probiotics

The World Health Organization (WHO) has defined probiotics as "live microorganisms which, when administered in adequate amounts, confer a health benefit on the host".[23] When sugared food or drinks.

The multi factorial caries requires multifaceted approach for a caries free future.[9] are ingested at a frequent interval, the oral environment becomes increasingly aciduric and acidogenic. Over a period of time, these changes in the oral environment causes a gradual shift of the demineralization-remineralization cycle towards loss of mineral ions from the tooth leading to development of a carious lesion. Several strategies have been applied for preventing dental caries until recently when live organisms seen to have beneficial impact like preventing cellular adhesion and pathogenic bacterial invasion in the digestive system may be used for oral health care to reduce incidence of caries, control plaque pH, diminish bacterial count in saliva.[24] Though conventional probiotics comprised of lactobacilli and bifidobacterium which were mainly of intestinal origin; the contemporary probiotic research has brought forward that oral diseases like dental caries, periodontal problems and candida infections have direct link with oral microbial disequilibria.[23] *Streptococcus salivarius* strains K12, M18 and JH have been currently employed as probiotics. *Streptococcus thermophilus* are considered effective oral probiotics.[25] In a study conducted by *Schwendicke et al. Streptococcus thermophilus* showed due to its urease activity, it is acidic in nature which increases in acidic environment. It would let *Streptococcus thermophilus* to replace lactobacilli like other carcinogenic microbes from biofilms, thus limiting the cariogenic effect. It have also limited and inhibited MS growth bio-film formation ability in comparison to lactobacilli probiotic strain.[26] In a studies conducted by *Burton et al., Di Pierro et al. Streptococcus salivarius* M18 strain taken twice regularly showed reduction of plaque formation and caries development in young children.[27,28] *Walker et al.* in his study showed that *S. salivarius* JH strain can produce SalE, a new bacteriocin, and EPS-hydrolyzing enzyme — dextranase. Bacteriocin has anti-MS activity while dextranase causes increased matrix penetration and better efficiency of killing in some anti-bacterial agents, has relatively broad anti-bacterial spectrum towards oral pathogenic microbes like *S. mutans* and *S. sobrinus* in vitro.[29,30] *Alamoudi NM et al.* conducted a study on the effect of probiotic Lactobacillus reuteri and concluded that the consumption of a probiotic lozenges improved salivary buffer capacity and reduced cariogenic bacterial counts and plaque accumulation.[31]

4. Interventive Approaches: (Table 4)

Restorative intervention of proximal dental lesions also intervention of cavitated and actively progressing lesions to minimize or arrest the continual caries development.[2]

5. Conclusion

It is essential to explore the various ways to prevent ECC rather than conducting trials to test the effectiveness of interventions against pre-identified risk factors of childhood caries.[32] A promising new approach towards primary prevention of the disease is to target the infectious

component of Early Childhood Caries by preventing or delaying acquisition of *Streptococcus mutans* at an early age by suppressing the infection in the mothers. Another approach is to prevent the accumulation of S. mutans to pathologic levels by topical application of antimicrobial agents.[33] Cleaning children's teeth with fluoride toothpastes has been clearly shown to provide protection against childhood caries. There is some evidence that the use of xylitol gum by mothers may also offer protection to their children.

6. Source of Funding

None.

7. Conflict of Interest

None.

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