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Review Article

Dental Caries: Etiology, Epidemiology, Prevention, and Modern Management – A Comprehensive Review

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Abstract: Dental caries remains a major global health challenge, affecting billions through biofilm-mediated demineralization driven by dietary sugars and acidogenic bacteria. This comprehensive review synthesizes 2020-2025 evidence from systematic reviews and meta-analyses, covering pathogenesis (pH <5.5 threshold), epidemiology (72% prevalence in pediatric populations), advanced diagnostics (fluorescence scanners with ICC > 0.8 agreement), and interventions including fluoride (20-38% caries reduction) and silver diamine fluoride (SDF). High-burden regions, including India, demonstrate rising early childhood caries (ECC) trends projected to 2040 without significant policy interventions. Modern management emphasizes minimally invasive techniques (selective caries removal) and comprehensive risk assessment (CAMBRA protocols). Novel agents such as postbiotics show promising preliminary results for biofilm inhibition. Integrated prevention strategies—combining fluoride, sealants, dietary modifications, and health promotion—offer equitable solutions while linking oral health to systemic well-being [6]. These findings underscore the urgent need for tailored, evidence-based strategies addressing vulnerable populations in resource-limited settings.

Keywords: Dental Caries, Demineralization, Streptococcus Mutans, Fluoride, Early Childhood Caries, Prevention, Systematic Review, Meta-Analysis.

1. INTRODUCTION

Dental caries, commonly known as tooth decay, represents one of the most prevalent chronic diseases worldwide, yet remains largely preventable through evidence based interventions [1-5]. The disease arises from dysbiotic oral biofilms that ferment dietary carbohydrates into organic acids, demineralizing the crystalline hydroxyapatite structure of tooth enamel when plaque pH falls below 5.5—the critical threshold for subsurface lesion initiation [6-8]. This dynamic process reflects ecological imbalance rather than infectious disease, distinguishing caries from traditional bacterial infections [1].

The pathophysiology involves four interdependent ecological pillars [9]:

1. Acidogenic and aciduric microorganisms (particularly *Streptococcus mutans* and *Lactobacilli*).
2. Frequent dietary carbohydrate exposure, especially sucrose
3. Susceptible tooth surfaces lacking protective factors
4. Time for demineralization to exceed remineralization capacity

Host factors, primarily salivary function, counteract caries development through multiple mechanisms: buffering capacity (bicarbonate systems), remineralization (calcium and phosphate ions), and antimicrobial peptides (lysozyme, lactoferrin) [10]. However, xerostomia induced by medications, radiation, or systemic disease shifts the balance decisively toward caries progression [8].

Epidemiologically, dental caries affects approximately 2.3 billion people globally with untreated disease, surpassing many other chronic conditions in prevalence [4]. Early childhood caries (ECC), affecting 50-60% of children in low socioeconomic status (SES) populations, represents a critical public health emergency [11]. The burden disproportionately affects low- and middle-income countries (LMICs), including

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India, where rural disparities, limited fluoridation, and dietary sugar increases compound the problem [12].

The clinical paradigm has shifted dramatically since the early 2000s from reactive, restorative "drill-and-fill" approaches toward patient-centered, prevention-focused frameworks such as Caries-Risk Assessment and Management by Risk (CAMBRA) and International Caries Consensus Collaboration Management System (ICCMS) [13]. This evolution reflects accumulating evidence that managing risk and preventing lesions is more effective and equitable than treating advanced disease [14].

This comprehensive review synthesizes peer-reviewed evidence from 2020-2025, prioritizing systematic reviews and meta-analyses to provide clinicians, public health officials, and educators with current, actionable insights into caries etiology, epidemiology, diagnosis, prevention, and management. Particular emphasis is given to vulnerable populations and India-relevant applications, recognizing geographic and socioeconomic disparities in caries burden and intervention access [12].

2. PATHOGENESIS AND ETIOLOGY

2.1 Biofilm Ecology and the Caries Process

Dental caries fundamentally represents a dysbiotic shift in oral biofilm ecology, not merely bacterial infection by pathogenic species [15]. The oral microbiome in health comprises a balanced consortium including *Streptococcus sanguinis*, *Actinomyces*, and *Corynebacterium* species that resists colonization by cariogenic organisms [1]. However, sustained exposure to fermentable carbohydrates, particularly sucrose, shifts ecological pressure toward acidogenic (acid producing) and aciduric (acid tolerating) bacteria [16].

Streptococcus mutans, the archetypal caries pathogen, survives in this acidic environment through multiple adaptive mechanisms: it produces extracellular polysaccharides (dextrans, levans) that enhance biofilm structural integrity and bacterial adhesion; it secretes organic acids (lactate, formate, acetate) that lower pH; and it possesses membrane-bound F-ATPase pumps that extrude protons to maintain intracellular pH despite external acidification [17].

The Stephan Curve, described by Stephan in 1944, elegantly demonstrates the pH dynamics of caries initiation [8]:

- At baseline, plaque pH rests around 7.0 (neutral) in unstimulated saliva.
- Upon sugar intake, bacterial fermentation rapidly drops pH to <5.5 within 1-3 minutes.
- Peak demineralization occurs at pH 4.5-5.0.
- With saliva flow (parotid stimulation) and buffering, pH recovers to ~6.8 within 30-60 minutes.
- Repeated cycles exhaust buffering capacity and remineralization, cumulative damage progresses [1-18].

2.2 Demineralization and Remineralization

The demineralization process begins subsurface in enamel, where acids penetrate through surface layers while mineral loss occurs deeper—paradoxically preserving outer surface integrity while internal structure weakens [10]. Clinically, this manifests as the "white spot lesion" (opaque subsurface zone) visible on smooth surfaces [8].

The demineralization equation, simplified:

$$Ca_{10}(PO_4)_6(OH)_2 + 8H^+ \rightarrow 10Ca^{2+} + 6H_2PO_4^- + 2H_2O$$

The phosphate buffer equation governing plaque pH:

$$pH = 6.35 + \log \frac{[HPO_4^{2-}]}{[H_2PO_4^-]}$$

Remineralization is the reversal of early enamel lesions and depends critically on:

- Elevated plaque pH (>7.0) via saliva buffering
- Available calcium and phosphate ions
- Fluoride presence (hydroxyapatite → fluorapatite, more acid-resistant)
- Time and frequency of neutral pH periods [1]

Fluoride incorporation forms fluorapatite, which has a critical pH of 4.5 compared to 5.5 for hydroxyapatite—providing substantial resistance [8]. This is the principal mechanistic basis for fluoride's 20-38% caries reduction efficacy [19].

2.3 Lesion Progression

Caries progression follows a generally predictable sequence, though individual variation is marked:

1. **White spot (Non-cavitated)**: Subsurface demineralization with intact surface; reversible if intervention occurs.
2. **Brown stain**: Chromogenic bacterial colonization; incipient cavitation risk.
3. **Cavitated enamel**: Surface integrity lost; dentin exposure imminent.
4. **Dentinal caries**: Faster progression (dentin is 5x less mineralized); spreads laterally beneath enamel.
5. **Pulpal involvement**: Pain, necrosis if untreated; endodontic therapy required [20].

On smooth surfaces, lesions progress slowly (months-years); in fissures, progression accelerates due to biofilm protection and mechanical inaccessibility [1].

2.4 Host and Modulating Factors

Beyond the four ecological pillars, individual susceptibility varies significantly:

Salivary Factors

Unstimulated salivary flow <0.1 mL/min (xerostomia) elevates caries risk 3-4 fold; medications (anticholinergics, antidepressants, antihistamines) reduce flow [21]. Salivary buffering capacity (bicarbonate concentration) and calcium/phosphate saturation predict remineralization success [10].

Enamel Defects

Molar incisor hypomineralization (MIH) affecting 10-15% of children in developed countries creates rough, discolored, structurally weak enamel highly susceptible to rapid caries [22]. Amelogenesis imperfecta and fluorosis represent extreme variants with profound caries implications [8].

Genetic Susceptibility

Variants in genes encoding ameloblastin (AMBN), enamel matrix protein 1 (ENAM), and tuftelin (TUFT1) associate with altered enamel properties and caries risk [23]. Twin studies suggest heritability of 40-50% for caries experience [24].

Vitamin D Status

Recent meta-analyses reveal vitamin D deficiency (25-hydroxyvitamin D <20 ng/mL) increases caries risk by 16-22% in children under 19 years [25]. Proposed mechanisms include:

- Impaired calcium absorption and mineralization.
- Reduced antimicrobial peptide expression (cathelicidin).
- Altered immune tolerance and inflammation regulation [25].

Systemic Conditions

Type 1 diabetes mellitus increases caries risk through altered salivary composition, impaired immune function, and dysbiotic microbiota shifts [26]. Gastroesophageal reflux disease (GERD) and bulimia expose enamel to hydrochloric acid, causing erosion and secondary caries [27].

3. EPIDEMIOLOGY AND GLOBAL BURDEN OF DISEASE

3.1 Global Prevalence and DMFT Indices

Dental caries remains the most common chronic disease globally, with profound disparities by region, age, and socioeconomic status [11-28]. The World Health Organization (WHO) and Global Burden of Disease (GBD) studies document this burden systematically.

Epidemiological Metrics

The DMFT Index (Decayed, Missing, Filled Teeth) quantifies caries experience in permanent dentition:

- D = decayed (untreated cavities)
- M = missing (due to caries)
- F = filled (restored)
- Range: 0 (no caries) to 32 (all teeth affected)

The dmf index (lowercase) applies to primary (baby) teeth; DMFS/dmfs measure surfaces rather than teeth [8].

Recent meta-analyses report:

Population	DMFT Range	Prevalence	Source [Reference]
Arab children (permanent)	2.1-5.06	72.62%	[29]
Global children (5-12 yr)	2.5-4.0	53-65%	[11]
Indian adults	3-7	60-70% (rural higher)	[12]
Low-income countries	3.5-6.0	70%+	[4]
High-income countries	0.8-1.5	15-25%	[30]

These disparities reflect cumulative effects of access to fluoridated water, fluoride toothpaste availability, dietary patterns, preventive behaviors, and health system capacity [12].

3.2 Early Childhood Caries (ECC)

Early childhood caries, defined as presence of one or more cavitated/non-cavitated/missing primary teeth due to caries in children under 6 years, represents a particularly severe public health crisis [31].

Epidemiology of ECC:

Global disease burden analyses (1990-2021) project:

- Current prevalence: 46-72% in LMICs, 15-30% in high-income countries.
- Projected increase to 2040: Rising trends in Africa, South Asia, Southeast Asia without intervention.
- Socioeconomic gradient: 60% prevalence in children from low-SES families vs. 20% high-SES [4].
- India-specific data: ECC prevalence 20-50% urban, 40-70% rural (limited fluoridation)[12].

Risk factors for ECC:

- Early colonization by S. mutans (vertical transmission from mother/caregiver).
- Prolonged bottle-feeding, especially with sugared liquids (milk with honey, juice).
- Limited parental oral health knowledge and behavioral capacity.
- Poor access to preventive care (no fluoride, sealants).
- Nutritional deficiencies (vitamin D, calcium) affecting enamel mineralization.

- Low birth weight and prematurity (enamel defects).

3.3 Regional and Socioeconomic Disparities

Caries exhibits stark socioeconomic gradients. Children from low-SES families experience:

- 3-4 times higher untreated caries burden.
- Later presentation (advanced disease) due to delayed care-seeking.
- Higher extraction rates (missing teeth in DMFT).
- Compounded by co-occurring nutritional deficiencies [12][28].

Geographic disparities reflect access infrastructure. Regions lacking:

- Centralized water fluoridation infrastructure (much of sub-Saharan Africa, rural India)
- Reliable fluoride toothpaste supply chains
- School-based prevention programs
- Preventive / restorative dental workforce demonstrates 3-5 fold higher disease burden [4][11].

3.4 Impact on Oral Health-Related Quality of Life

A 2025 meta-analysis of 10 studies found that dental caries significantly impairs quality of life in adolescents, with:

- Prevalence ratios (PR) of 1.66-1.72 for Child Oral Impact on Daily Performances (Child-OIDP) scale.
- Impacts include: difficulty eating/drinking (most common), pain, emotional distress, school absence.
- Heterogeneity reduced after sensitivity analysis excluding very small / high-bias studies [32].

4. DIAGNOSIS AND RISK ASSESSMENT

4.1 Clinical Diagnosis: ICDAS Coding System

The International Caries Detection and Assessment System (ICDAS II) provides standardized, evidence-based criteria for lesion detection and monitoring [33]:

Table 1: ICDAS Codes for Caries Assessment

Code	Lesion Type	Clinical Description
0	No caries	Sound surface
1	Initial change	White/brown discoloration (non-cavitated, dry)
2	Moderate change	Discoloration + microcavitation (not into dentin)
3	Advanced change	Obvious cavity, into dentin, < 50% surface
4	Severe change	Obvious cavity, into dentin, > 50% surface
5	Severe change	Cavity involves incisal / cusp edges
6	Severe change	Extensive cavity / missing tooth due to caries

Codes 1-2 are non-cavitated (reversible if managed early); codes 3-6 are cavitated (require restoration or extraction) [33].

4.2 Advanced Diagnostic Technologies

Fluorescence-Enhanced Intraoral Scanners

Digital intraoral scanners with fluorescence technology (e.g., TRIOS 4/5) detect caries by exciting enamel at 415 nm, visualizing early demineralization as altered fluorescence patterns [34]. A 2025 diagnostic agreement study reported:

- Intra-class correlation (ICC) >0.80 comparing scanner detection vs. visual ICDAS assessment for non-cavitated lesions
- Superior detection of interproximal caries (often missed visually)
- Real-time 3D visualization enabling patient education [34].

Laser Fluorescence

DIAGNOdent (655 nm laser) detects caries by photothermal radiometry; carious lesions emit higher fluorescence. Sensitivity 85-90%, specificity 80-85% for cavitated lesions; less sensitive for non-cavitated [8].

Transillumination

Fiber optic Transillumination (FOTI) and digital imaging fiber optics (DIFOTI) visualize lesions by light diffraction differences; particularly effective for interproximal detection [1].

4.3 Caries-Risk Assessment: CAMBRA Framework

The Caries-Risk Assessment and Management by Risk (CAMBRA) system, developed by the University of California, stratifies patients as low/moderate/high risk based on biomarkers and clinical indicators [35]:

High-Risk Indicators (any 2+ = High Risk):

- Visible plaque on teeth at baseline.
- Interproximal restorations or cavitated smooth surface lesions.
- Frequent snacking (4+ times daily) on sugary foods/drinks.
- Salivary flow <0.5 mL/5 min (unstimulated).
- Salivary buffering capacity (pH <6.0).
- High mutans streptococci (>10^5 CFU/mL) or lactobacilli counts.
- Recent caries activity (any lesion in past 3 years).

- Xerostomia or medications with xerostomic effects.

Risk stratification guides intervention intensity:

- **Low risk**: Fluoride toothpaste, dietary counseling.
- **Moderate risk**: Above + fluoride varnish (2x / year).
- **High risk**: Above + antimicrobial rinses, frequent professional fluoride, SDF, intensive behavioral support [35].

4.4 Predictive Risk Models for Pediatric Caries

A 2025 systematic review examined risk prediction models for caries in children and adolescents, identifying 45 models across 32 studies [36]. Key findings:

- **Validation adequacy**: Only 35% of models underwent external validation; most rely on development cohorts.
- **Performance**: Sensitivity 60-85%, specificity 65-80% (considerable variation).
- **Missing factors**: Few models integrate socioeconomic/behavioral elements; dominated by clinical/microbiological predictors.
- **Implementation barriers**: Complex models with laboratory testing not feasible in public health settings.

Recommendations: Models requiring chair-side simplicity and socioeconomic inclusion for practical utility [36].

5. PREVENTION STRATEGIES

5.1 Water Fluoridation and Community-Level Interventions

Water fluoridation remains the most cost-effective public health measure for caries prevention, with 70+ years of evidence demonstrating safety and efficacy [37].

Effectiveness Metrics:

A 2025 global meta-analysis in Sage Journals synthesized community water fluoridation (CWF) studies:

- **Caries reduction**: 20-38% in permanent teeth (varies by baseline prevalence).
- **Cavitation prevention**: 40% reduction in untreated cavitated lesions.
- **Socioeconomic benefit**: Highest benefit in low-SES populations with limited access to other preventive sources [37].

Optimal Fluoride Concentration:

WHO recommends 0.7-1.0 mg/L, adjusted for climate:

- Cooler climates: 0.9-1.0 mg/L (lower water intake).
- Warmer climates: 0.7-0.8 mg/L (higher water intake, greater dental fluorosis risk at higher concentrations) [8].

India's Status:

- Fluoridated water coverage: <5% of population (contrast: 70% USA, 60% Australia).
- Rural areas overwhelmingly unfluoridated; some regions (especially West Bengal, Assam) suffer endemic dental/skeletal fluorosis from naturally elevated fluoride [12].
- Policy opportunity: Targeted CWF in high-risk ECC regions [12].

5.2 Topical Fluoride Application

Toothpaste:

- Standard formulation: 1000-1500 ppm fluoride (equivalent to 1-1.5 mg F^- per gram).
- Efficacy: 24% caries reduction in children using twice-daily 1500 ppm vs. placebo [8].
- Age-specific: Children <3 years: smear amount (tiny dab); 3-6 years: pea-size; > 6 years: standard pea-size to avoid fluorosis risk [8].

Professional Varnish (5% NaF):

- Frequency: High-risk patients 2-4 times/year; moderate-risk 1-2 times/year.
- Efficacy: 33-46% caries reduction (interproximal better than free smooth surfaces) [19].
- Mechanism: High fluoride concentration (22,600 ppm) penetrates lesions, forms CaF_2 reservoir [19].

Gels and Rinses:

- 0.4% stannous gel (40,000 ppm): Professional application, 37-50% efficacy, staining risk.
- 0.05% sodium fluoride rinse (230 ppm): Daily home use, 30-35% efficacy [8].

5.3 Sealants

Dental sealants like resin or glass ionomer (GIC) barriers mechanically block fissures and pits where caries initiates 80-90% of cavities in children [38].

Efficacy:

10-year meta-analysis showed equivalent caries prevention: resin sealants 72-86% effective; GIC sealants 60-76% effective [39]. Retention matters unsealed fissures regain caries risk [39].

Indications:

- First molars (age 6-7) and second molars (age 11-13).
- High caries risk, deep pits/fissures, newly erupted teeth [8].
- Occlusal surfaces exclusively; no evidence for smooth surface sealants [39].

5.4 Silver Diamine Fluoride (SDF)

Silver diamine fluoride—a topical antimicrobial/remineralizing agent has emerged as a low-cost, minimally invasive option for ECC and high-risk patients [40].

Mechanism:

- Silver ions: Bactericidal, inhibit glycolytic enzymes, prevent biofilm reformation [40].
- Fluoride: Remineralizes incipient lesions, forms fluorapatite [40].

Efficacy for ECC:

Arrest rates: 70-80% (vs. <40% for fluoride varnish alone) [40]. Cost: <USD 1 per application (vs. >USD 100 for resin restoration) [40].

Practical Advantages:

- No drilling/injections required.
- Single visit (retention to lesion surface ~24 hours).
- Feasible in resource-limited settings, rural India [40].

Limitations:

- Stains carious lesions black (cosmetic concern for visible teeth).
- Parent/patient education essential [40].

5.5 Postbiotics and Novel Antimicrobial Agents

Postbiotics—microbial metabolites and cell components—inhibit S. mutans biofilm formation and adhesion in vitro [41]. A 2024 systematic review identified emerging compounds:

- **Probiotic Derived Postbiotics** (*Lactobacillus reuteri*, *Bacillus subtilis*): Reduce mutans counts 30-50% in biofilm models [41].
- **Plant Derived Polyphenols** (cranberry, green tea): Inhibit *Streptococcus mutans* exopolysaccharide synthesis [41].
- **Histatins and other Salivary Peptides**: Antimicrobial efficacy in preliminary studies [41].

Current status: Promising preclinical/in vitro evidence; clinical trials in children lacking. Not yet ready for routine recommendation [41].

5.6 Behavioral and Dietary Interventions

Sugar Restriction:

WHO guideline: Free sugars <5% of daily energy intake (equivalent to <25 g/day or <6 teaspoons) [42]. Meta-analyses show:

- Each additional sugar exposure daily increases caries risk 16% in children [42].
- Frequency (not total amount) predicts caries; 4+ daily exposures elevates risk 3-4 fold [42].

Xylitol: A sugar substitute fermented by fewer oral bacteria, xylitol reduces S. mutans counts and caries incidence when dosed 5-10 g/day [8]. However:

- Laxative effect at high doses [8].
- Cost higher than conventional prevention [8].
- Not recommended as primary prevention (insufficient evidence base) [43].

Oral Health Education:

Behavioral counseling addressing:

- Parental attitudes/practices (critical in ECC prevention) [44]
- Tooth brushing technique (twice daily, 2 minutes, pea-size paste) [8].
- Dietary knowledge (labels, hidden sugars in yogurts/juices) [44].
- Access facilitation (referrals, transportation assistance) [44]

Effectiveness Meta-Analysis: Behavioral interventions reduce DMFT by 0.5-1.0 units when sustained >12 months [44].

6. MANAGEMENT AND THERAPEUTICS

6.1 Minimally Invasive Caries Management

The paradigm shift from complete excavation to selective caries removal represents evidence-based advancement, particularly for deep lesions approaching the pulp [45].

Selective Caries Removal vs. Complete Excavation:

A 2024 meta-analysis of 15 RCTs compared techniques:

Table 2: Selective vs. Complete Caries Removal Outcomes [45]

Outcome	Selective Removal	Complete Excavation
Pulp vitality preservation	95%	82%
Pulp hypersensitivity (post-op)	8%	22%
Restoration longevity (5-yr)	87%	84%
Treatment time (minutes)	32	42

Selective removal involves excavating only grossly softened dentin and the outermost layer of hard carious tissue, halting when color/hardness indicates viable dentin [45]. This preserves pulpal vitality and reduces sensitivity [45].

6.2 Atraumatic Restorative Treatment (ART)

ART hand instrument excavation without local anesthesia or high speed equipment suits:

- Primary dentition (temporary restoration acceptable) [46].

- Access-limited settings (rural, refugee camps) [46].
- Anxiety-prone patients [46].

ART Efficacy:

3-year restoration survival: 80% in primary molars; 75% in primary premolars [46]. Materials: Glass ionomer cement (GIC) preferred for fluoride release and adhesion [46]. Cost-effectiveness: ART <USD 10/restoration vs. conventional >USD 30[46].

6.3 Silver Diamine Fluoride (SDF) for Non-Restorative Management

For ECC and patients declining treatment, SDF offers arrest without restoration:

- Application: 38% SDF topically applied annually [40].
- Mechanism: Silver arrests caries; fluoride remineralizes [40].
- Efficacy: 70-80% arrest in 12 months [40].

Guidelines recommend SDF for:

- Primary teeth with extensive caries.
- Uncooperative/anxious children.
- Remote communities without restorative capacity [40].

6.4 Restorative Materials

Glass Ionomer Cement (GIC):

Advantages: Fluoride release, micromechanical adhesion (no etching required), low cost [47]. Limitations: Lower wear resistance, susceptibility to moisture contamination during placement [47].

Resin Composite:

Advantages: Superior aesthetics, wear resistance, longevity [47]. Limitations: Technique-sensitive (moisture isolation critical), higher cost, no fluoride release (unless specially formulated) [47].

Atraumatic Restorative Treatment Materials:

High-viscosity GIC or composite-modified GIC deliver improved properties vs. traditional GIC while maintaining ART compatibility [46].

6.5 Endodontic Considerations

When caries reaches the pulp chamber, pulpotomy (partial pulp removal) or pulpectomy (complete pulp removal, root canal treatment) becomes necessary [48].

Pulpotomy in Primary Teeth:

- Materials: Zinc oxide eugenol (ZOE), MTA (mineral trioxide aggregate), iodoform pastes [48].
- Success rate (6-12 months): 85-92% with proper technique [48].
- Simplified alternatives: Laser-assisted pulpotomy shows promise in preliminary studies [48].

7. SPECIAL POPULATIONS AND CHALLENGES

7.1 Early Childhood Caries: Management in Infants and Toddlers

ECC management integrates prevention, diagnosis, and age-appropriate intervention [49]:

Prevention-First Approach:

- **Primary Prevention**: Maternal oral health, dietary guidance (bottle-feeding reduction by age 12 months, no night bottles) [49].
- **Secondary Prevention**: Topical fluoride (varnish 2x/year or SDF annually) starting at eruption [49].
- **Early Detection**: 6-month screening after first tooth eruption [49].

Management in Symptomatic ECC:

- **Pain Management**: Topical anesthetics (benzocaine gel), systemic analgesia if needed.
- **Emergency Arrest**: SDF application for temporary management.
- **Definitive Treatment**: Modified/full sedation for comprehensive restorative care in hospital setting if severe [49].

7.2 Carries in Type 1 Diabetes Patients

Type 1 diabetes mellitus increases caries risk 2-3 fold through:

- Altered salivary composition (higher glucose, lower buffering) [26].
- Dysbiotic microbiota shift toward acidogenic species [26].
- Impaired neutrophil function, hyperinflammatory response [26].

Management intensification:

- Stringent control of glycemia (HbA1c <7%) improves salivary function [26]
- Increased fluoride (varnish 3x/year vs. 2x in non-diabetics) [26]
- Frequent caries monitoring (6-monthly vs. 12-monthly) [26]

A 2024 meta-analysis documented altered oral microbiota—reduced Streptococcus sanguinis, elevated lactobacilli—in diabetic children, with corresponding DMFT elevation [26].

7.3 Challenges in Low-Resource Settings: India as Case Study

India faces unique caries epidemiological challenges [12]:

Infrastructure Barriers:

- Water fluoridation: <5% coverage (vs. >70% developed nations) [12].
- Dentist density: 1 per 10,000 population rural vs. 1 per 1,000 urban [12].
- Fluoride toothpaste availability: Inconsistent supply chains; cost barriers for poorest [12].

Behavioral/Cultural Factors:

- Limited oral health literacy, particularly among mothers/caregivers [12].
- Dietary patterns: Increasing sugar consumption with industrialization [12].
- Health system engagement: Low preventive care-seeking; presentation at advanced disease stages [12].

Policy Opportunities:

- School-based fluoride varnish programs (proven cost-effective in Thailand, Kenya) [50].
- Integration with AYUSH systems (Ayurveda, Yoga, Unani, Siddha, Homeopathy) for health promotion [51].
- Task-shifting to auxiliary nurses/health workers for basic prevention and fluoride application [50].
- Targeted SDF programs in high-burden ECC regions [40].

7.4 Caries and Systemic Health Integration

Emerging evidence links oral caries to systemic conditions:

- **Cardiovascular Disease**: Inflammatory burden from untreated oral disease elevates CVD risk [52].
- **Type 2 Diabetes**: Bidirectional relationship; poor oral health impairs glycemic control [52].
- **Respiratory Infections**: Oral biofilms aspiration risk, particularly in frail elderly [53].
- **Early childhood Development**: ECC-related malnutrition, pain, school absence impair cognitive outcomes [54].

This 'chronic disease model' justifies integrating oral health into primary care and chronic disease prevention platforms [7-52].

8. CONCLUSION

Dental caries, despite being largely preventable, remains a global public health crisis affecting billions across age groups and geographies, with disproportionate burden in vulnerable populations including infants with early childhood caries, low-income communities, and rural populations in developing nations [11, 12]. The accumulated evidence from 2020-2025 systematic reviews and meta-analyses provides robust guidance for prevention and management.

Key evidence based conclusions:

1. **Prevention is Paramount**: Integrated strategies combining water/topical fluoride (20-38% caries reduction), sealants, dietary modifications, and behavioral support achieve 30-50% burden reduction in population trials and remain far more cost-effective than treatment [19-37].
2. **Risk Stratification Improves Outcomes**: CAMBRA frameworks and validated predictive models, when implemented with adequate decision support and care coordination, enable targeted interventions matching intensity to risk, improving resource efficiency [35, 36].
3. **Innovation in Diagnosis and Minimally Invasive Management**: Fluorescence-enhanced scanners (ICC >0.80), selective caries removal (pulp preservation), and silver diamine fluoride (70-80% arrest rates) expand evidence-based options suited to diverse clinical and resource contexts [34-45].
4. **Special attention to ECC and Vulnerable Populations**: Early childhood caries prevalence of 50-72% in LMICs, with projected increases to 2040, demands coordinated maternal/early childhood health programs, school-based prevention, and task-shifted service delivery [4-49].
5. **Addressing Determinants**: Socioeconomic gradients, limited fluoridation infrastructure (particularly in India with <5% coverage), behavioral barriers, and low health literacy require multisector action beyond the dental profession—involving education, food policy, water systems, and primary care integration [12-37].
6. **Global Equity Imperative**: Disparities in caries burden correlate directly with income and health system development. Expanding access to fluoride, sealants, preventive education, and minimally invasive care through public health programs and policy advocacy remains critical for achieving health equity [11-28].

Future research priorities include:

- Validation of pediatric risk prediction models for implementation in low-resource settings [36].
- Large-scale trials of postbiotics and novel antimicrobials for clinical efficacy [41].
- Implementation science studies of school-based and community health worker-delivered prevention in high-burden regions [50].
- Integration frameworks linking oral health to chronic disease prevention platforms [52].

For India specifically, the opportunity is immediate: targeted water fluoridation in high-ECC burden regions, school-based varnish programs,

integration with ASHA (Accredited Social Health Activist) networks, and SDF-based programs in remote areas could prevent an estimated 40% of childhood caries burden within 5 years [12-50]. This requires policy commitment, stakeholder coordination, and sustained investment yet the evidence base and tools exist [12]. Dental caries will remain preventable only through sustained public health commitment and equitable access to evidence-based interventions. This review provides clinicians, policymakers, and educators with current evidence to guide decisions.

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