### DENTAL CARIOLOGY- CONCEPTS AND TRENDS IN INDIA

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### **Abstract**

Dental Caries is a pandemic and is one of the most important global oral health problems in the world today. It's an infectious disease characterized by a multifactorial etiology and slow evolution that leads to the destruction of dental hard tissues. The implementation of preventive measures, the need of investing in education for the correct maintenance measures of oral health, associated with preventive medical and dental care, are key to the awareness of populations of its existence and to the decline of its prevalence in India. The current paradigm for management of dental caries is evidence based and favours non-invasive therapies to prevent and/or arrest the progression of the disease. This article focuses on the concepts and trends of dental caries in India.

Keywords: Dental Caries, Prevalence, Etiology, Management, Preventive measures, Paradigm shift.

## Introduction

Dental Caries is one of the most prevalent health problems in India. It is a major public health oral disease which hinders the achievement and maintenance of oral health in all age groups. In spite of knowledge explosion in dental cariology science, dental caries still remains a misunderstood phenomenon by the dental health professionals in India. Prevention of this disease is not only affordable but also predictable rather than curing for the oral health status of an individual. The term 'Dental Caries', originates from the Latin word "Caries" which stands for 'rotten'1. WHO pointed that the global problem of the oral disease still persists despite great improvements in the oral health of population in several countries. It is defined as an infectious microbiological disease of the teeth that results in the localized dissolution and destruction of the calcified tissues of the teeth and demineralization of the organic substances of the tooth<sup>2</sup>.

### **Prevalence**

There are practically no geographic areas in the world whose inhabitants do not exhibit some evidence of dental caries. Worldwide, approximately 2.43 billion people; i.e. 36% of the population have dental caries in their permanent teeth. In India, the prevalence rate has been reported to be similar at 5 years and 12

years of age (49%) while it shows a steady increase from 15 years (60%) to 35-44 years (78%) and peaks at 60-74 year group (84%). Males have slightly higher prevalence at 5 and 12 years of age and females have a higher prevalence in the older age group3. A survey conducted by DCI in 2004 suggests an increase in prevalence rate of dental caries from 51.9% in 5-year old children to as high as 85.0% in adults aged 65-74 years. It also suggested dental caries to be the prime cause of edentulism in almost 30% of the senior citizens<sup>4</sup>.

### Classification

Dental Caries can be classified in a number of ways. According to the morphology or anatomical site of the lesion it can be classified as pit and fissure caries and smooth surface caries (Fig. 1).

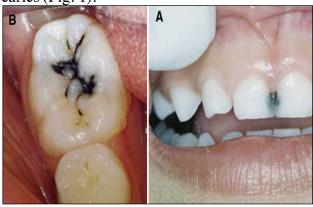


Fig.1- (A) Smooth surface caries (B) Pit and Fissure caries

Depending on the dynamics it can be initial, moderate and advanced dental caries(Table.1); while on the basis of caries activity, it can be active and inactive(Table.2). If we take

chronology as a differeTable. 1 American Dental Association Caries Classification System (ADA CCS).ntiating factor it can be nursing bottle caries and adolescent caries.

	AMERICAN DENTAL ASSOCIATION CARIES CLASSIFICATION SYSTEM										
	Sound	Initial	Moderate	Advanced  Enamel is fully cavitated and dentin is exposed. Dentin lesion is deeply/ severely demineralized.							
Clinical Presentation	No clinically detectable lesion. Dental hard tissue appears normal in color, translucency, and gloss.	Earliest clinically detectable lesion compatible with mild demineralization. Lesion limited to enamel or to shallow demineralization of cementum/dentin. Mildest forms are detectable only after drying. When established and active, lesions may be white or brown and enamel has lost its normal gloss.	Visible signs of enamel breakdown or signs the dentin is moderately demineralized.								
Other Labels	No surface change or adequately restored	Visually noncavitated	Established, early cavitated, shallow cavitation, microcavitation	Spread/disseminated, late cavitated, deep cavitation							
Infected Dentin	None	Unlikely	Possible	Present							
Appearance of Occlusal Surfaces (Pit and Fissure)**†	ICDAS 0	ICDAS 1 ICDAS 2	ICDAS 3 ICDAS 4	ICDAS 5 ICDAS 6							
Accessible Smooth Surfaces, Including Cervical and Root <sup>8</sup>				8							
Radiographic Presentation of the Approximal Surface <sup>5</sup>	E0 <sup>®</sup> or RO <sup>®</sup> No radiolucency	E1 <sup>1</sup> or RA1 <sup>*</sup> E2 <sup>1</sup> or RA2 <sup>*</sup> D1 <sup>1</sup> or RA3 <sup>*</sup> Radiolucency may extend to the dentinoenamel junction or outer one-third of the dentin. Note: radiographs are not reliable for mild occlusal lesions.	D2 <sup>®</sup> or RB4 <sup>®</sup> Radiouscry extends into the middle one-third of the dentin	D3 <sup>4</sup> or RC5 <sup>4</sup> Radiolucency extends into the inner one-third of the dentin							
† The ICDAS notation by the ICDAS Foun management syste Methods for stagin Longbottom C. ICC ‡ "Cervical and root" § Simulated radiogra § E0-E2, D1-D3 notal	dation over the last decade; ICD/ m ICCMS. (Pitts NB, Ekstrand KR, g of the caries process and enablii MS Guide for Practitioners and E- includes any smooth surface lesi phic images. tion system. <sup>53</sup>	If pit-and-fissure caries, pearance of occlusal caries lesions with the histologically deter Sc also has a menu of options, including 3 levels of caries let International Caries Detection and Assessment System (ICDA ing dentists to manage caries. Community Dent Orol Epidemio ducators. Available at: https://www.icdas.org/uploads/ICCMS on above or below the anatomical crown that is accessible ti scoring system (RC6 = into pulp). (Pitts NB, Ismail AI, Martignia)	mined degree of dentinal penetration us sion classification, radiographic scoring a SJ and its International Caries Classificat I 2013;41[1]:e41-e52. Pitts NB, Ismail Al, -Guide - US, polf. Accessed horough hrough direct visual/tactile examination.	ing the evidence collated and published and an integrated, risk-based caries ion and Management System (ICCMS): Martignon S, Ekstrand K, Douglas GAV, ill 13, 2015.)							

Table. 1 American Dental Association Caries Classification System (ADA CCS).

# **Etiology**

Caries results from an ecological imbalance in the equilibrium between the tooth minerals and oral biofilms(plaque). The biofilm is characterized by microbial activity, resulting in fluctuations in the plaque pH. As the pH falls below critical value, the demineralization of enamel, dentine or cementum occurs. The microbial community of caries is diverse and contains bacteria belonging to the genera Streptococci, Actinomyces, Eubacterium, Enterococci, Lactobacillus, Propionibacterium etc<sup>5</sup>.

Many theories have evolved through years of investigation and observation related to dental caries, namely, the acidogenic theory(Miller's chemico-parasitic theory), the proteolytic theory and the proteolysis- chelation theory;

which are among many which have stood the test of time6. Bacteria metabolize sugars and produce acid which leads to decrease in pH and the enamel demineralizes. There is always a battle between demineralization and remineralization(See Saw theory).

	Caries lesion activity assessment descriptors						
Activity assessment factor	Likely to be inactive/arrested	likely to be active					
Location of the lesion	Lesion is not in a plaque stagnation area	Lesion is in a plaque stagnation area (pit/fissure, approximal, gingival)					
Plague over the lesion	Not thick or sticky	Thick and/or sticky					
Surface appearance	Shiny; color: brown-black	Matte/opaque/loss of luster; color: white-yellow					
Tactile feeling	Smooth, hard enamel/hard dentin	Rough enamel/soft dentin					
Gingival status (if the lesion is located near the gingiva)	No inflammation, no bleeding on probing	Inflammation, bleeding on probing					

Table.2- Active and Inactive Lesions

Dental caries is a multifactorial disease in which there is interplay of three primary factors: the host, the microbial flora and the substrate. In addition a fourth factor- the amount of time the tooth is exposed to these adverse conditions must be considered in any discussion of the etiology of caries<sup>7</sup>.

In other words, caries requires a susceptible host, a cariogenic flora and a suitable substrate that must be present for a sufficient length of time. Conversely, caries prevention is based upon attempts to increase the resistance of the host, lower the number of microorganisms in contact with the tooth, modify the substrate by selecting noncariogenic foodstuffs; and reduce the time that the substrate is in the mouth by limiting the frequency of intake. The mere presence of microorganisms and a suitable substrate at a given point on a tooth surface is apparently insufficient to establish a carious lesion in all individuals. Several factors like composition of saliva, diet intake, pH, tooth morphology, genetic predisposition, quantity of saliva, age group, etc also contributes to the progression of dental caries<sup>8</sup>.

## Diagnosis of dental caries

Early detection of dental caries have been emphasized to understand the nature of the caries process. The first sign of tooth demineralization is observed as a small "white spot" i.e. initial/incipient caries which is not yet a cavity. The progression of non cavitated lesions seems to be slower, allowing preventive strategies to be implemented when the lesions have the greatest opportunity to arrest. Thus, early and accurate detection and diagnosis of dental caries are an important component of the overall management of dental patient. Some behaviour or systemic diseases like rheumatoid arthritis, uncontrolled diabetes, smoking/tobacco use, obesity, micronutrient deficiencies, etc are also common risk factors for dental caries9.Out of which inappropriate feeding practices, poor resourcesand malnutrition are common predisposing factors responsible for caries in India<sup>10</sup>.

Conventional diagnosis of caries was done using Visual inspection, Tactile sensation,

Radiography, Caries detecting dyes etc. While these methods gave satisfactory results in detection of cavitated lesions (Table.3), they are usually inadequate for the detection ofinitial lesions11. Newer technologies like Digital imaging, Fiber optic transillumination, Digital fiber optic transillumination imaging, Xeroradiography, Subtraction radiography, Mini-D, Fluorescence, DIAGNOdent (Fig.2), Carbon dioxide laser, Endoscope, Cone beam computed tomography, Optic coherence tomography, Terahertz imaging, Multiphoton imaging, Ultrasonics, Tuned Aperture Computed Tomography, Infrared fluorescence, Infrared thermography, Cariescan (Fig.2) etc have been developed to provide us a chance to aid in early and better diagnosis and this research is still continued<sup>12</sup>.





Fig.2- DIAGNOdentCariescan

Table.3- Performance summaries for various methods for the detection of carious lesions (James D. Bader, 2001)

Method Surface Extent of Lesion	Number of Studies	Number of Examiners mean median		Lesion Prevalence mean median		Quality Score mean median		Sensitivity mean median range			Specificity mean median range		
Visual													
occlusal surfaces													
cavitated	4	1	1	56%	51%	45	42	63	51	53	89	89	22
dentinal	10	9	4	50%	44%	50	45	37	25	92	87	91	59
enamel	2	9	2	21%	21%	48	48	66	66	12	69	69	7
any	4	12	7	78%	75%	48	43	59	62	62	72	74	39
proximal surfaces													
cavitated	1	1	•	nr*	•	50	-	94	-	•	92	-	
Visual-Tactile													
occlusal surfaces													
cavitated	1	1	-	nr-	0	50	-	92	-	-	85	-	
dentinal	2	12	6	29%	29%	45	45	19	19	10	97	97	7
any	2	4	4	40%	40%	45	45	39	39	44	94	94	13
proximal surfaces													
cavitated	3	3	3	5%	6%	62	65	52	32	64	98	99	2
dentinal	1	3	-	nr	•	35	-	50	•	-	71	•	-
Radiographic													
occlusal surfaces		100							_				
dentinal	26	4	3	54%	55%	47	45	53	54	79	83	85	50
enamel	4	2 5	2	18%	18%	48	48	30	28	25	76	76	10
any	7	5	4	82%	84%	49	50	39	27	67	91	95	18
proximal surfaces	_	•	•	120/	00/						0.5		
cavitated	7	3	3	13%	9%	63	60	66	66	63	95	97	1.
dentinal	8	39	5	27%	25%	53	55	38	40	42	95	96	7
enamel	2	10	10	25%	25%	60	60	41	41	11	78	78	4
any	11	6	3	62%	66%	50	50	50	49	85	87	88	2
Electrical Conductance													
occlusal surfaces	14	2	1	200/	270/	27	45	0.4	91	39	70	00	20
dentinal	14	1	1	38%	37%	37	45	84	91	39	78	80	3
enamel	8	į.	1	24% 69%	64%	50 29	37	65 73	70	21	73 87	85	2
FOTI any	0			09%	0470	29	3/	/3	70	21	0/	03	2.
occlusal surfaces													
dentinal	1	1		36%		60	121	14	12		95		
enamel	i	i		24%		55		21			88		
proximal surfaces				2470		33		21			00		
cavitated	1	4		6%		70	-	04			100		
Laser Fluoresence	•	7.8		070		, 0		0.					
occlusal surfaces													
dentinal	2	1		36%	36%	30	30	80	80	8	86	86	3
Combination Visual/Rad	-	•		55,0	55,0	50		-			-		,
occlusal surfaces	0. 1												
dentinal	3	10	10	61%	61%	47	45	67	65	37	75	74	2

Caries riskassessment plays a vital role to predict future caries development before the clinical onset of the disease (Table.3).

	S		A		F	E	R		
Caries Risk Level	Sealants	Saliva	Antibacterials	Fluoride (Topical)	Factors favorable for remineralization (pH, Ca <sup>2+</sup> & PO <sub>4</sub> <sup>3-</sup> )	Effective Lifestyle Habits	Radiographs	Recare	
Low Risk	Not indicated (optional for primary prevention of at risk deep pits and fissures)	Saliva testing is optional or may be done for purposes of baseline records	Not indicated	OTC fluoride toothpaste used bid.	Recession or sensitive roots may indicate need for supplementation.	Encourage healthy dietary habits, low frequency of fermentable carbohydrates, adequate protein intake & effective oral	Every 24–36 mo	Every 6 mo	
Moderate Risk	Sealants are	Measure resting and stimulated flow and pH especially if hyposalivation is	Xylitol therapy 2-3 timesiday for a total daily dose of 6-10 grams	OTC fluoride toothpaste used bid. 0.05% NaF rinse bid. Varnish applied every 4 to 6 mo.	Low resting pH, low stimulated flow or pH may indicate need for supplementation.	hygiene practices using motivational interviewing techniques. Substitute xylitol for sucrose.	Every 18-24 mc	Every 4-6 mo	
High Risk	recommended per ICDAS code (see table 3) for secondary prevention	DAS code able 3) for Objective with the following agents it must be understood that the evidence is ondary measurement of very limited for antifactorials & of very limited for antifactorials & of		5000 ppm toothpaste used gd or bid. 0.05%	Consider supplementing if topical fluoride alone is not effective		Every 6–18 mo	Every 3–4 mo	
Extreme Risk			NaE rinse bid. Vamish applied every 3 to 4 mo.	Required if xerostomia is 3present		Every 6 mo until no new caries lesions.	Every 3 mo		

SAFER guidelines. (From Glassman P. A manual of hospital dentistry. 10th edition. SanFrancisco (CA)

Various tests for caries risk assessment are also available for measuring carious activity like lactobacillus colony test, swab test, buffer capacity test, salivary reductase test,

etc13. Caries activity test establishes the need for personalized preventive measures and monitoring the effectiveness of education program.

Prevention and management of dental caries The control of dental caries presents one of the greatest objectives that must be met today by the dental profession and it leads to paradigm shift for approaching its management (Table.4).

Surgical approach (old)	Minimally invasive approach (new)
Review health history	Review health history
Dental examination	medication (xerostomic inducing)
Gingival probing as needed	Caries risk assessment
Oral hygiene instructions/diet counseling	Check saliva pH/flow/quality
Full set of radiographs initial visit	Check bacterial activity
Use sharp instrument to detect soft areas	Oral hygiene instructions/diet counseling
Fillings any soft area or suspicious stain	treat etiology: biofilm and environment
Cleanings 2/y	Dental examination (no sharp instrument but
Recall bitewing radiographs every year	dry teeth, observe texture and color)
New full set radiographs every 3 y	Diagnose: etiology/consider remineralize strategy
	Treat biofilm (antimicrobial)
	Gingival probing as needed
	Dental Cleaning
	Fillings with appropriate dental material
	Recall and radiographs based on caries risk

Table.4- Dental treatment paradigm shift comparison

It would be inappropriate to focus only on perfecting the techniques to repair the occurred damage and not focusing enough to perfect the measures to prevent it. The deepening of evidence regarding the dynamic process of demineralization and remineralization has led to a consensus that the resulting dental destruction due to bacterial action can be stopped or reversed by taking several preventive measures 14. This disease, like most infectious diseases, occurs on surfaces, specifically teeth, bathed by external secretions, in whichthe principal immunoglobulin isotype present is secretoryIgA (SIgA)15. Therefore, immunization procedures which resultin the induction of salivary SIgA antibodies would most likely beeffective means for inducing caries immunity. Vaccines are an immunobiological substance designedto produce specific protection against a given

disease. Bacterial components likeGlucan binding proteins, glucosyltransferases can make the most effective vaccines which can be administered through various routes 16. Integrating the caries vaccine after its development into publichealth programs could be beneficial in bringing dental caries toa minimal level. As a known fact 'Prevention is better than cure', several methods have been suggested for caries control which may be chemical measures, nutritional measures and mechanical measures 17. Chemical measures in India include fluoridation of water, fluoride supplements, fluoride dentifrices, silver nitrate, bis-biguanides, vitamin-K, antibiotics like penicillin, tetracycline and various plaque controlling agents (Table.5).

Table.5- Studies of the efficacy of caries prevention in high caries risk individuals(James D. Bader, 2001)

Study Reference	Quality Score	Treatment	Percent Reduction	p Value	Number Needed to Treat
Fluoride A	gents				
51	60	0.04% NaF rinse, once per day	15%	>.05	2.5
52	50	2.2% F varnish (Duraphat), twice yearly	30%	<.001	1.6
52	50	0.7% F varnish (FluorProtector), twice yearly	11%	ns*	5.4
53	55	2.2% F varnish (Duraphat), four times per year	7%	>.05	4.3
53	55	0.2% Ferric Aluminum F topical, four times per year	13%	>.05	2.5
54	80	1.23% APF gel, twice yearly	9%	>.05	6.7
55	55	1.1% F varnish (Duraphat), three times per year	0%	_	_
56	60	1% Amine F rinse, twice per year	24%	not rptd+	10.2
57	50	0.1% F varnish (FluorProtector), twice yearly	25%	<.05	3.5
Chlorhexi	dine Agents				
58	40	1% CHX <sup>2</sup> gel, whenever ms > 2.5*10 <sup>5</sup>	26%	ns	2.0
59	60	1% CHX gel, four times per year	44%	not rptd	1.5
53	55	1% CHX gel, eight times in two days, whenever ms > 2.5*105	52%	<.001	0.6
60	70	0.05% CHX rinse, twice daily for five days, every third week	3%	ns	27.5
61	25	CHX varnish, three times in eight months	25%	not rptd	_
62	55	CHX varnish, twice yearly	33%	<.05	2.8
62	55	CHX varnish, twice yearly	-9%	>.05	_
Combinati	ion Agents				
51	60	1% CHX / NaF rinse, once per day	43%	<.001	0.9
63	45	1% CHX gel once per day for two weeks every four months			
		when ms > 2.5*105, and occlusal sealants	81%	<.001	0.2
64	45	1% CHX gel as needed, and NaF topical and NaF gel	89%	<.05	0.7
60	70	0.05% CHX / 0.04 NaF /500 ppm Sr rinse, twice per day			
		for five days every third week	8%	>.05	9.2
60	70	0.05% CHX / 0.04F twice per day for five days			
		every third week	34%	>.05	2.1
65	40	1% CHX rinse, and 0.2%F rinse twice yearly to mothers	13%	ns	33.5
66	65	1%CHX / 0.1% NaF varnish, twice yearly	-26%	ns	_
Other Age	ents				
67	40	5% Kanamycin gel, twice/day for one week, repeated once	46%	not rptd	1.6
68	65	Occlusal sealants applied as needed, no repair	88%	not rptd	4.4
19	70	Xylitol gum, 3.5 g three times per day	55%	<.001	1.4
70	60	dentist directed to use high risk protocol	13%	ns	5.9
71	65	0.9% alum rinse, once per day	23%	ns	2.2
72	65	sorbitol / manitol / aspartame gum, three times per day	11%	.003	3.0

<sup>\*</sup>ns=reported as not statistically significant

<sup>\*</sup>not rptd= no statistical testing reported \*CHX=chlorhexidine

Nutritional methods comprises of phosphate diets18with less sugar intake with low frequency. Mechanical measures include prophylaxis by the dentist, tooth brushing, use of dental floss, chewing gums etc19. Various caries remineralizing agents are also used to slow down the process of demineralization 20 like Bioactive glass, Nanohydroxyapatite, Silver diamine fluoride, Theobromine, Self assembling peptides, Tricalcium phosphate products,etc21. The Government of India has started certain programmes and health related schemes like 'Ayushmann Bharat' and 'National Oral Health Program'. In remote areas where dental equipments are not available, a conservative approach i.e. Atraumatic Restorative Treatment(ART) is introduced which is based on removing decalcified tooth tissue using only hand instruments and restoring the cavity with an adhesive filling material22. The demand of restorative treatment in the developing countries is higher than the resources available for public health programs<sup>23</sup>.

Now, this greatest challenge of executing this knowledge into the large population of Indian masses having great diversity in eating habits and behavioral practices, persons relying on dental myths can be overcome by organizing workshops, seminars, webinars, live demonstrations. Occasional visits to schools to educate children about the proper brushing techniques and importance of oral hygiene can be organized.

### Conclusion

It should be emphasized that a good oral hygiene is as crucial as maintaining one's overall health24. Dental caries should be seen as a condition that can greatly affect the health and quality of life, so it is extremely important to increase the knowledge towards its development, focusing on prevention and the correct therapeutic approach. So, prevent dental caries and keep smiling forever.

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