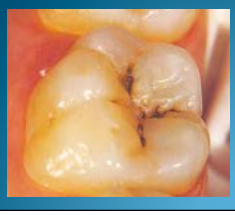
DENTAL CARIES

EARLY THEORIES, CLINICAL FEATURES AND HISTOPATHOLOGY



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INTRODUCTION

Dental caries has been recognized throughout history and exists around the world, although the prevalence and severity varies in different populations. Caries is one of the most common of all diseases and partly because of its relatively rapid progress. Ultimate effect of caries is to breakdown enamel and dentin and to open a path for bacteria to reach the underlying tissues. This causes infection and inflammation of the pulp and later of the periapical tissues.

EARLY THEORIES

THE LEGEND OF WORMS

- Ancient Sumerian text; 5000 BC
- Discovered on clay tablets excavated within Euphrates valley of the lower Mesopotamian area .
- Toothache caused by a worm that drank the blood of teeth and fed on the roots of the jaws.
- Idea that caries is caused by a worm was universal , as evidenced by the writings of Homer.

ENDOGENOUS THEORIES

HUMORAL THEORY

- Advanced by Greek physicians
- Persons physical and mental constitution was determined by relative proportion of four elemental fluids of body which are :

  Blood, phlegm, black bile, and yellow bile

  Corresponding to four humors-

  sanguine, phlegmatic, melancholic, & choleric
- All diseases including caries could be explained by an imbalance of these humors

VITAL THEORY

- End of 18th century
- Dental caries originates within the tooth itself, similar to bone gangrene
- A clinically well known type of caries is characterized by extensive penetration into the dentin, and even into the pulp, but with a barely detectable catch or a fissure.

EXOGENOUS THEORIES

CHEMICAL THEORY

- Parmly;1819
- Dental decay affected externally, not internally.
- Unidentified "chemical" agent responsible for caries.
- Caries began on the enamel where the food putrefied and acquired sufficient dissolving power to produce the disease chemically.
- Robertson(1835) said dental decay caused by acid formed by fermentation of food around teeth.

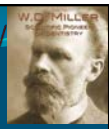
PARASITIC THEORY

- Erdl;1843-described filamentous organisms in the " surface membrane" of teeth.
- Ficinus;1847- 'denticolae' – decay related microorganisms. He implied that these bacteria caused decomposition of the enamel and then the dentin .
- Neither Erdl nor Ficinus explained how these organisms destroyed tooth structure.

SEPTIC THEORY

- Underwood, Milles; 1880- acid capable of causing decalcification was produced by bacteria feeding on organic fibrils of dentin.

CHEMICOPARASITIC THEORY / ACIDOGENIC THEORY



- Willoughby D. Miller; 1890
- Caries is caused by acids produced by microorganisms in mouth
- consists of 2 stages:
  1. Decalcification & destruction of enamel and dentin
  2. Dissolution of the softened residue

The process is supported by the presence of carbohydrates, microorganisms and dental plaque.

PROTEOLYTIC THEORY

- Gottlieb; 1944
- Organic elements are the initial pathway of invasion.
- initial action was due to proteolytic enzymes attacking the organic structures(lamellae ,rod sheaths ,tufts, dentinal tubules walls)
- acid formation accompanied the proteolysis, dissolving inorganic salts.

PROTEOLYSIS-CHELATION THEORY

- Schatz et al; 1955
- Challenged chemo-parasitic theory; acid may prevent tooth decay by interfering with growth and activity of proteolytic bacteria
- Simultaneous microbial degradation of the organic components & dissolution of minerals, by a process called Chelation
- Chelation: complexing of metallic ion to a complex substance through a coordinate covalent bond, resulting in a highly stable, poorly dissociated, weakly ionized compound

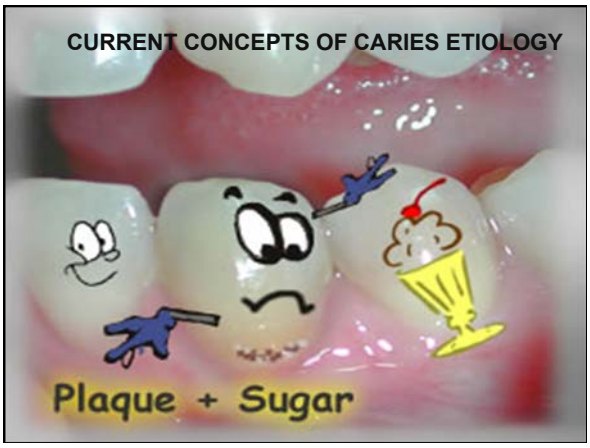
- Chelation is independent of the PH of the medium.

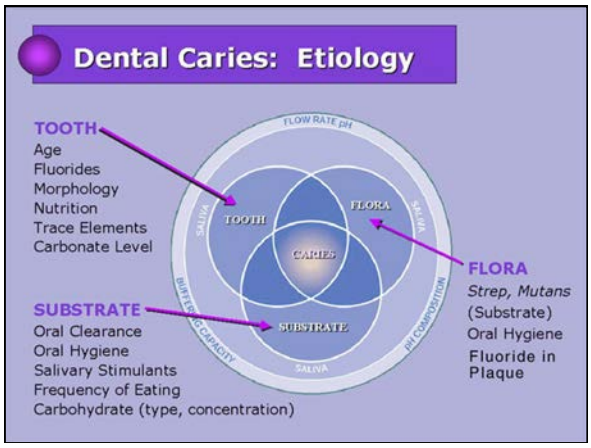
Bacterial attack on enamel surface - initiated by keratinolytic microorganisms - result in the breakdown of the protein chiefly keratin and formation of soluble chelates which decalcify enamel even at neutral PH

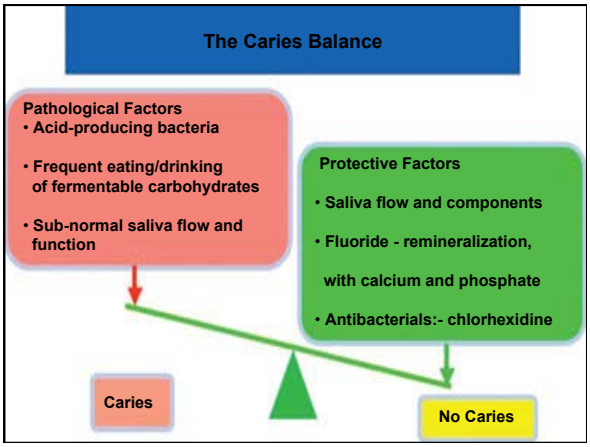
SUCROSE CHELATION THEORY

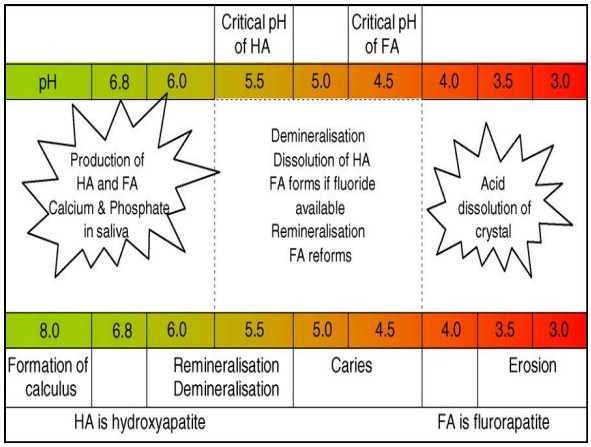
- Egglers-Lura; 1967
- High sucrose concentrations often encountered in the mouths of caries active individuals form calcium saccharate; thus a direct interaction between sucrose and calcium takes place
- Unlikely to be a significant process because of the rapidly with which sucrose is metabolized to acid and polysaccharide, and because calcium saccharates can only form at high pH, above the range usually found in the mouth.

- **ETIOLOGY OF DENTAL CARIES**









CLINICAL FEATURES OF DENTAL CARIES

- Incipient caries: Early carious lesion manifested as chalky white, opaque region- 'white spot' - best demonstrated when area is air dried
- Some enamel demineralization has occurred; absence of cavity or major histologic change
- As lesion demineralizes, will eventually turn into a cavitation
- A brown spot which is dull in appearance is probably a sign of active caries
- Affected areas change colour ; soft to touch.

- When decay passes through enamel, dentinal tubules becomes exposed, cause tooth to become symptomatic.
- Pain may worsen with exposure to heat, cold, or sweet foods and drinks
- Caries advances more rapidly in dentin than enamel; dentin provides much less resistance to acid attack
- Pulp dentin complex reacts to caries attack by attempting to initiate remineralization & blocking off open tubules

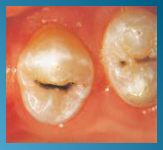
G.V.BLACK'S CLASSIFICATION
(based on treatment and restoration design)

CLASS I:

Caries in structural defects of the teeth i.e pits, fissures, grooves.

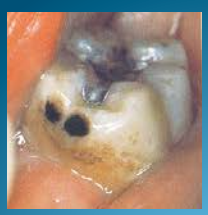
Locations-

- occlusal surfaces of premolars and molars
- occlusal 2/3rd of the buccal and lingual surfaces of molars and premolars
- lingual surfaces of maxilary anteriors



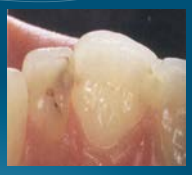
CLASS II:

- Proximal surfaces of posterior teeth



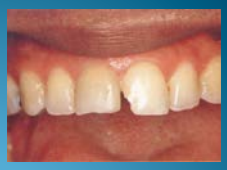
CLASS III:

Proximal surfaces of anterior teeth without involving incisal angle



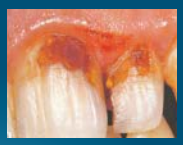
CLASS IV:

Proximal surfaces and incisal angles of anterior teeth



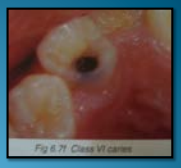
CLASS V:

Cervical 3rd of anterior and posterior teeth



CLASS VI:

Occlusal cusps of posterior teeth, incisal edges of anterior teeth



CLINICAL CLASSIFICATION OF DENTAL CARIES

1). According to Morphology i.e. according to anatomical site of the lesion
-pit and fissure caries
-smooth surface caries
-root caries

2). According to Dynamics, i.e. according to severity and rate of progression of lesion
-acute dental caries
-chronic dental caries
-arrested caries

3). According to whether lesion is a new or recurrent one
-primary(virgin) caries
-secondary (recurrent) caries

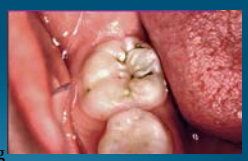
4). According to Pathway Of Caries Spread
-Forward caries
-Backward caries

5). According to Number Of Tooth Surfaces Involved
-Simple caries
-Compound caries
-Complex caries

6). According to Chronology
-Nursing bottle caries
-Adolescent caries
-Adult caries

PIT AND FISSURE CARIES

-Occlusal surface of molars & premolars
-Buccal pits of molars
-Lingual surfaces of maxillary anterior teeth



•Most common surface involved in modern humans

•Deep, narrow pits- poor self cleansing features- most prone for caries

•Appear brown/black, soft, and 'catch' a fine explorer point

•Enamel may become bluish-white; lateral spread of caries at DEJ

Noncavitated (caries free) -

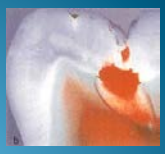
-Deep grooves may be present
-Superficial staining may be present in grooves
-Mechanical binding of explorer may occur

Cavitated (diseased) -

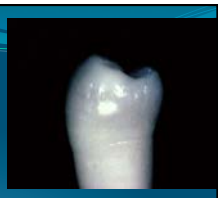
-Chalkiness of enamel on walls & base of pit or fissure
-Softening at base of a pit or fissure
-Brown- gray discoloration under enamel adjacent to pit /fissure

Occult Caries / Hidden Caries

- A large carious lesion with a small opening; detected radiographically
- Increased Fluoride exposure – remineralization & slows down caries progression in pit & fissures in enamel while it continues in dentin.
- May give way under mastication forces
- Earlier mistaken as 'internal caries'
- According to Seow(2000),Prevalence – 0.8% in premolars in 14-15 years old;upto 50% in 20 year olds
- 'Fluoride bomb' , 'Fluoride syndrome'



SMOOTH SURFACE CARIES



- Location:
  -proximal surface of teeth
  -Gingival third of buccal, lingual surfaces
- Preceded by dental plaque formation
- Small opaque white region showing contrast with translucency of adjacent enamel- 'white spot lesion'
- More advanced lesion- extensive in outline- follow curvature of cervical border of interdental facet –
  kidney shaped contour

- Enamel surface -**hard and shiny;**
  Similar to adjacent sound enamel when examined with explorer
- Surface lesion may appear brownish in color - Brown spot lesions
- Staining depend on the degree of exogenous material absorbed by the porous region; length of time the lesion present

Noncavitated (caries free) -

- Surface intact
- Opacity of proximal enamel may be present
- Marginal ridge is not discolored
- Opaque area may be seen

Cavitated (diseased) -

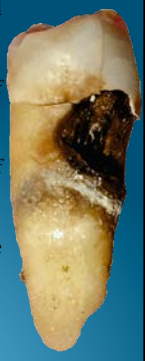
- Surface broken
- Marginal ridge may be discolored
- Opaque area in dentin
- Reduced density of sub-surface, detectable with transillumination

ROOT CARIES

(cemental caries, radicular caries, senile caries)

- Carious lesions that initiate at the dentinal – root portion
- Dentitions of older age groups with gingival recession and exposed roots
- 60% Root Caries – 50 to 59 years (Banting & Ellen)
- Most frequently on buccal and lingual surface of roots in contemporary populations
- In ancient populations root lesions predominated on proximal surfaces

- No symptoms; pain may be there in advanced lesions
- Soft in consistency at an early stage of development
- Ill-defined area, often discoloured, destruction of cementum with penetration of underlying dentin
- Mostly starts adjacent to gingival crest where dental plaque accumulates



Diagnosis of root caries, David.W. Banting
Journal of dental education, Oct 2001, vol 65, no.10: 991-996

- As it progresses it extends circumferentially rather than in depth; seldom more than 0.5-1mm in depth; U-shaped in C/S
- Mandibular molars most frequently followed by mandibular premolars, maxillary canines; mandibular incisors least involved
- Most predominant microorganism: A. viscosus
- Root caries is alarming because:
  - often asymptomatic
  - rapid progression
  - close to pulp
  - difficult to restore

- Cementum begins to demineralise at 6.7 pH, higher than enamel's critical pH; root more vulnerable to demineralization than enamel
- Easier to arrest the progression of root caries than enamel caries because roots have a greater reuptake of fluoride than enamel

Root Caries.
Oper Dent. 2004 Nov-Dec;29(6):601-7.

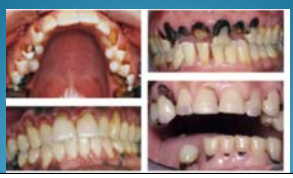
Based On Speed Of Caries Progression

- Acute Caries
- Chronic Caries
- Arrested Caries

RAMPANT CARIES

Suddenly appearing, widespread, rapidly progressing type of caries with early pulpal involvement

- A caries increment of 10 or more new carious lesions over a period of one year.
- Seen in individuals with xerostomia, poor oral hygiene, due to drug-induced dry mouth, large sugar intake
- Most often seen in primary dentition & permanent dentition of teenagers 11 -19 yrs.

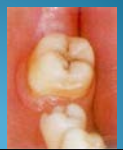


CHRONIC CARIES

- Slow progressing , long standing caries & involves the pulp later than acute caries.
- Deposition of secondary dentin.
- Hard in consistency & dark in color.
- Usually patient does not complain of any symptoms; detected during examination

ARRESTED CARIES

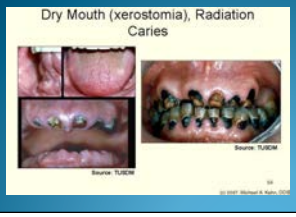
- Appears brown and shiny; suggests caries was once present but the demineralization process has stopped, leaving a stain.
- A change in the oral environment can result in arrest of the caries process.
- E.g.: If a crown is lost due to caries, remaining part of tooth becomes self cleansing(direct access by saliva to the carious dentin), the caries process gets arrested, and dentin rehardened.



Xerostomia induced caries (radiation caries)

common complication of radiotherapy of oral cancer – xerostomia – rampant caries

Appear as early as 3 months after onset of xerostomia.



Caries susceptibility of jaw

Caries is usually bilateral

Affects maxillary teeth more than mandibular

Caries susceptibility of individual tooth surfaces

- Occlusal surfaces –maximum involvement
- Buccal surface more than lingual in mandibular molars
- Palatal surface more than buccal in maxillary molars
- Mesial more than distal surface