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DENTAL CARIES: A BRIEF REVIEW

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Abstract: Dental caries is defined it as a microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth. **[1]**

IndexTerms**:** Dental caries

I. Introduction

Dental caries is a chronic, transmissible infection in which there is an altered tooth structure due to loss of chemicals resulting from the metabolic activity and also occurrence of dental biofilm on the surface of the tooth. Complications may include inflammation of the tissue around the tooth, tooth loss and infection or abscess formation. **[2]**

II. Classification

- G.V. Black Caries Classification **[3]**
  1. Class I: Cavity in pits or fissures on the occlusal surfaces of molars and premolars; facial and lingual surfaces of molars; lingual surfaces of maxillary incisors
  2. Class II: Cavity on proximal surfaces of premolars and molars
  3. Class III: Cavity on proximal surfaces of incisors and canines that do not involve the incisal angle
  4. IV: Cavity on proximal surfaces of incisors or canines that involve the incisal angle
  5. Class V: Cavity on the cervical third of the facial or lingual surfaces of all teeth
  6. Class VI: Cavity on incisal edges of anterior teeth and cusp tips of posterior teeth

- According to: **[4]**
- Location:
  1. Pit and fissure caries- caries on the pits' (small hollows) and 'fissures' (grooves) on their biting surfaces.
  2. Smooth surface caries- caries on the smooth surface of the teeth, usually between teeth and along the gumline
- Rate of progression:
  1. Rampant- caries which occur rapidly involving a large number of teeth
  2. Arrested- caries that have stopped progressing and are inactive
  3. Incipient- caries lesion which occur in the early and is white and chalky in appearance
  4. Recurrent- caries that occur under existing dental restorations
- Extent:
  1. Enamel caries
  2. Dentinal caries
  3. Root caries
- Direction of progression:
  1. Forward caries- caries which proceeds from enamel to dentin in a triangular pattern with the base of the triangle towards enamel and apex towards the dentin
  2. Backward caries- caries which proceeds from dentin to enamel in a triangular pattern with the base of the triangle towards dentin and apex towards the enamel

III. Theories of Dental Caries [5]

1. Miller's chemo-parasitic theory or the Acidogenic theory

➢ Theory- Caries is caused by acids produced by microorganism of the mouth

- Given by Willoughby D. Miller in 1882.

➢ According to his hypothesis, dental decay is a chemo parasitic process consisting of 2 stages: -

a) Decalcification of enamel and dentin (preliminary stage)
b) Dissolution of the softened residue (subsequent stage)

Acids resulting in primary decalcification are produced by the fermentation of starches and sugar from the retaining centres of teeth.

Miller found that incubating sugar, meat and bread in vitro at body temperature produces enough acid within 48 hours to decalcify sound dentin.

Miller also believed that caries was caused by a variety of microorganisms after isolating acidogenic and proteolytic microorganisms. He assigned an essential role to 3 factors in caries: -

a) Oral microorganisms in acid production and proteolysis
b) The carbohydrate substrate
c) Acid which causes dissolution of tooth minerals

➢ Millers's theory couldn't explain: -

a) Predilection of specific sites on a tooth to caries
b) Initiation of smooth surface caries
c) Caries free population
d) Phenomenon of arrested caries

2. The proteolytic theory

➢ Theory- Organic or protein elements of a tooth are the initial pathway of invasion by microorganisms.

- Enamel lamellae are pathways for organisms in the progress of dental caries.

➢ Gottlieb and Gottlieb, Diamond and Applebaum: Caries is essentially a proteolytic process- the microorganisms invade the organic pathways and destroy them in advance. Acid formation accompanies proteolysis.

- Drawbacks- No satisfactory evidence to support the claim that the initial attack on enamel is proteolytic. Gnotobiotic studies: Caries can occur in the absence of proteolytic organisms.

➢ Conclusion- Proteolysis in the initiation of dental caries is likely to be of no significance, but its role in the progression of the more advanced carious lesion cannot be ruled out.

3. The proteolysis- chelation theory

➢ Theory- simultaneous microbial degradation of the organic components and the dissolution of the minerals of the tooth by the process known as chelation.

- Chelation- A process involving the complexing of a metallic ion to a substance through a covalent bond which results in a highly stable, poorly dissociated or weakly ionized compound.

➢ Effects of chelation

a) Independent of pH of medium
b) Removal of metallic ions such as calcium from a biologic calcium- phosphorus system may occur at a neutral or even alkaline pH
c) The proteolysis- chelation theory resolves the argument to as whether the initial attack of dental caries is on the organic or inorganic portion of enamel by stating that both may be attacked simultaneously.

IV. Aetiology

Dental caries is a multifactorial disease with an interplay of 4 primary factors- **[6]**

- Host

1. Tooth structure

The structure of enamel, and of dentine in root caries, is important: some areas of the same tooth are much more susceptible to carious attack than others, possibly because of differences in mineral content (especially fluoride).

2. Flow rate and composition of saliva

The mechanical washing action of saliva is a very effective mechanism in the removal of food debris and unattached oral microorganisms. It has a high buffering capacity, which tends to neutralize acids produced by plaque bacteria on tooth surfaces, and it is supersaturated with calcium and phosphorus ions, which are important in the remineralization of white-spot lesions. Saliva also acts as a delivery vehicle for fluoride

- Microbial flora
  Two species of the '*mutans streptococci'* viz. *Streptococcus mutans* and *Streptococcus sobrinus* are the foremost cariogenic pathogens in tooth decay. They are highly acidogenic, producing short-chain acids which soften hard tissues of teeth. Three isozymes of glucosyltransferases catalyze and metabolize sucrose to synthesize insoluble extracellular polysaccharides like glucan, which increase their adherence to the tooth surface and serve as a nutritional source and a matrix which persuade biofilm formation.

- Steps of biofilm formation **[7]**

a. Association – Dental pellicle forms on the tooth & provides bacteria surface to attach.

b. Adhesion – Within hours, bacteria loosely binds to the pellicle.

c. Proliferation – Bacteria spreads throughout the mouth & begins to multiply.

d. Microcolonies – Microcolonies are formed, streptococci secrete protective layer (slime layer).

e. Biofilm formation – Microcolonies from complex groups with metabolic advantages.

f. Maturation – The biofilm develops a primitive circulatory system.

S. mutans is more cariogenic than *S. sobrinus* because of the presence of specific cell-surface proteins, which assist in its primary attachment to the tooth.

Lactobacillus and *Actinomyces* are also associated with caries.

Lactobacilli are dominant part of the flora, considered as pioneer microbes in the development of caries particularly in dentin. It inhabits the deep cavities, and their number correlates with the quantity of carbohydrates.

Actinomyces odontolyticus colonizes infants before eruption of teeth. Some root caries lesions are dominated by *Actinomyces naeslundii, A. israelii* and *A. gerencseriae*. The other significant species involved in caries includes *Streptococcus mitis, Bifidobacterium* and *Actinomyces* , a group of 'low pH' aciduric isolates which have been isolated from white spot lesions in humans. **[8]**

Different microorganisms can exist in single or poly-microbial communities in caries. i.e.

• Gram positive cocci: - Streptococcus mutans, S. mitis, S. salivarus, S. sanguis, S. intermedius, S. vestibularis, Staphylococcus aureus, Atopobium *spp*, Peptostreptococcus *spp*, Enterococcus fecalis

• Gram positive rods: - Actinomyces odontolyticus, A. naeslundii, A. viscosus, A. israelii, Lactobacillus fermentum, L. acidophillus, Bifiodobacterium dentium, Propionibacterium spp.

• Gram negative cocci: - Veillonella parvula, Nesseria spp.

• Gram negative rods: - Bacteriodes denticola, B. melaninogenicus, Fusobacterium necrophorum, F. mortiferum, Escherishia coli, Klebsiella pneumoniae, Enterobacter aerogens, Citrobacter freundi, Pseudomonas fluorescence, Haemophilus spp, Prevotella *spp*, Leptotrichia spp.

• Yeasts: - Candida albicans, C. tropicalis, *C.* glabrata **[9]**

- Diet

There is a direct relationship between dental caries and the intake of carbohydrates. The most cariogenic sugar is sucrose,

Sucrose is highly soluble and diffuses easily into dental plaque, acting as a substrate for the production of extracellular polysaccharides and acids. The relationship between sucrose and dental caries is complex and cannot be simply explained by the total amount of sugar consumed. The frequency of sugar intake rather than the total amount of sugar consumed appears to be of decisive importance. Also relevant are the stickiness and concentration of the sucrose consumed, both factors influencing the period for which sugar is retained in close contact with the enamel surface.

Carbohydrates other than sucrose, e.g. glucose and fructose, are also cariogenic, but less so than sucrose. Polyol carbohydrates, 'sugar alcohols' (e.g. xylitol), with low cariogenicity have been produced and are sought after as sugar substitutes in products such as chewing gum and baby foods. **[10]**

- Time

While the shift in microflora can occur over a fairly short period, a significant amount of time is needed for demineralization to lead to the development of white-spot and/or carious lesions. Acid production does not instantly trigger tooth decay, and in the early stages, remineralisation can restore enamel, keeping the effects of dental caries at bay. **[11]**

V. Risk factors

1. Sugar consumption:

   The growth rate of many oral bacteria increases and changes the composition of the microflora in a caries-promotion with sucrose- rich diet. Acid-producing micro-organisms, such as the *mutans streptococci* in dental plaque, play a crucial role in the caries development.

2. Tooth location:

   Molars and premolars are more susceptible towards tooth decay as these teeth have lots of grooves, pits and crannies that can accumulate food particles. As a consequence, they're difficult to maintain hygienic and clean. Plaque can fabricate and bacteria can increase between back teeth, producing the acid that demolishes tooth enamel.

3. Socio-economic factors:

   Low socio-economic status ultimately influence oral hygiene standards and attitudes to tooth care in children and adolescents

4. Age:

   Cavities are more frequent in children and teenagers and older adults also are at elevated jeopardy. Teeth can wear down and gums may recede, making teeth more vulnerable to root decay by overtime. Older adults can possibly use supplementary medications that decrease saliva flow, escalating the risk of dental caries

5. Dry mouth:

   Dry or dehydrated mouth is caused by a deficiency of saliva. Substances found in saliva also assist to oppose the acid produced by bacteria and can help in restore early caries. Certain medications, some medical conditions, radiation to head or neck, or certain chemotherapy drugs can augment risk of cavities by reducing saliva production

1. Dental fillings or dental devices:

   Dental fillings can weaken, begin to break down or develop rough edges over the years which allow plaque to build up more simply and make it harder to remove. Dental devices can also stop fitting well, allowing decay to begin beneath them

2. Eating disorder:

   Significant tooth erosion and cavities can also be due to anorexia and bulimia. Stomach acid from repeated vomiting (purging) cleanses over the teeth and instigates melting the enamel. Eating disorders can also hinder with saliva production

3. Heartburn:

Gastroesophageal reflux disease (GERD) or heart burn can cause stomach acid to flow into mouth (reflux), wearing away the enamel of teeth leads to significant tooth damage

4. Genetic factors

The presence of bacteria and carbohydrates are necessary for caries to develop. Whether this actually happens depends upon the inherited or acquired resistance of the teeth

5. Malnutrition

Children who are malnourished pre-, peri- or post-natally and/or who are of low birth weight are likely to have hypomineralised or hypoplastic primary teeth. These teeth have a higher risk of becoming carious and are more susceptible to mutans streptococci colonization **[12]**

VI. Caries mechanism

Susceptible tooth surface—formation of biofilm and microbial deposits—acid production and pH change—shift in dynamic change of mineral equilibrium—dissolution of minerals—initiation of caries. **[13]**

VII. Signs and symptoms

- The earliest sign of a new carious lesion is the appearance of a chalky white spot on the surface of the tooth, indicating an area of demineralization of enamel referred to as a white spot lesion or an incipient carious lesion.
- As the lesion continues to demineralize, it can turn brown but will eventually turn into a cavitation. Before the cavity forms, the process is reversible, but once a cavity forms, the lost tooth structure cannot be regenerated.
- A lesion that appears dark brown and shiny suggests dental caries were once present but the demineralization process has stopped, leaving a stain. Active decay is lighter in color and dull in appearance.
- As the enamel and dentin are destroyed, the cavity becomes more noticeable. The affected areas of the tooth change color and become soft to the touch.
- Once the decay passes through enamel, the dentinal tubules, which have passages to the nerve of the tooth, become exposed, resulting in pain that can be transient, temporarily worsening with exposure to heat or cold.
- A tooth weakened by extensive internal decay can sometimes suddenly fracture under normal chewing forces.
- When the decay has progressed enough to allow the bacteria to overwhelm the pulp tissue in the centre of the tooth, a toothache can result and the pain will become more constant. **[14]**

VIII. Prevention

- Oral hygiene

  Personal hygiene care consists of proper brushing and flossing daily. The purpose of oral hygiene is to minimize any etiologic agents of disease in the mouth. The primary focus of brushing and flossing is to remove and prevent the formation of plaque. A toothbrush can be used to remove plaque on most surfaces of the teeth except for areas between teeth. When used correctly, dental floss removes plaque from areas which could otherwise develop proximal caries. Other adjunct hygiene aids include interdental brushes, water picks, and mouthwashes.

  Professional hygiene care consists of regular dental examinations and cleanings

- Dietary modification

  For dental health, the frequency of sugar intake is more important than the amount of sugar consumed. In the presence of sugar and other carbohydrates, bacteria in the mouth produce acids which can demineralize enamel, dentin, and cementum. The more frequently teeth are exposed to this environment, the more likely dental caries are to occur. Therefore, minimizing snacking is recommended, since snacking creates a continual supply of nutrition for acid-creating bacteria in the mouth. Also, chewy and sticky foods tend to adhere to teeth longer, and consequently are best eaten as part of a meal.

  Also, chewing gum containing xylitol is widely used to protect teeth. Xylitol's effect on reducing plaque is probably due to bacteria's inability to utilize it like other sugars. Chewing and stimulation of flavour receptors on the tongue are also known to increase the production and release of saliva, which contains natural buffers to prevent the lowering of pH in the mouth to the point where enamel may become demineralised.

- Other preventive measures

  The use of dental sealants is a good means of prevention. Sealants are thin plastic-like coating applied to the chewing surfaces of the molars. This coating prevents the accumulation of plaque in the deep grooves and thus prevents the formation of pit and fissure caries.

Fluoride therapy is often recommended to protect against dental caries. It has been demonstrated that water fluoridation and fluoride supplements decrease the incidence of dental caries. Fluoride helps prevent decay of a tooth by binding to the hydroxyapatite crystals in enamel. The incorporated fluoride makes enamel more resistant to demineralization and, thus, resistant to decay. Topical fluoride is also recommended to protect the surface of the teeth. This may include a fluoride toothpaste or mouthwash.

IX. Diagnosis

Diagnosis is usually by a combination of: **[16]**

1. Direct observation.
2. Probing
3. Radiographs.
4. Experimental methods- laser fluorescence for diagnosis of buccal and lingual caries and electrical impedance (resistance) to detect occlusal caries.
5. Microbiological tests

X. Treatment

Destroyed tooth structure does not fully regenerate, although remineralisation of very small carious lesions may occur if dental hygiene is kept at optimal level. For the small lesions, topical fluoride is sometimes used to encourage remineralisation. For larger lesions, the progression of dental caries can be stopped by treatment.

Anaesthetics — local, nitrous oxide, or other prescription medications — may be required in some cases to relieve pain during or following treatment or to relieve anxiety during treatment. A dental hand piece is used to remove large portions of decayed material from a tooth. A spoon excavator is used to remove decay carefully and is sometimes employed when the decay in dentin reaches near the pulp. Once the decay is removed, the missing tooth structure requires a dental restoration of some sort to return the tooth to functionality and aesthetic condition.

Restorative materials include dental amalgam, composite resin, porcelain, and gold. When the decay is too extensive, there may not be enough tooth structure remaining to allow a restorative material to be placed within the tooth. Thus, a crown may be needed.

In certain cases, root canal therapy may be necessary for the restoration of a tooth and is recommended if the pulp in a tooth dies from infection by decay-causing bacteria or from trauma. During a root canal, the pulp of the tooth, including the nerve and vascular tissues, is removed along with decayed portions of the tooth. The canals are instrumented with endodontic files to clean and shape them, and they are then usually filled with gutta percha. Upon completion of a root canal, the tooth is now non-vital, as it is devoid of any living tissue.

An extraction can also serve as treatment for dental caries. The removal of the decayed tooth is performed if the tooth is too far destroyed from the decay process to effectively restore the tooth. **[17]**

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