Lec. 2
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Oral pathology

Dental caries

Dental caries is a multifaceted disease involving interplay among the teeth, the oral host factors of saliva and microflora, and the external factor of diet. The disease is a unique form of infection in which specific strains of bacteria accumulate on the enamel surface, where they elaborate acidic and proteolytic products that demineralize the surface and digest its organic matrix.

Once penetration of the enamel has occurred, the disease progress through the dentin to the pulp. If the process is not stopped, the tooth becomes destroyed.

Factors affecting caries prevalence: -

1- race: people living in same geographical area but belonging to different race have differing caries incidence. Generally, Chinese, blacks, Indians have lesser caries incidence than the Caucasian whites.

2. Age: dental caries more prevalent in children up to 12 years. Incidence decreases somewhat in younger and middle age group. Incidence increases again by the older age.

3. Gender: incidence of caries is significantly higher in females than males. This may be due to the fact that teeth in females erupt earlier compared to males.

4. Familial: there appears to be heredity involved. Children of parents with low caries experience also show lesser caries incidence and vice versa.

Loss of tooth substance may result from the action of oral microorganism in dental caries (bacterial causes), or may be due to non- bacterial causes, which include:

A- Mechanical factors associated with attrition and abrasion.

B- Chemical erosion.

C- Pathologic resorption.

Primary causes of dental caries

Dental plaque, dietary carbohydrates, tooth (susceptible tooth surface) and time.

The carious process : bacteria in dental plaque fermentable carbohydrates such as sugars (sucrose & glucose). .production of acids causing the plaque ph to fall below 5 .Repeated fall in ph in time may result in the demineralization of susceptible site on the tooth surface initiating the carious process.

Dental caries follows the interaction of four main factors, the host, bacteria, food (diet) and time for the process to develop.

Food + bacteria acid +tooth D.C.

Caries is one of the most common of all diseases and still a major cause of loss of teeth.

Dental caries is the most prevalent chronic disease in man throughout the world, 95% of the population have decay or will have it before they die. The only way to control the disease is through the use of systemic and topical fluoride, furthermore dental caries can be controlled by controlling the four main factors that are related to it: -

1- Host (tooth): administration of fluoride (as tables, or fluoride-containing diet), and fissure sealant (seal deep fissures in tooth surface to prevent accumulation of plaque).

2- Microorganism (bacterial flora): their action is hindered by active and passive immunization, and reduces the intake of sugars.

3- Diet (food):- reduction in consumption of cariogenic sugar like sucrose, fructose, maltose, glucose, both intrinsic sugar (from fruits and vegetables) and extrinsic sugars (added sugars, milk, fruit juices).

sucrose is considered as the most cariogenic type of sugar because

(1) it is readily fermented by bacterial plaque, and

(2) its easily converted to extracellular glucans by bacterial glucosyltransferase.

Glucans act as glue for bacteria helping their adherence to tooth surface.

4- time: frequent sugar intake between primary meals, as well as stopping teeth brushing for 12-14 hours will permit formation of bacterial plaque.

There are other indirect factors that have a role in the development of dental caries, such as:-

Tooth: regarding its

・Composition; (less fluoride, iron, zinc, magnesium make tooth more susceptible to dental caries).

・Morphology; (deep pits and fissures can seat more bacterial plaque).

・Position; (malposed tooth can hold more bacterial plaque).

Saliva: regarding its:

・Composition; inorganic constituents are more beneficial than organic constituents.

・ph; the higher the ph the less the action of bacteria.

・Quantity; the more the best washing action of plaque out of embrasures, fissures and pits.

・Viscosity; the more watery the best for the removal of plaque.

・Other antibacterial factors that prevent the proliferation of bacterial flora.

Diet (food): regarding its: Physical factors: quantity of diet.

Local factors: carbohydrate content, fluoride content, vitamin content.

Soft sticky food enhances the formation of plaque, and consequently caries. Refined carbohydrates, especially sucrose, are more likely to cause caries than raw products.

Systemic factors

Heredity: - racial tendency for high or low caries may be explained by heredity. However, local factors like change in dietary habits can change this tendency. Possible that caries tendency may be inherited through tooth form & structure

Pregnancy & lactation: - commonly observed that during pregnancy, women tend to neglect their oral health owing to all her attention being diverted to that of care for the newborn. Thus increased caries incidence during pregnancy & lactation is more a problem of neglect.

Etiology and pathogenesis:

Etiology is still controversial and not clear, due to its being complicated by many direct and indirect factors. Many theories were postulated in order to explain dental caries. Most noticed theories are:

1) ***Acidogeneic theory (miller s chemoparasitic theory 1890):-*** it is the most accepted and supported theory, because it is based on experimental studies; made later by Orland and his workers in 1954, showed that in germ free oral hygiene in some laboratory animals ,even with administration of sugar; there is no dental caries in these animals. Thus, dental caries is produced by chemical action of acids produced by micro flora.

Miller s theory suggests that dental caries develop in two phases. In the first phase, microflora attack the inorganic structure, where decalcification of enamel and dentin is carried out by means of acids produced as a result of fermented sugar accumulating in retaining spots on tooth surface .in the second phase, dissolution of the soft organic part is carried out.

Miller isolated numerous microorganisms from the oral cavity; most important species are lactobacillus acidophilus, streptococcus mutans , streptococcus sanguis , and streptococcus salivarius.

In his hypothesis, miller assigned essential roles to 3 factors:

1. Carbohydrate substrate.

2. Acid which caused dissolution of tooth minerals.

3. Oral microorganisms which produce acid and also cause proteolysis.

2) proteolytic theory (bodecker 1878) :-

Main suggestion of this theory is that microorganism attack the organic part of enamel, leaving the generated acid responsible for further decalcification of inorganic part.

3-proteolysis – chelation theory:-

Schatz et al in 1955 proposed that caries occurred as a result of simultaneous degradation of organic substances (proteolysis) and dissolution of tooth minerals by a process called chelation.

Histopathology of Caries

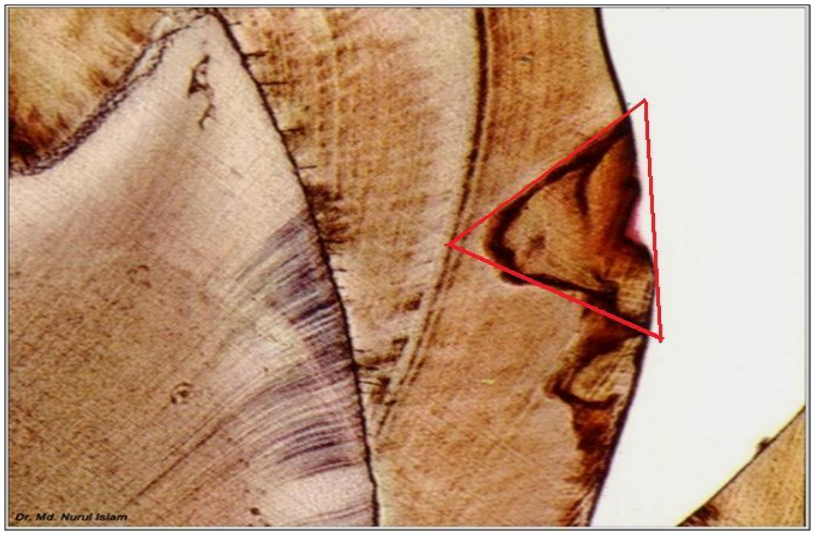
The histopathology of caries varies in both enamel and dentinal lesions. They are made up of several zones that differ in properties and appearance. Here we will discuss the zones present in these lesions.

Enamel Caries (Pre-cavitation)

- Normal enamel is composed of carious structures – enamel prisms (basic unit), prism borders, striations, incremental growth bands (Striae of Retzius) and the surface zone. The changes in these determine the lesion.
- Common sites – smooth surfaces (particular below contact points), in pits and fissures and recurrent around the edges of existing restorations.
- Lesions on smooth surfaces differ slightly from those in pits/fissures pre-cavitation.

Smooth surface Caries

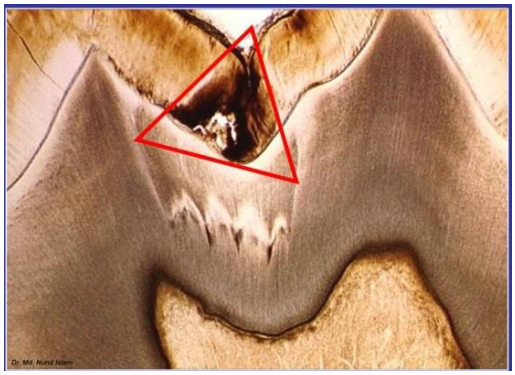
- Site – usually below contact points
- Appearance – surface typically hard and shiny. Usually a white opaque lesion, sometimes brown spot lesion
- Macroscopically, as the lesion progress through the enamel, we see the following:
    - A conical lesion with the apex towards the amelo-dentinal junction (ADJ)
    - Lateral spread at the ADJ
    - A larger dentinal lesion
- These changes are thought to be due to a combination of loss of interprismatic material, roughening of enamel rods and enhancement of Striae of Retzius.



Ground section demonstrating smooth surface enamel caries – apex extending towards the ADJ.

Pit/Fissure Caries

- Site – pits and fissures
- Appearance – commonly sticky brown/yellow lesions, may be chalky white lesions
- Macroscopically, as the lesion progress through the enamel, we see the following:
    - Lesion arising from fissure walls, not base
    - A conical lesion with the base towards the ADJ – the opposite of smooth surface, with the caries following the enamel rods
    - Lateral spread at the ADJ – more tubules involved as base is wider
    - A larger dentinal lesion
- These changes are thought to be due to a combination of loss of interprismatic material, roughening of enamel rods and enhancement of Striae of Retzius.



Fissure caries with base towards ADJ.

Microscopic Features of Enamel Caries

Within the enamel lesions described above, there are 4 zones identified pre-cavitation. These are:

1. Translucent zone – the advancing edge of the lesion
2. Dark zone
3. Body of the lesion
4. Surface zone

1) Translucent Zone

This is the advancing edge of the enamel lesion. It is only present in 50% of lesions.

- Pore volume of 1% (compared to 0.1% of normal enamel) – usually at prism boundaries and junctional sites
- Increased concentration of fluoride ions
- Preferential loss of magnesium and carbonate ions
- No protein loss

2) Dark Zone

Lies adjacent and superficial to the translucent zone. Present in up to 95% of lesions.

- Pore volume of 2-4% – smaller pores sit over the larger pores of the translucent zone. Briefly, medium can't penetrate the smaller pores and so they remain air-filled. This leads to light being scattered when trying to pass through the zone on ground section examination, giving a dark appearance.
- Re-precipitaton of minerals lost in the translucent zone
- Variable thickness – a wider dark zone indicates a slower advancing lesion

3) Body of the lesion

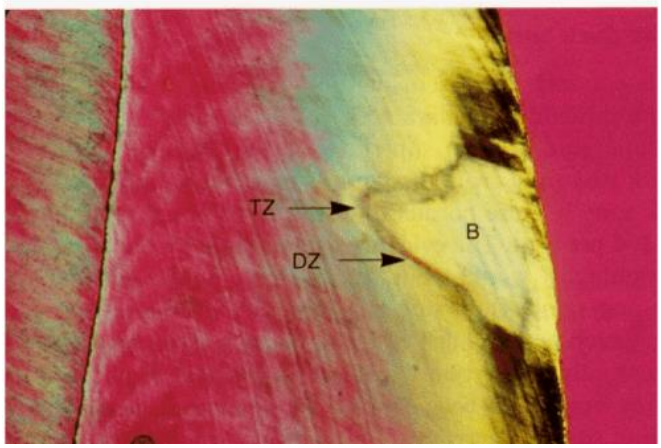
The largest part of the lesion and lies between the surface zone and dark zone.

- Area of greatest demineralization
- Pore volume of 5% (peripheries) to 25% (at center)
- Prominent Striae of Retzius
- Appears transparent on assessment

4) Surface Zone

This zone appears almost unaffected in the superficial layers

- Minimal demineralization (1-4%) until dentine is involved
- Pore volume of less than 5%
- Calcium and phosphate ions re-precipitate and remineralise from deeper zones
- Higher concentration of fluoride also favours remineralization



Demonstration of zones of enamel caries – body (B), dark zone (DZ) and translucent zone (TZ).

Dentinal Caries

Dentine can be affected both pre-cavitation and post-cavitation of the lesion. Unlike enamel, the presence of alive odontoblasts allows for reparative and protective changes to occur. **Due to lateral spread at the ADJ, a larger cavity can form very quickly (particularly larger than the apparent enamel lesion).** The general shape is a triangle with the apex towards the pulp.

Pre-cavitation

- Acids diffuse through the porous enamel and reach the dentine
- This triggers the pulp to respond to 'attack'
- 2 main zones form
    - **Sclerotic zone** – advancing edge with peri-tubular dentine walling off the lesion
    - **Body of the lesion** – present between the sclerotic zone and the ADJ

Essentially, there is minimal evidence of bacteria within the dentine at this point – only acid. Any bacteria that have managed to gain access are the pioneering bacteria. These changes are more prominent in slowly advancing lesions.

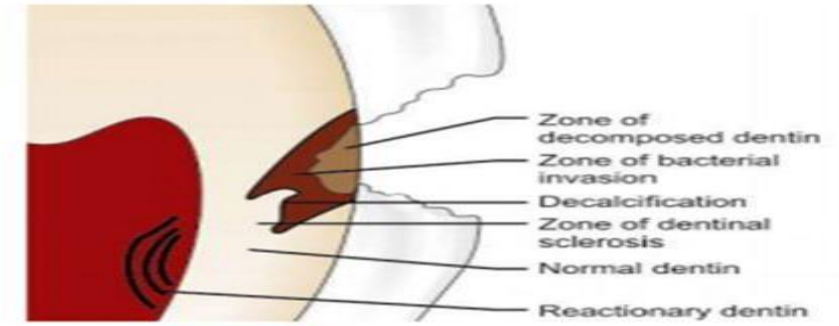


Figure 5.39: Zones of dentinal caries

Zones of cavitated lesion in to dentine

Post-cavitation

This allows bacteria to readily access the dentine via the tubules. As a result, the lesion forms new zones:

- **Sclerotic zone** – still the advancing edge with peri-tubular dentine being deposited
- **Body**, divided into 3 further zones, from innermost to outermost:
    - **Zone of demineralization/decalcification** – no/minimal bacteria are present, with acid attacks
    - **Zone of penetration** – bacteria lie within the dentinal tubules and multiply. This causes widening and softening of the tubules. Dentine typically becomes decalcified in this zone.
    - **Zone of destruction** – dentine decomposition – grossly discolored with severe breakdown of tooth structure. There is evidence of liquefaction foci (collapse of tubules due to bacteria multiplying) and transverse clefts (expansion and joining of foci to form large necrotic areas of dentine)

Throughout this process, secondary dentine is also laid down. The quicker the lesion progresses, the more irregularly this secondary dentine is. As the lesion progresses closer to the pulp, the pulp begins to get inflamed and starts to become responsive with changes such as dead tract formation and tertiary dentine deposition. Proteolytic bacteria are more common deep in the lesion, whilst acidogenic are present more superficially due to the presence of carbohydrates.