Operative Dentistry

Lecture (6) Dr. Raghad Noori

Dental caries

Dental caries: is an infectious microbial disease of the teeth that results in localized dissolution and destruction of the calcified tissues. It is essential to understand that cavitations in teeth are signs of bacterial infection (mostly *Mutans Streptococci* and *Lactobacilli*). Carious lesions only occur under a mass of bacteria (dental plaque) capable of producing a sufficiently acidic environment to dissolve tooth structure. The plaque bacteria metabolize refined carbohydrates for energy and produce organic acids as bi-products. The acids produced may then cause a carious lesion by dissolution of the tooth's crystalline structure.
Traditional caries management has consisted of the detection of caries lesions followed by immediate restoration. In other words, caries was managed primarily by restorative dentistry. However, when the dentist removes tooth structure with a hand piece, an irreversible process begins. Placing a restoration does not guarantee a sound future for the tooth; on the contrary, it may be the start of a restorative cycle in which the restoration will be replaced several times. Caries is like every other bacterial disease, and restoration without removal of the causative factors is like placing a new roof on a burning house.

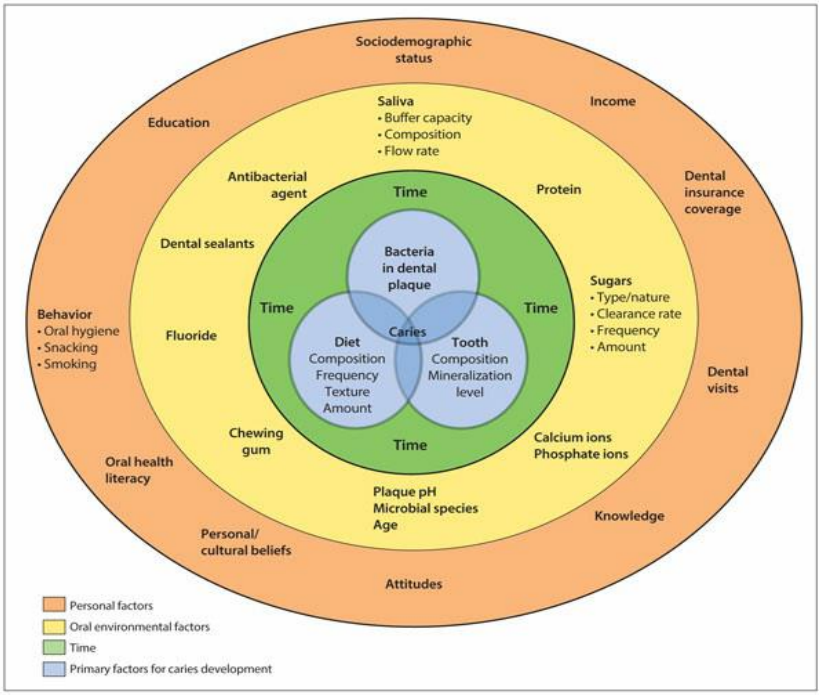
Dental caries is an ecological disease in which the diet, the host and the microbial flora interact in a way which increases demineralization of the tooth structure with resultant caries formation. The difference in occurrence of dental caries in different individuals of same age, sex, race and geographic area, diet, and similar living conditions is because of various factors that manipulate the etiology of caries.

The Caries Process:

Is a dynamic process of demineralization and remineralization of dental hard tissue which may result in progression, regression (reversal), or arrest (stabilization) of the lesion. When **demineralization** (loss of minerals "hydroxyapatites crystals") exceeds **remineralization** (gain of minerals) caries will progress, which occur in case of frequent eating (which lead to continuous reduction in pH of oral environment) and inadequate use of fluorides.

The initiation of the development of dental caries by the combined action of three primary factors (the tooth, dental plaque, and diet).

Also affected by other factors such as time, fluoride, saliva, social and demographic factors, and subject.



➢ 1-Host Factors

A-Teeth:

The teeth vary in their susceptibility to dental caries from one surface to another and from one subject to another. There are several factors affecting tooth susceptibility as:

- ***Morphology of teeth***:

Susceptible sites on the tooth, which favour plaque retention and stagnation, are prone to decay. These include:

1- Enamel pits and fissures.
2- A proximal enamel smooth surfaces.
3- Cervical margin of teeth.
4- Exposed root surfaces because of gingival recession.
5- Deficient or over hang restoration (recurrent caries).
6- Tooth surfaces adjacent to denture and bridges.

- ***Composition of teeth***:

Teeth are composed mainly of inorganic elements (96% in enamel, 70% in dentin by weight), in addition to organic elements and water. Composition of teeth is effected by environmental factors (water, diet, and nutrition).

Inorganic components: involve major elements as calcium, phosphate, hydroxyl group these are the constituents of hydroxyapatite crystals.

As the pH decreased, calcium phosphate minerals become susceptible to demineralization. As the environmental pH recovers, dissolved calcium &

phosphate can re-precipitate and the process called (remineralization). Remineralization is a slower process than demineralization.
Dentin is more vulnerable than enamel because of structural differences and greater numbers of impurities in the crystal lattice, which facilitate its degradation in an acidic environment.

B- Saliva:
Saliva can affect caries etiology and progression through the:

- rate of secretion
- composition.

Effect of saliva involve:

- ✓ Saliva affects the integrity of teeth by the composition of buffer system (calcium and phosphate).
- ✓ By the cleansing action of saliva (oral clearance), it can reduce the number of oral micro-organisms and food debris from the mouth.
- ✓ Saliva can provide antibacterial agents through *oral immune system* (specific and non-specific) that can minimize the number of cariogenic bacteria.

C-Subject:
The behavior, attitude and dental knowledge can affect the caries initiation and progression. These can influence the oral hygiene of the person as well as his dietary habits.

D- Social & demographic factors:
Many studies have shown that dental caries is more prevalent in the lower socioeconomic categories.

E- Fluoride:
It reduces tooth demineralization through the formation of fluorhydroxyapatite and fluorapatite crystals, and through accelerating and promoting reminerlization of a previously demineralized tooth structure.

➢ 2-Dental plaque:

A gelatinous mass of bacteria adhering to the tooth surface. Carious lesion only occurs under a mass of bacteria capable of producing a sufficiently acidic environment to demineralize tooth structure.
Plaque quantity and quality greatly influence caries etiology. Bacteria adhere to tooth surface and ferment carbohydrate causing release of acid that demineralizes the tooth surfaces. Cariogenic bacteria include *mutans streptococci, Lactobacilli* and others.
Mutans Streptococci ➡ early carious lesions of enamel.
Lactobacilli ➡ dentinal caries.

➢ **3- Diet:**

Dietary carbohydrates are necessary for the bacteria to produce the acids that initiate demineralization.

➢ **4-Time:**

This factor was introduced to indicate that the substrate (dietary sugars) must be present for a sufficient length of time to cause demineralization, in fact caries lesions do not develop overnight but take time; it may take years for cavitation to occur. This may provide the patient and the dentist enough time for preventative measures

CLASSIFICATION OF DENTAL CARIES

Caries lesion can be classified according to:

❖ **A. Location of caries lesion:**

The characteristics of the carious lesion vary with the nature of the surface on which the lesion develops

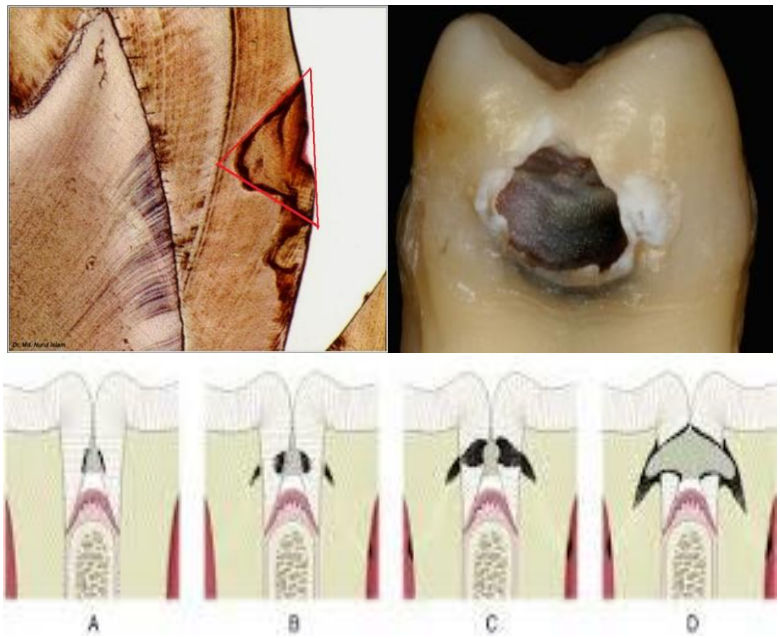
1. *Developmental pits and fissure caries:* The first and most susceptible site in the enamel. The shape of pits and fissures contributes to their high susceptibility to caries. Pit and fissure caries expand as it penetrates into the enamel affects a greater area of DEJ. Thus the entry site may appear much smaller than the actual lesion. In cross section, the gross appearance is an inverted V (or triangle) with a narrow entrance and a progressively wider area of an involvement closer to the DEJ.



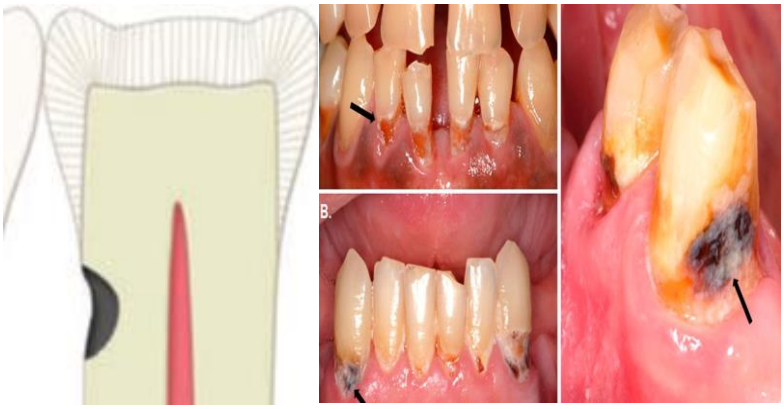


2. *Smooth enamel surfaces caries:* The second susceptible site to caries were contour or tooth position protects plaque against the rubbing action of some foods and often from being loosened by toothbrush, these include the areas of contacting proximal surfaces which are gingival to the contact area. Other

susceptible smooth enamel surfaces are those areas gingival to the height of contour of the facial and lingual surfaces.
Lesions starting on smooth enamel surfaces have a broad area of origin and a conical, or pointed, extension toward the DEJ. A cross section of the enamel portion of a smooth surface lesion show a V shape (or triangle) with a wide area of origin and the apex of the V directed toward the DEJ. After caries penetrates the DEJ, softening of the dentin spread rapidly laterally and pulpally.



3. *Root surface caries:* The third site where caries may attack. The root surface is rougher than enamel and readily allows plaque formation in the absence of good oral hygiene. The cementum covering the root surface is extremely thin and provides little resistance to caries attack. Root caries lesions have well defined margins, tend to be U-shaped in cross section, and progress more rapidly due to the lack of protection from an enamel covering; in addition to the fact cementum begins to demineralize at pH 6.7, which is higher than the enamel's critical pH of 5.5. It has been notable increase in prevalence of root caries, probably due to the increasing number of older persons who experience gingival recession and usually have cariogenic plaque on exposed root surface.



❖ **B. <u>The severity of the lesion:</u>**

1. ***Acute caries "rampant"*****:** is a rapid progressing involving a large numbers of teeth. The acute lesions are lighter colored than other lesion, being light brown or gray and their carious consistency makes the excavation difficult. Pulp exposures are often observed in patient with rampant caries.



2. ***Chronic caries*****:** these lesions are usually of long standing involvement affect of fewer numbers of the teeth and are smaller in size than acute caries.



❖ **C. <u>Whether the lesion is new or under previous restoration</u>:**

1. ***Primary caries "Initial":*** is one in which the lesion constitutes an initial attack on the tooth surface. It designated as primary because of the initial location of the lesion on the surface rather than on the extended damage.

2. ***Secondary caries "Recurrent"*****:** this type is observed around the edges of restoration. Surface overhanging margin and fracture on the surfaces in posterior teeth that are naturally prone to caries because of difficult in cleaning.

PROGRESSION OF CARIES:

The progression and morphology of caries lesion is variable depending on the site of origin and the conditions in the mouth. The time for progression from incipient caries to clinical caries (cavitation) on smooth surface is estimated to be 18 ± 6 months. Peak rate for the incidence of new lesion occurs 3 years after the eruption of the tooth.
Occlusal pit and fissure lesions develop in less time than smooth surface caries. Both poor oral hygiene and frequent exposures to sucrose containing food can produce *incipient* (white) lesions (first clinical evidence of demineralization) in as little as three weeks.
The volume and buffering capacity of saliva available to tooth surfaces has a major role in caries protection. The buffering capacity of saliva is primarily determined by the concentration of bicarbonate ion. The benefit of the buffering is to reduce the potential for acid formations.
Once the <u>pH falls below 5.5</u>, ⟹ tooth minerals begins to dissolve and the calcium and phosphate ions leaves enamel surface towards the adjacent plaque. When the <u>pH returns high (above 5.5)</u>, ⟹ remineralization of damaged tooth structure can occur before the initiation of cavitation.
Radiation induced xerostomia (dry mouth) can lead to clinical caries development in as little as three months from the onset of radiation. Thus caries development in healthy person is slower in comparison to the rate in compromised persons.

CLINICAL CHARACTERISTIC OF ENAMEL CARIES

On clean, dry teeth, the earliest evidence of caries on smooth enamel surface of crown is a white spot. These lesions are usually observed on the facial and lingual surfaces of the teeth. White spot are chalky white, opaque areas that are revealed only when the tooth is dried, and termed ***incipient caries***. These areas of enamel lose their translucency because of the extensive subsurface porosity caused by demineralization.

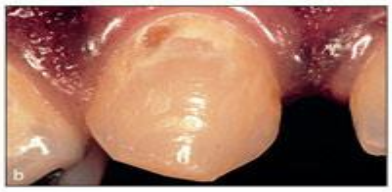


*(Care must be exercised to distinguish white spots of incipient caries from ***developmental white spot hypocalcifications*** of enamel).
Incipient caries partially or totally disappear visually when the enamel is hydrated (wet), while hypocalcified enamel is unaffected by drying and wetting. Hypocalcified enamel does not represent a clinical problem except when its appearance is objectionable esthetically).

Table 5-4	**Differences between white spots due to caries or developmental hypomineralization**	
Characteristic	**Caries**	**Hypomineralization**
Appearance and texture	Opaque, chalky, and matte (rough) surface when air dried	Smooth, glossy, and less opaque surface when air dried
	May be discolored by extrinsic stain	No extrinsic discolorat
	Feels rough when the tip of a CPITN probe or blunt explorer is moved gently across the surface	Feels smooth when the tip of a CPITN probe or blunt explorer is moved gently across the surface
Location and distribution	Located in plaque stagnation areas	Located mainly in self-cleansing areas
		Bilateral or multiple corresponding teeth; eg, canines

A more advanced lesion develops a rough surface that is softer than the unaffected, normal enamel, softened chalky enamel that can chipped away with an explorer is a sign of ***active caries.***





Incipient caries can be remineralized. Non cavitated enamel lesion retain most of the original crystal framework of the enamel rods and the etched crystallites serves as nucleating agents for remineralization.
Calcium and phosphate ions from saliva can then penetrate the enamel surface and precipitate on the highly reactive crystalline surfaces on the enamel lesion. The change in color (to brown or black spots) is due to trapped organic debris and metallic ions within the enamel. These lesions, which are termed***" Arrested caries",*** are more resistant to subsequent caries attack than the adjacent unaffected enamel. They should not be restored unless they are esthetically objectionable.



CLINICAL CHARACTERISTIC OF DENTINAL CARIES

Dentinal caries is a V- shaped in cross section with a wide base at the DEJ, and the apex directed pulpally. Caries advance more rapidly in dentin than in enamel because dentin provides much less resistance to acid attack. Caries produces a variety of responses in dentin, including pain, demineralization, and remineralization.
Early TEM and biochemical characterization of carious dentin revealed that the most superficial carious layer is necrotic, highly decalcified, and contains irregularly scattered granular crystals and irreversibly denatured collagen fibrils. Underneath this "caries infected" dentin, the deeper "caries affected" dentin layer exhibits decreased collagen crosslinks, but comprises needle-like apatite crystals, regularly attached to collagen fibrils with no signs of bacterial invasion. This deep carious layer has the potential to repair under the correct conditions as the collagen is not denatured.



Histopathology of dentinal caries

Dentin caries begins with the natural spread of the process along the DEJ and rapid involvement of the dentinal tubules. The dentinal tubules act as tracts leading to the pulp (path for micro-organisms).

In the earliest stages, when only few tubules are involved, microorganisms may be found penetrating the tubules. This initial decalcification involves the walls allowing them to distend as the tubules are packed with microorganisms. Each tubule is seen to be packed with pure forms of bacteria, eg., one tubule packed with coccal forms the other tubule with bacilli.

As the microorganisms proceed further they are distanced from the carbohydrates substrate that was needed for the initiation of the caries. Thus the high protein content of dentin must favor the growth of the microorganisms. Therefore proteolytic organisms might appear to predominate in the deeper caries of dentin while acidophilic forms are more prominent in early caries.

Advanced Dentinal Changes: decalcification of walls, confluence of the dentinal tubules, and tiny "liquefaction foci" are formed by the focal coalescing and breakdown of dentinal tubules. These are ovoid areas of destruction parallel to the course of the tubules which filled with necrotic debris and increase in size by expanding. The adjacent tubules are distorted and their course is bent due to this expansion. The destruction of dentin by decalcification and then proteolysis occurs in numerous focal areas leading to a necrotic mass of dentin of a leathery consistency. Clefts present in the carious dentin that extends at right angles to the dentinal tubules, accounts for the peeling off of dentin in layers while excavating.

