Operative Dentistry

Lecture (5)

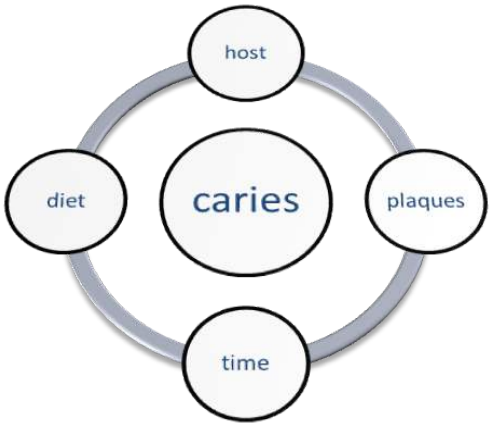
Management of dental caries in enamel and dentin (classification, diagnosis, prevention, and treatment)

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.

Dental caries is a multi factorial disease; it is the result of complex interaction between ***Host, Plaque, Diet,*** and ***Time***.

The initiation and progression of caries is affected by other factors such as saliva, fluoride, preventive measures, etc..



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- Approximal 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 of inorganic elements (96% in enamel, 70% in dentin), 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).

B-Saliva: Saliva can affect caries etiology and progression through the *rate of secretion* and *composition*.

- 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 behaviour, 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.

F-Fluoride: it reduces tooth demineralization through the formation of fluoro-hydroxyapatite 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.

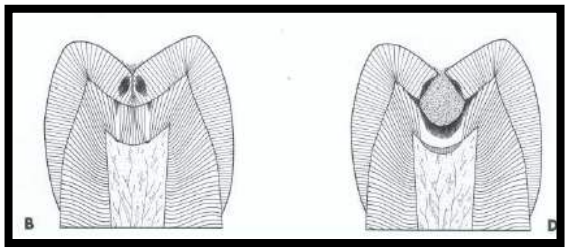
Lactobacilli dentinal caries.

3- Diet: dietary carbohydrates are necessary for the bacteria to produce the acids that initiate demineralization.

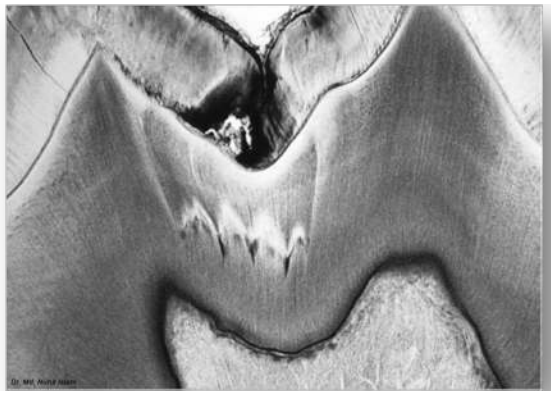
CLASSIFICATION OF DENTAL CARIES

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

1. The first and most susceptible site is in the *developmental pits and fissure* of the enamel.

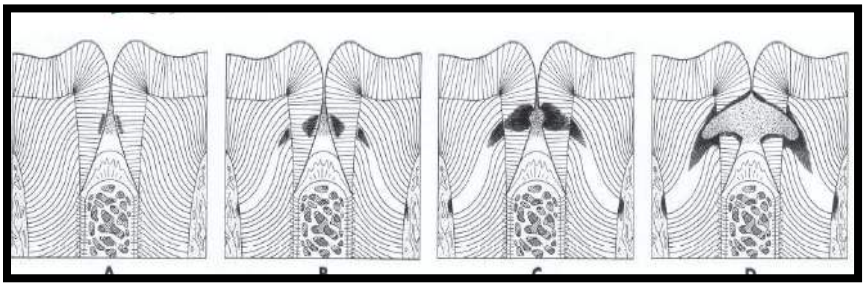


The shape of pits and fissures contributes to their high susceptibility to caries. Pit and fissure caries expand as it penetrates into the enamel affects a greater area of DEJ. Thus the entry site may appear much smaller than the actual lesion. In cross section, the gross appearance is an inverted V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



2. The second site is on certain areas of the *smooth enamel surfaces* 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. The third site where caries may attack is the *root surface*. 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.

In addition, caries could be classified according to the type and severity of the lesion into:

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.
3. *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.
4. *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

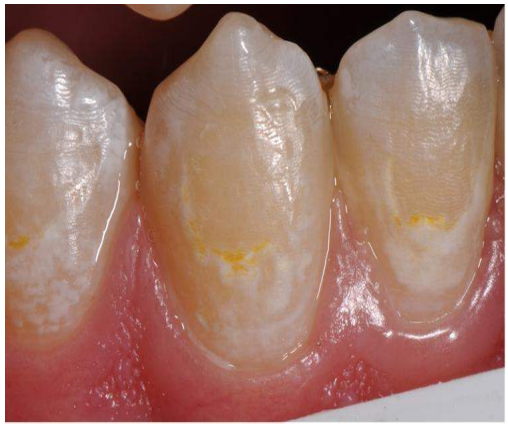
The progression and morphology of caries lesion is variable depending on the site of origin and the conditions in the mouth. The time for progression from incipient caries to clinical caries (cavitation) on smooth surface is estimated to be 18 ± 6 months. Peak rate for the incidence of new lesion occurs 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 pH falls below 5.5, tooth minerals begins to dissolve and the calcium and phosphate ions leaves enamel surface towards the adjacent plaque. When the pH returns high (above 5.5), 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

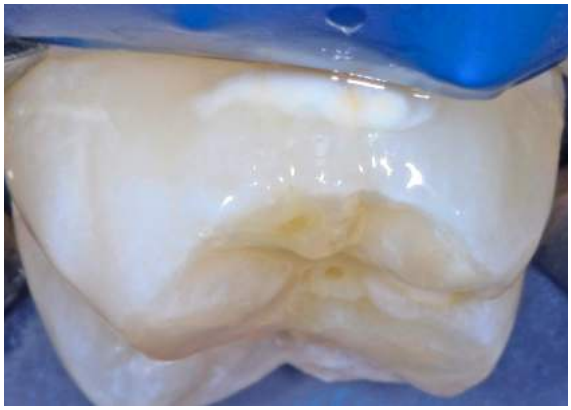
On clean, dry teeth, the earliest evidence of caries on smooth enamel surface of crown is a white spot. These lesions are usually observed on the facial and lingual surfaces of the teeth. White spot are chalky white, opaque areas that are revealed only when the tooth is dried, and termed ***incipient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

A more advanced lesion develops a rough surface that is softer than the unaffected, normal enamel, softened chalky enamel that can chipped a way 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.



Often, pain is not reported even when caries invades dentin, except when deep lesions bring the bacterial infection close to the pulp. Once bacterial invasion of the dentin is close to the pulp, toxins and even a few bacteria inter the pulp, resulting in inflammation of the pulpal tissues.

Initial pulpal inflammation is thought to be evident clinically by production of sharp pains for only a few seconds (10 or less) in response to a thermal stimulus. A short, painful response to cold suggests ***reversible pulpits*** or ***pulpal hyperemia***. Reversible pulpitis is a limited inflammation of the pulp from which the tooth can recover if the caries producing the irritation is eliminated timely by operative treatment.

When the pulp becomes more severely infected, a thermal stimulus will produce pain that continues after termination of the stimulus, typically longer than 10 seconds (partial or total pulp necrosis). This clinical pattern suggests ***irreversible pulpitis*** which need endodontic treatment.