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**Preventive dentistry**

**Dental caries development**

Dental caries: is a chronic disease involving a large number of populations. The term dental caries (tooth decay) is used to describe the results – the signs and symptoms – of a localized chemical dissolution of the tooth surface caused by metabolic events taking place in the biofilm (dental plaque) covering the affected area. It is a disease of dental calcified tissue , a multi-factorial disease characterized by “demineralization of the mineral components and dissolution of the organic matrix”.

The caries process affects the mineralized tissues of the teeth, enamel dentine and cementum and caused by the action of micro-organisms on fermentable carbohydrates in the diet.

The disease is often described as progressive disease, if it is not treated may expand in size and progress to the pulp leading to pulp inflammation thus pain, discomfort, and the end result will be loss of vitality then loss of tooth

Carious process is complicated process, there should be interaction of several etiological and predisposing factors for caries t occur. ***This may be explained the variation in the caries susceptibility of individuals to caries process***

Dental caries process is the result of an interaction of the following:

1. **Host**
2. **Plaque.**
3. **Diet**

***Host factor***

* **susceptible tooth**
* **saliva**
* **The subject his or her self.**

**Tooth Morphology:** such as presence of deep pits and fissures with a sharp cusp,

**1- Tooth Morphology:** Dental caries lesions may develop at any tooth site in the oral cavity where a biofilm develops and remains for a period of time. Such sites include pits, grooves and fissures in occlusal surfaces, especially during eruption, approximal surfaces cervical to the contact point/area and along the gingival margin. Insertion of foreign bodies to the dentition (e.g. fillings with inappropriate margins, dentures, orthodontic bands) may also result in such ‘protected’ sites.

Morphology of tooth are mostly occurred due to hereditary factors while in the same time there is some environmental factors which affected the type of pits and fissure like the presence of some trace elements in the environment especially strontium which make effect on tooth morphology and make the cusps more rounded and shallower pits and fissures, these areas are relatively protected from mechanical influence from the tongue, the cheeks, abrasive foods and, not least, tooth brushing. These are the sites where lesion development is more likely to occur because the biofilm is allowed to stagnate there for prolonged time.

Sites on the tooth, which favor plaque retention and stagnation, are prone to decay: these are:

1. Enamel Pits and fissure
2. Proximal enamel surfaces.
3. Cervical margin of teeth.
4. Exposed root surface because of gingival recession
5. Deficient or over hang restoration (recurrent caries).
6. Tooth surfaces adjacent to denture and bridges

**Tooth Position**:

*Posterior* teeth are more susceptible than *anterior* teeth because of the heavy contact, presence of stagnation areas, this point is definitely hereditary. But early loss of deciduous teeth leading to space loss, mal position of teeth, leading to crowding which increase susceptibility to dental caries

***Composition of the tooth***

The tooth is composed mainly of inorganic materials, 96%in enamel and 70% in dentin and remaining organic materials and water. Composition of teeth is affected by environmental factors (water, diet and nutrition).

**The inorganic components are divided into**:

***Major elements***

Calcium phosphorous, hydroxyl group, and bicarbonate group, (hydroxy apetite crystal. {Ca10(PO4)6(OH)2}. the Ca\P ratio is 2.15. any changes in this ratio is an indication of presence of other types of crystals.

***Minor elements****:*

There are elements in the teeth are Zinc, copper, strontium, magnesium and fluoride, - -etc.

These elements may incorporate the enamel crystal in substitutions with one of its major elements as for example substitution of Ca ions by Mg, (Ca9Mg (PO4)6F2, or substitution of hydroxyl group by fluoride ion and formation hydroxy appetite crystals Ca10 (PO4)6F2. Certain elements(zinc, fluoride, iron, chloride)accumulate in the enamel surface, while others are sparse in surface as compared with subsurface enamel. Changes of the enamel (decrease in density and permeability, an increase in fluoride content) occur with age.

Some of these elements may incorporate either in pre-eruptive stage including all layers of enamel and dentin or in post eruptive stage involving the outer enamel surface only.

Some of these elements may increase the resistance of teeth to dental caries as fluoride ions, tin, zinc, strontium and molybdenum, while others increase susceptibility to dental caries as magnesium, however the role of other elements may not well substantiate as K, Mn and Al

The organic constituents and water of both enamel and dentin may act as a diffusion pathway for bacterial acids increasing the tooth destruction. In other way, they permit the penetration of ions for physiological remineralization-demineralization process. Such voids in enamel as well as proteins act as a caution for intense biting pressure to prevent fracture.

***Saliva:***

Saliva affects the caries etiology through the rate of secretion and composition.

Saliva affects the integrity of teeth by the composition of (buffer system, calcium and phosphorous content). By cleansing action of saliva (oral clearance), it can affect he number of microorganisms and food debris from the mouth, the oral immune system (specific and non-specific).

***Subject:***

The behavior, attitude and dental knowledge affect caries etiology. These can influence the oral hygiene of the person as well as his dietary habits.

***Dental Plaque:***

Plaque quantity and quality greatly influence caries etiology. Bacteria adhere to tooth surface and ferment carbohydrate causing release acid thus demineralization of tooth surface. Cariogenic bacteria involve mutans streptococci, lactobacilli and others

***Diet***

Sweet consumption especially between meals may lead to continuous drop of Ph and not allowing the enough time for the PH to return to normal, thus demineralization of teeth.

**Terminology of caries:**

* **Primary Caries:** lesions on natural un restored, intact tooth surface.
* **Pits and fissure caries:** are a lesion affected tooth occlusally.
* **Recurrent or secondary caries:** is a lesion developing at tooth surface adjacent to margins of an old restored area.
* **Arrested (inactive) carious lesion:** A lesion that may have formed years previously and then stopped further progression.
* ***Rampant caries:*** Is the name given to sudden rapid destruction of many teeth frequently involving surface of teeth that are usually caries free. It may be seen in the permanent teeth of teen agers and is usually due to taking frequent cariogenic snacks and sweet drink between meals, it's also seen in mouths where there is a sudden marked reduction in salivary flow ( xerostomia).
* ***Nursing caries:*** Is a particular from of rampant caries in the primary dentition of infants and young children, it is found in an infant or toddler who falls asleep sucking a bottle (called nursing bottle). Which has changed from conditions predisposing to caries to conditions that tend to arrest the lesion.

**Dynamics process of de-\re-mineralization**

**Dynamics Process of De-/Remineralization**

Dental caries is a disease that is manifested as a dynamic process of

de/remineralization in the mouth (Enamel sieve concept). Demineralization is a continual imbalance between pathological and protective factors that results in the dissolution of apatite crystals and the net loss of calcium, phosphate, and other ions from the tooth. The first stage of demineralization is occurring at the atomic level far before it can be seen visually as gross demineralization. During this step, fermentable carbohydrates are metabolized by bacteria in dental plaque to produce organic acids. The acids diffuse into the dental hard tissue through the water among the crystals and could reach a susceptible site on a crystal surface. Calcium and phosphate are dissolved into the surrounding aqueous phase between the

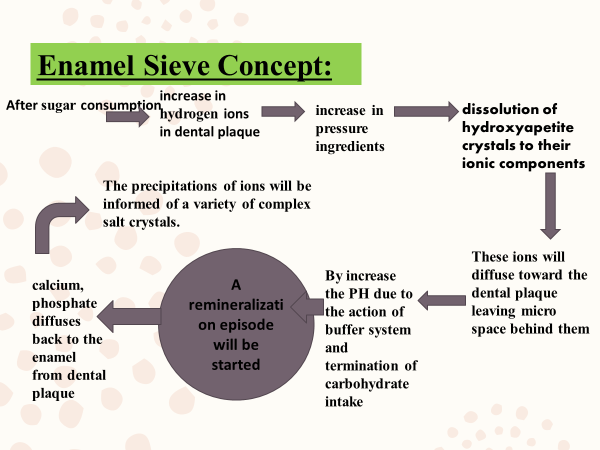
crystals. This is considered as the first step in the continuum of the dental caries process which can eventually lead to cavitation.

The oral fluids (saliva, biofilm fluid) have calcium (Ca) and phosphate (P) insupersaturated concentrations with respect to the mineral composition of enamel.

At physiological conditions (a neutral pH of 7), low ion concentrations are sufficient to keep dental hard tissues in equilibrium. If the pH drops because ofacid produced by the dental plaque, higher ion concentrations are needed toprevent dissolution of dental hard tissue. Calcium (Ca) and phosphate (P) ions are continually deposited on the enamel surface or are redeposit in enamel areas where

they were lost. At a pH of 5.5, under saturation begins, that is, the calcium and phosphate ion concentrations in the plaque fluid are not sufficient to maintain the enamel in stable equilibrium; thus, the enamel starts to dissolve. The term “remineralization” is used to described mineral gain. Remineralization isthe body’s natural repair process for subsurface non-cavitatedcarious lesions. In the process of remineralization, calcium and phosphate ions are supplied from a source external to the tooth to promote ion deposition into crystal voids in demineralized enamel to produce net mineral gain.

De-/remineralization cycles continue in the mouth as long as there are factors including cariogenic bacteria, fermentable carbohydrates, and saliva present. The balance between pathological factors and protective factors determines whether demineralization or remineralization is proceeding at any one time.



**The development of carious lesion ocuurs in three stages**:

-The earliest stage is the incipient lesion; macroscopically evidenced on the tooth surface by the appearance of an area of opacity (the white spot lesion), which is accompanied by histologic changes of the enamel at the microscopic level and is well established with a number of recognizable zones.

- The second stage includes the progress of the demineralization front toward the dentino- enamel junction and/or into the dentin; the affected dentin displays discoloration from brown to dark brown or black, microscopic changes of dentil showed different zones.

*-* The final phase ofcaries development is the development of the overt, or frank lesion, which is characterized by actual cavitation.

**Root caries**

Root caries differs from coronal caries (enamel and dentin) in several aspects (mineralization and bacterial invasion).

Clinical appearance: Root-surface caries comprises a continuum of clinical manifestations ranging from small, slightly softened and discolored areas to extensive, yellow-brown soft or hard areas, which may eventually encircle the entire root surface. The lesions may or may not be cavitated. However, even in the case of rather extensive lesions, cavitation does not necessarily involve the pulp, loot-surface caries lesions may be classified as:

I - An active root-surface lesion is a well-defined, softened area on the root surface that shows a yellowish or light- brown discoloration. The lesion is likely to be covered by visible plaque. Some slowly progressing lesions may be brownish or black and reveal a leathery consistency on probing with moderate pressure.

I - An arrested (inactive) root-surface lesion appears shiny and is relatively smooth and hard on probing with moderate pressure. The color may vary from yellowish to brownish or black. In both active and inactive lesions, cavity formation may be observed, but in the latter case the margins appear smooth.