Operative

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

Dental caries is an infectious microbiological 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 stryptococci and Lacto bacilli). 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 a bi-product. The acids produced may then cause a carious lesion by dissolution of the tooth's crystalline structure.
So there should be four etiological factor for dental caries initiation which are (1) host (tooth), (2) diet (such as sucrose and carbohydrate), (3) bacterial plaque, and (4) time.

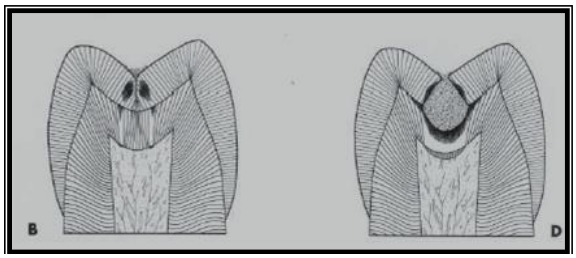
Classification of dental caries

A) *<u>According to the clinical sites for caries initiation</u>*

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.

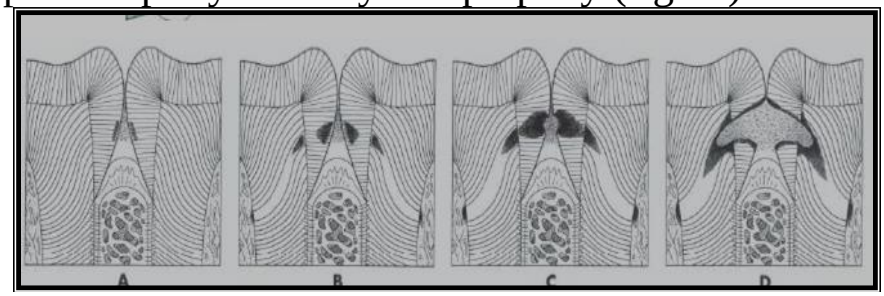
The shape of pits and fissures contributes to there high susceptibility to caries. Pit and fissure caries expand as it penetrates into the enamel affects a greater area of DEJ. Thus the entry site may appear much smaller than the actual lesion. In cross section, the gross appearance is an inverted V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



(Fig. 1): pit and fissure caries

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 e enamel surfaces are those areas gingival to the height of contour of the facial and lingual surfaces.

Lesions starting on smooth enamel surface s 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 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 (fig. 2).



(fig.2): proximal smooth surface caries

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. 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 (mostly A. viscosus) on exposed root surface.

Also we have another classification according to the location; Cl I, Cl II, Cl III. Cl IV, Cl V, and Cl VI.

B) *According to the severity of caries*

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 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 over hanging 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 month, plus or minus 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 mineral is dissolved and the calcium and phosphate ions are loses into the plaque. When the pH is high (above 5.5) the remineralization of damaged tooth structure will occur before the occurrence of cavitation (incipient lesion).

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 slow in comparison to the rate of possible 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 are termed ***incipient caries***. These areas of enamel loss 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 remineralize, non activated 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 (brown or black spots) is due to tapped organic debris and metallic ions within the enamel. ***Arrested caries*** are more resistance 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 through 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, 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 pulp extirpation and root canal filling.

Caries diagnosis

Caries diagnosis and treatment has traditionally been limited to the detection and restoration of cavitated lesions. This "drill and fill" approach is simply symptomatic treatment and fails to deal with the underlying etiological factors. Undoubtedly, unaffected teeth are superior to restored teeth. Therefore, early detection of incipient caries and limitation of caries activity prior to significant tooth destruction are primary goals of an effective diagnosis and treatment program.

Varieties of diagnostic methods are available such as inspection, radiographic, and dye uptake. However, a single test for caries diagnosis usually cannot be used alone because such test may not be sufficient for accurate caries diagnosis. Therefore, multiple diagnostic criteria should be adjusted according to the patient's overall risks (age, gender, fluoride exposure history, general health, and ability to maintain good oral hygiene).

Chemical dye also used (such as acidic red dye) that selectively stain the outer layer infective dentin (demineralized and infected with bacteria) that should be removed, while the inner affected dentine (demineralized but not infected) will not stain with red dye, the affected dentine should not be removed decause it may remineralized.

Pits and fissures

Cavitation at the base of pit and fissure sometimes can be detected tactilely as softness or by binding of the explorer tip. However, mechanical binding of an explorer tip may be due to non-carious cause such as the shape of fissure, sharpness of the explorer, and the force of application. Thus, other factors should be observe such as softening at the base of the pit or fissure, opacity surrounding the pit or fissure, and softened enamel that may be flaked away by the explorer.

Smooth surfaces

Bitewing radiographs are the most effective method for evaluation of the proximal smooth surfaces for evidence of

demineralization because these areas are not readily assessed visually or tactilely. Proximal radiolucencies detectable on bitwing radiographs should be examined clinically because not all proximal radiolucencies are associated with cavitation of the surface and therefore may not need restorative treatment since most incipient proximal lesions in healthy patients end up as arrested lesions.
It is equally important to detect smooth surface lesions on facial or lingual surfaces as soon as possible, because lesions on these surfaces are almost always seen in individuals with high caries activity. The diagnosis is confirmed when the affected area is rehydrated (wetted) and the chalky area (white spots) partially or totally disappears. These incipient lesions have intact surfaces and care should be given to avoid damaging the surface with an explorer.

Root surfaces

Root surfaces exposed to the oral environment, usually due to gingival recession, are at risk for caries and should be examined visually and tactilely. Discoloration of such areas is common and usually is associated with remineralization. Generally the darker the discoloration, the greater the remineralization. On the other hand, active, progressing caries shows little discoloration and is primarily detected by the presence of softness and cavitation.

Caries prevention and treatment

Caries preventive treatment is a complex process involving multiple interrelated factors. Maintaining of good oral hygiene "brushing and flossing", diet containing sucrose and carbohydrate control, and fluoride treatment; all these factor can affect carious lesion initiation and also remineralization the incipient carious lesion specially in smooth surfaces to arrested carious lesions. Pit and fissure sealant is the most effective method in preventing pit and fissure caries.

Once caries has produced cavitation of the tooth surface, preventive measures are usually inadequate to prevent further progression of caries. So, cavity preparation and restoration are

needed. Once the pulp is dead partially or completely; root canal filling become necessary to avoid tooth extraction.