ORAL PATHOLOGY

Dental Caries

**Lecture 1**

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# Definition

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***Dental caries*** defined as a bacterial disease of the calcified tissues of the teeth characterized by demineralization of the inorganic and destruction of the organic substance of the tooth.

Dental caries is a complex and dynamic process involving :

∗ Physicochemical processes associated with the movements of ions across the interface between the tooth and the external environment.

 ∗ Biological processes associated with the interaction of bacteria in dental plaque with host defense mechanisms.

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.

Loss of tooth substance may result from the action of oral microorganisms as in dental caries or be due to non-bacterial causes that include mechanical factors associated with attrition and abrasion, chemical erosion, and pathological resorption.

# Etiology of Dental Caries

Various theories for the aetiology of dental caries have been proposed, but there is now overwhelming support for the **acidogenic theory**.

This theory, which has remained unchanged since first postulated by Miller in 1890, proposes that acid formed from the fermentation of dietary carbohydrates by oral bacteria leads to a progressive decalcification of the tooth substance with a subsequent disintegration of the organic matrix.

Essential requirement for development of dental caries:

1. Cariogenic (acidogenic) bacteria.
2. Bacterial plaque.
3. Susceptible tooth surfaces and stagnation areas.
4. Fermentable bacterial substrate (sugar).
5. Time for process to develop.

Factors influencing site attack and rates of progression in dental caries are:

# Factors intrinsic to the tooth

🗷 Enamel composition - There is an inverse relationship between enamel solubility and enamel fluoride concentration.

🗷 Enamel structure - Developmental enamel hypoplasia and hypomineralization may affect the rate of progression but not the initiation of caries.

🗷 Tooth morphology - Deep, narrow pits and fissures favour the retention of plaque and food.

🗷 Tooth position - Malaligned teeth may predispose to the retention of plaque and food.

# Factors extrinsic to the tooth

 **Saliva -** Flow rate, viscosity, buffering capacity, availability of calcium and phosphate ions for mineralization, and the presence of antimicrobial agents such as immunoglobulins may affect caries pattern.

 **Diet -** The most important factor is the frequency of intake of sugary foods and drinks.

🗷 **Use of fluoride -** Fluoride readily enters bacterial cells and can inhibit enzymes involved in the metabolism of sugar.

🗷 **Immunity–** IgA is secreted in saliva but small amount of IgG enter the mouth from the gingival crevice.

# CLINICAL CLASSIFICATION OF DENTAL CARIES

Clinically, dental caries may be classified according to the location of the lesion on the tooth and to the rate of attack,

according to whether lesion is new (primary) or under previous restoration (secondary).

## Classification by site of attack

Pit and fissure caries:

It is the most common type and appears at an early age on the occlusal and buccal surfaces of the molars of the primary and secondary dentition.

This form of caries is the most destructive, because it quickly goes deeply into the dentin, remains hidden as it undermines the enamel, and becomes clinically evident as pain caused by pulpal involvement or as a large cavity when a substantial portion of the tooth crumbles.

The characteristic shape of lesions demonstrating a small triangle shaped lesion in the fissure of the occlusal enamel (gray-black) that appears narrow at the surface but wider at the DE junction to provide an even greater involvement of dentin (dark brown). The pulp of the tooth reacts with the deposition of reparative dentin.

**Smooth-surface caries:**

It is occurs on the interproximal (contact) areas of the teeth that are not self-cleansing and on the gingival third of the buccal and lingual surfaces.

A proximal caries begins just below the contact point as well-demarcated chalky-white opacity of the enamel.

The characteristic triangular shape of these lesions that occur corresponds to the orientation of the enamel rods and the dentinal tubules.

**Cemental or root caries:**

This occurs when the root face is exposed to the oral environment as a result of periodontal disease followed by bacterial colonization. Root caries is clinically diagnosed by a softening and brownish discoloration of the tissues.

The cavities, which may be extensive, are usually shallow, saucer shaped, with ill-defined boundaries. Sclerosis may lead to arrested lesions and the surface of the exposed dentine may be covered by a hypermineralized layer.

**Recurrent caries:**

This secondary caries occurs around the margin or at the base of a previously existing restoration.

## Classification by rate of attack

**Rampant or acute caries:**

This is rapidly progressing caries involving many or all of the erupted teeth. The rapid coronal destruction leads to early involvement of the pulp.

Young patients are most susceptible to acute or rampant caries, because they have teeth with large pulpal chambers, wide and short dentinal tubules containing little or no sclerosis. In these patients is often combined with a diet high in refined carbohydrates and less than adequate oral hygiene.

When caries is present on the labial surfaces of the primary teeth of infants, it is nearly always caused by a habit of leaving the feeding bottle containing milk or juice in the infant’s mouth when sleeping.

**Slowly progressive or chronic caries:**

This is caries that progresses slowly and involves the pulp later. It is most common in adults and the slow progress allows time for defence reactions of the pulpodentinal complex to develop in which teeth have smaller pulpal chambers with additional deposits of a denser and less tubular dentin on the pulpal walls (sclerosis and reactionary dentine formation).

**Arrested caries:**This is caries of enamel or dentine, including root caries that becomes static and shows no tendency for further progression.

**Enamel:** Arrest of a **proximal smooth** surface lesion prior to cavity formation can occur when the adjacent tooth is lost so that the lesion becomes accessible to plaque control. Remineralization may then occur from saliva.

Arrest of **coronal dentinal** caries may occur in lesions characterized by early dentinal sclerosis which limits the rate of inward spread of the caries. Such dentine is deeply pigmented brown-black in colour. Its surface is hypermineralized due to remineralization from oral fluids.

Arrested lesions of **root caries** have a similar clinical appearance and develop in a similar manner following loss of the superficially softened cementum.

**Enamel caries**

Smooth-surface enamel caries is most commonly located on the mesial and distal surfaces at the point of contact with the adjacent tooth (interproximal caries). The less common lesions on the buccal and lingual surfaces have a similar microscopic appearance.

Early caries may be detected clinically by brown or black discoloration of a fissure in which a probe 'sticks'. The enamel directly bordering the pit or fissure may appear opaque, bluish-white as it becomes undermined by caries.

Because the enamel is composed primarily of inorganic salts, the process results in the production of a cavity because of demineralization.

**HISTOPATHOLOGY**: The early lesion (white spot lesion) in smooth surface enamel caries is cone-shaped, with the base of the cone on the enamel surface and the apex pointing towards the dentinoenamel junction.

When a thin ground section of an early lesion (before cavitation) is viewed with light microscopy, four zones can be identified:

* 1. Translucent zone, the advancing front of initial demineralization; It is more porous than normal enamel.

Dissolution of mineral occurs from the junctional areas between the prismatic & interprismatic enamel and a magnesium & carbonate rich mineral is dissolved in this zone.

* 1. Dark zone, where the previously liberated salts are redeposited; some remineralization has occurred due to reprecipitation of mineral lost from the translucent zone. The dark zone becomes more porous.
	2. Body of lesion, containing the region of maximal

demineralization and contains apatite crystals result from the reprecipitation of mineral dissolved from deeper zones.

* 1. Surface zone, remaining relatively unaffected until it is sufficiently undermined to collapse, resulting in cavitation and being more highly mineralized.



Diagrammatic representation of the development of fissure caries.

In their early stages, pit and fissure enamel caries have histologic zones similar to smooth surface carious lesions.

The shape of the lesion differs because of the different angulations of the enamel rods; those lesions will be wider in the deeper portion and will be a wider area of involvement of the dentinal tubules at the DE junction than at the surface.

The enamel is much thinner at the base of the pit or fissure, results in the lesions progressing at a much faster rate than the smooth-surface type.

# Dentine caries

Dentine differs from enamel in that it is a living tissue and as such can respond to caries attack.

In dentine caries it is necessary to consider both:

* + 1. The defense reaction of the pulpodentinal complex and
		2. The carious destruction of the tissue which involves acid demineralization followed by proteolytic breakdown of the matrix.

Caries of the dentine develops from enamel caries: when the lesion reaches the DE junction, lateral extension results in the involvement of great numbers of tubules.

The early lesion is cone-shaped with the base at the DE junction.

Larger lesions may show a broadening of the apex of the cone as it approaches the circumpulpal dentine.

Dentin caries progresses at a much faster rate than enamel caries. It is more porous, because it contains dentinal tubules and is less densely mineralized.

In the teeth of younger patients the dentinal tubules are less densely mineralized, shorter in length, and wider in diameter, allowing for ease of penetration and progression of the invading microorganisms.

In older patients deposits of calcifying salts usually narrow the dentinal tubules making the teeth less porous.

In acute, rapidly progressing caries the necrotic dentine is very soft and yellowish white; in chronic caries it has a brownish-black colour and is of leathery consistency.

In decalcified sections show a zoned lesion from pulpal side outward into:

The deepest zone **Zone 1** fatty degeneration reflects the earliest changes of caries infection where the bacteria in the dentinal tubules, causing a breakdown of the organic component of the dentin- liberating lipid.

**Zone 2**, translucent zone, is a band of

hypermineralized dentin in which the dentinal tubules are sclerotic. This zone is regarded as a vital reaction of odontoblasts to irritation.

Two patterns of mineralization have been described:

* The first is the result of acceleration of the normal physiological process of peritubular dentine which eventually occludes the tubules.
* In the second, mineral first appears within the cytoplasmic process of the odontoblasts and the tubule is obliterated by calcification of the

odontoblast process itself.

**Zone 3,** demineralization, is composed of dentin that is softer than normal because of the initial action of the bacterial enzymes.

**Zone 4,** Zone of bacterial invasion; brown discoloration, is due to a reduction in the mineral content and the presence of distended dentinal tubules packed with bacteria. This zone is usually soft enough to be removed with a hand instrument.

**Zone 5,** cavitation is present no mineralization and the organic component is partially dissolved by the bacteria. This zone is the clinically observed base of a large cavity. It has a brown coloration and easily peels away in layers along the incremental lines of growth.

## Defence reaction of pulpodentinal complex:

The defense reaction may begin before the carious process reaches the dentine, because of irritation to the odontoblasts transmitted through the enamel, however, in progressive lesion the defense reaction is over taken by the carious process as it advances towards the pulp.

In addition, the dentin will be thicker because of the production of additional normal and abnormal secondary dentin on the pulpal walls. This defensive mechanism results in:

1. Sclerosis (Development of dentinal sclerosis).
2. Reactionary dentine formation (Sealing of dead tracts).
3. Secondary dentin (tubular dentin separated from primary dentin by hyperchromatic zone).

***Reactionary (or tertiary) dentine***

A layer of reactionary (or tertiary) dentine is often formed at the surface of the pulp chamber deep to the dentine caries, this dentine being localized to the irritated odontoblasts.

It varies in structure but the tubules are generally irregular and fewer in number than in primary dentine, or may even be absent. Its formation effectively increases the depth of tissue between the carious dentine and the pulp, and in this way delays involvement of the pulp.