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Non-Cavitated Lesions: A Brief

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Abstract

Recent paradigm shift has shifted the focus from G.V.Black's "extension for prevention" to a minimal intervention strategy. Accurate and reliable diagnosis of white spot lesions is crucial for diagnosing enamel demineralization at its early stages. New diagnostic devices can help dentists detect and treat lesions early, promoting remineralization and preserving tooth structure. Preventive treatments are more cost-effective than surgical or restorative procedures to address high caries rates. Modern dentistry aims to manage white lesions non-invasively using remineralization to avoid disease development and improve tooth strength, aesthetics, and function. The present literature review was envisaged with a view for early diagnosis and management of non-cavitated carious lesions.

Keywords: non-cavitated carious lesions, white spot lesions, dental caries, demineralization, remineralization

Introduction

Dental caries is a localized chemical dissolution of the tooth surface caused by metabolic events taking place in the biofilm covering the affected enamel, dentin and

cementum. It begins when bacteria in acidogenic dental plaque—mainly Streptococcus mutans, Streptococcus sobrinus and Lactobacillus acidophilus—ferment carbohydrate in the diet producing organic acids that act on hydroxyapatite crystals, freeing the calcium and phosphate mineral content and thereby, initiating the process of cavity formation. These lesions are often capable of being reversed, arrested or progressing to cavitation. Nevertheless, they are active lesions that are confined to the enamel and are sometimes referred to as smooth surface caries or non-cavitated lesions; more commonly referred as white spot lesions.[1]

A constant cycle of demineralization followed by remineralization takes place on the tooth surface because of continual fluctuations in the intraoral pH levels. However, the tooth surface is protected to a degree by the oral biofilm, the combination of pellicle and plaque, which closely adheres to the tooth surface, because this helps to prevent total loss of calcium and phosphate ions from the immediate environment. Even though ions are released from the enamel in the presence of a lowered pH, many may remain trapped within the biofilm and are available to return into the tooth surface as the pH level rises again.[2,3]

The bacteria that invade the incipient lesion reach the deepest layers or enamel without cavities, all the way to the amelo-dentinal limit and may hide the lesion that penetrate dentin. Cavities that appear clinically healthy and apparently intact may hide lesions; these changes have important implications for diagnosing and managing incipient lesions. Thus, early detection and determination of depth of the lesion should be the prime consideration because they can lead to shift from surgical intervention to preventive treatment. For diagnosis of early carious lesions without cavities there should be an ideal diagnostic method that should offer a high level of sensitivity and specificity to prevent false negative and false positive findings.[4,5]

The present literature review was envisaged with a view for early diagnosis and management of non-cavitated carious lesions.

The Demineralization - Remineralization Cycle:

Bio-mineralization is a dynamic, complex, lifelong process by which living organisms control precipitations of inorganic nano-crystals within organic matrices to form unique hybrid biological tissues, for example, enamel, dentin, cementum. Enamel, dentin, cementum, and bone are natural composites of both organic and inorganic components. Bone, cementum, and dentin are specialized connective tissues, while enamel has an ecto-dermal origin. For the specialized connective tissues (bone, cementum, and dentin), collagen type I constitutes ~90% of their organic component; noncollagenous proteins form the remaining. On the other hand, enamel has little or no collagen, and its organic matrix is made up of noncollagenous protein, which is 90% amelogenin. Modifying factors can result in variations in the biochemical characteristics of an intrinsic or extrinsic factor, behavioral patterns such as regular tooth brushing, some of which can also be classed as socio-economic factors.[6,7]

The inorganic component of these hard tissues consists of biological apatite, $Ca_{10}(PO_4)_6$ $(OH)_2$. Enamel has more inorganic content (~90% prismatic crystals) than dentin and bone (~70%) and cementum (45%). The unit cell of biological apatite is hexagonal in shape; repetitions of the unit cells produce crystals of various sizes. In dentin, the crystals are plate like of 50 nm length, 20 nm width, and 2–5 nm thickness. However, they are bigger and highly oriented in enamel than in

bone and dentin, making it the hardest tissue in the body. The carbonate content of bone and teeth HA is 4%–8%; with age, the carbonate content increases but the hydrogen phosphate decreases. The ratio of inorganic-to-organic contents varies according to the tissue; such variation reflects the properties of each tissue. With high organic content, however, dentin is more resilient than enamel and therefore provides a resilient layer under enamel and cementum. Enamel lacks regenerative capacity while dentin regenerates by formation of secondary and tertiary dentin. Mineralization is a process, in which an inorganic substance precipitates onto an organic matrix and collagen fibrils form a scaffold for a highly organized arrangement of uniaxially organized calcium phosphate crystals.[8]

In enamel, immediately after initial dentin mineralization at the DEJ, ameloblast cells secrete enamel matrix proteins (eg, amelogenin, ameloblastin, and enamelin) and proteinases (matrix metalloproteinase-20 and kallikrein-related peptidase-4) at the dentin surface. These proteins and proteinases are responsible for immediate mineralization of ~30% of enamel. The first formed enamel crystals (ribbons) grow between the existing dentin crystals by mineralizing around dentin proteins. These crystal ribbons further elongate at the mineralization front where enamel proteins are secreted. During their movement from the dentin surface, the ameloblast start secreting large amounts of enamel matrix proteins. When the entire thickness of enamel is formed, the ameloblasts become protein-resorbing cells; and therefore, additional mineral is required to coincide with the bulk removal of enamel proteins and water to produce enamel with 95% mineral content. The formed crystals are long, thin, and parallel ribbons of 26×368 nm^2 ; ~10,000–40,000 ribbons at packing density of 550 crystallites/µm2 form a rod (prism) with ~5 µm diameter.[9] Each ameloblast produces one rod; all rods are organized in a three-dimensional structure. The mineral crystals formed within the enamel rods grow in c-axis length parallel to each other from DEJ to the tooth surface, while those developed between the rods have limited lengths and always ordered at angles relative to the rod crystals. Since this process of enamel formation and maturation is a cell-mediated process, completion of mineralization is associated with several morphological changes in ameloblasts; hence, the matrix removal and crystal growth occur efficiently. For calcification, the influx of calcium from the blood to the enamel matrix involves intercellular and transcellular routes. Unlike collagen-based mineralized tissues, no matrix vesicles are associated with mineralization of enamel. The mineral content is reduced from the enamel surface toward the dentinoenamel junction. The position of hydroxyapatite (HA) is located between the nanospheres of amelogenin. Therefore, ameloblasts are not only responsible for secreting the enamel matrix proteins and proteinases but also induce mineral formation and finally organize these minerals into rod and interred patterns.[10]

Role of calcium and phosphate ions in teeth and bone. Calcium phosphate is fundamental for the formation of bone and teeth and is essential for achieving optimal peak bone mass in the first 2–3 decades of life and for the maintenance of bone in later life. The HA in teeth varies from empirically derived HA, and HA found in bones, as the dental version, is often calcium deficient due to fluorine substitutions – Equations 1 and 2 – that shows the stoichiometric formula of HA (Equation 1). The formula of HA shows the sites for atomic substitution (Equation 2). This HA is calcium deficient and carbonated. X, calcium substitution with metal

cation; Y, phosphate substitution with carbonate; and Z, hydroxide substitution with fluoride.[11,12]

$Ca_5 (PO4)_3 (OH)$ (1)

Ca_{10}-X NaX (PO_4) 6-Y (CO_3) $(OH)_2$-Z FZ (2)

Calcium-deficient carbonated HA comprises the major substitution activity that takes place. Other much smaller number of substitutions occur where calcium ions, ~1%, is replaced by other metal ions, including potassium, sodium, and magnesium. The presence of carbonates and other ionic substitutions significantly disrupts the crystal lattice in HA. This weakens the HA, increasing its susceptibility to acid attack and solubility. The carbonate content of dentine is 5%–6%, while in enamel it is 3%, and the HA crystal size in dentine is much smaller than those in enamel, thus making dentinal matrix much more vulnerable to acidic attack.[13]

With age, the crystallinity of dental HA decreased but the carbonate content increased. The α-lattice constant, associated with the carbonate content, decreased while the c-lattice, associated with hydroxyl sites, does not change significantly with age. Increased crystal structure disorder and reduction in crystallinity are expected with higher number of planar carbonate ions substituting for tetrahedral phosphate ions in the apatite structure. Both A- and B-type of carbonate substitutions are present but the B-type (carbonate for phosphate) is greater than the A-type (carbonate for hydroxyl). Since c-lattice parameter is nearly age independent; this indicates that the phosphate tetrahedron represents the main site of carbonate substitution in the apatite lattice. A decrease in crystallinity and increase in carbonate content favor the dissolution of dental apatite. This is a change in material phase and the composition of dental mineral, while also reducing crystal size. The carbonate content has a significant effect on the reactivity and solubility of physiological HA. Calcium, phosphate, and fluoride ions play an important role in the demineralization and remineralization cycle and accordingly modify the susceptibility of tooth to caries progression. During demineralization, calcium release precedes phosphate release from enamel, dentin, and cementum. Therefore, using calcium rather than phosphate to suppress the demineralization process is effective.[14]

Role of Dental Biofilm in Demineralization of Teeth –

A dental biofilm is defined as a microbial community growing on a tooth surface. It is easily disclosed when the mouth is rinsed with a disclosing solution On a clean surface, single cells (cocci) attach to the pellicle (the proteinaceous saliva film) within 12 hours and the cells start to multiply and form microcolonies within 24 hours. At this stage, the surface may feel matt or rough when moving the tongue tip over the surface of the teeth. If left undisturbed there is a microbial succession, continued growth and an increased species diversity resulting in a ‘mature’ or climax type of biofilm within a week. This structure is often referred to as dental plaque.[15]

It is highly diverse in composition with numerous microcolonies of different species. Close to the tooth surface the packing of microcolonies is very dense, with many of the colonies separated by channels of intermicrobial matrices. Such protein matrices are partly formed by the microorganisms and partly derived from saliva. The surface of a dental biofilm is much more open in structure with new cocci constantly attaching and detaching. Saliva, particularly at the opening of the major salivary glands into the oral cavity, is supersaturated with respect to calcium and phosphate. Due of this, par of biofilm close to the opening of the salivary glands (buccal to the upper first and second

molars and lingual to the lower incisors) may become mineralized as dental calculus. This may happen even in biofilms that are a few weeks old.[16]

Metabolism in The Dental Biofilm

A Stephan curve is a pH curve measured consistently for 30 minutes in dental plaque after a washing with 2% sucrose for 1 or 2 minutes. Changes in nutritional circumstances, such as adding excess dietary carbohydrates like glucose, fructose, and sucrose, can drastically boost metabolism. [17]

Any shift in pH will influence the chemical composition of the biofilm fluid and the relative degree of saturation of this fluid with respect to the minerals is important for maintaining the chemical composition of the tooth surface. When the cumulative result of the numerous pH fluctuations over months or years is a net loss of calcium and phosphate, of an extent that makes the enamel sufficiently porous to be seen in the clinic, it may be diagnosed as 'a white spot lesion'. When sections are cut through an active, so-called white spot lesion, and examined, either by light microscopy or by micro-radiography, it is apparent that the loss of mineral occurs in the subsurface enamel and that the thickness of the surface zone may vary considerably. When the enamel surface is examined in a scanning electron microscope, active enamel caries surfaces appear moth-eaten with partial dissolution of crystals so that there is an enhanced spacing between crystals.[18]

Saliva, fluoride therapy, diet control, and probiotic bacteria are all preventative measures for dental demineralization. To reverse demineralization, neutralize the pH and ensure enough Ca_2+ and PO_4+ ions are present in the surrounding area. Apatite dissolving products can be neutralized through buffering or inhibited by Ca_2+ and $(PO_4)_3$ ions in saliva via the common ion effect. This process, known as remineralization, allows for the regeneration of partially disintegrated apatite crystals. Rapid deposition of fluorapatite will form a firm surface layer, which is notably more resistant to further demineralization but at the same time is resistant to penetration of the calcium and phosphate ions required to rebuild the lesion in depth. The result is often the visible persistence of the white spot lesion, including some level of stain uptake that emphasizes its presence.[19,20]

Remineralization of white-spot lesions may be possible with a variety of currently available agents containing fluoride, bioavailable calcium and phosphate, and casein phosphopeptide in-amorphous calcium phosphate, self-assembling peptide. Non-cavitated enamel lesions retain most of the original crystalline framework of the enamel rods, and the etched crystallites serve as nucleating agents for remineralization. Calcium and phosphate ions from saliva can penetrate the enamel surface and precipitate on the highly reactive crystalline surfaces in the enamel lesion. The supersaturation of the saliva with calcium and phosphate ions serves as the driving force for the remineralization process. Remineralized (arrested) lesions can be observed clinically as intact, but discolored, usually brown or black spots. The change in color is presumably due to trapped organic debris and metallic ions within the enamel. These discolored, remineralized, arrested caries areas are intact and are more resistant to subsequent caries attack than the adjacent unaffected enamel. They should not be restored unless they are esthetically objectionable.[21]

Zones of Non-Cavitated Lesions: [22,23]

Zone 1: Translucent Zone

- The translucent zone is the deepest zone and represents the advancing front of the enamel lesion

showing structureless appearance when perfused with quinoline solution and examined with polarized light.

- In this zone, the pores or voids form along the enamel prism (rod) boundaries, presumably because of the ease of hydrogen ion penetration during the carious process. When these boundary area voids are filled with quinoline solution, which has the same refractive index as enamel, the features of the area disappear.
- The pore volume of the translucent zone of enamel caries is 1%, 10 times greater than normal enamel.

Zone 2: Dark Zone

- The next deepest zone is known as the dark zone because it does not transmit polarized light. This light blockage is caused by the presence of many tiny pores too small to absorb quinoline.
- These smaller air-filled or vapor-filled pores make the region opaque. The total pore volume is 2% to 4%. There is some speculation that the dark zone is not really a stage in the sequence of the breakdown of enamel; rather, the dark zone may be formed by deposition of ions into an area previously containing only large pores.
- The size of the dark zone is an indication of the amount of remineralization that has recently occurred.

Zone 3: Body of Lesion

- The body of the lesion is the largest portion of the incipient lesion while in a demineralizing phase. It has the largest pore volume, varying from 5% at the periphery to 25% at the center. The striae of Retzius are well marked in the body of the lesion, indicating preferential mineral dissolution along these areas of relatively higher porosity. The first penetration of caries from the enamel surface is via the striae of Retzius.
- The inter-prismatic areas and these cross-striations provide access to the rod (prism) cores, which are preferentially attacked. Bacteria may be present in this zone if the pore size is large enough to permit their entry. Studies using transmission electron microscopy and scanning electron microscopy show the presence of bacteria invading between the enamel rods (prisms) in the body zone.

Zone 4: Surface Zone

- The surface zone is relatively unaffected by the caries attack. It has a lower pore volume than the body of the lesion (<5%) and a radiopacity comparable to unaffected adjacent enamel.
- The surface of normal enamel is hypermineralized by contact with saliva and has a greater concentration of fluoride ion than the immediately subjacent enamel. It has been hypothesized that hypermineralization and increased fluoride content of the superficial enamel are responsible for the relative immunity of the enamel surface.
- Removal of the hypermineralized surface by polishing fails, however, to prevent the reformation of a typical, well-mineralized surface over the carious lesion. The intact surface over incipient caries is a phenomenon of the caries demineralization process rather than any special characteristics of the superficial enamel.
- Nevertheless, the importance of the intact surface cannot be overemphasized because it serves as a barrier to bacterial invasion. As the enamel lesion progresses, scanning electron microscopy shows conical-shaped defects in the surface zone.

- These are the first sites where bacteria gain entry into a carious lesion. Arresting the caries process at this stage results in a hard surface that may at times be rough, although self-cleansable.

Diagnosis Of Non-Cavitated Lesions: [24,25]

Newer Technologies In Diagnosis Include:

1. Multiphoton imaging
2. Infrared fluorescence
3. Infrared thermography
4. Terahertz imaging
5. Optical coherence tomography
6. Polarized Raman spectroscopy
7. Modulated (frequency-domain) infrared photo thermal radiometry.

Mechanism of Light in Detection of Dental Caries:

- The internal structure of a tooth guarantees that light propagates well through the crystalline enamel and dentin tubules, and any disruption to the tooth's structure increases the risk of scattering. The uptake of fluid into demineralized pores, as well as the uptake of foreign stain, bacterial breakdown products, and other contaminants produced by the caries process, will alter the usual interaction of light with tooth structure. In addition to scattering, these changes will involve absorption and fluorescence. A variety of procedures employ one or more of these interactions to identify dental caries.

Management of Non-Cavitated Lesions: [26,27,28]

Primary Prevention of Dental Caries

Initiation of community and individual caries prevention strategies, particularly water fluoridation and the use of fluoridated toothpaste, as well as the use of other remineralization strategies, oral hygiene, patient education, and preventive programs (tooth brushing programs, sealants/mouthrinses), which have proven to be very successful in many countries over the last 30 years.

Education and motivation of patient - It is important to educate patient regarding early carious lesion and motivation to maintain proper oral hygiene so that these lesions can be prevented.

Mechanical/chemical plaque control

Mechanical/chemical removal of plaque (oral hygiene). Traditional physical/mechanical methods of caries prevention includes oral hygiene procedures (tooth brushing, flossing, and professional tooth debridement). The preventive strategies to be effective in high-risk people along with oral hygiene are also likely to be effective for arresting and reversing lesions. The use of anti-plaque agents, such as chlorhexidine, cetylpyridinium chloride, delmopinol, hexitidine, Sanguinaria extracts, triclosan, casein phosphopeptideamorphous calcium phosphate (CCP-ACP) are proved to be effective in reversing the incipient caries. Use of fluorides in the form of community-based fluoride interventions, self-applied methods of fluoride delivery, such as (a) fluoride dentifrices, (b) fluoride rinses, (c) fluoride gels and foams, (d) fluoride chewing gum, professional fluoride delivery methods, such as (a) fluoride gels (b) varnishes.

Remineralizing agents - These are the agents which are commonly used to manage early carious lesions or white spot lesion through a remineralization approach. They increase oral calcium and phosphate levels and shifts the equilibrium toward remineralization. Apart from white spot lesion management, they are also used in patients undergoing orthodontic treatment and bleaching procedure to reduce decalcification risk.

Conclusion

It is of utmost significance to understand the origin and evolution of subsurface lesions, as well as the limits of modern technologies and their clinical applications, to tailor preventive measures for high level caries risk people. The balance between demineralization and remineralization influences the evolution of an array of carious lesions. Newer enamel remineralization methods are gaining attention, leading to a shift in our understanding of dental caries. Recent research focuses on calcium phosphate-based technologies that boost fluoride's ability to rebuild tooth mineral.

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