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REVIEW ARTICLE

Management of Enamel White Spot Lesions

Surabhi Joshi, Chintan Joshi

ABSTRACT

Dental caries is a highly prevalent multifactorial disease and is a major public health problem. A goal of modern dentistry is to manage enamel white spot lesions noninvasively and effectively an attempt to prevent disease progression and improve esthetics, strength and function. The progression of caries has been tried to be curbed at initial stage only but for that only use of fluoride application was suggested but with recent developments in dental materials other remineralization options as well as noninvasive masking procedure can be performed to attain best result. This article reviews all the materials and techniques mentioned in the literature to manage the world's most common disease in its initial stage only.

Keywords: White spot lesion, Remineralization, Fluoride, CCP-ACP, Resin infiltration, Nanohydroxyapatite, Hard tissue lasers.

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INTRODUCTION

The initial carious lesions are the so-called 'white spot' lesions which imply that there is a subsurface area with most of the mineral loss beneath a relatively intact enamel surface. A cross-section of the white opaque spot reveals the characteristics of carious enamel and this means that dental caries is essentially an enamel defect with a relatively intact surface layer and some subsurface damage due to acid formed from plaque on tooth surface.[1] The enamel demineralization defect has a lower mineral distribution in the surface layer in comparison to the adjacent sound enamel and also a lower interprismatic mineral content. The first stage of enamel demineralization is characterized by removal of interprismatic mineral content and in the subsequent stages a well-defined surface layer formation occurs which constitutes early caries lesion.[2]

The main types of enamel demineralization include incipient lesions and 'surface-softened defect' which are also some of the various terms that have been used to describe early caries lesions. It is important to differentiate incipient lesion from arrested lesions. Incipient lesions are active lesions which continue to progress under acid attack whereas an arrested lesions does not progress. *In vivo* ultrastructural studies by Thylstrup and Fredebo led them to conclude that there were wide variations between active and arrested lesions. 'Micro-scars' were seen on active lesions while microcavitation was usually seen on arrested lesions.[1]

In context of presently available literature, the management of enamel white spot lesion can be mainly divided into intervention through remineralization techniques, noninvasive infiltration and inhibition and surface alteration of the lesion. This article attempts to review all presently available methods and their philosophy behind management of enamel white spot lesion.

REMINERALIZATION

Traditionally, the strategy to manage white spot lesion has been remineralization of the lesion through fluoride application, recently the use of CCP-ACP, Galla chinensis and nanohydroxyapatite have been suggested for the same.

Fluoride Application

The unmasking of enamel structure as seen in microradiographs suggests that acid initially removes mineral from some sites and not from others. In addition, both microradiography and polarized light microscopy has revealed the existence of a relatively highly mineralized surface layer in many enamel lesions. Chemical analysis has shown that decayed 'white spot' enamel has not only less calcium and phosphate but also less carbonate and magnesium, indicating a preferential removal of these mineral ions. On the other hand, carious enamel has more organic material, a higher proportion of phosphate in the form of HPO_4^{2-} and a higher concentration of fluoride. This increase in fluoride (F) is most pronounced in the well-mineralized surface layer.[3]

The apparent preservation of the surface zone initially suggested that its character *per se* renders it less susceptible to acid attack. It contains, e.g. high concentrations of fluoride, which stabilizes apatite and low carbonate and low magnesium, which have a reverse, destabilizing effect. This would favor a lower acid solubility for mineral in this tissue region, effectively protecting it from dissolution. At the same time, penetration of acid into the deeper, more soluble, layers would remove interior mineral in preference to the outer tissue. The outer tissue could then continue to accumulate fluoride and become even more acid-resistant.

The normal presence of organic material on or in the enamel surface (the pellicle) has also been suggested as a contributor to surface zone formation by reducing mineral loss or acting as a permeable selective barrier. Organic components, mainly proteins from the saliva, such as those seen in pellicle, may not only affect transport into and out

of the enamel but also together with components, such as fluoride and, e.g. pyrophosphates. While it is clear that fluoride provides less soluble apatite and will facilitate redeposition, it will also facilitate the hydrolysis of acidic calcium phosphate phases, such as dicalcium phosphate dihydrate (DCPD) and octocalcium phosphate (OCP), to the more stable fluoridated apatite.

Discriminating between the effects of rendering enamel mineral less acid-soluble and facilitating redeposition is clearly difficult. For fluoride stimulated remineralization, the situation is less straightforward. Fluoridated mineral will have a lower solubility product and will tend to precipitate readily, mainly at the surface. If blocking of surface porosity occurs, the repair process would be restricted to the surface layer. In this sense, fluoride could be said to be less effective at facilitating remineralization than inhibiting demineralization, since it would not lead to repair deep within the lesion.[4]

Quantitative microradiographic studies after application of higher fluoride concentration showed an increase in remineralization in the outer lesion and a decrease in demineralization in the inner part, resulting in a significant increase in mineral gain. It has recently been discussed that with elevated external F-levels, the F-gradient might be higher, driving the fluoride deeper into the advanced lesion, in spite of the F-diffusion being slowed by adsorption onto and reaction with hydroxyapatite crystallites.[5] The bioavailability of fluoride is important for its effect in the prevention of caries. However, this bioavailability depends on its solubility in the compounds and on the way it adheres to the compounds of the surface. In a study done by Santos et al, the fluoride products reduced the depth of the artificial carious lesions but they did not completely prevent their development so it was emphasized that other preventive methods, such as controlling diet and plaque, should be considered in the prevention and/or control of dental caries disease.[6]

In a study, comparison between low fluoride mouthrinse/ toothpaste (<50 ppm) combination compared with a nonactive control combination showed there was a general exponential reduction in demineralized white lesion area but failed to show any differences or therapeutic affect.[7] In two different studies conducted by Sano et al and karlinsey et al, it was concluded that 500 ppm F containing dentrifices will have remineralization of early carious lesion as well as caries inhibiting effect.[8,9]

Casein Phosphopeptides and Amorphous Calcium Phosphate (CPP-ACP)

The anticariogenic properties of milk and milk products, such as cheese have been studied previously in animal models. This activity has been attributed to the direct chemical effects of phosphoprotein casein and calcium phosphate components in cheese. It has been suggested that casein phosphopeptides (CPPs) have the ability to stabilize calcium phosphate (ACP) with their multiple phosphoserine residues, thereby allowing the formation of small CPP-ACP clusters. CPP-ACP might prevent tooth erosion by suppressing demineralization, enhancing remineralization of these two processes.[10]

In a study by EC Reynolds, it was found that CPP-stabilized calcium phosphate solutions remineralize subsurface lesions in human enamel *in vitro*. The more effective remineralizing solutions were those with the higher concentrations of CPP stabilized free calcium and phosphate ions, which were the 0.5 and 1.0% CPP solutions at pH 7.0. The CPP can stabilize over 100 times more calcium phosphate than in normally possible in aqueous solution at neutral or alkaline pH before spontaneous precipitation. The majority of the calcium phosphate in the CPP-stabilized solutions was in the form of ACP bound by the CPP, the solutions still contained CPP-stabilized free calcium and phosphate ions at very high activities, far exceeding normal solubilities, resulting in supersaturation with respect to the amorphous and crystalline calcium phosphate phases. Notwithstanding this highly supersaturated state, the CPP stabilized the solutions, preventing spontaneous precipitation.

The results of the study therefore suggested that the remineralization process involves diffusion of $CaHPO_4(0)$ and associated calcium and phosphate ions through the protein/H20-filled pores of carious surface enamel into the body of the enamel lesion. Once in the body of the enamel lesion, these calcium and phosphate species, by dissociation, would increase the activities of Ca^{2+} and PO_4^{3-}, thereby increasing the degree of saturation with respect to HA. The formation of HA in the lesion would lead to the generation of acid and phosphate, including the neutral $H3PO_4(0)$, which would diffuse out of the lesion down a concentration gradient. The CPP, by stabilizing calcium phosphate in a metastable solution, facilitate high concentrations of calcium and phosphate ions, including $CaHPO_4(0)$, which can diffuse into the enamel subsurface lesion. The CPP will also maintain the high activities of the free calcium and phosphate ions during remineralization through the reservoir of bound ACP. The bound ACP, by being in dynamic equilibrium with free calcium and phosphate ions, will maintain the concentrations of the species involved in diffusion into the lesion. Furthermore, dissociation of the CPP-bound ACP will be facilitated by the acid generated during enamel remineralization. This would explain why the CPP-supported metastable calcium phosphate solutions are such

efficient remineralizing solutions, since they would consume the acid generated during enamel lesion remineralization by generating more calcium and phosphate ions, including $CaHPO_4(0)$, thus maintaining their high concentration gradients into the lesion.[11]

In two independent studies conducted by Llena et al and Jayarajan et al, it was concluded that significantly high levels of calcium and phosphate have been found in both biofilm and subsurface incipient caries lesions and in lower level demineralization of enamel or dentine surfaces previously treated with CPP-ACP based compounds.[12,13]

Galla Chinensis (G Chinensis)

It is a traditional Chinese herb investigated as an anticarious drug during recent years. In a study done by cheng and Ten cate, it was found that the combination of Galla chinensis extract with the enamel surface could reduce the demineralization of dental enamel. After demineralization, the mineral structure of the surface layer was not destroyed too greatly, so that deposit of mineral on remaining hydroxyapatite would occur slowly on the surface layer. On the other hand, the remineralization of enamel crystals on the surface would also be slowed by the combination with Galla chinensis extract. And thus, more calcium and phosphate could enter into the lesion body but would not first precipitate in the layers closest to the surface.[14]

Various studies were done to confirm the reports on remineralization and to understand the mechanism which is still unknown. Studies by Cheng et al showed that Galla chinensis extract could affect the mineral ions deposit on the surface layer and then modified the remineralization of initial dental caries.[15] A study by Zang et al shows similar finding but with the organic matrix of enamel shown also playing a substantial role.[16] Chemical and crystallographic and atomic force microscopy studies by Zang et al provide evidence by showing that crystallinity was increased and there was a change in surface topography of lesion.[17,18]

Nanohydroxyapatite

Recently, some role of nanohydroxyapatite has also been studied. In a study where role of nanohydroxyapatite alone on remineralization was done by zuang et al, it was found during the scanning electron microscope analysis that nano-hydroxyapatite particles were regularly deposited on the cellular structure of the demineralized enamel surface, which appeared to form new surface layers. It was concluded that nanohydroxyapatite had the potential to remineralize initial enamel lesions and a concentration of 10% nano-hydroxyapatite might be optimal for remineralization of early enamel caries.[19] In one another study by Zuang et al, the combined effect of nanohydroxyapatite and Gal chinensis was studied and it was found that more mineral deposition occurred in the lesion body, and lesion depth was reduced significantly and it was concluded that there was a significant synergistic effect of combined Galla chinensis extract and nanohydroxyapatite treatment on promoting the remineralization of initial enamel lesion.[20]

NONINVASIVE INFILTRATION

Resin Infiltration Technique

With the recent advancement in dental material and laser technology attempts have also been made to 'restore' or treat the lesion. In this direction, resin infiltration technique is novel concept in which the pores within enamel lesions provide diffusion pathways for acids and dissolved minerals, the resin infiltrates these pathways aiming at occluding the pores and thus preventing acid infiltration into the lesion. [21]

In a study done by Jin-ho Phark, the protocol for resin infiltration has been discussed in which it has been mentioned that to achieve good adhesion and penetration of the resin into the more porous subsurface lesion body of the artificial lesions, the pseudo-intact surface layer was etched using phosphoric acid. This layer is formed by precipitation of minerals on the enamel surface and has a much lower pore volume compared to the lesion body, thus inhibiting the penetration of the resin. However, there are structural differences between artificial and natural enamel lesions. The surface layers of natural lesions are more inhomogeneous and may show greater thickness and mineral content compared to artificial lesions because of alternating demineralization and remineralization cycles in the oral cavity. Therefore, penetration of adhesives into natural lesions even after 2 minutes of etching with phosphoric acid was only superficial. For that reason, an alternative etching protocol using 15% hydrochloric acid for 2 minutes was developed. In addition, the rheologic properties of regular adhesives do not allow sufficient penetration into the porous lesion. Therefore, resins with low viscosity (infiltrants) with improved rheologic properties were developed. The combination of etching with hydrochloric acid and the application of low-viscosity infiltrants allowed a nearly complete penetration of natural enamel carious lesions.[22]

A study done by Belli et al evaluated the wear and morphology of infiltrated white spot lesion and concluded that the procedure ensured improved surface stability depending on infiltration quality[23] In another study done by Rocha et al, it was found that the group infiltrated with low viscosity resin presented the lowest means of color change compared to one treated by fluoride application.[24]

SURFACE ALTERATION

Hard Tissue Lasers

Since last 40 years, development of potential of hard tissue lasers is being studied and its role in not only being an invasive tool but also a preventive device has been studied. Studies have been done using CO_2 lasers, the basic mechanism described in prevention of lesion is that the carbon dioxide laser wavelengths are efficiently absorbed by the carbonated apatite mineral of the tooth and that the absorbed light is rapidly transformed to heat near the surface, causing loss of carbonate from the mineral with a subsequent marked decrease in acid reactivity. The variables involved are wavelength, pulse width, incident and absorbed pulse energy, beam diameter, number of pulses, repetition rate and irradiation intensity.[25] the reason hypothesized for increase in acid resistance is due to the melting and fusion of enamel hydroxyapatite (HA) crystals and/or the subsequent sealing of the enamel surface. However, a cross-sectional TEM examination revealed that the melting of the enamel surface was not homogenous and usually occurred in limited areas. A significant increase in inter- and intra-crystalline voids occurred beneath the melted surfaces. The new chemical products in the melted enamel structure, which include alpha- and beta-tricalcium phosphate (TCP) and tetra-calcium phosphate, are less resistant to acid attack than is enamel hydroxyapatite.[26]

Further to decrease the risk of increase in temperature and subsequent harmful effect to the pulp, effects of pulsed mode over continuous mode were also studied in one such study it was concluded that pulsed CO_2 laser treatment of enamel surfaces can inhibit as much as 87% of subsequent caries like lesion progression keeping pulses less than 25 was found to be optimal.[27]

With the emergence of so many concepts and promising results, it helps us now to better understand the factors which promote remineralization, arrest caries progression and produce morphologically stable surface. In future, we can only hope that, with this knowledge, we will be better able to treat white spot lesion either with single treatment option or combination therapy, for which further research is required.

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ABOUT THE AUTHORS

Surabhi Joshi

Lecturer, Department of Periodontics, Karnavati School of Dentistry Gandhinagar, Gujarat, India

Chintan Joshi (Corresponding Author)

Reader, Department of Conservative and Endodontics, Karnavati School of Dentistry, Gandhinagar, Gujarat, India, e-mail: drchintanjoshi@rediffmail.com