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White spot lesions and their management

Dr. Ravi Shanthraj raviortho11@gmail.com JSS Dental College and Hospital, Mysore, Karnataka

Ashwini B drashwini2007@gmail.com Faroqia Dental College and Hospital, Mysore, Karnataka

Biju Jose dr.bijojose@gmail.com JSS Dental College and Hospital, Mysore, Karnataka

ABSTRACT

White spot lesions develop as a result of a dietary carbohydrate and saliva modified bacterial infection, resulting in an imbalance between demineralisation and remineralisation of enamel. The lesion is caused by the accumulation of plaque and bacteria along with insufficient oral hygiene. High plaque accumulation can increase the risk of white spot lesion formation. The dynamic balance between demineralization and remineralization determines the progression of white spot lesion. The diagnostic armamentarium includes novel technologies and non-invasive techniques like fiber-optic transillumination and electrical resistance methods which are very useful in detecting posterior approximal dentinal caries and occlusal caries. Radiographs and direct digital imaging are still important tools in the estimation of caries. A clear understanding of the mechanism of subsurface lesion formation and progression, possibilities, and limitations of newer methods and their clinical applications need to be recognized by the dentist to direct preventive strategies to the high caries risk individuals.

Keywords: White spot lesions, Caries, Demineralization, Remineralization

1. INTRODUCTION

Dental caries is a major public health problem in the whole world. It is becoming a controlled problem with fluoride enriched water and personal hygiene applications¹

Caries lesions are the clinical manifestation of a pathogenic process that may have been occurring on the dental surface over months or years. The first step of cariogenicity is that oral bacteria start to decay the food interacting with mucopolysaccarides like sucrose on the enamel surface. Dental plaque bacteria metabolize dietary sugars to produce organic acids that solubilize tooth enamel's hydroxyapatite crystals. During the exposure of the enamel to organic acids, solid calcium phosphate is solubilized to free calcium. This process is called demineralization and it is reversible to some point by the presence of salivary sodium bicarbonate aiding remineralization²

White spot lesions (WSLs) are defined as a "subsurface enamel porosity from carious demineralization" that presents as "a milky white opacity when located on smooth surfaces.³ The White spot lesions (WSLs) are the first visible evidence of caries in the enamel, characterized by demineralized lesion underneath an intact surface⁴

White spot lesions develop as a result of a dietary carbohydrate and saliva modified bacterial infection, resulting in an imbalance between deminaralisation and reminaralisation of enamel. The lesion is caused by the accumulation of plaque and bacteria along with insufficient oral hygiene. High plaque accumulation can increase the risk of whitening spot lesion formation⁵

2. DEVELOPMENT OF WHITE SPOT LESIONS

WSLs develop as a result of prolonged plaque accumulation on the affected surface, commonly due to inadequate oral hygiene⁶. With the maintenance of these conditions, acids diffuse into the enamel and begin the demineralization of subsurface enamel. If the demineralization process is not stopped, the intact enamel surface eventually collapses and cavitates. These lesions are characterized by a white, chalky, opaque appearance and are commonly located in pits, fissures, and smooth surfaces of teeth.

However, after the placement of fixed orthodontic appliances, there is an increasing number of plaque retention sites due to the presence of brackets, bands, wires, and other applications, which make oral hygiene more difficult and limit naturally occurring self-cleansing mechanisms. As a consequence, there is an increased risk of demineralization and, conclusively, of WSLs forming on smooth surfaces, if there is no effective plaque removal.8

The clinical characteristics of these lesions include loss of normal translucency of the enamel because of altered light properties with a chalky white appearance, particularly when dehydrated; a fragile surface layer susceptible to damage from probing,

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particularly in pits and fissures; increased porosity, particularly of the subsurface, with increased potential for uptake of stains; reduced density of the subsurface, which may be detectable radiographically, with transillumination or with modern laser detecting devices; and potential for remineralization, with an increased resistance to further acid challenge particularly with the use of enhanced remineralization treatments.⁹

The white appearance of early enamel caries is due to an optical phenomenon which is caused by a mineral loss in the surface or subsurface enamel. Enamel crystal dissolution begins with subsurface demineralization, creating pores between the enamel rods. The resultant alteration of the refractive index in the affected area is then a consequence of both surface roughness and loss of surface shine and alterations in internal reflection, all resulting in greater visual enamel opacity.¹⁰

The overall prevalence of WSLs amongst orthodontic patients has been reported as anywhere between 2% and 97%. Its presence can be detected as early as 4 weeks into orthodontic treatment. usually, orthodontic patients have a significantly higher incidence of WSLs compared to non-orthodontic patients and may also exhibit esthetic changes a few years after treatment. Approximately, 50% of patients develop more than one WSL during orthodontic treatment. In orthodontic patients, 5.7% of the teeth are affected. 12

WSLs can seriously jeopardize the esthetic outcome of the treatment. WSLs lesions have a limited ability to improve after appliance removal and white spots can sometimes be detectable even 12 years after treatment. Although primary prevention must be in focus, two major strategies on how to deal with existing lesions after debonding have been suggested; remineralizing or masking the lesions.¹³

The first is based on secondary prevention and reversing the lesions through remineralizing agents like topical fluoride, amorphous calcium phosphate, or self-assembling peptides. The second strategy aims to mask and improve the esthetic appearance of the teeth through minimal-invasive measures, such as bleaching, micro-abrasion, or resin infiltration.¹⁴

3. DETECTION OF WHITE SPOT LESIONS

The traditional methods of detecting early lesions include visual inspection and radiography. In visual observation, reflected light is used to detect changes in color, texture, and translucency of the tooth substance. However, these traditional methods for early caries diagnosis have been found to be inaccurate and insensitive.¹⁵

Recent advances in detection of white spot lesions are

- 1) Quantitative Iight-induced Fluorescence (QLF) which measures enamel auto-fluorescence can detect differences in remineralization of early enamel caries, 16
- 2) Polarization Sensitive Optical Coherence Tomography (PSOCT) system has also been used to study the spatially resolved scattering and polarization phenomena of teeth which are known to have strong polarization effect.¹⁷
- 3) Digital Imaging Fiber-Optic Transillumination (DIFOTI) uses images of teeth obtained with a digital CCD camera, which are sent to a computer for analysis with dedicated algorithms for location and diagnosis of carious lesions by the operator -in real time, thereby providing a quantitative characterization for monitoring of approximal, occlusal, and smooth-surface caries.¹⁸
- 4) Laser Fluorescence Device Diagnodent has been used to detect occlusal caries and has more sensitivity and specificity than radiographic examination.¹⁹

3.1 Remineralizing agents for the treatment of white spot lesions

3.1.1 Fluorides

In the oral cavity, the presence of fluorides decreases the development/progression of dental caries by 3 different mechanisms: inhibition of demineralization of the enamel, increase in remineralization of the enamel, and inhibition of the bacterial enzyme producers of acids.²⁰ The professional application or prescription of fluorides for home use includes: gels and toothpaste (maximum 5000 ppm), mouthwashes (223 ppm), and varnishes (23,000 ppm).²¹

The fluoride ions are revealed in 3 ways: sodium monofluorophosphate, sodium fluoride, and amine fluoride. It has been described in the literature that high fluoride concentrations promote WSL remineralization for hypermineralization. However, it occurs in the enamel surface and inhibits the ions' movement through the subsurface, affecting the subsurface remineralization and therefore, the light reflection.²²

3.1.2 Casein Phosphopeptides

The casein phosphopeptides (CPPs) have the remarkable ability to stabilize the calcium and phosphate ions present in the solution, creating the casein phosphopeptides with amorphous calcium phosphate (CPP-ACP complex). This complex increases the calcium and phosphate levels, promoting the remineralization process. Although this does not occur without the presence of fluorides, the CPP ACFP complex (casein phosphopeptides with amorphous calcium phosphate and fluoride) exists commercially for this.²³

The CPP has the ability to bind and stabilize calcium and phosphate in solution, as well as to bind dental plaque and tooth enamel. Through their multiple phosphoryl residues, CPPs bind to form clusters of ACP in metastable solution, preventing their growth to the critical size required for nucleation and precipitation. The proposed mechanism of anticariogenicity for the CPP-ACP is that it localizes ACP in dental plaque, which buffers the free calcium and phosphate ion activities, thereby helping to maintain a state of

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supersaturation with respect to tooth enamel depressing demineralization and enhancing remineralization. The CPPs have been shown to keep fluoride ions in solution, thereby enhancing the efficacy of the fluoride as a remineralizing agent.²⁴

3.1.3 Amorphous Calcium Phosphate

The ACP technology requires a two-phase delivery system to keep the calcium and phosphorous components from reacting with each other before use. The current sources of calcium and phosphorous are two salts, calcium sulfate, and dipotassium phosphate. When the two salts are mixed, they rapidly form ACP that can precipitate onto the tooth surface. This precipitated ACP can then readily dissolve into the saliva and can be available for tooth remineralization.²⁵

3.1.4 Sodium Calciumphosphosilicate (Bioactive Glass)

When bioactive glass comes in contact with saliva, it rapidly releases sodium, calcium, and phosphorous ions into the saliva that are available for remineralization of the tooth surface. The ions released form hydroxycarbonate apatite (HCA) directly. They also attach to the tooth surface and continue to release ions and remineralize the tooth surface after the initial application.²⁶

3.1.5 Xylitol Carrier

The use of chewing gum carrying xylitol increases salivary flow rate and enhances the protective properties of saliva. This is because the concentration of bicarbonate and phosphate is higher in stimulated saliva, and the resultant increase in plaque pH and salivary buffering capacity prevents the demineralization of tooth structure. Moreover, the higher concentration of calcium, phosphate, and hydroxyl ions in such saliva also enhances remineralization.²⁷

3.1.6 Nano-Hydroxyapatite

A study was done to determine the effect of nano-HAP concentrations on initial enamel lesions under dynamic pH-cycling conditions. It was concluded that nano-HAP had the potential to remineralize initial enamel lesions. A concentration of 10% nano-HAP may be optimal for remineralization of early enamel caries.²⁸

3.1.7 The Trimetaphosphate Ion

The potential mode of action of trimetaphosphate ion (TMP) is likely to involve in adsorption of the agent to the enamel surface, causing a barrier coating that is effective in preventing or retarding reactions of the crystal surface with its fluid environment, and hence reducing demineralization during acid challenge.²⁹

3.1.8 Alpha-Tricalcium Phosphate

It is used in products such as Cerasorb, Bio-Resorb, and Biovision. Tricalcium phosphate (TCP) has also been considered as one possible means for enhancing the levels of calcium in plaque and saliva.³⁰

3.1.9 Infiltrative Resins

Infiltrative resins had been commercialized as a minimal invasive restorative treatment which involves the resin penetration inside the body of the WSL, with minimal enamel loss. This technique uses etch-and-rinse acid to remove the superficial layer of enamel, exposing the WSL demineralization, infiltrating the lesion with a low viscosity resin. The therapeutic purposes are the mechanic stabilization of the hydroxypatite structure instead of WSLs cavitation.³¹

4. CONCLUSION

The dynamic balance between demineralization and remineralization determines the progression of white spot lesion. The diagnostic armamentarium includes novel technologies and non-invasive techniques like fiber-optic transillumination and electrical resistance methods which are very useful in detecting posterior approximal dentinal caries and occlusal caries. Radiographs and direct digital imaging are still important tools in the estimation of caries. A clear understanding of the mechanism of subsurface lesion formation and progression, possibilities, and limitations of newer methods and their clinical applications need to be recognized by the dentist to direct preventive strategies to the high caries risk individuals.

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