WHITE ENAMEL LESIONS ASSOCIATED WITH FIXED ORTHODONTIC APPLIANCES

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ABSTRACT

Appearance of white demineralized lesions in the vicinity of bonded and banded attachments is a common occurrence during fixed orthodontic treatment. It seems to be a big challenge for every treating orthodontist. The prerequisite of tremendous oral hygiene practice during active fixed orthodontic treatment must be conveyed to the patient and parents apiece. Strict preventive measures are to be reinforced. Awake up call and still more to adapt various ways to prevent this terrible condition from endangering our net orthodontic outcome.

Key words: *Orthodontic attachments, white lesions, Plaque, Fluoride.*

INTRODUCTION

White opaque lesion on the surface of a tooth is caused by decalcification of enamel. Its appearance around orthodontic attachments is a common problem during and following fixed orthodontic treatment. Decalcification occurs due to plaque accumulation promoted by orthodontic attachments and bonding materials. The acid produced by the bacterial plaque result in changes in the appearance of the enamel surface. These opaque, white spots are caused by mineral loss from the surface and subsurface of the enamel. If mineral loss is not arrested, then a frank cavity is produced. Clinically, white spots around brackets appear as early as 4 weeks into treatment and their prevalence among orthodontic patients ranges from 2-96%.[1] The labio-gingival area of the lateral incisors is the most vulnerable site for white lesions while the maxillary posterior teeth are the least effected teeth in the oral cavity. Incidence of white spot lesions is predominant in male than in female patients' This problem has been identified and many efforts are being already made, but it still continues to menace and plague the den· tal aesthetics of our youth in an unnoticed manner.

Etiology: Bacterial plaque, fermentable carbohydrates, susceptible tooth surface, and substantial span of time are the main factors.

It has been established that smooth surface caries is associated with *Streptococcus mutans* and its prevalence is directly related to the incidence of caries and its index.' *Streptococcus mutans* prefer to colonize over the retentive areas and thus their presence on these surfaces is a reflection of increased caries risks. After the onset of carious lesion, its progress is fueled by *Lactobacillus*. High index oflactobacillus indicates that an amicable environment for the ignition of dental caries is existing. Increased proliferation of these microorganisms and new sites of plaque deposition on the enamel surrounding the orthodontic attachments is common in patients undergoing fixed orthodontic treatment. This may be compounded by the total treatment span and the configuration and number of orthodontic attachments present in the oral cavity.

Saliva plays a pivotal role that influences the dynamics of demineralization and remineralization at the enamel-plaque interface. The amount and rate ofenamel demineralization, and the chances of remineralization is affected by salivary pH[3], rate of flow and buffer potential. Exposure of the tooth surface to the fermentable carbohydrates, pH of the plaque and microbial composition of the plaque are controlled by the saliva. It transports and delivers fluoride ions both to the plaque and enamel.

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Tooth surfaces that are more exposed to dietary carbohydrate with less exposure to saliva are the common sites of demineralization. Thus the site with highest incidence of demineralization, that occurs during treatment, is the maxillary anterior teeth. The lingual surface of the lower incisors where salivary flow is adequate is often the site of calculus formation, indicating mineralization.' This implies that sufficient flow of saliva acts as a major contributing factor in preventing demineralization. It is well documented that salivary flow rate can influence both caries risk and caries activity.' Adequate flow of saliva helps in the physical cleansing of carbohydrates from tooth surfaces, maintains its buffering capacity and anti-microbial activities. Low pH of plaque leads to enamel demineralization. This acidic pH is usually countered by the alkaline pH and buffering capacity of the saliva. The pH and buffering capacity of the saliva is, however maintained by the rate of salivary secretion. An intraoral environment with low pH favors colonization of the cariogenic bacteria, whereas a high salivary pH maintains a higher buffering capacity. There is also an important negative correlation between the buffering capacity of the saliva and the frequency of carious lesions.[4]

Oral hygiene: Orthodontic attachments make tooth cleansing more difficult and cumbersome thus predisposes tooth surface to plaque accumulation. Appliances also hamper the self-cleansing action of the tongue, lips and cheek to remove food debris from the tooth surface. Therefore, accumulated food debris, particularly fermentable carbohydrates, encourage growth of the cariogenic bacteria. Most harbor areas are usually found under the wings of brackets and near the edges of bands, where there may be five fold increase in the lactobacillus count in patients undergoing active orthodontic therapy.

Diet: Following the ingestion of fermentable carbohydrate, acids are produced inside the plaque and plaque pH falls, and it recovers as salivary buffering occurs. However, as the frequency of carbohydrate intake increases, the enamel surface may be exposed to repeated attacks of acid without intervening repair, leading to a net loss of minerals from the surface of enamel.

Orthodontic attachments

They create new stagnation areas. Plaque deposition is greater on the composite resin than on enamel and also more on the gingival side of all bonded brackets. Thus, the introduction of fixed appliances into the oral cavity appears to alter the normal microbial ecology and introduces another variable into the system. The contribution of arch wire ligation materials to plaque accumulation, bacterial colonization and enamel decalcification has also been evaluated.[5]Teeth ligated with elastomeric modules exhibited a greater number of cariogenic microorganisms than teeth ligated with stainless steel ligatures. However, recent studies do not find any change in the number of microorganisms around the brackets ligated with either ligature wire or elastomeric modules.[6,7]

It is evident from various studies that the resting salivary flow increases during fixed orthodontic therapy.[8] Since the salivary pH and buffering capacity increases with the increase rate of salivary flow, it offsets the demineralization tendency that arise during fixed orthodontic treatment. This could be the reason why in some patients there are less white spot lesions around orthodontic attachments despite plaque accumulation in those regions.

It seems justified that at the outset of fixed orthodontic treatment, each patient prone to enamel demineralization is to be thoroughly assessed. We recommend a range of factors to be kept in mind at the time of screening all risky patients. These factors include; History of past enamel caries, caries activity tests, plaque score, caries incidence over the past year, evaluation of rate of salivary flow, dietary habits, fluoridated or non-fluoridated community.

Prevention & Treatment of white spot lesions

White spot lesions can be prevented by either eliminating the plaque deposition by improving patient oral hygiene or by augmenting the enamel resistance by using topical fluoride. At times, maintenance of optimum oral hygiene is frequently inadequate. Therefore fluoride is used as a principal ingredient for the prevention of enamel demineralization. Fluoride not only inhibits the development of white spots, it also reduces the size of the white spots.[9] Topical fluoride fortifies enamel remineralization following orthodontic treatment. It has been documented that a high fluoride concentration in the enamel is less important than a moderate increase in fluoride concentration in oral fluid.10 For maximum caries inhibition, continu-

ous presence of fluoride, even at low concentrations, in saliva and plaque fluid is the need of the hour. Proper oral hygiene maintenance, combined with daily home use of topical fluoride, is found to significantly reduce enamel decalcification.

Daily mouthrinse with sodium fluoride (.05% or 0.2%) and/or weekly with acidulated phosphate fluoride (1.2%) rinse have been found to reduce the incidence of enamel demineralization during active fixed orthodontic treatment. Use of stannous fluoride gels (0.4%), during active orthodontic treatment decreases enamel decalcification.

Routine use of fluoride toothpaste is preferred for orthodontic patients. Toothpastes containing sodium fluoride are found most effective against the development of white spot lesions. Fluor Protector, a polyurethane varnish containing 0.7% diflurosilane was found to decrease white lesion if used under the molar bands.[11] Recently, chlorohexidine varnish was also suggested for reducing plaque accumulation and enamel decalcification.[12]

Pit and fissure sealant: Frazier et. al. applied light cured pit and fissure sealants on the labial enamel surface adjacent to the bonded orthodontic attachment and found it satisfactory in preventing enamel demineralization without patient compliance.[13] However, the main hitch of placing sealants in a patient's mouth is that it is very technique sensitive, and mechanical and chemical breach in the sealant barrier may ensue enamel decalcification directly under its inner surface.

Fluoride in luting cement: Glass ionomer cements have been shown to decrease enamel decalcification when compared with zinc phosphate and zinc polyacrylate cements. A study revealed that fluoride releasing cements like zinc polycarboxylate and resin modified glass ionomer cement demonstrated less enamel demineralization than the zinc phosphate cement.[14] Millett et. al. also found less severe enamel decalcification around orthodontic brackets with glass ionomer cement when compared with composite resins.1[5]

Fluoride in bonding agents: Bonding agents containing fluoride have the potential for decreasing enamel decalcification. Fewer white spot lesions were found with the fluoride containing composite as compared to conventional bonding agents. Glass ionomer cement used for bonding brackets significantly reduced enamel demineralization around orthodontic brackets.[16] Recently, it was suggested that resin-modified glass ionomer cement efficiently reduced enamel demineralization around the bonded brackets." It was concluded that the fluoride release is greater with resin modified glass ionomer cements and also over prolonged period, as compared to the fluoride containing composite resins .[18]

Fluorides in elastomeric modules: Many investigations advocate that fluoride-releasing elastomeric modules are effective in reducing plaque accumulation and enamel decalcification around the brackets.[19]However, recently Benson, Shah and Campbell concluded that fluoridated elastomers had no effect on the quantity of disclosed plaque around orthodontic brackets .[2°] Joseph, Grobler and Rossouw reported that fluoride release from a fluoride containing elastic chain was high for the first week and decreased significantly after that. It was further found that the fluoride release, in vivo, is about 7 times more as compared to in vitro during one week period.[21] In the presence of fluoridated toothpaste and mouthrinse the fluoride release is also significantly more. Thus, fluoridated elastomers may imbibe fluoride from the oral environment.[21]

Argon laser: Role of argon laser in the prevention of enamel decalcification has been suggested. It alters the crystalline structure of the enamel. Blankenau et. al. for the first time found an average of 29.1% reduction in the depth of enamel decalcification with argon laser irradiation.[22] Many other studies have also revealed significant reduction in the lesion depth after argon laser irradiation of ename1.[23] Thus, it should be considered as an effective method in reducing enamel decalcification during orthodontic treatment.

Mechanical plaque control: Since plaque is the primary cause of demineralization, thorough mechanical control is of paramount importance. It is found that tooth brushing is the most practical and acceptable method. Proper method of tooth brushing during fixed orthodontic treatment has been recommended. A special type of orthodontic toothbrush has been suggested to patients with fixed orthodontic appliances. Use of disclosing solutions or tablets is also helpful for self monitoring of oral hygiene. Use of a power toothbrush

or daily water irrigation in combination with manual tooth brushing may be a more effective method than manual tooth brushing alone. Bracket attachment by direct bonding exposes the proximal surfaces to enamel demineralization because of the difficulty in accessing the areas under the archwires. Flossing is the best choice in interproximal cleaning. A floss threader can be used for threading the floss under the main archwire. A soft rubber interdental stimulator or brush can be helpful in massaging and cleaning the interproximal areas respectively.

CONCLUSION

White spot lesions are the common complications of fixed orthodontic treatment. It is the responsibility of an orthodontist to minimize the risk of every patient having decalcification as a consequence of orthodontic treatment. A protocol of excellent oral hygiene practice, during fixed orthodontic treatment, must be explained and executed. Patients must be given a vigorous fluoride supplement in the form of mouth rinsing in preventing and eliminating enamel decalcification during and after fixed orthodontic treatment.

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