ISSN 2229-5518

Iatrogenic effects of Orthodontic treatment – Review on white spot lesions

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Abstract— Demineralization is an inevitable side effect associated with fixed appliance orthodontic treatment, especially associated with poor oral hygiene. Fixed orthodontic appliances create several retentive areas for the accumulation of bacterial plaque. The acidic byproducts of these bacteria are responsible for the subsequent enamel demineralization and formation of white spot lesions (WSL), causing caries therefore leading to poor esthetics, patient dissatisfaction and legal complications. This highlights the need for assessing the saliva, oral hygiene status and caries rate before beginning of treatment and initiating preventive measures. Orthodontists must take up active responsibility to educate the patients about the importance of maintaining good dietary compliance and excellent oral hygiene regime. Depending on the oral environment, WSL can develop into cavities, stay stable for a long time, or heal to a certain extent. Thus, the prevention of WSL is crucial to prevent tooth decay as well as minimize tooth discoloration that could compromise the treatment results.

Index Terms— Iatrogenic effects, White spot lesion, Incidence, Orthodontic Treatment, Duration, Oral hygiene, Etiology, Prevention, Fixed Appliances, Demineralization.

1 INTRODUCTION

White spot lesion (WSL) is a common iatrogenic effect seen in patients undergoing orthodontic treatment with fixed appliances (Fig.1).[1,2] Individuals with malocclusions often have many plaque retention sites due to the irregularities of their teeth (Fig.2). Orthodontic treatment with fixed appliances and complex loop designs further increases the risk for development of WSL due to the creation of additional retention sites on surfaces generally not susceptible to caries.[2] Hence a strong co-relation exists between oral hygiene and caries incidence in orthodontic patients as compared to in non orthodontic individuals.[3] Despite intensive efforts to educate patients about effective oral hygiene procedures, WSL associated with fixed orthodontic appliances remains a significant clinical problem (Fig.3). This clinical problem has increased since the advent of directly bonded orthodontic brackets.[4] Appearance of these spots after the completion of orthodontic treatment can lead to patient dissatisfaction and legal complication.[5] The formation of WSL after completion of orthodontic therapy is discouraging to a speciality whose goal is to improve esthetics in the dento-facial region. Orthodontists should be proactive and take active responsibility to prevent the development of WSL by educating their patients about the importance of maintaining an excellent dietary compliance and oral hygiene regime. Oral hygiene regime must include topical fluoride agents such as fluoridated toothpaste, fluoride-containing mouth rinse, gel and varnish to prevent or minimize the formation of WSL during orthodontic treatment.[6]

DEFINITION

The term white spot lesion was defined as 'the first sign of a caries lesion on enamel that can be detected with the naked eye'.[7]

The WSL has also been defined as 'subsurface enamel porosity from carious demineralization' that presents itself as 'a milky white opacity when located on smooth surfaces'.[8]

CLASSIFICATION OF WHITE LESIONS ON ENAMEL

White discolorations of enamel can be classified as dental fluorosis, opacities, or WSL.[6] A set of criteria has been developed to differentiate between fluorosis and opacities.[9] Fluorosis (Fig.4) is a white/yellowish lesion that is not well defined, blends with normal enamel, and has symmetrical distribution in the mouth. Nonfluoride opacities have a more defined shape, are well differentiated from surrounding enamel, often located in the middle of the tooth, and randomly distributed (Fig.5).

INCIDENCE

Orthodontic patients have significantly more WSL than non-orthodontic patients and these WSL may present esthetic problems years after treatment.[3,10] A recent review of literature[11] showed variations ranging from 2% to 97%, for WSL prevalence associated with orthodontic treatment[3,10,12-17]. This high prevalence is attributed to the difficulties in performing oral hygiene procedures on bonded dental arches along with long-time accumulation and easier retention of bacterial plaque on tooth surfaces around fixed orthodontic appliances.[15,18] The variation in WSL prevalence among studies could be attributed to differences in the number of teeth ex-

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amined, the methods and the standardizations in examinations, the location of the study sample (cultural differences), time era of the study, age at the start of treatment, treatment duration, and materials (banding vs bonding).[19] In general, the prevalence of WSL in patients after orthodontic treatment varies from 15% to 85%[13], with most studies reporting 50% to 70%.[10,15-17,20-22] It is reported that any tooth in the mouth can be affected by the process with the common ones being maxillary lateral incisors, maxillary canines, and mandibular premolars.[15] The incidence was highest in the labio-gingival area of the maxillary lateral incisors (Fig.6) and lowest in the maxillary posterior segment. The reported incidence and prevalence of WSL between males and females have been found to be inconclusive.[10,11,14] No significant differences between the right and left sides of the maxilla and mandible were noted.[10,14]

MECHANISM OF FORMATION OF WSL

WSL can occur on any tooth surface in the oral cavity where the plaque is allowed to develop and remain for a period of time (Fig.7). The naturally occurring self cleansing mechanisms of the oral musculature and saliva are limited by the irregular surface of brackets, bands, and wires.[23] The composition of the bacterial flora of the plaque shows a rapid shift following the placement of orthodontic appliances. Patients undergoing treatment with fixed orthodontic appliances have a rapid increase in the volume of dental plaque (with a lower pH) than that in non-orthodontic patients.[24,25] The levels of acidogenic bacteria, especially Streptococcus mutans and lactobacillus, are significantly elevated.[26] Both *S.* mutans and lactobacilli are often associated with caries development. Streptococcus mutans colonize over the retentive areas of orthodontic appliances and surrounding enamel surfaces. Lactobacillus is responsible for the progression of the carious lesion. Their presence in large numbers is indicative of the necessary condition for dental caries to exist.[27] However, the association between caries and bacteria is not straightforward. The prediction of caries development based on bacterial counts is uncertain and of minor clinical significance.[28] S. mutans and lactobacilli produce organic acids in the presence of fermentable carbohydrates and this is responsible for lowering the pH. Sucrose plays an important role in plaque formation inducing the formation of a cariogenic plaque.[29] There is a direct relationship between plaque pH and total plaque fluoride. Total plaque fluoride levels are low in areas of low pH. The lowest pH (as low as 4) during resting and fermenting conditions was observed in the plaque of the bonded upper incisors.[30] After bonding, resting pH is lowered. In the patient with good oral hygiene, fluoride is able to prevent lesions to develop by increasing remineralization and inhibiting demineralization. With poor oral hygiene, plaque builds up around the appliance and the resting pH may reach the limit of the fluoride effect at pH 4.5. During an acid attack, caries and even erosions develop.[29] Carious decalcification occurs when the pH drops below the threshold for remineralization and creates an alteration in the appearance of the enamel surface which is visualized as WSL.[25,31] Such lesions have been clinically noticed within a short span of 4 weeks[2]. If these are not treated, they progress to a cavitated carious lesion.[32] WSL makes the affected area softer than the surrounding sound enamel, making the tooth more prone to caries[33]. There is about 10% reduction in the mineral content of enamel in these incipient carious lesions. This leads to their increased abrasion in vivo.[34] This makes the affected teeth more susceptible to enamel loss while debonding.[35] Fast developing white spots may remineralize almost completely within a few weeks of the removal of the cariogenic challenge. However, lesions that develop slowly take a longer period to remineralize.[36] Micro-leakage around orthodontic brackets can be another cause for the formation of WSL(Fig.8). [37] The teeth expand and contract when they are heated and cooled by the ingestion of hot or cold foods[38]. The linear thermal coefficient of expansion of enamel, ceramic / metal brackets and the adhesive systems do not match.[39] This repeated expansion and contraction at different coefficients results in fluids being sucked in and pushed out at the margins of the bracket. In comparison with ceramic brackets, the metal brackets are associated with more micro-leakage (Fig.9).[40] Metal brackets contract and expand more than ceramic brackets, enamel, or the adhesive systems, producing microgaps between the bracket and the adhesive system causing leakage of oral fluids and bacteria beneath the brackets, leading to the formation of WSL.[41]

RISK FACTORS FOR WSL

Formation of WSL is primarily due to the subsurface demineralization resulting in porosities and a change in the optical properties. If the surface of porous enamel remains intact, there is a possibility of arrest / remineralization of the lesion due to the buffering action of the saliva. If the pH of plaque remains low for a prolonged period of time, the environment becomes conducive for long periods of demineralization with short periods of remineralization, resulting if frank carious lesions. Risk factors for the development of incipient caries during orthodontic treatment are young age (preadolescents), number of poor oral hygiene citations during treatment, unfavorable clinical outcome score, white ethnic group, and inadequate oral hygiene at the initial pretreatment examination.[42]

Factors such as the patient's medical history, dental history, medication history, diet; salivary flow rate, levels of calcium, phosphate, and bicarbonate in saliva, fluoride levels and genetic susceptibility also play an important role.[23,43,44] There is a poor correlation between length of treatment time and the incidence of number of white spot formations.[14]

PREVENTION OF WSL

Studies have shown that decalcification is a significant risk during fixed orthodontic treatment.[3,10,14,1745] Current evidence suggests that topical fluoride treatment (TFT) is beneficial in preventing the development of WSL during orthodontic treatment.[46] When topical fluoride is applied on the tooth surface (enamel / dentin), a calcium fluoride-like material (CaF2) builds up in plaque, or in incipient lesions which acts as a reservoir and releases fluoride ions when the pH is lowered during a caries attack.[47] Implementing a good oral hygiene regimen including proper tooth brushing with a fluoridated dentifrice is the most important prophylactic

measure to prevent the occurrence of WSL in orthodontic patients. Fluoride concentrations of less than 0.05% are beneficial in reduction of the carious lesions.[48] Evidence suggests that reduced demineralization and enhanced remineralization can occur with a toothpaste containing 5000ppm fluoride.[49,50]

When fluoride ions are incorporated into the surface of enamel, it forms a fluoroapatite crystal structure that has lower solubility in the oral environment compared with hydroxyapatite. Fluoroapatite helps in reducing tooth decay by remineralization of small decalcified areas and reduction in the formation of new lesions.[51] In addition, stannous fluoride may have a plaque-inhibiting effect by interfering with the adsorption of plaque bacteria to the enamel surface.[52,53] Atoms of tin in stannous products block the passage of sucrose into bacterial cells and thus inhibit acid production. The use of a fluoridated antiplaque dentifrice may reduce enamel demineralization around brackets more than the use of a fluoridated dentifrice alone.[54] Enamel dissolution occurs rapidly around orthodontic brackets even during regular use of a fluoride dentifrice[31]. Thus, supplemental sources of fluoride are suggested.

Fluoridated mouth rinses containing 0.05% sodium fluoride used daily have been shown to significantly reduce lesion formation beneath bands. Chemical agents such as chlorhexidine or benzydamine used in the form of mouth rinses or oral sprays are useful adjuncts in plaque and inflammation control.[55] These mouth rinses have been combined with antibacterial agents such as chlorhexidene, triclosan, or zinc to improve their cariostatic effect.[33] When patients have been noncompliant with other oral hygiene regimens, chlorhexidine mouthwashes might be beneficial in preventing white spot caries lesions as an intensive, short-term regimen. Chlorhexidine mouthwash used as a complement to fluoride therapy has demonstrated demineralization-inhibiting tendencies in patients with fixed orthodontic appliances.[56] The main goal of antimicrobial therapy is to achieve a shift from an ecologically unfavorable to an ecologically favorable biofilm.[57] Patients are instructed to use chlorhexidine rinse (available in nonalcohol formulations for patients with xerostomia or saliva dysfunction) for 30 seconds once a day, preferably before bedtime, because saliva flow diminishes overnight and the concentration of the drug in the oral cavity remains high until morning.[58] A 14-day regimen is usually recommended.[58] While these products provide the patient with increased caries protection, patient compliance is mandatory. A fluoride mouth rinse will work best if it is used regularly by the patient. Studies have showed that less than 15% of orthodontic patients rinsed daily as instructed but patients who were more compliant had fewer WSL.[15]

An in-office application of a high concentration of fluoride in the form of a varnish can be beneficial for the less compliant patients. It eliminates the need for patient cooperation that is required with fluoride rinses. The American Dental Association's Council on Scientific Affairs recommends application of 'in-office fluoride varnish at six-month intervals for moderate and high-risk patients'. Although varnish application is associated with the temporary discoloration of the teeth and gingival tissue, it has been reported that the application of a fluoride varnish resulted in a 44.3% reduction in enamel demineralization in orthodontic patients.[59] Acid-resistant coatings of calcium fluoride or titanium fluoride on the enamel surface and the use of fluoride in combination with different antimicrobials have been suggested to improve the cariostatic effect of fluoride at low pH.[60] Varnish forms of the other antibacterial solutions such as benzydamine, triclosan, and xylitol could be helpful for suppressing levels of oral mutans or the other microbes for long periods, when used before the placement of fixed orthodontic appliances. In contrast to one-time topical application in high doses, a long-term, low-dose fluoride availability might increase the caries-resistant fluorapatite concentration in enamel, helping the prevention and reduction of demineralization.[48,51]

Unfortunately, preventive and chemoprophylactic products, such as high-fluoride toothpaste or gel, fluoride varnish, and chlorhexidine rinse, gel, or varnish, are rarely prescribed by orthodontists. It was reported that 95% of orthodontists provide oral hygiene instructions, while only 52% prescribe fluoride mouth rinse[61].

Xylitol, a polyol (a type of carbohydrate) that does not act as a metabolizing substrate for Streptococcus mutans, can be used as a low-calorie sugar substitute to prevent caries.[62] Xylitol has been used as a caries preventive agent in form of gum and mints. It is noncariogenic and appears to have antimicrobial properties that help to inhibit S mutans attachment to the teeth. The salivary pH remains stable as there is no metabolism by bacteria, and the environment does not favor acidogenic bacteria.[63] Additionally, the consumption of chewing gum and mints has been demonstrated to result in increased production of stimulated saliva containing more calcium and phosphate ionic concentrations when compared with non-stimulated saliva.[63] The systematic use of xylitol chewing gum can significantly reduce the risk of caries compared with gums that contain sorbitol and sucrose.[64] Chewing xylitol gum thrice a day for 5 minutes has shown positive results.[65] However, long-term clinical trials with a standardized methodology are needed. Moderate and high-risk adult patients are recommended to chew 2 pieces of xylitol gum for 10minutes at least, 3 to 5 times a day.[66] Therapeutically, 6gm/day of xylitol is recommended for adults.[67] However, xylitol can cause diarrhea if the recommended doses are exceeded.[63]

Enamel demineralization might be prevented by the application of products containing casein phosphopeptides-amorphous calcium phosphate (CPP-ACP). CPP-ACP is a nanocluster that binds calcium and phosphate ions in an amorphous form. CPP-ACP has been shown to adhere to the bacterial wall of microorganisms and tooth surfaces.[68,69] When an intraoral acid attack occurs, the calcium and phosphate ions are released to produce a supersaturated concentration of ions in the saliva, which then precipitates a calcium-phosphate compound onto the exposed tooth surface.[69] However, there is insufficient clinical trial evidence to make a recommendation regarding its long-term effectiveness.[63]

The prolonged duration of orthodontic treatment places the patient at an increased caries risk. This risk can be

minimized by a continuous fluoride release from the bonding system around the bracket base. The introduction of fluoride-releasing adhesive systems, resin composites, and glass ionomer cements for bracket bonding offered a means of fluoride delivery adjacent to bracket-enamel interface independent of patient cooperation. However, the ability of these materials to reduce decalcification clinically remains equivocal.[70] Glass ionomer cements(GIC) do not provide complete caries protection under loose bands or in areas of missing/dissolved cement.[71] However the shear bond strength (SBS) of GIC was not adequate for bonding brackets. In an attempt to increase the bond strength of GIC's, resin particles were added to their formulation to create resin modified Glass Ionomer (RMGI) bonding systems. These adhesives release fluoride like conventional GIC's and can also be used successfully to bond orthodontic brackets because of their relatively higher SBS.[72-83] Additionally, in vivo studies have shown no significant differences in bracket failure rates between the RMGI's and composite adhesives.[84] Because of the recent improvements in the fluoride-releasing capabilities and the SBS of RMGI, it has been suggested that these adhesives should be used more widely in bonding orthodontic brackets in the future.[85] However a recent study concluded that it is impossible to make recommendations on the use of fluoride-containing orthodontic adhesives during fixed orthodontic treatment.[86] The authors found sufficient evidence to suggest that GIC is more effective than composite resin in preventing white spot formation, but further research is required to determine the effectiveness of the various fluoride-containing orthodontic adhesives.

A fluoride releasing antibacterial bonding agent has been developed by combining the physical advantages of dental adhesive technology and antibacterial effect. The antibacterial activity of 12-methacryloyloxydodecyl-pyridinium bromide (MDPB) incorporated in the antibacterial adhesive systems demonstrated inhibition of caries formation, especially along the enamel margins.[87] Incorporating MDPB into self-etching primer and adhesive resin has demonstrated in vitro antibacterial activity, bonding ability, cytotoxicity, and pulpal response. It was confirmed that MDPB-containing primer has got antibacterial effects in vivo when used in animal models.[88,89]

Other fluoride-release mechanisms like fluoride- releasing elastic ligature and power chains have been tried. Research has shown that fluoride-releasing elastomeric ligatures were effective in reducing plaque accumulation and decalcification around the brackets.[16,90,91] However, later investigations reported that fluoride-releasing elastomeric ligatures did not reduce the amount disclosed plaque around the brackets.[92] Research has shown that the fluoride release was high in the first week but decreased significantly in the subsequent weeks.[93]

Finally, use of argon laser to cure composite resins has demonstrated its ability to alter the enamel, rendering it less susceptible to demineralization. It was also shown that combining laser irradiation with fluoride treatment can have a synergistic effect on acid resistance preventing formation of WSL and dental caries.[94] Research has shown that exposing the teeth to an argon laser for 60 seconds at the time of appliance placement reduced lesion depth by 91.4% and lesion area by 94.6% when compared with untreated control teeth.[95]

Clinical Significance

The authors recommend the following measures to prevent WSL in orthodontic patients:

1. Educate and motivate the patients at every visit to maintain optimal oral hygiene around the appliances to obtain the full effect of fluoride.[29]
2. Daily brushing with fluoride toothpaste (1500ppm or more) twice a day.[56] Use of interdental brushes to remove plaque around the brackets.
3. Daily use of a fluoride mouth rinse (0.05% NaF).[29,31,55]
4. Performing oral prophylaxis (scaling) and reinforcing instructions at each appointment in non-compliant patients.
5. Use of chlorhexidine mouth rinse at night for 2 weeks in patients with poor oral hygiene.
6. Use of topical fluoride in the form of solutions, varnishes, or gels around the brackets of non-compliant / high risk patients at 6 months interval.
7. Cementing the bands with good quality GIC.

CONCLUSION

WSL on the enamel surface adjacent to fixed orthodontic appliances is an important and prevalent iatrogenic effect of orthodontic therapy. The components of the appliance and the bonding materials create stagnation areas for plaque accumulation and bacterial colonization. The subsequent acid production by the acidogenic bacteria leads to enamel decalcification. The orthodontist must educate the patient regarding the importance of maintaining good oral hygiene and dietary regime. Fluoride is the most important agent to prevent decalcification and restrict lesions from progressing. Oral hygiene regime must include topical fluoride agents such as fluoridated toothpaste, fluoride-containing mouth rinse, gel and varnish to prevent or minimize the formation of WSL during orthodontic treatment.

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FIGURES FOR THE ARTICLE (1 Figure per page)

Iatrogenic effects of Orthodontic treatment – Review on white spot lesions

Fig.1 Cervical carious lesions observed after orthodontic treatment

Top

Fig.2 Bacterial plaque accumulations in areas of crowding in natural dentition.

Top

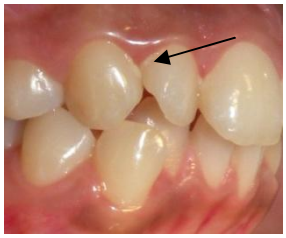

Fig.3 Inaccessible areas created due to orthodontic appliances

Top

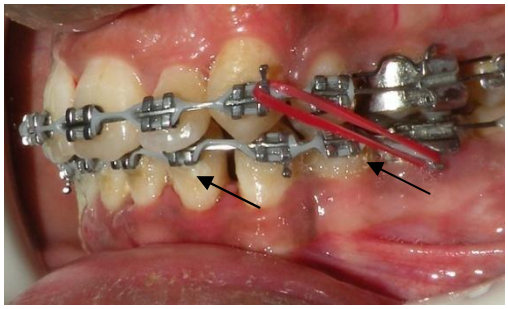

Fig.4 Fluorosis showing white and yellowish-brown areas

Top

Fig.5 Non specific white opacities on the distal tip of the left central incisor

Top

Fig.6 White spot lesions with lateral incisor

Top

Fig.7a,b Bacterial plaque accumulations in cervical region of canine

Top 7a

Top7b

Fig.8 Arrows showing micro-leakage around the brackets

Top

Fig.9 Least amount of micro-leakage around ceramic brackets

Top