Indian Journal of Health Sciences and Care
Vol. 3, No. 2, August, 2016, pp- 85-95
DOI: 10.5958/2394-2800.2016.00016.X

Review Article

White Spot Lesions: Fixed Orthodontic Treatment Scars - A Review

Ashish Dabas[1]*, Nupur Dabas[2], Maninder Singh Sidhu[3], Mona Prabhakar[4], Seema Grover[4], Sandeep Kumar[5]

[1]Reader, [3]Professor & Head, [4]Professor, [5]Senior Lecturer, Department of Orthodontics and Dentofacial Orthopedics, Faculty of Dental Sciences, SGT University, Budhera, Gurgaon
[2]Reader, Department of Prosthodontics, Faculty of Dental Sciences, SGT University, Budhera, Gurgaon

*Corresponding author email id: dabasashish@yahoo.co.in

ABSTRACT

White spot lesions (WSLs) are one of the common iatrogenic effects seen in patients undergoing orthodontic treatment with fixed appliances. Fixed orthodontic appliances act as food traps leading to prolonged plaque accumulation. The acidic by products of the bacteria in plaque are responsible for the subsequent enamel demineralization and formation of white spot lesions. These orthodontic scars lead to poor esthetics, patient dissatisfaction and legal complications. This concern raises the need for assessing the saliva, oral hygiene status and caries rate before beginning of treatment and initiating preventive measures. Orthodontists must take up the active responsibility to educate patients about the importance of maintaining good dietary compliance and excellent oral hygiene regime. A review of literature suggests that topical fluoride agents such as fluoridated toothpaste, fluoride-containing mouth rinse, gel and varnish prevent or minimize the formation of WSLs during orthodontic treatment.

Keywords: White spot lesions (WSLs), Plaque, Demineralization, Prevention

INTRODUCTION

White spot lesions (WSLs) are one of the common iatrogenic effects seen in patients undergoing orthodontic treatment with fixed appliances. These orthodontic scars develop into caries thereby leading to poor esthetics, patient dissatisfaction and legal complications. WSLs develop in association with brackets, bands, arch wires, ligatures and other orthodontic devices that complicate conventional oral hygiene measures, leading to prolonged plaque accumulation. The acidic byproducts of the bacteria in plaque are responsible for the subsequent enamel demineralization and formation of white spot lesions. This concern raises the need for assessing the saliva, oral hygiene status and caries rate before beginning of treatment and initiating preventive measures. Orthodontists must take up the active responsibility to educate patients about the importance of maintaining good dietary compliance and excellent 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 WSLs during orthodontic treatment[1,2].

Despite intensive efforts to educate patients about effective oral hygiene procedures, WSL associated with fixed orthodontic appliances remains a significant clinical problem. Tufekci *et al.*[3] concluded in his clinical study that a sharp increase in the number of WSLs occurred during the first 6 months of treatment that continued to rise at a slower rate to 12 months, thus in initial months of the treatment critical evaluation of oral hygiene is recommended.

DEFINITION

The term white spot lesion (WSL) was defined as “the first sign of a caries lesion on enamel that can be detected with the naked eye”. 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[2].

DIFFERENTIAL DIAGNOSIS

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

INCIDENCE AND PREVALENCE

Orthodontic patients have significantly more WSLs than non- orthodontic patients and these WSLs may present esthetic problems years after treatment. A recent review of literature showed variations ranging from 2% to 97%, for WSL prevalence associated with orthodontic treatment. 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[5,6]. The variation in WSL prevalence among studies could be attributed to differences in the number of teeth examined, 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). In general, the prevalence of WSL in patients after orthodontic treatment varies from 15% to 85%, with most studies reporting 50% to 70%. 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. The incidence was highest in the labio-gingival area of the maxillary lateral incisors and lowest in the maxillary posterior segment. The reported incidence and prevalence of WSL between males and females have been found to be inconclusive. No significant differences between the right and left sides of the maxilla and mandible were noted.

Studies have shown that white spot lesions can take only one month to develop[1,7,8]. A clinical study[9] reported the prevalence at 50%, while recent investigations put the incidence of white spot lesions in the orthodontic populations studied at 73-95%[10,11]. Orthodontists and patients will notice these lesions after removal of the fixed appliances, especially since the white spots tend to form in the maxillary esthetic zone[9,12]. While some studies have reported a decrease in the display of white spot lesions over time (post orthodontic treatment), these unesthetic spots tend to remain unless they are resolved with more aggressive treatment, such as minimally invasive or even full restorative dentistry[13,14].

ETIOLOGY

The appearance of WSL on the enamel surface during fixed orthodontic treatment is due to a multiplicity of factors. Co-existence of the four factors namely, bacterial plaque, fermentable carbohydrates, a susceptible tooth surface and a sufficient period of time are necessary for WSL to develop.

Microbial factors

The presence of *Streptococcus mutans* and *Lactobacillus* and new sites of plaque appearance on the enamel surrounding the orthodontic attachments is common in patients undergoing fixed appliance therapy. This may be further influenced by the duration of the orthodontic treatment and the number of orthodontic attachments[15].

Salivary factors

The amount and rate of enamel demineralization and the likelihood of enamel remineralization is influenced by salivary factors such as pH, rate of flow and buffer capacity. Saliva also acts as a vehicle for the delivery of fluoride ions to the enamel and plaque. Adequate flows of the saliva helps in physical cleansing of carbohydrates from tooth surfaces, maintains its buffering capacity and anti-microbial activities. Therefore, adequate flow of the saliva is considered as an important factor for prevention and management of enamel demineralization. Tooth surfaces that are more exposed to dietary carbohydrate with less exposure to saliva are common sites for demineralization to occur (maxillary anterior teeth). The lingual surface of the lower incisors where salivary flow is adequate is often the site of calculus formation, indicating mineralization. Enamel demineralization is caused by the low pH of the plaque when not compensated by the buffering capacity of the saliva.

Oral hygiene

Fixed orthodontic appliances make tooth cleaning more difficult and also restrict the self-cleansing action of the tongue, lips and cheek to remove food debris from the tooth surface. Therefore, accumulated food debris, particularly of the cariogenic bacteria is seen in patients undergoing active orthodontic treatment

Diet

As the frequency of carbohydrate intake increases, the enamel surface may be exposed to overlapping episodes of acid without intervening repair, resulting in a net loss of minerals over time making it prone for enamel demineralization.

Alteration of the oral environment

Insertion of the fixed orthodontic appliance into the oral cavity creates new stagnation areas that in the presence of carbohydrate and the reduced access by saliva encourages the colonization of *S. mutans* and *Lactobacilli*. It has been found that plaque deposition is greater on resin bonded material than on enamel. Even plaque deposition is greater on gingival side of brackets. Teeth ligated with elastomeric rings exhibited a greater number of cariogenic microorganisms than the teeth ligated with stainless steel ligature wires. However, few studies failed to find an alteration in the number of *S. mutans* around the orthodontic brackets ligated with either ligature wire or elastomeric rings[16,17].

Studies have shown that the resting salivary flow increases during fixed orthodontic treatment in few patients. Since the salivary pH and buffering capacity increases with the increase rate of salivary flow, it counteracts the demineralization tendency that arises during fixed orthodontic treatment. This could be the reason why in some patients there are little white spots around orthodontic appliances despite moderate plaque accumulation[18].

Thus, at the beginning of fixed orthodontic treatment, an assessment of patient's susceptibility to enamel demineralization seems logical. Many authors[19-21] recommended a range of factors to be examined in order to identify patients at risk of developing demineralization. These factors include assessment of salivary flow rate, history of past enamel caries, caries incidence over the past year, plaque scores, caries activity tests, dietary pattern and resident as a part of fluoridated or non-fluoridated communities.

Mechanism of Formation of White Spot Lesions [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 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 in frank carious lesions. Risk factors[22] 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. 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.

There is a poor correlation between length of treatment time and the incidence of number of white spot formations.

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. The naturally occurring self cleansing mechanisms of the oral musculature and saliva are limited by the irregular surface of brackets, bands, and wires. 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[23-27] have a rapid increase in the volume of dental plaque (with a lower pH) than that in non-orthodontic patients. The levels of acidogenic bacteria, especially Streptococcus *mutans* and lactobacillus, are significantly elevated. 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. However, the association between caries and bacteria is not straight forward. The prediction of caries development based on bacterial counts is uncertain and of minor clinical significance. S. *mutans* and lactobacilli produce organic acids in the presence of fermentable carbohydrates and this is responsible for lowering the pH[28].

Sucrose plays an important role in plaque formation inducing the formation of a cariogenic plaque. 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. 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. 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.

Such lesions have been clinically noticed within a short span of 4 weeks. If these are not treated, they progress to a cavitated carious lesion. WSL makes the affected area softer than the surrounding sound enamel, making the tooth more prone to caries. There is about 10% reduction in the mineral content of enamel in these incipient carious lesions. This leads to their increased abrasion *in vivo*. This makes the affected teeth more susceptible to enamel loss while debonding. Fast developing white spots may re-mineralize almost completely within a few weeks of the removal of the cariogenic challenge. However, lesions that develop slowly take a longer period to re-mineralize. Micro-leakage around orthodontic brackets can be another cause for the formation of WSL. The teeth expand and contract when they are heated and cooled by the ingestion of hot or cold foods. The linear thermal coefficient of expansion of enamel, ceramic or metal brackets and the adhesive systems do not match. 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. 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.

PREVENTION AND MANAGEMENT

The risk of enamel demineralization during fixed orthodontic treatment can be prevented by:

1. Improving oral hygiene using mechanical plaque control methods
2. By enhancing the enamel resistance to the microbial acid by using topical fluoride
3. By additional methods using different mechanisms.

Mechanical plaque control

Mechanical plaque control by proper tooth brushing is of paramount importance. A modification of the standard toothbrush, use of disclosing solutions, use of floss can help our patients in attaining good oral hygiene. Use of a power toothbrush or daily water irrigation in combination with manual tooth brushing may be a more effective method in reducing plaque accumulation than manual tooth brushing alone. Bracket attachment by direct bonding exposes the proximal surfaces to enamel demineralization because of the difficulty in maintaining oral hygiene with arch wires in place. Dental flossing has proved helpful in interproximal cleaning. A floss threader can be used for threading the floss under the main arch wire. A soft rubber interdental stimulator can also be helpful in cleaning and massaging the interproximal areas.

Enhancing enamel resistance using topical fluorides

Fluoride enhances enamel remineralization following orthodontic treatment. The cariostatic effect of topical fluoride is primarily due to calcium fluoride (CaF_2) formation. 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. Proper oral hygiene maintenance, combined with daily use of topical fluoride, is found to significantly reduce enamel decalcification. Home use of topical fluoride agents needs patient compliance. As a result, different non-compliant topical fluoride delivery measures have been implemented to prevent enamel demineralization around orthodontic brackets. When topical fluoride is applied on the tooth surface (enamel/dentin), a CaF_2 like material 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[29]. Different modes in which fluorides has been documented to prevent WSL are as follows:

Fluoride mouth rinse

Daily mouth rinse with sodium fluoride (NaF) (0.05% 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.

After a systematic review, Benson *et al.*[30] recommended that the best method to prevent enamel demineralization during fixed orthodontic treatment is daily use of 0.05% NaF mouth rinse. However, Hirschfield and Johnston[31] advocated the use of an APF mouth rinse to make enamel more resistant to orthodontic induced decalcification. Geiger *et al.*[32] reported 25% reduction in the number of WSL using fluoride rinse. It was also found that following 2 weeks use of NaF mouth rinse, with one rinse per day, fluoride concentration in the saliva increased significantly.

Fluoride gel

Many investigators have tried Stannous fluoride gels (0.4%) during orthodontic treatment and reported decreased enamel decalcification. Currently, Boyd and Chun[33] compared the use of a 1100 ppm fluoride toothpaste alone or together with either a daily 0.05% NaF rinse or a 0.4% stannous fluoride gel applied twice daily by toothbrush. He found that both the gel and rinse provided additional protection against decalcification when compared with toothpaste alone, but neither was superior.

Fluoride toothpaste

The regular use of fluoride toothpaste is a very common recommendation by the orthodontist, but if used alone it is shown to be inefficient inhibiting remineralisation of initial enamel lesions[34].

Fluoride varnish

Duraphat (5%NaF), fluorprotector (1% difluorosilane and 0.1% F), duraflor (5% NaF) are the commonly used Fluoride varnishes. Azarpazhooh and Maim[35] concluded that over the 3-year follow-up period, application of fluoride varnish every 6 months was the most cost-effective method for high and medium-risk group. He also concluded that the slow release of fluoride was seen for periods of up to 6 months with Durafluor and Duraphat and the greatest release occurred in the first 3 weeks and more gradual release thereafter. On the basis of this observation, he supported the recommendation of twice-yearly application of single-dose preparations. In contrast, some studies advocated that applying every 90 days (tri-monthly) would be sufficient to promote adequate protection. Demito *et al.*[36] found there was the increase of 32% in deminerization in areas where varnish was not applied in comparison with 30-50% reduction in WSL's in areas where Duraphat was applied twice annually. Use of Fluor Protector (polyurethane varnish) decreased WSL formation under molar bands. Recently, chlorhexidine varnish was also suggested for reducing plaque accumulation and enamel decalcification.

Pit and fissure sealant

Light cured pit and fissure sealants applied on the labial enamel surface adjacent to the bonded orthodontic brackets were found to be effective in preventing enamel demineralization without patient compliance. The disadvantage being its technique sensitive and mechanical and chemical breaks in the sealant layer may lead to enamel decalcification under the sealant. According to Benham *et al.*[37]. highly filled flowable composites, which are resistant to micro abrasion, when applied gingival to the bonded brackets significantly reduced the WSL's. On the other hand, in another study, it was found that dual-cured lightly filled bisphenol A glycidyl methacrylate, BIS-GMA fluoride releasing

sealant did not provide added protection to enamel in comparison with control because material might not be resistant to mechanical and chemical wear. According to Salar *et al.*[38] fluoride releasing sealants demonstrated decreased WSL's when compared to conventional sealant, but potency was still lesser than high fluoride release glass ionomer cement (GIC). As per Soliman *et al.* [39], the fluoride-containing sealant like Pro Seal had the ability to be recharged with fluoride ions when introduced into a foaming solution of acidulated phosphate fluoride acting like a fluoride pump. Discs of the said sealants released fluoride ions in a sustained but significantly decreasing amounts from a high concentration in the 1st week to a low concentration at 17th week, whereas composite sealant neither released fluoride nor were recharge with fluoride solutions.

Fluoride in luting cement

It has been advocated to use cements containing fluoride for banding like GIC. It had been suggested that fluoride releasing cements such as zinc polycarboxylate and resin modified GIC demonstrated less enamel demineralization than the zinc phosphate cement.

Fluoride in bonding agents

Bonding agents containing fluoride have the potential for decreasing enamel decalcification. It was concluded that the fluoride release is greater with resin modified GIC and also over a prolonged period, as compared with the fluoride containing composites. Bonding with GIC showed less WSL in 12 year follow-up in comparison to conventional composite material. However, traditional fluoride releasing cements, glass-ionomer cements and resin-modified GIC have bond strengths that are substantially lower than those of conventional resins. Bioactive glass (BAG) materials have recently been incorporated into the field of dentistry and are surface active materials known to successfully release ions (calcium, phosphate and fluoride ions) in simulated body fluid. Manfred *et al.*[40] found that BAG-Bond adhesives outperformed conventional composites at maintaining superficial enamel hardness surrounding orthodontic brackets and released reservoir ions that decreased the chances of WSL around brackets. Thus, these adhesives hold the potential to be biomimetic bonding agents.

Fluorides in elastomers

Many investigations have also suggested that fluoride-releasing elastomeric modules were effective in reducing plaque accumulation and enamel decalcification around the brackets. However, Doherty *et al.*[41], using an in situ model, found that Fluoride-releasing ligatures do not provide a significant anti-cariogenic benefit in patients undergoing orthodontic treatment. However, they might affect the local environment surrounding the bracket.

Use of fluoride containing antibacterial adhesives

The antibacterial activity of 12-methacryloyloxydodecyl-pyridinium bromide incorporated in the antibacterial adhesive systems demonstrated inhibition of caries formation, especially along the enamel margins[42].

ADDITIONAL METHODS TO PREVENT WHITE SPOT LESIONS

Intentional use of Hawthorne effect

Feil *et al.*[43] found that the home care of noncompliant adolescent orthodontic patients with "poor" oral hygiene could be improved through the use of a deception strategy designed to intentionally induce the Hawthorne effect. As per this effect, the thought of participating in and fulfilling the requirements of a study alters subjects' behavior, thereby contributing to improvement in oral health. Compared with the control group; experimental group showed a reduction in plaque scores from 72 to 52% after 3 months, whereas in the control group plaque scores were increased. The Hawthorne effect can have an effect on patients' behavior that lasts as long as 6 months; hence, they suggested to further evaluate its role in controlling WSL in orthodontic patients where treatment will last more than a year.

Essential oil mouth rinse

Essential oil mouth rinse like *Listerine* has recognizable bactericidal activity. According to Tufekci *et al.*[44], adding Listerine to the daily oral hygiene regimen reduces plaque and gingivitis development in orthodontic patients over a 6-month period in comparison with tooth brushing and flossing alone. As fixed orthodontic appliances may cause enamel decalcification because of plaque accumulation around the bracket base, it is recommended that orthodontists instruct their patients to rinse twice daily with 20 ml of Listerine in addition to brushing and flossing.

Argon laser

The mode of action of the argon laser for the prevention of enamel decalcification is by altering the crystalline structure of the enamel has been suggested[45]. Blankenau *et al.*[46]. for the first time found an average of 29.1% reduction in the depth of enamel decalcification with argon laser irradiation. Many other studies[47] have also reported a significant reduction in lesion depth after argon laser irradiation of enamel. Thus, argon laser irradiation can be considered as an effective method in reducing enamel decalcification during the orthodontic treatment.

Use of self-ligating brackets

Buck *et al.*[48], in a randomized clinical study found that most patients bonded with self-ligating brackets had fewer bacteria in plaque than did the teeth bonded with elastomeric ligated brackets both at 1 and 5 weeks after bonding. On contrary, Polat *et al.*[49], found no differences in terms of WSL formation between conventional straight wire and self-ligating brackets and WSL formation depend largely on patients oral hygiene status, not the type of bracket or ligation used. Further research is awaited on this topic before recommending self-ligating brackets over conventional brackets to reduce WSL.

Chewing gums containing xylitol

Xylitol, a polyol (a type of carbohydrate) that does not act as a metabolizing substrate for *S. mutans*, can be used as a low-calorie sugar substitute to prevent caries. It resulted in increased production of stimulated saliva containing more calcium and phosphate ionic concentrations when compared with non-stimulated saliva. Moderate and high-risk adult patients are recommended to chew two pieces of xylitol gum for 10 min at least, 3-5 times a day. Therapeutically, 6 g/day of xylitol is recommended for adults. However, xylitol can cause diarrhea if the recommended doses are exceeded[50,51].

Use of casein phosphopeptides amorphous calcium phosphate

Enamel demineralization might be prevented by the application of products containing Casein Phosphopeptides Amorphous Calcium Phosphate (CPP-ACP)[52]. For many years, it has been known that milk and its derivatives have a tooth protective effect. Ramalingam *et al.*[53] reported that CPP-ACP, which is derived from milk casein, was capable of being absorbed through the enamel surface and could affect the demineralization-remineralization processes. Recently, research has shown that this activity is due to a part of the casein protein called CPP, which carries calcium and phosphate ions 'stuck' to it, in the form of APP. This complex of CPP-ACP is an ideal delivery system for bio-available calcium and phosphate ions. The proposed anti-cariogenic mechanism of CPP-ACP involves the incorporation of the nanocomplexes into dental plaque and onto the tooth surface, thereby acting as a calcium and phosphate reservoir. CPP-ACP has been shown to adhere to the bacterial wall of microorganisms and tooth surfaces. 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. Few studies[54-58] showed that daily applications of the re-mineralizing cream could reverse the severity and visual appearance of post orthodontic WSL more effective than or at least as good as, fluoride toothpaste. The use of CPP-ACP can be more beneficial than fluoride rinse for post orthodontic remineralization. Uysal *et al.*[59] displayed reduced demineralization when ACP was incorporated in the orthodontic composite.

Microabrasion

Microabrasion is an effective treatment for cosmetic improvement of longstanding WSL's. Around 18 % hydrochloric acid was mixed with fine pumice powder to obtain a slurry form. The slurry was agitated into the tooth surface for 30 s and then washed off with an air-water spray. The cycle of microabrasion procedure and washing was repeated 3-4 times on each affected tooth. It was the most beneficial method among the three methods tested-fluoriderinse, CPP-APP with fluoride rinse and microabrasion procedure[60].

Chlorhexidine varnish

Beyth *et al.*[61] provided additional evidence that sustained release chlorhexidine varnish decreases *S. mutans* levels in orthodontic patients with fixed appliances and therefore

might be useful in preventing caries lesions. Kronenberg *et al.*[62] have shown *mutans streptococci* suppression and an enhanced prophylactic effect compared with non-protected teeth. When combining chlohexidine varnish with a fluoride varnish (Fluor Protector), the cariostatic effect was enhanced even further in comparison to ozone, which also has been shown to decrease *S. mutans* count.

Clinical implication

Based on this literature review, the following measures can be suggested to prevent WSL in orthodontic patients[63,64]:

1. Educate and motivate the patients at every visit to maintain optimal oral hygiene around the appliances to obtain the full effect of fluoride.
2. Advise daily brushing with fluoride toothpaste twice daily. Use of interdental brushes to remove plaque around the brackets can be advised.
3. Daily use of a fluoride mouth rinse (0.05% NaF) or essential oil mouth rinse can be prescribed.
4. Performing oral prophylaxis (scaling) when needed and reinforcing instructions at each appointment in non-compliant patients.
5. Use of topical fluoride in the form of varnishes, around the brackets of non-compliant/high risk patients at 6 months interval.
6. Cementing the bands with good quality resin modified glass-ionomer cement and applying fluoride releasing sealants around brackets.
7. CPP-ACP re-mineralizing creams are recommended for prolonged periods post debonding.

FATE OF ENAMEL SCARS

Several studies have reported that demineralization ceases following removal of fixed orthodontic appliances. This could be due to physical removal of the overlying acid-producing plaque, areas of plaque retention i.e., orthodontic attachments thereby improving accessibility to saliva. The demineralized enamel that appears as white spot may disappear either because of surface abrasion or result from a reparative precipitation of mineral deposits once the treatment is over. Fitzpatrick and Way[65] demonstrated that after acid etching, the return to a normal enamel surface was because of a filling-in of material and not because of wearing away of the etch. Some investigations also indicated that it was of an appatitic nature, with only minor amount of impurities. It has been suggested that polishing or abrasion of the dull and irregular enamel surface results in the exposure of the more tightly packed enamel crystals, which give a harder and glossier clinical appearance. It is important that remineralization is significantly enhanced by fluoride. Therefore, routine fluoride mouth rinse might serve a valuable purpose also in the time period after debonding. The need to prescribe an additional topical fluoride will be dependent upon the needs of the individual patient and clinical judgment. However, the appearance of white lesions that persists after orthodontic treatment can be improved by a hydrochloric acid-pumice micro-abrasion technique. Recently the studies of the effects of CPP-ACP have so far shown promising dose-related increases in enamel remineralization within already demineralized enamel lesions. The ability of CPP-ACP to prevent WSL formation has not as yet, been proven

CONCLUSION

WSL are one of the common complications of fixed orthodontic treatment. It is the responsibility of an orthodontist to minimize the risk of the patient having decalcification as a consequence of orthodontic treatment by educating and motivating the patients for excellent oral hygiene practice. Different regimens suggested by different authors can be prescribed to the patients to control WSL along with topical fluoride application.

REFERENCES

1. Ogaard B, Rolla G, ArendsJ. Orthodontic appliances and enamel demineralization. Part 1 Lesion development. Am J Orthod Dentofacial Orthop 1988; 94:68-73.
2. Summitt JB, Robbins JW, Schwartz RS. Fundamentals of Operative Dentistry.
3. Tufekci E, Dixon JS, Gunsolley JC, Lindauer SJ. Prevalence of white spot lesions during orthodontic treatment with fixed appliances. The Angle Orthodontist: 2011; 81(2):206-210.
4. Russsell AL. The differential diagnosis of fluoride and nonfluoride enamel opacities. J Public Health Dent 1961; 21:143-146.

5. Zachrisson BU, Zachrisson S. Caries incidence and oral hygiene during orthodontic treatment. Scand J Dent Res 1971; 79:394-401.

6. Ogaard B, Larsson E, Henriksson T, Birkhed D, Bishara SE. Effects of combined application of antimicrobial and fluoride varnishes in orthodontic patients. Am J Orthod Dentofacial Orthop 2001; 120:28-35.

7. White DJ, Featherstone JD. A longitudinal microhardness analysis of fluoride dentifrice effects on lesion progression in vitro. Caries Res 1987; 21(6):502-12.

8. Gorton J, Featherstone JD. *In vivo* inhibition of demineralization around orthodontic brackets. Am J Orthod Dentofacial Orthop 2003; 123(1):10-4.

9. Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. Am J Orthod 1982; 81(2):93-8.

10. Tymofiyeva O, Boldt J, Rottner K, Schmid F, Richter EJ, Jakob PM. High-resolution 3D magnetic resonance imaging and quantification of carious lesions and dental pulp in vivo. MAGMA 2009; 22(6):365-74.

11. Lovrov S, Hertrich K, Hirschfelder U. Enamel Demineralization during Fixed Orthodontic Treatment - Incidence and Correlation to Various Oral-hygiene Parameters. J Orofac Orthop 2007; 68(5):353-63.

12. Banks PA, Richmond S. Enamel sealants: a clinical evaluation of their value during fixed appliance therapy. Eur J Orthod 1994; 16(1):19-25.

13. Ogaard B. Prevalence of white spot lesions in 19-year-olds: A study on untreated and orthodontically treated persons 5 years after treatment. Am J Orthod Dentofacial Orthop 1989; 96:423-7.

14. Artun J, Thylstrup A. A 3-year clinical and SEM study of surface changes of carious enamel lesions after inactivation. Am J Orthod Dentofacial Orthop 1989; 95(4):327-33.

15. Mitchell L. Decalcification during orthodontic treatment with fixed appliances. Br J Orthod 1992; 19:199-205.

16. Lundstrom F, Krasse B. Streptococcus mutans and lactobacilli frequency in orthodontic patients;The effect of chlorhexidine treatments. Eur J Orthod 1987; 9:109-16.

17. Rosenbloom RG, Tinanoff N. Salivary Streptococcus mutans levels in patients before, during, and after orthodontic treatment. Am J Orthod Dentofacial Orthop 1991; 100:35-7.

18. Chang HS, Walsh LJ, Freer TJ. The effect of orthodontic treatment on salivary flow, pH, buffer capacity, and levels of mutans streptococci and lactobacilli. Aust Orthod J 1999; 15:229-34.

19. Bishara SE, Ostby AW. White Spot Lesions: Formation, Prevention and Treatment. Semin Orthod 2008; 14:174-182.

20. Scheie AA, Arneberg P, Krogstad O. Effect of orthodontic treatment on prevalence of Streptococcus mutans in plaque and saliva. Scand J Dent Res 1984; 92:211-7.

21. Sakamaki ST, Bahn AN. Effect of orthodontic banding on localized oral lactobacilli. J Dent Res 1968; 47:275-9.

22. Balanseifen JW, Madonia JV. Study of dental plaque in orthodontic patients. J Dent Res 1970; 49:320-4.

23. Lundstrom F, Krasse B. Caries incidence in orthodontic patients with high level of Streptococcus mutans. Eur J Orthod 1987; 9:117-21.

24. Turkkahraman H, Sayin MO, Bozkurt FY, Yetkin Z, Kaya S, Onal S. Archwire ligation techniques, microbial colonization, and periodontal status in orthodontically treated patients. Angle Orthod 2005; 75:231-6.

25. Sukontapatipark W, El-agroudi MA, Selliseth NJ, Thunold K, Selvig KA. Bacterial colonization associated with fixed orthodontic appliances-a scanning electron microscopy study. Eur J Orthod 2001; 23:475-84.

26. Chatterjee R, Klienberg I. Effect of orthodontic band placement on the chemical composition of human incisor plaque. Arch Oral Biol 1979; 24:97-100.

27. Gwinnett JA, Ceen F. Plaque distribution on bonded brackets: a scanning electron microscope study. Am J Orthod 1979; 75:667-677.

28. Fournier A, Payant L, Bouchin R. Adherence of Streptococcus mutans to orthodontic brackets. Am J Orthod Dentofacial Orthop 1998; 114:414-417.

29. Suri L, Huang G, English JD Jr, Owen S, Nah HD, Riolo ML, Shroff B, Southard TE and Turpin DL. Topical fluoride treatment. Am J Orthod Dentofacial Orthop 2009, 135:561-63.

30. Benson PE, Parkin N, Millett DT, Dyer FE, Vine S, Shah A. Fluorides for the prevention of white spots on teeth during fixed brace treatment. Cochrane Database Syst Rev 2004; (3):CD003809.

31. Hirschfield RE, Johnston LE. Decalcification under Orthodontic Bands. The Angle Orthodontist 1974; 44: 218-221.

32. Geiger AM, Gorelick L, Gwinnett AJ, Griswold PG. The effect of a fluoride program on white spot formation during orthodontic treatment. Am J Orthod Dentofacial Orthop. 1988; 93(1):29-37.

33. Boyd RL, Chun YS. Eighteen-month evaluation of the effects of a 0.4% stannous fluoride gel on gingivitis inorthodontic patients. Am J Orthod Dentofacial Orthop 1994; 105(1):35-4.

34. Vyavhare S, Sharma DS, Kulkarni VK. Effect of three different pastes on remineralization of initial enamel lesion: an in vitro study. J Clin Pediatr Dent 2015; 39(2):149-60.

35. Azarpazhooh A, Main PA Fluoride varnish in the prevention of dental caries in children and adolescents: a systematic review. J Can Dent Assoc 2008; 74(1):73-9.

36. Demito CF, Rodrigues GV, Ramos AL, Bowman SJ. Efficacy of a fluoride varnish in preventing white-spot lesions as measured with laser fluorescence. J Clin Orthod 2011; 45:25.

37. Benham AW, Campbell PM, Buschang PH. Effectiveness of pit and fissure sealants in reducing white spot lesions during orthodontic treatment. A pilot study. Angle Orthod 2009; 79(2):338-45.

38. Salar DV, García-Godoy F, Flaitz CM, Hicks MJ Potential inhibition of demineralization in vitro by fluoride-releasing sealants. J Am Dent Assoc 2007; 138(4):502-6.

39. Soliman MM, Bishara SE, Wefel J, Heilman J, Warren JJ. Fluoride release rate from an orthodontic sealant and its clinical implications. Angle Orthod 2006; 76(2):282-8.

40. Manfred L, Covell DA, Crowe JJ, Tufekci E, Mitchell JC. A novel biomimetic orthodontic bonding agent helps prevent white spot lesions adjacent to brackets. Angle Orthod 2013; 83(1):97-103.

41. Doherty UB, Benson PE, Higham SM. Fluoride-releasing elastomeric ligatures assessed with the in situ caries model. Eur J Orthod 2002; 24(4):371-8.

42. Pinto CF, Berger SB, Cavalli V, Da Cruz SE, Gonçalves RB, Ambrosano GM, Giannini M. In situ antimicrobial activity and inhibition of secondary caries of self-etching adhesives containingan antibacterial agent and/or fluoride. Am J Dent 2015; 28(3):167-73.

43. Feil PH, Grauer JS, Gadbury-Amyot CC, Kula K, McCunniff MD. Intentional use of the Hawthorne effect to improve oral hygiene compliance in orthodontic patients. J Dent Educ 2002; 66:1129-35.

44. Tufekci E, Casagrande ZA, Lindauer SJ, Fowler CE, Williams KT. Effectiveness of an essential oil mouth rinse in improving oral health in orthodontic patients. Angle Orthod 2008; 78:294-8.

45. Featherstone JD, Fried D, Bitten E, Machule D. Rationale for laser-induced inhibition of enamel demineralization (abstract). J Dent Res 1997; 176:47.

46. Blankenau RJ, Powell G, Ellis RW, Westerman GH. In vivo caries-like lesion prevention with argon laser: Pilot study. J Clin Laser Med Surg 1999; 17:241-3.

47. Hicks MJ, Flaitz CM, Westerman GH, Blankenau RJ, Powell GL, Berg JH. Enamel caries initiation and progression following low fluence (energy) argon laser and fluoride treatment. J Clin ediatr Dent 1995; 20:9-13.

48. Buck T, Pellegrini P, Sauerwein R, Leo MC, Covell DA Jr, Maier T, *et al.* Elastomeric-ligated vs self-ligating appliances: A pilot study examining microbial colonization and white spot lesion formation after 1 year of orthodontic treatment. Orthodontics Chic 2011; 12:108-21.

49. Polat Ö, Gökçelik A, Arman A, Arhun N. A comparison of white spot lesion formation between a self-ligating bracket and a conventional preadjusted straight wire bracket.World J Orthod 2008; 9:e46-50.

50. Stecksén-Blicks C, Holgerson PL, Olsson M, Bylund B, Sjöström I, Sköld-Larsson K, *et al.* Effect of xylitol on mutans streptococciand lactic acid formation in saliva and plaque from adolescents and young adults with fixed orthodontic appliances. Eur J Oral Sci 2004; 112:244-8.

51. Sengun A, Sari Z, Ramoglu SI, Malkoç S, Duran I. Evaluation of the dental plaque pH recovery effect of a xylitol lozenge on patients with fixed orthodontic appliances. Angle Orthod 2004; 74:240-4.

52. Shen P, Cai F, Nowicki A, Vincent J, Reynolds EC. Remineralization of enamel subsurface lesions by sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. J Dent Res 2001; 80:2066-70.

53. Ramalingam L, Messer LB, Reynolds EC. Adding casein phosphopeptide-amorphous calcium phosphate to sports drinks to eliminate *in vitro* erosion. Pediatr Dent 2005; 27:61-7.

54. Sudjalim TR, Woods MG, Manton DJ. Prevention of white spot lesions in orthodontic practice: A contemporary review. Aust Dent J 2006; 51:284-9.

55. Bailey DL, Adams GG, Tsao CE, Hyslop A, Escobar K, Manton DJ, *et al.* Regression of post-orthodontic lesions by a remineralizing cream. J Dent Res 2009; 88:1148-53.

56. Beerens MW, van der Veen MH, van Beek H, ten Cate JM. Effects of casein phosphopeptide amorphous calcium fluoride phosphate paste on white spot lesions and dental plaque after orthodontic treatment: A 3-month follow-up. Eur J Oral Sci 2010; 118:610-7.

57. Bergstrand F, Twetman S. A review on prevention and treatment of post-orthodontic white spot lesions-evidence-based methods and emerging technologies. Open Dent J 2011; 5:158-62.

58. Akin M, Basciftci FA. Can white spot lesions be treated effectively? Angle Orthod 2012; 82:770-5.

59. Uysal T, Amasyali M, Ozcan S, Koyuturk AE, Akyol M, Sagdic D. In vivo effects of amorphous calcium phosphate-containing orthodontic composite on enamel demineralization around orthodontic brackets. Aust Dent J 2010; 55:285-91.

60. Waggoner WF, Johnston WM, Schumann S, Schikowski E. Microabrasion of human enamel in vitro using hydrochloric acid and pumice. Pediatr Dent 1989; 11:319-23.

61. Beyth N, Redlich M, Harari D, Friedman M, Steinberg D. Effect of sustained-release chlorhexidine varnish on

Streptococcus mutans and Actinomyces viscosus in orthodontic patients. Am J Orthod Dentofacial Orthop 2003;123:345-8.

62. Kronenberg O, Lussi A, Ruf S. Preventive effect of ozone on the development of white spot lesions during multibracket appliance therapy. Angle Orthod. 2009; 79:64-9.

63. Gaworski M, Weinstein M, Borislow AJ, Braitman LE. Decalcification and bond failure: A comparison of a glass ionomer and a composite resin bonding system *in vivo*. Am J Orthod Dentofacial Orthop1999; 116:518-21.

64. Manfred L, Covell DA, Crowe JJ, Tufekci E, Mitchell JC. A novel biomimetic orthodontic bonding agent helps prevent white spot lesions adjacent to brackets. Angle Orthod 2013; 83:97-103.

65. Fitzpatrick DA, Way DC. The effects of wear, acid etching, and bond removal on human enamel. Am J Orthod 1977; 72(6):671-81.