White Spot Lesions
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Abstract White spot lesions are an early stage of tooth decay formation. White spot lesions are commonly associated with the fixed orthodontic treatment and it poses a significant problem in the clinical setup. These lesions are caused by tooth demineralization which results in visible enamel changes. Accumulation of plaque around the brackets and its improper removal is the main etiological factor behind the incidence of white spot lesions. The purpose of this study is to review the literature on the prevalence of white spot lesions and methods with which it can be prevented.

Keywords Fixed Orthodontic appliances; Plaque accumulation; Fluoride application

Introduction White spot lesions are defined as subsurface enamel porosity from carious demineralization that presents as a milky white opacity when they are located on smooth surfaces. Fixed orthodontic treatment alters the oral environment and increases the accumulation of plaque. Gingivitis and enamel decalcification around fixed appliances occur since cleaning becomes difficult with the presence of the orthodontic appliance and their components (Arends and Christofferson, 1986). Bands and brackets increase the retention of food and plaque on smooth tooth surfaces, which would otherwise tend have low prevalence of caries (Bishara and Ostby, 2008). The predictors for white spot lesions at debonding are visible plaque and streptococcus mutans. However, the removal of stagnant plaque alone does not achieve complete repair of white spot lesions and some spots that are secondary to debonding can last from 5 to 12 years (Willmot, 2008). Natural mineralization through saliva involving mineral gain in the surface layer of white spot lesions has little improvement on the aesthetics and structural properties of deeper lesions.

The fixed orthodontic treatment alters the amount of plaque and changes the composition of the oral flora since it becomes more difficult for the patient to clean the teeth. Despite many attempts at prophylaxis, the prevalence of white spot lesions remains as high as 61% after debonding (Pender, 1986). The facial surfaces of the lateral incisors and the canine are more severely affected, followed by the premolars and the central incisors. There was no difference in white spot lesion incidence in the right and left sides (Ogaard, 1989; Cochrane et al., 2010). The variation in WSL prevalence among studies could be due to the differences in number of teeth examined, the location of the study sample, the methods and standardizations of the examinations, treatment duration and materials used.

There is a higher risk of white spot lesion incidence in the preadolescent age than in the older patients due to differing brushing habits, treatment duration and phase-2 treatment (Chapman et al., 2008). In a study conducted, the use of a combination of antimicrobial and fluoride varnish significantly reduced formation of new lesions in the first 48 weeks of the treatment. However it did not result in less development of white spot lesions on the labial surfaces (Gorelick et al., 1982; Yeung et al., 1989).

Plaque accumulation around the brackets was generally higher in the first four months and gradually reduced during the course of the treatment because of the improved oral hygiene maintenance of the patient after they overcome the initial difficulties related to
fixed appliance (Pender, 1986; Dalessandri et al., 2012).

Visual assessment of WSLs by clinical or photographic examination can be used to quantify the severity of WSLs, but it is not sensitive to small changes (Gorelick et al., 1982).

**Prevention**

During orthodontic treatment, white spot lesions can be prevented by Fluoride application, calcium-phosphate based remineralising agents.

**Fluoride varnish**

Fluoride has been shown to arrest development and progression of carious lesions. Regular application of topical fluoride varnish during treatment may reduce the development of white spot lesions adjacent to the bracket base (Stecksen-Blicks et al., 2007). Although the remineralising capacity of fluoride on enamel is accepted, the evidence is not sufficient to support the effectiveness of fluoride in remineralising post orthodontic WSL (Heymann and Grauer, 2013). Fluoride varnishes can be used in the presence of plaque and thorough cleansing of the tooth surface is not required before application of the varnish (Ogaard et al., 2001).

**Fluoride toothpaste**

Toothpastes with high fluoride concentration (1 500–5 000 ppm) have been reported to show greater inhibition of demineralization though the conventional fluoride concentration of 1 000 ppm is efficient in reducing incidence of new caries (Heymann and Grauer, 2013). However, high concentration fluorides should be used in case of completely arrested lesions rather than active lesions as they lead to discolouration in the latter lesion (Ay et al., 2007; Ogaard et al., 2001).

**Fluoride releasing bonding agents**

Glass ionomer and resin modified glass ionomer cements though mild, provide resistance to white spot lesions compared to other acrylic based cements. Glass ionomers show an initial burst of fluoride release but rapidly in decrease to levels that are unlikely to have any effect on caries prevention (Ogaard et al., 2001).

**Casein phosphopeptide amorphous calcium phosphate (ACP)**

Casein phosphopeptide amorphous calcium phosphate works by increasing the levels of calcium and phosphate ions and thus enhances the incorporation of fluoride (Beerens et al., 2010). The use of indirect bonding technique for the placement of brackets revealed lesser accumulation of plaque and lesser presence of white spot lesions than direct bonding technique during the first four months of treatment (Dalessandri et al., 2012).

**Xylitol**

Xylitol is a polyol carbohydrate that cannot be metabolized by Streptococcus mutans. It has been known to reduce the risk of caries by preventing the attachment of Streptococcus mutans on tooth surfaces (Heymann and Grauer, 2013). Chewing xylitol gum increases the production of saliva that has high phosphate and calcium levels (Ay et al., 2007).

**Diet**

Refined carbohydrates and carbonated soft drinks have been well documented to play a major role in the development of caries. Sucrose has a negative effect on the development as the biofilms formed in the presence of sucrose have lower concentrations of calcium, phosphate and fluoride which are necessary for remineralisation.

**Carbamide peroxide**

The urea present in carbamide peroxide helps increase the pH of plaque and saliva. This increase in pH and the antimicrobial action of hydrogen peroxide helps to diminish plaque formation.

**Post orthodontic Treatment**

**Vital bleaching**

Whitening of the teeth affected by the lesion lightens the healthy enamel as well as the affected enamel. However, the healthy enamel appears to increase in lightness more than the white spot lesion which gives less contrast between the unaffected enamel and the lesion.

**Natural resolution by remineralisation**

After orthodontic treatment, visible white spot lesions diminish in area and improve in appearance after 1 to 2 years from debonding. Active lesions have a better prognosis compared to completely arrested lesions.
The use of high concentration fluorides on active lesions arrest the lesions and result in discoulouration of the lesions.

Resin infiltration-
The white opaque appearance of white spot lesions is due to the difference in the refractive index of the healthy enamel and demineralised enamel. The porous nature of an active white spot lesion allows low viscosity resins to permeate into the previously demineralised enamel matrix and fill in the voids with resin (Heymann and Grauer, 2013). This creates a refractive index that resembles that of healthy enamel and therefore there is an improvement in the appearance of the tooth. Resin infiltration is more successful in lesions that are not completely arrested.

Indirect porcelain restorations or direct resin restorations-
They can be given for cavitated lesions and for severe forms of white spot lesions that do not show significant improvement after conservative aesthetic treatment (Dalessandri et al., 2012).

Table 1 Preventive measures, intratreatment and postorthodontic treatment of white spot lesions

<table>
<thead>
<tr>
<th>Fluoride varnish</th>
<th>Every 4 months</th>
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<tbody>
<tr>
<td>Fluoride toothpaste</td>
<td>1 000 ppm fluoride toothpaste twice daily</td>
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<tr>
<td>Fluoride releasing bonding agents</td>
<td>Glass ionomer cement</td>
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<tr>
<td>Casein phosphopeptide amorphous calcium phosphate</td>
<td>MI paste (900 ppm fluoride) nightly for 3-5 min after brushing</td>
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<tr>
<td>Xylitol</td>
<td>3-5 pieces a day for 10 min</td>
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<td>Carbamide peroxide</td>
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<tr>
<td>Prophylaxis</td>
<td>Every 3 months</td>
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<td>Post orthodontic treatment:</td>
<td></td>
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<tr>
<td>Resin infiltration</td>
<td>For cavitated or severe white spot lesions</td>
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<tr>
<td>Natural remineralisation</td>
<td>If lesion doesn’t exceed 0.2-0.3 mm in depth</td>
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<td>Vital bleach</td>
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<td>Indirect Resin restorations</td>
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<td>Micro abrasion</td>
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Conclusion

White spot lesions that are associated with orthodontic treatment can be minimized with proper prevention, management and treatment procedures.

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